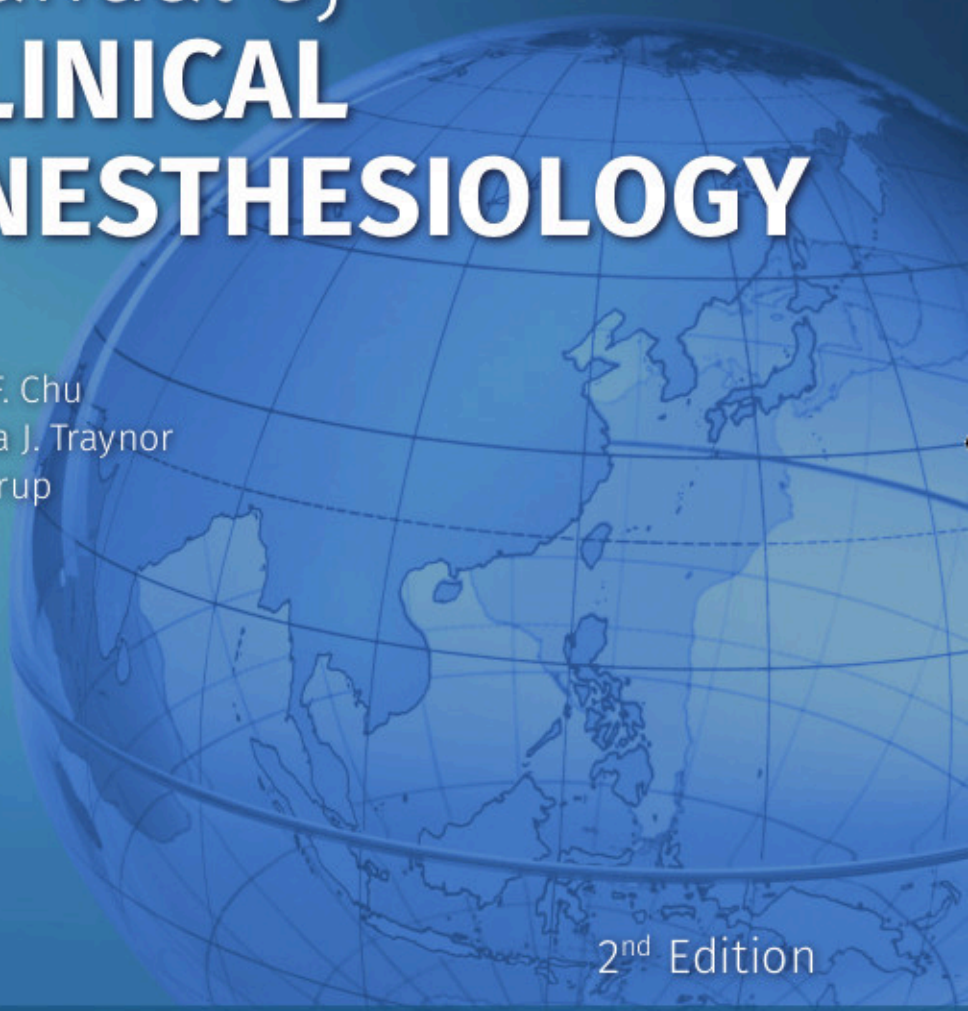
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South Asian Edition of

Manual *of* CLINICAL ANESTHESIOLOGY

Larry F. Chu
Andrea J. Traynor
Viji Kurup



2nd Edition

Wolters Kluwer

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CLINICAL ANESTHESIOLOGY

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Second Edition

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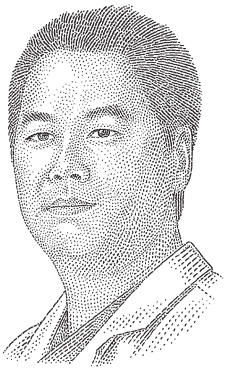
To our patients, colleagues, communities and families. We dedicate our efforts to improving patient-centered anesthesia care and gratefully acknowledge the support and trust you have given us along the way.
(L.C.)

To the patients who trust us with their lives
(A.T.)

To my parents who gave up everything for me and to whom I owe everything, my husband who has been a pillar of support at every step, and my kids who inspire me to be my best self everyday
(V.K.)

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PREFACE



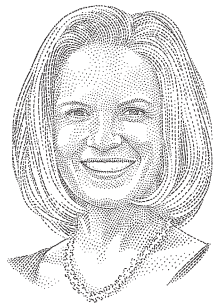
Larry F. Chu, MD, MS

Bringing a book together in the middle of a global COVID-19 pandemic is certainly a challenging experience. Leading people to a shared goal through a crisis requires the ability to navigate uncertain times. It reminded me of all the reasons I envisioned creating this book in the first place: to ensure those at the frontlines of anesthesia care are empowered with the tools they need to perform their roles effectively.

Our focus in creating this book is on your success at the frontlines. Whether in routine practice or during a critical crisis, communication skills and techniques that are taught in crisis resource management are important for optimal patient-centered anesthesia care. We have also prioritized access to information early enough to make a difference. We have organized this textbook in a way that is highly visual and portable so that it can serve as a practical aid at the point of care.

This second edition builds on the success of our prior edition by including even more cognitive aids, clinical algorithms, and visual atlases of anesthesia practice. I could not have asked for better collaborators in this second edition than Dr. Andrea J. Traynor and Dr. Viji Kurup. They have worked tirelessly with me to bring the vision of a practical yet comprehensive point-of-care manual into your hands.

Larry F. Chu, MD, MS
Stanford, California



Andrea J. Traynor, MD

The practice of anesthesiology requires us to think "on the fly." We are often faced with clinical challenges late at night, emergently, and in patients with coexisting illnesses. The goal of this book was to provide a reliable place to turn at the point of care. We went to great lengths to recruit contributors and editors from every area of the United States and the world in an effort to reflect a broad view of the specialty and not just an institutional or regional approach. It is intended for anyone who practices general anesthesiology, whether he or she has been practicing for 3 days or 30 years.

I would like to express my gratitude to some people who have made this project possible. Thanks to Dr. Viji Kurup for joining this effort. She is a very special person and brought our team together with her calm, compassionate, diligence, and positive attitude. Thank you to Dr. Larry F. Chu, whose unique ability to condense complex topics into visual resources makes this book special. I am sincerely grateful to my colleagues and fellows in the Stanford OB Anesthesiology group, who teach me every day and make practicing cutting-edge anesthesiology so much fun. I thank Brian, Henry, and Benji for inspiring and encouraging me to work hard, stay focused, and be positive every day. Thank you to my friends and family, for their unwavering support. Thank you to all my patients for trusting me. You are all inspiring and have helped me become a better doctor and a better person.

Andrea J. Traynor, MD
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Viji Kurup, MD

In a world where information is available at your fingertips, the challenge is in making sense of this information and having it when and where you need it. This book was conceived to help the practitioner at the point of care. I am incredibly grateful to Dr Larry F. Chu and Dr Andrea J. Traynor who after a highly successful first edition of this book, invited me to coedit the second edition of this book. We focused our efforts on making sure that the information presented was visually appealing and cognitively coherent. Medicine is an ever-changing field, and we hope that this book will be a partner to learners in the field of anesthesiology whether they are medical students, residents, fellows, or practicing anesthesiologists.

My gratitude also to my kids, Anirudh and Anisha, who delight me everyday and have taught me more than I could ever teach them, my patients, who make everything I do worthwhile, my teachers who were my role models, and my incredible group of friends who have been unwavering in their support and encouragement at all times.

Viji Kurup, MD
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Online resources are not available with this text.

A L G r a w a n y

HOW TO USE THIS BOOK

This book is designed to be used at the point of clinical care by practicing anesthesiologists. It is not a textbook of anesthesiology. There are already many excellent texts that provide detailed explanations of the principles and practice of perioperative medicine.

This is a **manual** of clinical anesthesiology.

We have designed it from the beginning to be a highly visual manual of anesthetic practice that brings practical information where it is needed most: in the operating room, on the wards, and at the patient bedside.

We have incorporated cognitive aids, clinical algorithms, and visual atlases of anesthesia practice into a single portable reference text that is intended to be used at the point of care.

In order to facilitate rapid reference of material, we have utilized several graphical conventions throughout this book.

You may already have noticed the use of color to provide emphasis to important ideas and information within the text.

Chapters are also easily identified by numbers that are printed in large, easy-to-read type at the top of each chapter heading.

Sections of the book are also immediately identified by color bars that are printed on the edge of the page, making it easy to rapidly reference sections of the text.

Finally, we have three main graphical enhancements: (1) the read more arrow to direct you to related material, (2) the skull and bones to alert you to important clinical information, and (3) pearls, which help highlight interesting and useful facts and information.

Monitoring Equipment and Procedures Subspecialty Anesthesia

16

Chapter numbers in large font with bold formatting. Size and color enhance rapid-reference readability.

READ MORE

Chapter 74, Overview of Pediatric Anesthesiology, page XX

These icons are used to link to other chapters in the book with related information. This helps direct readers to useful content quickly.



The needle used to treat a tension pneumothorax should be left in place until a tube thoracostomy is performed.

The Skull & Bones icon is meant to highlight extremely important clinical information that may help prevent adverse events.



Using clinical signs alone for placement of a Double-lumen endotracheal tube is an important clinical skill.

These icons are used to identify useful clinical pearls to the reader.

Color-coded heading bars bled to the edge of the page help readers quickly identify relevant sections of the book.

INTRODUCTION

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1 Anesthesiology and Person-Centered Anesthesia Practice

Amy Price • Alicia C. Staley • Larry F. Chu

Effective anesthesia care meets the goals, preferences, and values of each person at each point in the perioperative patient journey. Research shows that patients' satisfaction with anesthesia care depends on the perioperative physician designing an anesthesia care plan that accounts for these preferences. When asked, patients place a high value on whether their concerns were identified and addressed during the preoperative phase, on being spoken and attended to during the operative phase, and on being given appropriate information before and after discharge. Effective patient-anesthesiologist communication has been shown to decrease postoperative pain, the length of hospital stay, and the cost of care. Mastery of perioperative medicine requires empathy for the patient experience and an understanding of how to implement a **person-centered anesthesia practice**.

1) **Empathy for the patient experience**

Anesthesiologists are perioperative physicians. We shepherd people through the most vulnerable moments of their lives, as they journey from consciousness to unconsciousness, from incision to closure, and from sickness to health. We are the voice of the patient and their champion when they are asleep, and we continue care until they are safely through the surgery and in the recovery room. We are the last person they see before they go to sleep and the first person they see when they awaken from surgery. As one of many members of a team, we work in concert together with the surgeon and patient, along the perioperative care journey. As you begin your training in the field of anesthesiology, we believe it is paramount to understand the patient journey and experience of anesthesia. Several patient experts, coauthors of this chapter, will share their own experiences of anesthesia that can help shape your development as future perioperative clinicians.

a) **Advice for new anesthesiologists from your future patient**

Dr. Amy Price: *"As the anesthesiologist you set the scene for a journey I have to make. I am afraid, and yet I need you. When you choose to extend your hand gently and consider how to walk with me, the journey becomes easier, the fear of the unknown dissipates and my helplessness scatters, peace comes and hope rises, I am no longer alone, you remain my anchor and my representative for this short journey. It helped when you saw me as a person and not just the patient. You were interesting, curious, and kind. Your words were the last ones I heard. I respect that it cost you time to make eye contact, and to prepare me for the journey. It was great that you reassured my husband and told him where he could find me. Thank you for sharing outside news and making me laugh. You respected and informed me. You set the stage for the team. My advice to you as you start your career is don't underestimate how much patients value your role or how your leadership can change the tone in the room. And please do the same for me when we meet someday".*

b) **Advice for new anesthesiologists from your future patient**

Alicia Staley, MBA: *"The anesthesiologist plays an extraordinary role in a patient's surgical journey. The relationship that forms between the anesthesiologist and the patient, while*

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brief as it may seem to you, is sacrosanct to me. I've been fortunate to have had amazing anesthesiologists for all seven of my major surgeries, and I will never forget those ephemeral, significant moments. It's in these moments I know you, the future anesthesiologist, can learn from my journey. I remember the count downs, listening to beautiful music, the lights in the operating room, and the conversations exchanged. In those moments, you were the doctor closest to me and I found great comfort in focusing intently on your words and movements. You conducted the energy in the operating room and your positive, calming tone created the safe space I needed to allow myself to slip from conscious to unconscious. The start of the surgical journey flowed through you. I rested comfortably knowing you'd watch over me, guard my space, and be my champion while my eyes were closed. You gave a voice to the voiceless while my eyes were closed. When I began to emerge from the depths of this controlled state, your voice was the first I recognized, and it brought me back to the here and now. As an anesthesiologist, you will always be my guiding force."

2) **Partnership with patients in perioperative medicine**

Partnership in the clinical practice of perioperative medicine is essential for effective anesthetic care that meets the goals, preferences, and values of each person. Effective partnership with patients involves supporting them as valued members of the care team and aligning your practice and the work of the surgeon to meet the goals, preferences, and beliefs of each person. We call this person-centered anesthesia care.

a) Include patient preferences and goals in developing your perioperative care plan. Research suggests that patient satisfaction is dependent on the anesthesiologist designing an anesthesia regimen that closely fits a patient's preferences.[1]

b) Educational aids for patients[2] and their families[3,4] are used by people to help prepare for the perioperative care journey. Such aids may decrease the anxiety of patients coming for surgery and help them feel more informed about the perioperative process. It is important to note that some aids may contain outdated information or reference information that is not supported by scientific literature. In these cases, the perioperative physician should update knowledge whenever possible. Some patients may prefer citations that they can reference. It may be useful to print out the latest evidence from the point of care tool you are using.

c) Support affirmations for people who ask to engage. While the scientific evidence may not support claims of such statements improving healing, such affirmations can help calm patient anxieties and build trust in the collaborative care relationship between patient and physician.

d) **Advice for new anesthesiologists from your future patient**

Alicia Staley, MS MBA: *"In my last two surgeries, I co-wrote a "surgical affirmations" list with the help of my anesthesiologist and oncology social worker. As a team, we read a surgical preparedness book[2] and created my list of affirmatives that were used as part of my surgery. It was reassuring to hear my anesthesiologist read my surgical affirmations with me as I drifted off. "I'll wake up from this surgery feeling refreshed and rested. I'll be able to start my healing journey as soon as I wake up." Hearing such simple words over and over again help me stay focused and allowed me to feel prepared for the surgery. Emerging from these surgeries was easier for me, as I did feel rested and ready to go back to my hospital room. I credit my anesthesiologist for encouraging me to explore this option as part of my pre-surgery preparations. To me, this is the definition of person-centered anesthesia care."*

e) Communication between patient and anesthesia provider.

Enhance communication by taking time to be purposeful in defining collaboration set points with patients and establishing goal clarity for the anesthesia care plan. There is opportunity at each stage of anesthesia to use effective communication to improve patient-clinician partnership. Effective anesthesiologist-patient communication has been shown to reduce postoperative pain, length of hospital stay, and cost of care.[5,6]

i) Preoperative. An example might be establishing expectations about pain and sedation levels with a patient before a procedure and creating goal clarity for them to communicate with you when they have pain or discomfort.

"This is what we are going to do with your eye, this is how it is going to feel. When your eye is frozen, that sensation is completely gone. You can let me know at any time if you feel pain."

Research completed over 50 years ago by Egbert[7] has shown that anesthesiologist-patient communication in the preoperative visit had a greater effect on reducing anxiety than intramuscular injection of pentobarbital. Other studies have found that preoperative instructions on postoperative pain and its therapy combined with postoperative instructions significantly reduced postoperative narcotic requirements and reduced the hospital stay by 2.7 days.[8]

Induction. Several communication strategies can enhance anesthesiologist-patient partnership during the induction period:

(1) Observe that patients can feel foolish, anxious, or fearful as they move to the operating room table and are strapped down, and monitors are attached by masked operating room personnel. Many people feel a loss of autonomy or identity.

(2) You can assume the role of mentor and guide for the person by offering "small talk" at this time and anticipating foreign sensations by describing them which can help build trust and connection with your patient. "*Please move over to this bed, you'll feel a small gap and I'm going to reach over and untie your gown so it doesn't catch underneath you.*" Or "*The blood pressure cuff is going to squeeze your arm kind of tight right now, but it only does this the first time. Once it gets to know you, it won't squeeze so tight.*" Or "*I'm going to place some cold sticky EKG pads on you now.*" All of these statements reassure the patients that they would not be harmed and are trustworthy behaviors.

ii) Visit your patients postoperatively and find out how you can improve your practice. "How could I have made your anesthesia better today?"

3) **Sparking a joyful person-centered anesthesia practice**

Anesthesiology training is a unique opportunity to make people the center of your clinical practice. By centering your clinical practice around optimizing the lives of people, you will keep the joy of medicine top of mind and highlight the unique role of the perioperative physician in the healing process. Some specific ways you can maintain a person-centered anesthesia practice include the following:

a) Listen to your patient

i) Address your patient's significant concerns by listening carefully to them. "How can I make your anesthesia a great experience for you today?"

ii) Review the EHR beforehand or along with your patient, but then engage with your patient during the preoperative consult. "I have the information that I think I need from reviewing your electronic record, but I find that the patient often has more information that is helpful."

b) Engage in shared decision-making

i) Ask open-ended questions to find out what level of knowledge the patient has about the anesthetic. "How much do you know about the anesthesia that you are going to be given today?"

ii) Based on their prior knowledge, establish how much detail and information to provide in order for them to effectively participate in shared decision-making for the type of anesthesia they will be given and to consent for the procedure.

(1) This can include asking about specific fears or concerns about the anesthetic procedure.

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(2) Explain the procedure, how it will be performed and any associated risks in ways that are easily understood.
(3) The length of the procedure and/or associated effects of the anesthetic such as postoperative blocks.
(4) Inform the patient of any potential side effects and how they might be associated to the procedure.

iii) Ask about the patient's goals, preferences, and beliefs and incorporate these into your anesthetic plan.
(1) "Do you have any goals, like taking a plane trip right away that I should know about that could affect the way I do your anesthesia today?"
(2) "Do you have any preferences or beliefs like refusing blood transfusion that could potentially affect how I do my anesthesia today?"

c) Have considerate conversations
i) Consent is not always well understood. Take time to ensure that your patient understands the scope of the procedure, its associated risks, and has the capacity to consent.
ii) Carefully explain procedures. Some patients want to know all the details, while others are only interested in a general overview. Tailor your explanation to the needs of the person.
iii) Time-out: anesthesia time-out is a rare opportunity to recheck together with the patient what we are doing and verify any procedure concerns. It is also another opportunity to build trust in the patient-provider relationship. "Ms. Jones we are going to do an anesthesia time-out right now. You may hear us talking about you and the case. Feel free to listen in and let us know if there is anything we might have missed."
iv) Keep conversations, attitudes, and music positive: emotional processing can occur even at subconscious levels.
v) Introduce all members of the care team, including trainees such as medical students.

d) Maintain dignity of people
i) Comfort is important to the dignity of people. Ensure the comfort of people including the following:
(1) Adequate gown coverage and draping during procedures to help ensure the modesty of people according to their customs and culture.
(2) Offer a heated blanket while patients are waiting to go into operating room.
(3) In the waiting space between SAU and the operating room, people will be pleased if they are offered reading materials, music, their phones, or computers.
(4) Have your staff update your patient about delays.
ii) Be mindful of the environment and ask permission before performing procedures or asking health history questions in the presence of other people.
iii) Welcome supporters and caregivers to the perioperative environment as they provide important emotional support and caregiving resources to your patient.

e) Flatten power hierarchy and elevate respect hierarchies for the expertise each person brings to the care team. As a perioperative physician, you will need all available resources to optimize the care of your patient. Including caregivers and your patient themselves as members of the care team will ensure perioperative care plans are made that not only include the goals and preferences of the patient but align with the abilities of the caregiver and patient to carry them out at home.

f) Demonstrate trustworthy behavior
Trust is the ability to predict a person's behavior in a reliable way and know that their actions will not result in harm. Anesthesiologists should demonstrate trustworthy behavior in order to build trust with patients quickly in the perioperative period across multiple domains.

i) Communication: effective communication is important to demonstrate to patients that their input is valued, respected, and heard so that they understand harm will not result.
   (1) *Anesthesiologist*: "Do you have any allergies?"
   Patient: "I'm allergic to codeine."
   Anesthesiologist: "Did you go into anaphylactic shock?"
   Patient: "No, but I went to sleep for four days."
   Anesthesiologist: (Looks away at computer screen)
   Patient: (Looking concerned, expands on her comments) "The next time I was given a smaller dose and I had to have intravenous fluids and antiemetics."
   (2) In this example, the patient had a prior history of a severe adverse reaction to codeine and attempted to communicate a fear of repeated harm from the medication. The anesthesiologist's question, "Did you go into anaphylactic shock?" is a common method used to determine the type or severity of reaction. In this patient, it caused a feeling of devaluing and disrespecting her concerns and broke trust. She was not sure if the anesthesiologist might give her the drug when she was asleep and could not advocate for herself.
      (a) Open-ended questions create psychological safety and opportunities for people to express their concerns openly, "Can you tell me more about your allergy to codeine?"
      (b) Explicitly acknowledging harm or fears to your patient can help build trust, "I hear that codeine causes you a severe adverse reaction, and I'm going to make sure that you will not get any during your care here today."
      (c) Reconfirm and acknowledge to build trust, create a shared mental model, and improve patient safety. "Ms. Jones we are going to do an anesthesia time-out right now. You may hear us talking about you and the case. Feel free to listen in and let us know if we get anything wrong (speaking to OR staff). Ms. Jones has a patient-specific concern which is a severe adverse reaction to codeine. We will all make sure she will not receive any codeine during this procedure."

ii) Nonverbal communication: body language, facial expression, eye gaze, and blinking all provide nonverbal communication to patients that is an opportunity to demonstrate respect, allows the patient to judge motivations and identify any shared values, and is an opportunity to show trustworthy behavior.[9]
   (1) Open posture shows friendliness, openness, and a willingness for engagement.
   (2) When speaking with people, bring yourself to eye level. If your patient is lying in bed, sit in a chair or kneel so that you are at eye level with the person. This helps to level the power hierarchy, creates a deeper level of connection and engagement with the person, and helps the person to feel felt and heard.

iii) Keep your word: delivering on your promises (big or small) goes a long way to build trust with patients. Be realistic with what you can deliver and follow through. Broken promises can destroy a patient's trust.

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A L G r a w a n y

2 Learning and the Pursuit of Excellence

Viji Kurup

Being a practitioner of anesthesiology involves having a deep knowledge of anatomy, physiology, pharmacology, as well as clinical skills in virtually all specialties. No other practice needs knowledge of all specialties as does anesthesiology. Over the years, we have made the administration of anesthesia safe by designing safer machines and better tools in addition to adding checklists for critical scenarios. Yet, there are times when one must think on one's feet because seconds count in diagnosis and treatment. Therefore, the practice needs a constant maintenance of skills. There are evidence-based methods that facilitate retention of knowledge. This chapter discusses qualities important to have a successful career in medicine in general and anesthesiology in particular such as cultivating joy in learning, prioritizing personal wellness, and methods of developing curiosity and humility in the practice of anesthesia.

1) **Approaching learning with purpose and structure**
   a) Anesthesiology residency programs have dual goals. First, teachers must ensure that residents develop clinical skills, procedural competency, and expertise in the practice of anesthesiology. They also must ensure that residents have the information and resources to help them perform well on the written and oral board examinations. Evidence-based strategies for learning and retention exist, but not all faculty and residents are aware of them, and we hear the refrain "You need to read more" repeated to learners.
   b) A strong knowledge base is essential for building more advanced and critical thinking skills. There is no alternative to knowing facts; however there are techniques that can improve long-term content retention. A number of evidence-based methods have been described in the literature. We will describe two of them here, and the interested reader can refer to additional methods in the readings section at the end of this chapter.
      i) Spaced repetition:
         (1) This technique involves spacing out study episodes over time rather than studying in one long stretch.
         (2) Most students are familiar with "cramming" right before an exam, and although cramming can help get a grade, it is not conducive to long-term retention.
         (3) Humans forget learned content at a constant rate, and spaced repetition decreases the slope of the forgetting curve and results in longer retention of learned material in working memory.[1]
         (4) Adaptive learning software and computer algorithms designed to remind the learner relevant content at the appropriate time will assist with retention.
         (5) The spaced repetition technique involves planning and scheduling study sessions based on the necessary concepts to cover.
         (6) This may be different for the AKT, ITE, the Basic, and the Advanced exams. The content outline for these tests are available on the ABA Web site (http://www.theaba.org/).

ii) Retrieval practice:
  (1) This technique requires the learner to actively recall information from memory.[2]
  (2) This can be done by reading a section and then closing the book and recalling as much information as possible either by saying it aloud, writing it down, or telling someone about what was learned.
  (3) The act of testing improves retention and allows for meaningful interaction with the content.
  (4) Practice tests and postreading quizzes employ this method.
    (a) Using time for tests and quizzes has been shown to be more effective for retention than rereading material.[3]



Using spaced repetition and retrieval practice together is more time efficient and effective for content retention.

iii) Using spaced repetition and retrieval practice together is more time efficient and effective for content retention.

2) **Sparking curiosity** in the art and practice of anesthesiology
  a) Curiosity is critical for optimal learning. When we are curious about our environment and a topic, we probe the limits of our knowledge and skills to the point of failure, define our capacity and aptitude for a subject, and expand our perspectives freely.
  b) There are several factors that create a culture of curiosity in learning:
    i) Optimize psychological safety. Curiosity represents the opposite of fear. To be curious as learners, we must be absent of fear: fear of failure, of shaming or ridicule, of harm of reputation, or of hurting the ego.
      (1) Utilize simulation and case-based learning. These modalities of learning allow learners to explore high-stakes clinical scenarios as well as participate in educational activities for learning and ongoing maintenance of skills with high psychological safety. There is low risk of actual patients being harmed. Individual failure is minimized in the setting of team-based activities.
      (2) Defer judgment. Psychological safety is increased when we defer judgment of people and instead state clear observations, followed by a probing question that signals curiosity about the situation. A sample scenario might be as follows:
      Resident: "I saw that this patient has a serum potassium of 6.0 last night. I noticed that you have given me succinylcholine for induction. Is that what you would like me to use for this patient?"
      Attending: "Thanks for cross-checking that with me. The patient just had dialysis this morning and his stat potassium came back just now at 5.0. I think we are okay to proceed."
      (3) Give yourself permission to be imperfect. The practice of medicine reflects the process of constant learning and acquiring of a skill. There are several limitations in both the content and context of medicine. True masters seldom regard their learning as complete. Rather they remain curious about their craft and retain a need to continuously explore and expand their knowledge. As you advance in your journey toward mastering the art and practice of anesthesiology, you will undoubtedly encounter failure. Each failure will invariably come with a lesson. Give yourself permission to be imperfect. Embracing your vulnerability as a learner will create psychological safety to ask for help from your mentors and optimize your learning opportunities. Compassionate care for our patients must begin with compassion for ourselves as learners. We are human, we are imperfect, and we learn from our failures.
    ii) Be curious about people. People in your training program are an important resource for sparking curiosity in your learning. They can be important sources of information about your training program or hospital. And they can provide important feedback about your learning, provide important insights about

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pathways to success in a career in anesthesiology, and serve as critical resources and support networks.

(1) Consider asking people to help you gain feedback. We often can gain important insights about our skills and abilities through feedback from people around us who see our work and how we interact with those around us. Yet often, feedback is not freely offered unless prompted. Here are some tips for asking for feedback:
   (a) Don't wait. Ask for feedback while it is still fresh on your attending's mind. Or set expectations early in the day with your attending by asking for 5 minutes at the end of the day for feedback with your attending, "Could we set aside 5 minutes at the end of the day for feedback?"
   (b) Embed a compliment or context. "Thanks so much for the opportunity to work with you today. I've learned so much about thoracic anesthesia and how to manage one-lung ventilation. But, I'm wondering what you think I should work on to make these types of cases better next time?"
   (c) Be specific and/or narrow the scope. You will receive more valuable feedback the more specific you are with your feedback. A sample interaction may be as follows: "I noticed when we placed the bronchial blocker, we had great lung isolation. In the past I have not had the same experience. I'm wondering, what do you think I should work on to improve my ability to get optimal lung isolation with blockers?"
   (d) Ask "how" and "what" questions. Asking questions that begin with "how" and "what" are more likely to get useful feedback.
   (e) Avoid being defensive. Create psychological safety for your mentor so he or she can feel comfortable giving you feedback.
   (f) Accept a compliment and close with a thank you. Don't be afraid to graciously accept a compliment, and remember to thank your mentor for taking the time to give feedback.
   (g) Self-reflect at the end of the day. Think about what went well and what did not. Consider if you would make any changes in your practice or need to read up about any issues that came up during the case. Reflect on what you learned each day.

(2) Who can give feedback?
   (a) Near peers. Fellow trainees in your program who work with you on rotations together, senior residents who work with you on call.
   (b) Mentors. Clinical and research faculty at your institution within anesthesiology, perioperative, or pain medicine. Wellness advisors or faculty in your graduate medical education office.
   (c) Staff. Perioperative staff such as nurses, program administrators, and support staff such as anesthesia techs can provide helpful feedback.

3) **Cultivating joy in learning and practice**

a) We all chose to go into medicine for a reason. I encourage you to not lose sight of the incredible gift of being able to participate in the healing process of our patients.

b) There are several factors that may contribute to burnout and/or lack of balance in a physician's life.[4] Self-care is important and should be a priority for a physician.

c) Being an anesthesiologist means that we meet patients when they are the most vulnerable. They are anxious about their diagnosis and worried about the outcome of surgery. Therefore, your interactions with patients at this time can be very impactful, because your words and skills can make a significant difference in their experience of this big (and often life-altering) event.

d) We encourage you to extend that kindness and compassion to all the people you interact with and develop meaningful relationships with your colleagues. Make time to pursue hobbies, relationships, or activities that lend meaning to you personally.

Being a practitioner in anesthesiology means being a lifelong learner, being awake to the miracle of life, being reminded of our own mortality, and being an active participant in the journey our patients take. We have a social responsibility to take care of ourselves, to keep ourselves continuously updated with latest information, and to be competent in clinical skills so we can provide the best possible care to our patients and realize that when we say "I will take care of you," it is a solemn promise we make to our patients.

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A L G r a w a n y

3 The Anesthetic Plan and Induction of Anesthesia§

A. Katharine Hindle • Larry F. Chu

The anesthesiologist's role in the care of a patient for surgery begins with the preoperative assessment, continues with the induction and maintenance of anesthesia for the surgical procedure, and extends to the postoperative period until the effects of the anesthetic drugs and procedures have resolved and the patient has recovered. A comprehensive anesthetic plan describes how the anesthesiologist and perioperative care team will create a customized, tailored plan that considers the patient's medical history and overall condition and the impact of the anticipated procedure. This chapter discusses the development of an anesthetic plan and induction of anesthesia.

1) **Overview**
   a) The anesthetic plan is a guide to the perioperative care of patients undergoing anesthesia for surgery and consists of preoperative assessment and optimization, intraoperative management, and postoperative care.
   b) The development of a plan for the safe administration of anesthesia is customized to the individual patient and considers both the patient's medical history and physical examination findings, as well as surgical needs.
2) **Preoperative assessment and optimization**
   a) A history and physical examination can help identify relevant coexisting diseases and allow the anesthesiologist to assess the patient's anatomy and physical status to determine any potential challenges with planned anesthetic techniques.
   b) The anesthesiologist may choose to optimize certain coexisting diseases, such as hypertension or heart disease, prior to surgery.
   c) The preferences of the patient and surgeon with regard to anesthesia for the procedure should be discussed, in addition to consideration for the type, length, and complexity of the surgery.
   d) **Selection of anesthetic technique**
   The anesthetic plan should be discussed during the preoperative assessment, including the risks and benefits discussion with both the patient and surgeon in order to ensure that informed consent is obtained (**Table 3-1**).
      i) Types of anesthesia techniques. A combination of these techniques may also be used.
         (1) Local anesthesia
            (a) Infiltration or a field block typically done by surgeons, with or without patient sedation.
            (b) Many times, cases booked as "local anesthesia" without patient sedation are performed by surgeons without the involvement of an anesthesiologist.
         (2) Monitored anesthesia care (MAC)
            (a) Anesthetic drugs are administered to varying levels of sedation, analgesia, and anxiolysis as necessary for the procedure.
            (b) The provider must be prepared and qualified to treat hemodynamic changes, manage the airway, and convert to general anesthesia (GA) if necessary.

READ MORE
Difficult airway management, Chapter 21
Preanesthesia assessment, Chapter 4



Loss of consciousness or the ability for purposeful response during MAC is GA, irrespective of whether the airway is instrumented.[1]

TABLE 3-1 Anesthetic Plan Considerations

Perioperative Issue	Anesthetic Technique Considerations
Length of surgical procedure	• Long surgical cases may require GA, even with neuraxial or PNB techniques, because of tourniquet pain and uncomfortable positioning.
Potential for blood loss and/or hemodynamic instability	• Endotracheal intubation and mechanical ventilation may become necessary if a patient sustains loss of consciousness due to hemodynamic instability. • Consider GA or plan for conversion to GA if necessary.
Patient position	• Prone or lateral positioning is often uncomfortable for a patient and may require GA instead of RA or PNB techniques.
Airway anatomy	• PNB and RA techniques may allow avoidance of airway manipulation in patients with a difficult airway. • Consider definitively securing the airway and GA in an elective and controlled manner if the potential for conversion to GA is likely.
Anticipated postsurgical pain	• PNB and RA techniques are very effective in postoperative pain control, especially when high postoperative pain is anticipated (eg, knee replacement surgery).
Requirement for surgical neuromuscular blockade	• The need for neuromuscular blockade may require dense RA/PNB for extremity surgeries and GA with mechanical ventilation for abdominal surgeries.
Patient history of postoperative nausea and vomiting	• RA/PNB techniques provide excellent analgesia and reduce the need for emetogenic agents, such as opioid narcotics and volatile anesthetic gases.

(3) General anesthesia (GA)
  (a) GA involves loss of consciousness and is induced by anesthetic drugs that can be given by inhalation or intravenous (IV) injection.
  (b) The airway can be kept patent with an endotracheal tube (ETT) or supraglottic airway device (eg, laryngeal mask airway [LMA], oral or nasal airway) or can remain unsecured if the procedure is brief.

(4) Peripheral nerve block (PNB)
  (a) Anesthesia is provided by placing local anesthetic drugs near peripheral nerves using a needle.
  (b) Typically used for surgeries of the extremities, though truncal blocks such as TAP and paravertebral are also performed.
  (c) May be used as the primary anesthetic or as postoperative analgesia when administered in conjunction with other techniques such as GA.

READ MORE
Overview of peripheral nerve blocks, Chapter 44

(5) Neuraxial anesthesia
  (a) Anesthesia is provided by placing local anesthetic within the cerebral spinal fluid (spinal) or epidural space (epidural).
  (b) These techniques can provide dense surgical anesthesia for surgeries involving the abdomen and lower body.
  (c) May be used as the primary anesthetic or for postoperative analgesia or may be combined with MAC if anxiolysis or sedation is necessary.

3) **Intraoperative plan**
  a) **IV access (Table 3-2)**
    i) Most anesthetics are done with IV access because of the need to administer medications and fluids during most surgical procedures.
      (1) For healthy patients having procedures without significant potential for blood loss (eg, carpal tunnel, cataract surgery), one 20- or 22-G IV is likely adequate.

A L G r a w a n y

TABLE 3-2 Peripheral IV Catheters

Gauge size	Color	External Diameter (mm)[a]	Length (mm)[a]	Flow Rate (mL/min)[a]
24	Yellow	0.7	19	20
22	Blue	0.9	25	36
20	Pink	1.1	32	60
18	Green	1.3	32	90
16	Gray	1.8	45	180
14	Orange	2.1	45	240

[a]May vary by specific catheter and manufacturer.

(2) For patients with comorbidities and/or having a more invasive procedure, one to two 18- to 20-G IVs is likely adequate.
(3) An additional IV can be placed intraoperatively, provided there is reasonable access to the patient's arms, legs, or neck.
(4) Large-bore IV access (14-16 G) should be considered in situations with the potential for large blood loss or fluid shifts.

ii) For patient with difficult IV access, consider the following:
(1) Warming the extremity
(2) Ultrasound to assist in locating deeper veins
(3) External jugular or central venous access
(4) Inhalational induction with subsequent IV placement



For major surgery with significant potential for blood loss or fluid shifts, at least two large-bore IVs, 18-G or larger, should be placed.

b) **Selection of patient monitors**

i) Routine monitors
(1) ASA standard monitors should be used as minimum standards for all general, regional, and MAC anesthetic techniques. In addition to the continuous presence of a qualified anesthesia provider, ASA standard monitors include assessment of oxygenation, ventilation, circulation, and temperature.

ii) Special monitors
(1) Additional monitoring such as intra-arterial catheterization may be considered when consistent and continuous monitoring of blood pressure or frequent blood draws are needed (eg, hemodynamic management during neurosurgery, patients at risk for myocardial or end-organ ischemia). Arterial line monitors may also assist in goal-directed fluid therapy or an indirect assessment of cardiac function.
(2) Central venous access may be necessary to monitor central venous pressure (CVP) and to infuse pressors, fluids, or blood products. Central venous access also allows for placement of a Swan-Ganz catheter which may assist in assessment of cardiac function and pressure.
(3) Certain surgical procedures or patients may also require monitoring to assess neurologic function (eg, cerebral tumor resection, carotid surgery or certain spine procedures). The monitors may include EEG, EMG, SSEP, cerebral stump pressures, BIS, etc.



Poiseuille Law describes the flow of fluid through an IV catheter and states that the flow a fluid (Q) is related to the pressure gradient (P), the radius of the tube (r), the viscosity of the fluid (η), and the length of the tube (l).

READ MORE
Standard ASA monitors, Chapter 9
Invasive arterial blood pressure monitoring, Chapter 12
Intravenous induction agents, Chapter 30

c) **Induction of GA**

i) Induction of anesthesia is the administration of anesthetic drugs to induce a state of anesthesia.

(1) Typically, induction of anesthesia implies GA and inducing a state of unconsciousness.
(2) Alternatively, induction of anesthesia may be qualified as induction of neuraxial or regional anesthesia, where surgical anesthesia is induced but the patient remains conscious.

ii) Premedications prior to induction of GA
Whenever premedications are given, the patient should be in a monitored setting with emergency airway equipment and reversal medications available.
(1) Anxiolytic drugs (eg, benzodiazepines) can be administered to ease anxiety during transport to the operating room prior to induction.
(a) For pediatric patients, oral midazolam 0.5 mg/kg up to 20 mg. Some children may develop paradoxical hyperactivity.
(b) For adults, starting doses of 0.5-2 mg IV midazolam can be titrated upward to effect. Chronic benzodiazepine or heavy alcohol users may require larger doses. Decrease or consider omitting benzodiazepines in the elderly patients with obstructive sleep apnea (OSA) or those with neurological impairment.[2]
(2) Antiemetic drugs (eg, scopolamine) can be administered to prevent postoperative nausea and vomiting (PONV) in high-risk patients.
(a) Scopolamine transdermal patch (1.5 mg) is applied to the postauricular area to prevent PONV.
(i) It is an anticholinergic agent and common side effects include dry mouth, drowsiness, blurred vision, sedation, and dilated pupils.
(ii) It is contraindicated in patients <18 years of age and patients with glaucoma.[3]
(3) Antisialagogue drugs (eg, glycopyrrolate) can be useful for patients undergoing fiberoptic bronchoscopy (FOB).
(a) Glycopyrrolate is an anticholinergic (0.2-0.4 mg IV) used to decrease salivary, bronchial, and GI secretions.
(b) Unlike scopolamine, it is a quaternary amine, so it does not cross the blood–brain barrier and can cause central nervous system effects.[2]
(4) Ketamine can be used for its bronchodilating properties and therefore may be helpful for asthmatic patients. In addition, Ketamine has significant analgesic effects and may be especially useful in patients with chronic pain.
(5) Dexmedetomidine can cause significant bradycardia and hypotension but does not cause respiratory depression, so it can be a useful adjunct for an awake FOB.
(6) Agents can be administered prior to induction of GA to minimize the hemodynamic response to laryngoscopy and to improve postoperative pain control.[4,5]
(a) Opioids
(i) Fentanyl: 1.5-5 µg/kg IV bolus (titrate dose upward for chronic opioid users)
(ii) Remifentanil: 0.5-1 µg/kg IV bolus followed by infusion of 0.05-0.25 µg/kg/min
(iii) Alfentanil: 10-50 µg/kg
(b) Beta-adrenergic antagonists
(i) Esmolol: 0.25-0.5 mg/kg IV bolus
(c) Local anesthetics
(i) Lidocaine: 1 mg/kg may assist with both providing analgesia for tracheal intubation and decreasing the painful sensation of some induction agents (eg, propofol).

READ MORE
Opioids, Chapter 215
Endotracheal intubation and Fiberoptic intubation, Chapters 20 and 23

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(ii) Bupivacaine or ropivacaine: preoperative nerve blocks may be performed to improve postoperative pain and decrease the need for opioid medications.

(d) Acetaminophen or nonsteriodal anti-inflammatory drugs (NSAIDS) (eg, Celebrex) may be dosed po to improve postoperative pain and minimize the need for opioid medications.



Blood pressure should be measured every minute during induction to monitor for signs of hemodynamic instability.

iii) IV induction of GA

(1) A wide variety of IV induction agents are available, each of which have advantages and disadvantages that are reflective of the unique pharmacokinetic and pharmacodynamic differences.

(a) See chapter on Intravenous Induction Agents for a detailed discussion of these drugs.

(2) Standard IV induction of GA

(a) Place standard ASA monitors, special monitoring if indicated.

(b) Position patient in proper "sniffing position."

(c) Obtain baseline vital signs prior to induction. Consider measuring blood pressure every 1-2 minutes during induction and intubation.

(d) Confirm MSMAIDS algorithm (**Table 3-3**).

(e) Preoxygenate for eight deep breaths over 1 minute or 100% FiO_2 for 3 minutes to ensure denitrogenation (replacement of air with oxygen).[6,7]

(f) Titrate IV induction agent to effect.

(i) Initial induction dose administered is estimated based on weight and other factors such as age and premedication.

(ii) Additional small doses may be administered if the initial dose does not induce unconsciousness.

(iii) Testing of eyelash reflex (reflex motion of the eyelid to stroking of the eyelash) can aid in assessing level of consciousness.

(g) Neuromuscular blocking agent

(i) Unless a rapid sequence intubation (RSI) is being performed, confirmation of the ability to ventilate the patient with positive-pressure mask ventilation prior to administering neuromuscular blocking agent is advised.

TABLE 3-3 MSMAIDS Algorithm Prior to Induction

Machine checked	• Confirm high flow oxygen present • Ambu bag or device to provide positive-pressure mask ventilation present • Anesthesia machine functional
Suction on	• Yankauer suction catheter at patient's head • Audible and palpable suction present at catheter tip
Monitors on	• Current vital signs are displayed • Know patient's baseline vital signs • Noninvasive blood pressure every 1-2 min
Airway equipment	• Oral airways immediately available • Intubating equipment ready
IV access	• IV runs freely to gravity into patient • Adequate amount of IV fluid in bag
Drugs	• Induction agent • Neuromuscular blocking agent • Analgesic agent • Emergency drugs (eg, ephedrine, phenylephrine)
Special	• Any extra equipment that is anticipated for case

(ii) Supraglottic airway devices such as LMAs usually do not require neuromuscular blocking agents for placement.

(h) Mask ventilation

(i) Provide positive-pressure mask ventilation to ensure adequate oxygenation and ventilation during induction.

(ii) Please refer to mask ventilation cognitive aid for additional instruction.

READ MORE
Atlas of anesthesia procedures: mask ventilation, Chapter 160

(i) Airway instrumentation

(i) Once optimal intubating conditions have been achieved through adequate neuromuscular blockade, airway instrumentation and intubation can proceed.

(ii) Placement of supraglottic airway devices can proceed after adequate induction of anesthesia.

(3) Rapid sequence intubation (RSI)

(a) A method of rapidly inducing GA and neuromuscular blockade to quickly provide optimal intubating conditions while minimizing risk of aspiration.

(b) Indications for RSI include:

(i) Gastroparesis

(ii) Acute trauma

(iii) Ileus or "acute abdomen"

(iv) Full stomach (**Table 3-4**)

(v) Gastroesophageal reflux disease (GERD), for example, hiatal hernia and pregnancy beyond first trimester

(c) Performing RSI

(i) Premedication for aspiration prophylaxis in high-risk patients.

1. Nonparticulate antacid (eg, 0.3 M sodium citrate 30 cc)
2. Metoclopramide 10 mg IV
3. H_2 receptor antagonist (eg, ranitidine 50 mg IV)

(ii) Confirm MSMAIDS algorithm (**Table 3-3**).

(iii) Place standard ASA monitors, special monitoring if indicated.

(iv) Position patient in proper "sniffing position."

(v) Baseline vital signs should be obtained prior to induction. Consider measuring blood pressure every 1-2 minutes during induction and intubation.

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Endotracheal intubation and Fiberoptic intubation, Chapters 20 and 23

TABLE 3-4 ASA Fasting Guidelines to Reduce Risk of Pulmonary Aspiration

Ingested Material	Minimum Fasting Period (h)
Clear liquids	2
Breast milk	4
Infant formula	6
Nonhuman milk	6
Light meal[a]	6
Heavy meal[b]	8

[a]Clear liquid and toast.
[b]Fried or fatty foods.
Reprinted with permission from ASA Taskforce on Preoperative Fasting. Practice guidelines for preoperative fasting and the use of pharmacologic agents to reduce the risk of pulmonary aspiration: application to healthy patients undergoing elective procedures. *Anesthesiology*. 2017;126:376-393. Ref. [8]

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(vi) Preoxygenation for eight deep breaths over 1 minute or 100% FiO_2 for 3 minutes to ensure adequate denitrogenation.[6,7]

(vii) Apply cricoid pressure, which is a technique performed by an assistant and used to occlude the esophagus during intubation with the goal of preventing passive regurgitation of gastric contents and to decrease the possibility of pulmonary aspiration
1. It has been suggested to apply 10 Newtons (N) of force to the awake patient and 30 N to the unconscious patient.[9]
2. If cricoid pressure obstructs the laryngoscopic view, pressure should be released.



If cricoid pressure obstructs the laryngoscopic view of the ETT through the vocal cords into the trachea, cricoid pressure should be modified or released. In addition, cricoid pressure should be released with active emesis.

(viii) Administer precalculated dose of induction agent (ie, no titration).
1. Suggested dosing for hemodynamically stable healthy adults includes propofol 2-3 mg/kg IV.
2. For hemodynamically compromised patients, consider etomidate 10 mg IV.



The 10 N of force required for cricoid pressure is the amount of pressure required to depress the cartilage on the tip of your nose.

(ix) Follow immediately with neuromuscular blockade: succinylcholine if not contraindicated 0.6-1 mg/kg[10] or rocuronium 1.2 mg/kg IV.
1. Use of 1.5-2 times the effective intubating dose (ED_{95}) of a neuromuscular blocking agent can be used (at the risk/expense of prolonged paralysis—which may be an issue if the patient is difficult to intubate or mask ventilate).

(x) Mask ventilation is not performed prior to first laryngoscopy.

(xi) Cricoid pressure should not be released until ETT placement is confirmed by auscultation and capnography.

(4) Modified RSI
- (a) The potential risks of RSI (inability to ventilate, hemodynamic instability, trauma to the airway) may not be warranted in patients who are at low risk for pulmonary aspiration or other risks such as hemodynamic instability or a difficult airway are present.
- (b) In these cases, a modified RSI can be performed, which minimizes these risks to the patient.
- (c) The RSI technique is most commonly modified by
  - (i) Performing gentle positive-pressure ventilation before tracheal intubation.
  - (ii) Ideally, peak pressures should be <20 mm Hg (closing pressure of the lower esophageal sphincter).

iv) Inhalational induction
- (1) Anesthesia is induced by inhalation of volatile anesthetic gases.
- (2) Commonly used in pediatric patients who cannot tolerate IV line placement prior to induction of anesthesia.
- (3) May also be used in adult patients who present with:
  - (a) Difficult IV access
  - (b) Severe anxiety and phobia of needles precluding IV placement
  - (c) Airway anatomy such as airway tissues that may collapse and cause obstruction with loss of spontaneous ventilation
- (4) Cannot be used as the induction agent for RSI

(5) Technique
  (a) Follow standard IV induction technique as described above.
  (b) Instead of administering IV induction agent, slowly administer an increasing dose of volatile anesthetic.
    (i) Sevoflurane is a desirable agent because it has a less pungent odor than other volatile anesthetics and decreases airway irritation.
    (ii) Dose is gradually increased until patient loses consciousness.
    (iii) To avoid inadvertent overdose by forgetting to turn down the anesthetic dose after induction, it is advisable to maintain contact with vaporizer dial until GA is induced and then dial down anesthetic dose prior to removing your hand.
    (iv) A N_2O/O_2/sevoflurane induction can be used, which will provide a more rapid inhalational induction due to the second gas effect, but this must be weighed against the benefit of inducing a patient on 100% FiO_2.
  (c) Once the patient is induced, you may establish IV access and proceed with your anesthetic plan.



Maintain physical contact with the vaporizer dial any time your dial is set above 1.2 MAC

d) **Maintenance of anesthetic plan**
  i) Maintenance of the anesthetic plan involves maintaining the state of surgical anesthesia for the duration of the procedure. Plans should be made for the maintenance of anesthesia during the surgical procedure.
  ii) This usually involves administration of volatile or IV anesthetic agents.
  iii) Different methods for maintenance of anesthesia have benefits and limitations that may be better suited to certain patients and procedures. This topic is discussed in depth in the "Intraoperative Management and Maintenance of Anesthesia" of Chapter 5.

e) **Emergence from anesthesia**
  i) Emergence from GA involves waking the patient up at the end of the procedure and removing the airway device in a manner that is safe and minimizes postoperative complications. Plans for emergence from anesthesia should be discussed with the surgeon to minimize postoperative complications.
    (1) Concerns may include whether the patient should be extubated fully awake or unconscious during stage III anesthesia (deep extubation).
  ii) A detailed discussion of this topic is covered in Chapter 6, Emergence and Postoperative Issues in Anesthesia.
  iii) PONV prophylaxis should be considered prior to emergence in at-risk patients.
  iv) Postoperative pain control should be considered with multimodal medication techniques. Analgesic medications may be titrated to respiratory rate or end-tidal CO_2 levels (eg, an $ETCO_2$ level of 45-50 should demonstrate both a certain degree of patient comfort and adequate ventilation.

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Postoperative nausea and vomiting, Chapter 129

READ MORE
Acute pain management, Chapter 132

4) **Postoperative care plan**
  a) Postoperative care involves managing the patient's recovery from anesthetic drugs and procedures and managing postsurgical pain. Plans for postoperative care should be discussed with the surgeon and the postoperative care unit nursing staff. Enhanced recovery after surgery (ERAS) is a systematic process addressing many aspects of surgical and anesthetic care to improve patient recovery. ERAS protocols will likely

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A detailed discussion of ERAS is covered in the chapter on "Emergence and postoperative issues in Anesthesia, Chapter 6."

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become an increased part of anesthetic care with an emphasis on multimodal pain management. Potential concerns include the following[11]:

i) Treatment of postoperative anesthetic complications such as shivering or nausea and vomiting
ii) Pain control
iii) Discharge to appropriate facilities based on the patient's condition (eg, home, ward, monitored bed)

CHAPTER SUMMARY FOR THE ANESTHETIC PLAN AND INDUCTION OF ANESTHESIA

Anesthetic Plan	The anesthetic plan describes how the anesthesiologist will conduct an individual care plan that is tailored to the patient and surgeon's specific needs while also insuring a safe perioperative experience. A successful anesthetic plan requires consideration of preoperative, intraoperative, and postoperative management.
Induction of Anesthesia	Induction of GA involves administering anesthetic drugs to induce a state of unconsciousness.
Induction Agents	GA can be induced using IV drugs or inhalational agents. IV agents are most commonly used for adults.
Induction Techniques	Standard induction techniques involve preoxygenation, administration of IV induction agent ± neuromuscular blocking agents, and ventilation of the patient prior to airway management. Rapid sequence intubation techniques are used to protect the patient from aspiration.

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4 Preanesthesia Assessment

Lauren Sutherland • Jonathan Hastie • Danielle B. Ludwin

The preanesthesia assessment is an essential part of perioperative care. This assessment is conducted by the anesthesiologist and includes a review of the patient's medical history, the performance of a focused physical examination, and a consideration of the context and nature of the proposed procedure. An anesthetic plan is formulated only after completion of these steps.

Adequate preanesthesia assessment allows for optimization of the patient's medical status, which may occur over days or weeks, and may be overseen by an anesthesiologist or other physician. The preanesthesia assessment is also an opportunity to explore patients' goals and provide education to patients and their families. This chapter reviews indications for preoperative testing, guidelines for the prevention of pulmonary aspiration, and the American Society of Anesthesiologists' (ASA) physical status classification. It also describes the model of the perioperative surgical home (PSH).

1) **Overview:** The ASA has published basic standards for a preanesthesia evaluation.[1]
   These are considered to be minimal acceptable requirements and include
   a) **Determining the medical status of the patient**
      i) Reviewing available medical records
      ii) Performing a history and physical examination
      iii) Pursuing further studies or consultations as indicated
   b) **Developing an anesthesia plan, including the following:**
      i) Obtaining consent for anesthesia care
      ii) Proper documentation in the medical record
      iii) Ordering premedication
2) **The medical history** allows the physician to assess the patient's current medical condition. Essential features include
   a) **Signs and symptoms related to the planned operation**
   b) **Surgical and anesthesia history, including personal and family history of anesthetic complications**
   c) **Focused review of systems (Table 4-1).**
   d) **Medications and allergies**
      i) Documentation should be complete and updated.
      ii) Use of generic medication names facilitates communication, particularly with combination therapy.
      iii) Include both nonprescription and herbal therapies.
      iv) Consider potential for drug interactions and side effects.
      v) Allergy history should include both medications and latex.
         The reaction to each allergen should be noted.
         (1) Remote and uncertain reaction may be documented accordingly.



Formulating an anesthetic plan requires a history, physical examination, and consideration of the proposed procedure.

Consent for anesthesia care typically occurs immediately following the preanesthesia assessment.

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Anesthetic plan and induction of anesthesia, Chapter 3

Allergy and anaphylaxis, Chapter 49

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TABLE 4-1 Basic Review of Systems

Neuromuscular: History of stroke or seizure. History of weakness or nerve injury.
Respiratory: History of asthma, chronic obstructive pulmonary disease (COPD). Obstructive sleep apnea diagnosis or risk factors, such as snoring or observed apnea. Recent respiratory tract infection.
Cardiac: History of hypertension or CAD. Heart failure symptoms: shortness of breath, orthopnea, paroxysmal nocturnal dyspnea. Palpitations, chest pain, or chest pressure. Exercise tolerance.
Renal: History of kidney disease. If on dialysis: type, date of last treatment, presence of fistula or indwelling catheter.
Gastric: History of gastroesophageal reflux disease (GERD) or hiatal hernia. Severity of symptoms.
Liver: History of liver disease or hepatitis. History of pancreatic disease.
Endocrine: History of diabetes or abnormal thyroid function. Weight changes. Heat or cold intolerance.
Hematologic: History of blood disorders, blood clots, or on anticoagulant medication.
Obstetric: For female patient, history of pregnancy and last menstrual period.
Social History: Alcohol or recreational drug use. Tobacco use.

3) **The physical examination** should generally confirm the information learned in the medical history and further characterize disease. In addition, the examination may yield new diagnoses or concerns. It should include the following components:
   a) **Vital signs**
   b) **Global examination (Table 4-2)**
   c) **Comprehensive airway assessment** (see 7.a. "Specific Considerations: Airway Examination").
   d) **Neurological examination**
      i) Mental status
      ii) Gross sensory and motor examination
      iii) Closer examination of focal neurological deficits
   e) **Cardiac examination**
      i) Heart rate and regularity
      ii) Auscultation for quality of heart sounds
      iii) Presence of murmurs, gallops, or rubs
      iv) Presence of subcutaneous pacemaker or other implantable cardiac device
   f) **Pulmonary examination**
      i) Respiratory effort and rate
      ii) Auscultation of lung sounds for presence of wheezes, rhonchi, or rales



The physical examination confirms the medical history, characterizes disease, and may also yield new diagnoses to the astute clinician.

TABLE 4-2 Global Examination

On entering a patient's room, the following can often be assessed with a moment of observation:
- General health and appearance: posture, eye contact, skin, and muscle tone
- Level of consciousness, mood, and state of distress
- Presence of family, friends, or caretakers
- Accessory devices, such as a cane to assist ambulation or supplemental oxygen
- Inpatient therapeutic devices
  - Vascular access devices
  - Drains
  - Ventilator

g) **General examination may include the following:**
   i) Abdominal examination
      (1) Evaluate for presence of distension or ascites.
      (2) Auscultate for bowel sounds.
      (3) Assess for tenderness or guarding.
   ii) Extremity and skin examination
      (1) Signs of peripheral perfusion: temperature, capillary refill
      (2) Peripheral edema
      (3) Skin breakdown and rashes
      (4) Topical medications and patches
   iii) Musculoskeletal examination
      (1) Gait
      (2) Orthopedic injuries or braces
      (3) Prosthetic devices

4) **Review any investigations that are available and decide whether additional testing is necessary.**
   a) Preoperative tests should not be ordered routinely, especially in a generally healthy patient.
   b) Tests may be ordered on a selective basis for optimizing perioperative management (**Table 4-3**).
   c) Invasiveness of the surgery should be considered before ordering a test; preoperative testing is not generally indicated for low-risk procedures but may be reasonable prior to a high-risk procedure.
   d) Laboratory assessments are indicated for select patient groups, such as:
      i) An electrolyte panel for patients on diuretics or angiotensin-converting enzyme (ACE) inhibitors
      ii) Electrolytes with blood urea nitrogen (BUN) and creatinine for patients with cardiac, liver, or kidney disease
      iii) A complete blood count (CBC) for patients with hematologic disease or patients at the extremes of age
      iv) Electrolytes with BUN and creatinine, CBC, hepatic function tests, and coagulation studies for patients with liver disease
      v) Blood glucose level for patients with diabetes
   e) A chest radiograph should be considered for patients with acute or chronic pulmonary disease, patients who smoke, and those with recent upper respiratory infections.
   f) An electrocardiogram (ECG) should be obtained for patients with a known history of cardiovascular or pulmonary disease, for those with advanced age, and prior to more invasive surgeries. An ECG may be considered for patients with coronary artery disease (CAD) risk factors, such as hypertension, diabetes, or kidney disease.
   g) Other available imaging studies or cardiac testing results

5) **Review available prior anesthetic records for the following concerns:**
   a) **Airway complications**
      i) Difficulty securing the airway
      ii) Airway reactivity
      iii) Airway or dental injury
   b) **Anesthesia complications**
      i) Allergic and medication reactions
      ii) Hemodynamic instability
      iii) Intraoperative awareness



A chest radiograph should be considered for patients with cardiac or pulmonary disease.

An ECG should be obtained for patients with cardiovascular or pulmonary disease. An ECG is also indicated for patients with advanced age, and prior to more invasive surgeries.

Every investigation should be guided by an analysis of the potential benefits of additional information while weighing the cost and possible risks.



Always review any available anesthetic records for a history of difficult airway or other complications.

The urgency of a surgical procedure may preclude consultation with medical specialists.

A L G r a w a n y

TABLE 4-3 Responders to a Survey of ASA Members Typically Consider Ordering Tests Based on the Indications Listed

A. Electrocardiogram
   1. **Advanced age**
   2. **Known cardiac disease**
   3. **Known pulmonary disease**
B. Other cardiac evaluation (eg, stress test)
   1. Cardiovascular compromise
C. Chest radiograph
   1. Known pulmonary disease
   2. Smoking
   3. Recent respiratory tract infection
   4. Known cardiac disease
D. Pulmonary function tests
   1. **Reactive airway disease**
   2. **Chronic obstructive pulmonary disease (COPD)**
   3. **Restrictive lung disease, such as scoliosis**
E. Office spirometry (ie, portable spirometer)
   1. Reactive airway disease
   2. COPD
   3. Restrictive lung disease (eg, scoliosis)
F. Hemoglobin and hematocrit
   1. **Extremes of age (i.e., very young or old)**
   2. **Anemia**
   3. **Liver disease**
   4. **Bleeding disorders**
G. Coagulation studies
   1. Bleeding disorders
   2. Kidney disease
   3. Liver disease
   4. Anticoagulant medications
H. Serum electrolytes
   1. **Endocrine disease**
   2. **Kidney disease**
   3. **Certain medications**
I. Pregnancy test
   1. **Consider for *all* female patients of childbearing age**
   2. **Unclear pregnancy history**
   3. **Pregnancy likely by history**

The ASA Taskforce recommends testing based *only* on the **bolded** indications.

Adapted from Practice Advisory for Preanesthesia Evaluation. *Anesthesiology*. 2002;96:485-496.

iv) Delayed awakening
v) Postoperative nausea and vomiting
vi) Postoperative delirium

c) **Surgical complications**
i) Bleeding
ii) Infection

d) **Difficult postoperative pain management**

6) **Indications for additional information**
a) Further laboratory investigations, physiological tests, or imaging studies should only be requested as guided by the history and physical examination.
b) No specific study is required for every patient undergoing anesthesia care.
c) Every investigation should be guided by an analysis of the potential benefit of additional information (**Table 4-3**). This is weighed against the cost and possible risks, such as procedure delays or hazards inherent to the study.[1]

d) Consider consultation with medical specialists if the adequacy of disease management is unclear. The urgency of a procedure may preclude consultation.

7) **Specific considerations**

a) **Airway examination.** This remains the most crucial component of the physical examination because it determines how the airway is managed throughout the perioperative period.

i) Components and predictors of difficult airway

(1) High Mallampati classification (**Fig. 4-1**)

(2) Small mouth opening

(3) Prominent upper incisors

(4) Thyromental distance <6 cm

(5) Decreased neck extension

ii) Mallampati classification scheme (**Fig. 4-1**), which categorizes the approximate ratio of the size of the tongue to the oropharynx

iii) The anesthesiologist's composite impression of the airway is more predictive than any one component of the ease or difficulty in securing an endotracheal tube.[2]

iv) Other considerations

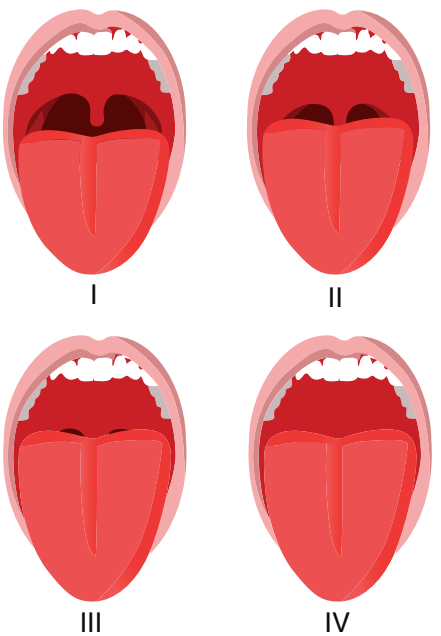


Consider a pregnancy test for all female patients of childbearing age.



The anesthesiologist is a perioperative medicine consultant who actively assesses disease severity, evaluates adequacy of management, and detects unrecognized disease.

FIGURE 4-1 Mallampati Classification Scheme



Mallampati classification scheme used to describe findings on the airway examination. Grade I—soft palate, uvula, and tonsillar pillars seen. Grade II—soft palate and uvula seen. Grade III—soft palate and base of uvula seen. Grade IV—hard palate only seen. Reprinted with permission from Johnson J. *Bailey's Head and Neck Surgery*. 5th ed. Philadelphia, PA: Wolters Kluwer; 2013.

A L G r a w a n y

(1) Consider cervical spinal cord compromise based on history and physical examination. If evidence of spinal cord involvement exists, care should be taken to protect the spinal cord during manipulation of the airway, such as by cervical spine collar, manual in-line stabilization, or fiberoptic intubation.

(2) Surgical procedure considerations should include route of intubation (nasal or oral). The type of endotracheal tube (e.g., RAE, reinforced, or laser-safe) will be determined by the procedure. Thoracic and procedures involving the descending thoracic aorta may require lung isolation with a double-lumen endotracheal tube.

(3) In patients with preexisting airways (such as an endotracheal or tracheostomy tube), document the following:
   (a) Indication and date of placement
   (b) Ease of placement
   (c) Size of device and depth of device
   (d) Presence or absence of inner cannula and cuff

READ MORE
Difficult airway management, Chapter 21



If evidence of cervical spinal cord compromise, the spinal cord should be protected during manipulation of the airway with techniques such as manual in-line stabilization, keeping a cervical spine collar in place, or fiberoptic intubation.

b) **Prevention of pulmonary aspiration**

i) Pulmonary aspiration of gastric contents may cause significant complications, including pneumonitis, superimposed infection, prolonged intubation, and death.

ii) The ASA has published guidelines for the prevention of aspiration of gastric contents (**Table 4-4**).[3]

iii) If surgery is urgent, a risk-benefit analysis should inform the decision to proceed or delay the operation. Clinical judgment in a particular context takes priority over guidelines. Appropriate documentation should be made.

iv) These guidelines are intended for healthy patients. Disease or physiological states that lead to increased gastric volume or acidity may prompt the anesthesiologist to practice with an increased level of caution.

TABLE 4-4 *Nil Per Os* (NPO) Guidelines and Pulmonary Aspiration Precautions, Summarized from ASA Published Practice Guidelines

Preanesthesia assessment: History, physical examination, review of available medical records. Screen for GERD, dysphagia, and diseases of gastric motility, which may increase the risk of aspiration.	
Fasting Guidelines	
Clear fluids	At least 2 h for all patients
Breast milk	4 h for neonates and infants
Infant formula	6 h
Light food (toast, crackers, fruit)	6 h
Heavy food (fatty foods, meat)	8 h
Medications: Not recommended for routine preoperative use in patients without increased risk of pulmonary aspiration.[3]	
Gastric motility agents	Reduce gastric volume, but not acidity
Inhibitors of acid secretion	Reduce gastric volume and acidity
Antacids	Reduce gastric acidity, but not volume. If indicated for other reasons, use *nonparticulate* antacids.
Antiemetics	Reduce nausea/vomiting after surgery
Anticholinergics	Do not reduce gastric volume or acidity

Adapted from Practice Advisory for Preanesthesia Evaluation. *Anesthesiology*. 2002;96:485-496.

8) **Cardiac disease (Table 4-5)**
   a) The American College of Cardiology (ACC) and American Heart Association (AHA) have jointly published guidelines for a perioperative cardiac examination for patients undergoing noncardiac surgery.[4]
      i) Urgency of a surgical procedure should be considered.
         (1) Emergent—one in which life or limb is threatened if not in the operating room within <6 hours.
         (2) Urgent—there may be limited time for a clinical evaluation, usually when life or limb is threatened if not in the OR within 6-24 hours.
         (3) Time-sensitive—one in which a delay of >1 to 6 weeks to allow for an evaluation and significant changes in management will negatively affect outcome.
         (4) Elective—one in which the procedure could be delayed for up to 1 year.
   b) A stepwise approach to assessment for risk of major cardiac adverse events (MACE) in patients with risk factors for or known CAD (**Fig. 4-2**):
      i) Emergency surgery should proceed with determination of clinical risk factors and appropriate monitoring and management based on this assessment.
      ii) Patients with acute coronary syndromes including myocardial infarction or unstable angina should be referred to cardiology for evaluation and management.
      iii) For patients with stable CAD risk factors, a risk assessment should be performed using a tool described in section (f) below. These tools use both surgical and patient factors.
         (1) Surgical risk is classified as either low risk or high risk:
            (a) Low risk—one in which the combined surgical and patient characteristics predict a risk of MACE < 1%
               (i) Examples include cataract and plastic surgery.
               (ii) Patients undergoing very-low-risk surgery, even with multiple risk factors, have low risk of MACE.

READ MORE
Ischemic heart disease and revascularization procedures, Chapter 98
Valvular heart disease, Chapters 99–102
Aortic valve disease, Chapter 100



Patients with good preoperative functional status have a lower risk of perioperative complications.

READ MORE
Pulmonary hypertension, Chapter 48

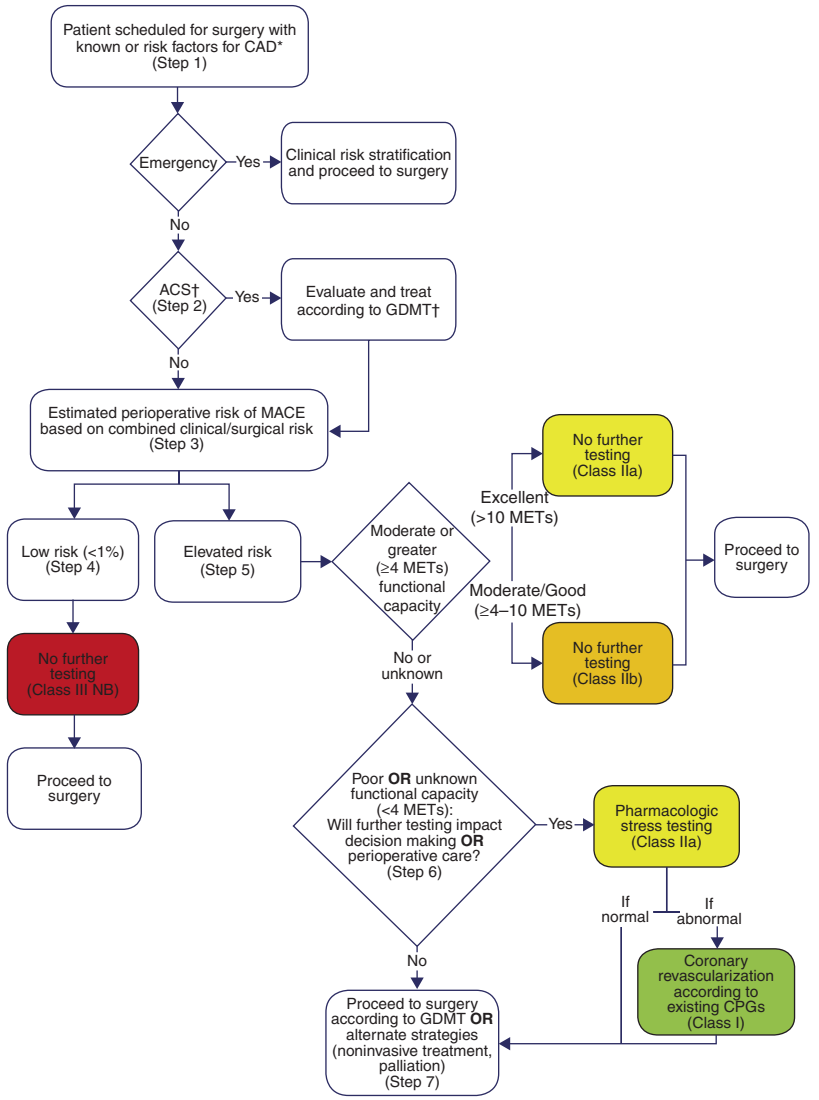
TABLE 4-5 Screening Evaluation for the Cardiovascular System

History:
- Uncontrolled hypertension
- Unstable cardiac disease
  - Myocardial ischemia
  - Congestive heart failure
  - Valvular heart disease
  - Cardiac dysrhythmias
- Symptoms including chest pain, dyspnea, or syncope
- Presence of coronary stents or implantable cardiac devices

Physical Examination:
- Blood pressure elevation
- Auscultation of the heart
- Carotid bruits
- Peripheral pulses

Reproduced from Hata TM, Moyers JR. Preoperative patient assessment and management. In: Barash PG, Cullen BF, Stoelting RK, eds. *Clinical Anesthesia*. 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2013:583-611, Ref.[5]

FIGURE 4-2 Stepwise Approach to Perioperative Cardiac Assessment for CAD



Reprinted from Fleisher LA, Fleischmann KE, Auerbach AD, et al. 2014. ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: a report of the American College of Cardiology/American Heart Association Task Force on practice guidelines. *J Am Coll Cardiol*. 2014;64(22):77, with permission from Elsevier.

(b) Elevated risk—one with a risk of MACE ≥ 1%

(i) A patient undergoing major vascular surgery, even with few risk factors, has an elevated risk of MACE.

(ii) Risk can be lowered by a less invasive approach (eg, endovascular repair of an abdominal aortic aneurysm).

(2) Functional status is a reliable predictor of perioperative and long-term cardiac events.

(a) Functional capacity is often expressed in terms of metabolic equivalents (METs), where 1 MET is the resting oxygen consumption of a 40-year-old, 70-kg man.

TABLE 4-6 METs of Various Activities That Should Be Used for Assessment of Exercise Tolerance

1 MET	Watching television
4 METs	General self-care activities such as dressing or using the toilet
5-10 METs	• Walk 1-2 blocks on level ground at a slow pace • Light housework • Climbing a flight of stairs • Heavy housework such as moving furniture or scrubbing floors • Moderate recreational activities such as golf, doubles tennis, or throwing a football • Vigorous exercise such as running or swimming

Adapted from Fleisher LA, Beckman JA, Brown KA, et al. ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol.* 2007;50:e159-e242, with permission from Elsevier.

(b) Functional capacity is classified as excellent (>10 METs), good (7-10 METs), moderate (4-6 METs), poor (<4 METs), or unknown (**Table 4-6**).
(c) Patients with reduced functional status preoperatively are at increased risk of complications.
(d) Conversely, patients with good functional status preoperatively are at lower risk. In these patients, it is often appropriate to proceed to surgery without further cardiac testing.

(3) In cases with poor or unknown exercise tolerance, pharmacological stress testing is reasonable if the result will impact decision-making or perioperative care.
(4) If stress testing is abnormal, coronary revascularization should be considered prior to proceeding with noncardiac surgery.
(5) Other preoperative assessment of heart function and stress testing prior to elevated-risk surgery is reasonable in patients with elevated risk, poor or unknown functional capacity, or a worsening in clinical status.

c) **Heart Failure:** Patients with clinical symptoms or physical examination findings of heart failure, as well as a severely decreased left ventricular ejection fraction (<30%), are at significantly elevated risk for perioperative complications in noncardiac surgery.
d) **Valvular Disease:** Significant valvular heart disease increases cardiac risk for patients undergoing noncardiac surgery.
i) Patients with suspected moderate or severe valvular abnormalities should undergo preoperative echocardiography to quantify severity of valve lesion, degree of ventricular dysfunction, and estimation of pulmonary arterial pressure.
ii) Perioperative risk of noncardiac surgery is decreased by performing indicated interventions on valves (eg, transcatheter aortic valve replacement).
iii) It may be reasonable to perform elective procedures with appropriate monitoring in patients with severe, asymptomatic valvular disease.
e) **Arrhythmia:** The presence of arrhythmia in the preoperative setting should prompt investigation into underlying cardiopulmonary disease, myocardial ischemia, drug toxicity, or metabolic derangements. However, it is unclear whether a preoperative arrhythmia increases perioperative risk.
f) **Validated tools for predicting MACE in noncardiac surgery:**
i) Revised Cardiac Risk Index (RCRI) is a simple, validated tool to assess perioperative risk of major cardiac complications.
(1) Six predictors are assessed:
(a) Serum creatinine ≥2 mg/dL
(b) History of heart failure
(c) Insulin-dependent diabetes mellitus

(d) History of cerebrovascular accident or TIA
(e) History of ischemic heart disease
(f) High-risk surgical procedure
(2) Risk assessment:
(a) The presence of 0 or 1 predictors confers low risk of MACE
(b) ≥2 predictors have elevated risk

ii) American College of Surgeons National Surgical Quality Improvement Program Myocardial Infarction Cardiac Arrest (NSQIP MICA) is a tool that specifically determines risk based on surgical site as well as increasing age, creatinine >1.5 mg/dL, and functional status.

iii) American College of Surgeons NSQIP Surgical Risk Calculator determines a procedure-specific risk assessment based on 21 patient-specific variables, and from these calculates the risk of MACE, death, and 8 other outcomes.

9) **Pulmonary disease**

a) A history, physical examination, and review of available records will give the anesthesiologist information about a patient's pulmonary status (**Table 4-7**).

b) The anesthesiologist should form an independent assessment of the presence and severity of disease based on all available information.

c) The presence of pulmonary disease has significant bearing on anesthetic technique selection and patient counseling about intraoperative risk and postoperative course.

d) A pulmonary function test is generally indicated for severe reactive airway disease or significant obstructive or restrictive pulmonary disease.[1]

e) For compressing intrathoracic or mediastinal masses, further workup is indicated, including a thoracic computed tomography and flow-volume loops.



Pulmonary function tests are generally indicated for severe reactive airway disease or significant obstructive or restrictive pulmonary disease.

10) **ASA physical status classification**[6]

a) At the conclusion of the preanesthesia assessment, the anesthesiologist should classify the patient according to his or her ASA physical status. ASA physical status definitions are broad by design; the anesthesiologist should exercise judgment and has some latitude in assigning classification.

i) ASA 1—Normal, healthy patient

The ASA physical status classification is a general assessment of a patient's health that is useful for concise communication.

TABLE 4-7 Screening Evaluation for the Pulmonary System

History
- Tobacco use
- Dyspnea (at rest or exertion)
- Exercise tolerance
- Cough, wheezing, or stridor
- Inhaler use
- Recent respiratory tract infection
- Snoring or sleep apnea

Physical examination
- Respiratory rate
- Chest excursion
- Use of accessory muscles
- Nail shape and color
- Ability to walk or talk without dyspnea
- Auscultation for decreased breath sounds, wheezing, stridor, or rales

Reproduced with permission from Hata TM, Moyers JR. Preoperative evaluation and management. In: Barash PG, Cullen BF, Stoelting RK, eds. *Handbook of Clinical Anesthesia*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2006:275, with permission.

ii) ASA 2—Patient with mild systemic disease (eg, well-controlled hypertension)
iii) ASA 3—Patient with severe systemic disease (eg, end-stage renal disease undergoing regularly scheduled dialysis)
iv) ASA 4—Patient with life-threatening systemic disease (eg, severe aortic stenosis)
v) ASA 5—Patient not expected to survive without the operation (eg, ruptured abdominal aortic aneurysm)
vi) ASA 6—Brain-dead patient for organ harvest

11) **The Perioperative Surgical Home**
a) The PSH is a patient-centered approach that aims to coordinate care and guide patients through the surgical experience.
i) The PSH is physician-led and incorporates a multidisciplinary team.[7]
ii) The goal is to increase the health care system's *value* by improving clinical outcomes and perioperative services at a lower cost.
iii) Variability in perioperative care increases the likelihood for errors and complications. Thorough perioperative planning and emphasis on continuity and coordination can decrease variability.
(1) One team, potentially led by an anesthesiologist, manages all aspects of care, from the decision to undergo surgery until up to 30 days following discharge.
(2) Patients are informed, educated, and involved in decision-making and treatment planning throughout the process.
iv) Key features of the PSH at different stages of the continuum include:
(1) Decision to Operate
(a) Patient information, education, and expectation management
(b) Shared decision-making to outline best treatment course
(c) Discharge planning
(d) Referral to classes for optimal healing strategies
(2) Preoperative
(a) Early anesthesia intervention (ie, preoperative health and risk assessment)
(b) Shared decision-making to outline the best course of anesthesia care, recovery, and pain management
(c) Preoperative prescriptions and instructions
(d) Tailored optimization of health conditions
(3) Intraoperative
(a) Standardized protocols for anesthesia care
(b) Standardized equipment and nursing protocols
(c) Infection prevention strategies
(d) Optimization of fluid management
(e) Minimal use of drains, tubes, and catheters
(f) Multimodal analgesia
(4) Postoperative
(a) Targeted recovery plan
(b) Early ambulation including physical and occupational therapy
(c) Multimodal analgesia
(d) Early removal of drains, tubes, and catheters
(e) Early intervention for deviations from recovery goals
(f) Discharge readiness protocols
(5) Postdischarge
(a) Personal recovery pathway
(b) Early remote follow-up
(c) Home health management
(d) Physical and occupational therapy
(e) Timely return to normal activity and work

CHAPTER SUMMARY FOR PREANESTHESIA EVALUATION

General Principles	A comprehensive evaluation, including history, review of medications and allergies, review of systems, and focused physical examination should be performed for all patients. The anesthesiologist should actively screen for disease during the preanesthesia evaluation. In the presence of known disease, an independent assessment of disease severity and management should be formed. Investigations (labs, imaging) should be guided by an analysis of potential benefits and weighed against the cost and possible risks. Specific consideration must be made to the airway examination to anticipate difficulty with endotracheal intubation. Prevention of pulmonary aspiration requires consideration of NPO guidelines. Screening for and managing cardiac and pulmonary disease requires special attention. All patients should be categorized according to the ASA physical status classification. The perioperative surgical home is an emerging framework to obtain patient satisfaction and desired outcomes for patient care in the perioperative period.
Elective Procedures	Require a comprehensive preanesthesia assessment. The patient's medical status should be optimal. However, minor procedures, such as ophthalmologic, endoscopic, or superficial procedures can often be performed without delay for optimization.
Urgent Procedures	Anesthesia assessment may be more focused, and management will require medical judgment. Decision-making should be described with appropriate documentation.

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5 Intraoperative Management and Maintenance of Anesthesia

Jed Wolpaw • Arthur Hertling

Intraoperative management occurs after induction and prior to emergence from anesthesia. The goals are to maintain surgical anesthesia and physiologic homeostasis. Multiple different techniques may be used and are discussed in this chapter.

1) **Overview**
   a) **Intraoperative management and maintenance** of anesthesia is defined as the management of physiological functions and the maintenance of surgical anesthesia following induction and until emergence.
   b) **Several goals include:**
      i) Maintenance of surgical anesthesia
      ii) Maintenance of physiologic homeostasis
         (1) Hemodynamic parameters (BP, HR) are optimized to ensure adequate tissue perfusion.
         (2) Patients under general anesthesia (GA) are unable to protect their airway and often have an endotracheal tube or a supraglottic airway inserted during induction. Adequate ventilation and oxygenation must be ensured.
         (3) An adequate urine output may be used as a surrogate for euvolemia and end-organ perfusion but should not be used in isolation as other factors can lead to reduced urine output, such as release of antidiuretic hormone during surgery and surgical compression of ureters.
         (4) Plasma electrolytes and glucose levels must be maintained within physiological limits, especially in diabetics and patients undergoing large fluid shifts.
         (5) Temperature maintenance is of critical importance. Active warming is used to compensate for the loss of thermal homeostasis under GA.
      iii) Monitoring is needed to ensure that these goals are met.
2) **Maintenance of surgical anesthesia** can be accomplished by the intravenous (IV) and/or inhaled administration of medications in order to provide temporary unconsciousness (hypnosis), analgesia, immobility, and, if needed, muscle relaxation. **Table 5-1** compares balanced and total IV methods of anesthesia maintenance.
   a) **Hypnosis**
      i) These include inhalation agents, benzodiazepines, propofol, and etomidate. These agents act primarily at the thalamic and cortical levels, and their main activity is on the GABA receptor (with the exception of ketamine, which acts as an *N*-methyl-D-aspartate (NMDA) receptor antagonist).
      ii) Amnesia accompanies unconsciousness, although benzodiazepines can provide amnesia while the patient is merely sedated.
   b) **Analgesia**
      i) Painful surgical stimuli have a number of physiological effects.



Maintenance of surgical anesthesia can be accomplished by the IV and/or inhaled administration of medications in order to provide temporary unconsciousness (hypnosis), analgesia, immobility, and, if needed, muscle relaxation.

TABLE 5-1 Comparison of Balanced and IV Techniques of Maintenance of General Anesthesia

Technique	Balanced Anesthesia	Total Intravenous Anesthesia
Inhaled vs IV	Combines inhaled and IV agents	Only IV agents
Pharmacokinetics	Inhalation anesthetic elimination is independent of hepatic or renal function	Hepatic or renal insufficiency can significantly prolong drug effect
Titration	Easily adjustable, especially with newer poorly soluble agents (desflurane, sevoflurane)	More difficult to titrate, need to adjust infusion rate over time, occasional delayed awakening
Post-op analgesia	Inhalation anesthetics do not provide postoperative analgesia	Postoperative analgesia by intermediate- or long-acting opioids
Effect on PONV, awakening	Higher incidence of PONV, emergence agitation, postoperative shivering	Recommended if history of PONV; smoother awakening
Cost	Low cost if low fresh gas flow is used	Higher cost
Environmental effects	Potential for atmospheric pollution, destruction of the ozone layer	Potential for water pollution by phenol metabolites

(1) They induce a neurohumoral response with activation of the sympathetic nervous system (hypertension, tachycardia), the coagulation cascade (hypercoagulable state), and the stress hormone release (hyperglycemia, protein catabolism, immunosuppression).
(2) Postoperative splinting and ileus and increased strain on the heart due to increased oxygen demand are also among the side effects of inadequate analgesia.



There are multiple detrimental physiologic effects of painful surgical stimuli, including sympathetic overdrive, hypercoagulability, hyperglycemia, and immunosuppression.

ii) The blockade of nociception can be achieved by a conduction block (peripheral nerve or central neuraxial blockade) or, pharmacologically, via agents acting primarily at the level of the spinal cord and the brainstem.
(1) Agents include opioids, nitrous oxide, central alpha-2 agonists, and ketamine.
(2) Regional anesthesia is superior to opioid administration to block the neurohumoral response to surgical stress.



Regional anesthesia is superior to opioid administration to block the neurohumoral response to surgical stress.

iii) At this time, however, there is no clinically validated monitor of the adequacy of analgesia provided during general anesthesia,[1] and clinicians have to rely on surrogates such as heart rate, blood pressure, patient movement, respiratory rate, pupillary size, and responsiveness.
iv) Analgesic administration should take into account the fact that analgesia should extend into the postoperative period.
(1) Short-acting opioids such as remifentanil may be adequate intraoperatively, but medications with a longer half-life may need to be titrated at the end of the procedure, depending on how much postoperative pain is expected and on whether the patient is likely to be tolerant to opioids.



ERAS protocols reduce length of stay and improve patient satisfaction in part by reducing opioid use.

v) Early recovery after surgery (ERAS) protocols use a multifaceted approach to improve outcomes, such as patient satisfaction, postoperative nausea and vomiting, hospital length of stay, and cost. One of the mainstays of these protocols is reducing opioid consumption through use of regional anesthesia; multimodal regimens utilizing nonopioid

medications, such as acetaminophen and nonsteroidal anti-inflammatory drugs (NSAIDs); and use of minimally invasive surgical techniques.[2]



Analgesic administration should take into account the fact that analgesia should extend into the postoperative period.

c) **Immobility** can be achieved with deeper planes of anesthesia that cause a decrease in muscle tone.
   i) Medications that block transmission at the neuromuscular junction, or neuromuscular blockade (NMB), are used to provide complete muscle relaxation.
   ii) NMB is useful for tracheal intubation and should be considered for any surgical procedure where movement of the patient could be detrimental (eg, cerebral aneurysm surgery). **Table 5-2** summarizes the main indications for NMB.

3) **Monitoring and maintenance of physiologic homeostasis**
   a) **Cardiovascular**
      i) For normotensive patients, euvolemia should be maintained, taking into account basic metabolism, blood loss, insensible fluid losses, and urine output.
      ii) Hypertensive patients tend to be volume depleted and become hypotensive as the vascular tone is reduced under GA.
         (1) In these patients, blood pressure is typically labile, and wide swings without clear triggering factors are common.
         (2) Diuretics should be held on the day of surgery to limit hypovolemia unless the patient is at high risk for volume overload and heart failure.
         (3) Hypotension in patients on ACE inhibitors can be refractory to vasopressors.[3]



Hypotension in patients on ACE inhibitors can be refractory to vasopressors.

      iii) Controlled hypotension may be requested by the surgeon to reduce bleeding for procedures such as total hip replacement or extensive spine instrumentation. These requests must be balanced with the risk of organ hypoperfusion when blood pressure is lowered.
         (1) Mean arterial pressure (MAP) should be maintained at a level adequate to maintain end-organ perfusion.
         (2) No single MAP has been shown to be ideal for all patients. Patients who are hypertensive at baseline likely require a higher MAP, while patients with lower baseline pressure can likely tolerate lower MAP intraoperatively. One approach is to keep patients within 10%-20% of their baseline (preoperative) pressure.[4]
         (3) General anesthesia can interfere with cerebral autoregulation. Additionally, chronic hypertension can shift the autoregulation curve to the right requiring higher MAP to maintain cerebral perfusion.



Placement of the BP cuff on the arm during surgery in the sitting position requires the clinician to be aware that the pressure in the brain is lower than the BP cuff readings. If an arterial catheter is placed, the transducer should be placed at the level of the circle of Willis during these procedures.

TABLE 5-2 Indications for Intraoperative Neuromuscular Blockade

- Abdominal surgery, laparoscopic surgery, intrathoracic surgery, robotic surgery
- Open eye surgery
- Orthopedic surgery with the need for reduction of bone fragments on which powerful muscles insert (eg, femoral shaft fracture)
- Facilitation of mechanical ventilation in patients with ALI or ARDS
- Need for imaging while holding ventilation (eg, angiography)

ALI, acute lung injury; ARDS, acute respiratory distress syndrome

iv) In patients with CAD, the oxygen supply-demand balance should be optimized. ST-segment monitoring allows detection of myocardial ischemia. Ischemic changes are best detected in lead V5 while arrhythmias are best seen in lead II. For this reason many anesthesiologists will display leads V5 and II on their monitor.



To determine the amount by which a BP cuff on the arm overestimates the pressure in the brain, measure the distance from the tragus to the BP cuff in centimeters. Then convert from cm H_2O to mm Hg by multiplying the number of centimeters by 0.7. The result should be deducted from the BP cuff reading to determine brain perfusion.

b) **Respiratory**

i) Controlled ventilation is typically used under GA with endotracheal intubation.

(1) Minute ventilation should be adjusted based on capnometry to maintain a partial pressure of end-tidal CO_2 ($PetCO_2$) and therefore a partial pressure of arterial CO_2 ($PaCO_2$) slightly below the patient's baseline.

(a) In general, maintaining tidal volumes at 6-8 cc/kg of predicted body weight is preferred for lung protection, and minute ventilation should be increased by increasing respiratory rate, not tidal volume.

(2) A high FiO_2 as well as positive end-expiratory pressure (PEEP) may be needed to maintain oxygenation in the face of atelectasis in the dependent regions of the lungs, but high levels of FiO_2 should be avoided whenever possible as long as the patient is not hypoxemic.

ii) When possible, spontaneous ventilation is more physiologic, with less atelectasis and a better ventilation-perfusion matching.

(1) Analgesia can be titrated to the respiratory rate if the patient is breathing spontaneously.

c) **Renal**

i) Urine output should be monitored for major procedures, as it can give information on end-organ perfusion. It should not, however, be used in isolation as factors other than hypovolemia can lead to reduced urine output, such as release of antidiuretic hormone during surgery and surgical compression of ureters.

ii) Patients with preexisting renal insufficiency and those who are hemodynamically unstable are at increased risk of renal failure.

iii) The kidney has a major role in maintaining electrolyte homeostasis.



Patients with preexisting renal insufficiency and those who are hemodynamically unstable are at increased risk of renal failure.

d) **Temperature**

i) Thermal control is impaired under GA, and patients will develop mild hypothermia unless active warming is instituted preoperatively to bring peripheral blood up to 37°C.

ii) Hypothermia impairs coagulation, causes increased blood loss and transfusion requirements, and leads to increased postoperative infection.

iii) In addition, hypothermia will cause shivering postoperatively, increasing severalfold the oxygen consumption and leading to myocardial ischemia in susceptible patients.

e) **Endocrine**

i) Blood glucose should be measured periodically in diabetic patients, except for short procedures.

ii) Insulin should be administered if needed, either subcutaneously or as an IV infusion, to maintain glucose, in most cases, below 180 mg/dL.



Hypothermia can impair coagulation causing increased blood loss and transfusion requirements.

f) **Neurologic**
   i) Monitoring of unconsciousness may be achieved with surface EEG signals processed via proprietary software incorporated into monitors (eg, BIS or Entropy).
   ii) Processed EEG readings yield a value between 0 and 100. Value ranges suggest depth of sedation.
      (1) Maintenance between 40 and 60 is thought to ensure an adequate depth of anesthesia and a rapid emergence. An exact range may vary by manufacturer.
      (2) Numerous causes of EEG signal errors have been described, the most prominent being the interference of muscular activity and a paradoxical increase in the presence of ketamine.
   iii) The EEG tracing may be more useful to the clinician than the index number itself, but training is required to interpret the EEG tracing

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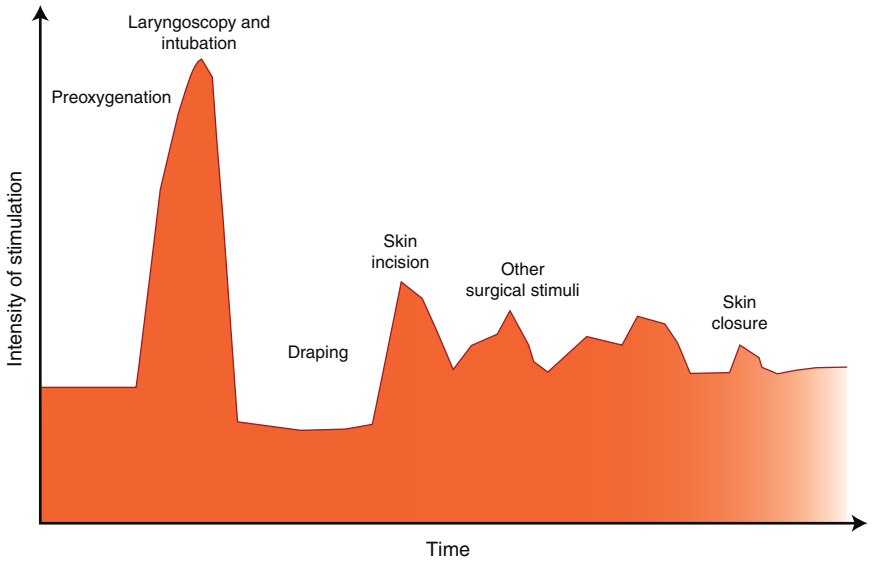
4) **Practical management**
   a) The choice of agents is based on the personal preferences and experience of the anesthesiologist, the patient's comorbidities (especially cardiovascular function), the procedure type and anticipated duration, and the postoperative plan (extubation or not, analgesic requirements, ambulatory or inpatient).
      i) Hypnotic agents are typically administered continuously, by inhalation or IV infusion.
      ii) Analgesics and NMB agents can be administered intermittently or by continuous infusion.



The depth of hypnosis, analgesia, and NMB has to be adjusted based on surgical stimulus and patient response.

   b) **The depth of hypnosis, analgesia, and NMB has to be adjusted based on surgical stimulus and patient response,**[6] titrated up before laryngoscopy and intubation, skin incision, etc., and titrated down when surgical stimulation is low (eg, draping, waiting for pathology). **Figure 5-1** illustrates the variations in the intensity of stimulation and thus the need for anesthetic depth, during a generic surgical procedure.
      i) Monitoring anesthesia depth may decrease the amount of hypnotics used, reducing side effects and recovery time while also preventing intraoperative awareness.
      ii) Analgesia titration currently relies on surrogate criteria such as hemodynamic response and respiratory rate in spontaneously ventilating patients.
   c) **Fluid and blood products should be administered to maintain euvolemia and the blood's oxygen-carrying capacity.**[7]
      i) The patient's baseline metabolic fluid requirement may be able to be estimated using the 4-2-1 rule. Add 4 mL/kg/h for the first 10 kg of body weight, 2 mL/kg/h for the next 10 kg, and then 1 mL/kg/h for the remainder of the body weight. For example, a 70-kg patient needs $(4 \times 10) + (2 \times 10) + (1 \times 50) = 110$ mL/h of crystalloids.
      ii) The fluid deficit due to the NPO period was once estimated by multiplying the baseline requirement by the number of hours the patient has been NPO, and this was then given to the patient during the course of the anesthetic. A better approach to fluid management is to use dynamic variables (pulse pressure variation, systolic pressure variation) to determine fluid responsiveness in a goal-directed approach.
      iii) Urine output should be taken into account when estimating volume status.
      iv) Insensible fluid loss depends on the procedure and can vary from 0 (carpal tunnel release) to 12 mL/kg/h (open major abdominal surgery). It is due to evaporation and third-space fluid loss because of surgical trauma. It should be taken into account when estimating the patient's volume status.
      v) Blood loss should be estimated and compensated when needed to maintain hemodynamics, using 3 mL of crystalloid or 1 mL of colloid for each mL of blood loss.

FIGURE 5-1 Schematic Representation of the Intensity of Stimulation and Need for Anesthetic Depth, During a Surgical Procedure.



For example only. Stimulation may vary with the surgical procedure.

vi) Red cell transfusion should be considered to maintain the hemoglobin above 8 in patients with CAD or 7 in patients without. Actively bleeding patients under anesthesia who are hemodynamically unstable may need red cell transfusion before a hemoglobin level can be checked.

d) As appropriate, plasma electrolytes and glucose levels should be monitored and corrected. Diabetic patients and those undergoing procedures with large fluid shifts are especially at risk. Transplant recipients can experience sudden hyperkalemia with cardiac arrest.

e) Ventilation can be controlled, or the patient may be allowed to breathe spontaneously, especially when a supraglottic device is used.
   i) Adjust ventilation based on capnometry. $PetCO_2$ in healthy patients is typically 5 mm Hg lower than $PaCO_2$ due to dead space gas which has little to no CO_2. This gradient can be significantly higher for patients with lung disease. The aim should be to maintain $PaCO_2$ just below the patient's baseline. Do not hyperventilate patients with chronic hypercapnia to a "normal" $PaCO_2$.
   ii) Debate exists about the ideal FiO_2. Patient's should receive adequate FiO_2 to avoid hypoxemia.

f) Hypothermia should be prevented using an active forced-air warming device. Fluid-warming devices are useful only if large amounts of fluid or blood products are to be administered.[8] **Table 5-3** lists causes of heat loss and methods of rewarming.



In healthy patients, $ETCO_2$ is typically 5 mm Hg lower than $PaCO_2$ due to dead space.



Do not hyperventilate patients with chronic hypercapnia to a "normal" $PaCO_2$.

TABLE 5-3 Causes of Intraoperative Heat Loss and Strategies for Rewarming

Causes of heat loss	• Radiation (accounts for 2/3 of heat loss) • Conduction • Convection • Evaporation
Methods of rewarming (approximate rewarming rate in degree Celsius per hour)	• Forced air warming (most effective noninvasive method) (up to 3.5) • Warm IV fluids, warm blankets, warm room (0.1-2) • Hemodialysis[2–4] • Thoracic lavage[3] • Venovenous ECMO[4] • Venoarterial ECMO[6] • Cardiopulmonary bypass[9]

From Danzl DF, Pozos RS. Accidental hypothermia. *N Engl J Med.* 1994;331:1756-1760.

CHAPTER SUMMARY FOR INTRAOPERATIVE MANAGEMENT AND MAINTENANCE OF ANESTHESIA

Definition	Management of physiological values and maintenance of surgical anesthesia following induction and until emergence
Maintenance Goals	Maintenance of surgical anesthesia Maintenance of physiologic homeostasis Hemodynamic parameters Respiratory Temperature Renal and electrolytes Monitoring is needed to ensure that these goals are met.
Maintenance Strategies	IV and/or inhaled administration of medications in order to provide unconsciousness (hypnosis), immobility, analgesia, and, if needed, muscle relaxation Balanced (combining inhaled and IV agents) or total IV methods of anesthesia maintenance can be used
Intraoperative Management Issues	The depth of hypnosis, analgesia, and NMB has to be adjusted based on surgical stimulus and patient response. Analgesic administration should take into account the fact that analgesia should extend into the postoperative period. Fluid and blood products should be administered to maintain euvolemia and the blood's oxygen-carrying capacity. Plasma electrolytes and glucose levels should be monitored and corrected. Ventilation can be controlled, or the patient may be allowed to breathe spontaneously, especially when a supraglottic device is used. Hypothermia should be prevented using an active forced-air warming device.

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6 Emergence and Postoperative Issues in Anesthesia

Emmanuel Nisimov • Arthur Hertling

Emergence is the transition process where a patient goes from general anesthesia to awake and spontaneously breathing. The patient's recovery during this process as well as in the postoperative care unit may present challenges to the anesthesiologist.

1) **Overview**
   a) **Emergence** is the critical period of recovery from general anesthesia, with the return of consciousness, neuromuscular conduction, and airway protective reflexes.
      i) Complications such as airway loss, respiratory failure, bronchospasm, laryngospasm, aspiration of gastric contents, delirium or agitation, postoperative nausea and vomiting, or acute kidney injury can occur.
   b) **A plan for emergence from anesthesia should be developed, considering the surgical needs and the patient's comorbidities.**
      i) Awake extubation is the most common choice: the trachea is extubated only when the patient responds to simple commands and is breathing spontaneously.
      ii) Deep extubation is planned when the presence of an ETT is to be avoided during emergence, to prevent "bucking" (patient-ventilator asynchrony or dyssynchrony) and straining, with the concomitant increase in intra-abdominal, intracranial, and intraocular pressures, and to decrease the risk of bronchospasm.
2) **Evaluation of the signs and stages of anesthesia**
   a) It is important to be able to evaluate a patient's response to anesthetic drugs in order to appropriately manage a patient during emergence from anesthesia. The following is the four-stage Guedel classification of anesthetic depth (**Table 6-1**).
   b) **Stage II is described as an excitement period with irregular breathing and possible agitation, laryngospasm, and regurgitation.**
   c) **Awake extubation should be performed only once Stage I is reached, that is, eyes have returned to a central position, breathing is regular, and patient is awake and responds to simple commands.**
      i) The main rationale is to keep the patient's airway secure and protected during Stage II, to avoid the risk of laryngospasm and vomiting.
3) **Preparation for emergence**
   a) **Estimate remaining duration of surgical procedure;** decrease concentration of inhaled anesthetic and/or rate of intravenous agent.
   b) **Do not readminister muscle relaxants;** give reversal medications.



Emergence is the critical period of recovery from general anesthesia, with the return of consciousness, neuromuscular conduction, and airway protective reflexes.



Stage II is described as an excitement period with irregular breathing and possible agitation, laryngospasm, and regurgitation.



Emergence complications include airway loss, bronchospasm, laryngospasm, and aspiration of gastric contents. Awake extubation should be performed only once Stage I is reached, that is, eyes have returned to a central position, breathing is regular, and patient is awake and responds to simple commands.

TABLE 6-1 Stages of Anesthesia According to Guedel

Stage I: Amnesia and Analgesia • From the beginning of induction of anesthesia to loss of consciousness
Stage II: Excitement/Delirium • From the loss of consciousness to the onset of automatic breathing. • The eyelash reflex disappears but other reflexes remain intact. • Coughing, vomiting, and struggling may occur. • Respiration can be irregular, with breath holding.
Stage III: Surgical Anesthesia Plane 1: Sleep • From the onset of automatic respiration to the cessation of eye movements. • Eyelid reflex is lost, swallowing reflex disappears, and marked eye movement may occur. • The conjunctival reflex is lost at the bottom of the plane. Plane 2: Sensory loss • From the cessation of eye movements to the beginning of intercostal muscle paralysis. • Laryngeal reflex is lost, although inflammation of the upper respiratory tract increases reflex irritability. • Corneal reflex disappears. • Tear secretion increases. • Respiration is automatic and regular. • Movement and deep breathing as a response to skin stimulation disappear. Plane 3: Loss of muscle tone • From the beginning to the completion of intercostal muscle paralysis. • Diaphragmatic respiration persists, but there is progressive intercostal paralysis. • Pupils are dilated, and light reflex is abolished. • The laryngeal reflex lost in plane 2 can still be initiated by painful stimuli. This was the desired plane for surgery when muscle relaxants were not used. Plane 4: Intercostal muscle paralysis
Stage IV: Medullary Paralysis • From complete intercostal paralysis to diaphragmatic paralysis (apnea)

c) **In case of patient movement or early awakening, with the need to increase the depth of anesthesia in the final 40 minutes of the case, administer an IV hypnotic such as propofol.** Avoid redosing NMB agents if possible.

d) **Titrate opioids to analgesia** (estimate requirements based on procedure, patient's weight, physiological status and opioid tolerance).
   i) Respiratory rate and tidal volume, which make up minute ventilation, can be useful indicators of adequate analgesia in a spontaneously ventilating patient. These parameters will vary with patient age (pediatric vs adult) and body weight.

e) **Administer a 5-HT3 antagonist if indicated to prevent PONV.**

f) **Administer 100% oxygen for 5-10 minutes, especially if N_2O used, to prevent hypoxemia.**
   i) If N_2O has been used and the patient is allowed to breathe room air, diffusion hypoxia can occur. N_2O has a low blood-gas partition coefficient and will diffuse rapidly from the blood into the alveoli once administration is discontinued, resulting in the displacement of oxygen in the alveoli.
   ii) Therefore, use supplemental O_2 after extubation for transport to PACU.



Administer 100% oxygen for 5-10 minutes prior to extubation, especially if N_2O used, to prevent hypoxemia.

g) **Return patient to supine or backup position prior to extubating.** It is possible to extubate in the lateral or even prone position, but have a plan to be able to reposition the patient supine emergently if needed.

TABLE 6-2 Extubation Criteria

Patient conscious and responding to simple commands ("open your eyes, squeeze my hand"). **If full stomach or high risk of regurgitation or if difficult intubation, extubate patient only when fully awake.**
Patient hemodynamically stable, normothermic, not having received massive amounts of fluids
Adequate spontaneous ventilation with tidal volumes >8 mL/kg and rate >8
Adequate reversal of NMB; **hand strength or TOF not reliable**; sustained head lift or sustained tetanus (painful) for 5 s

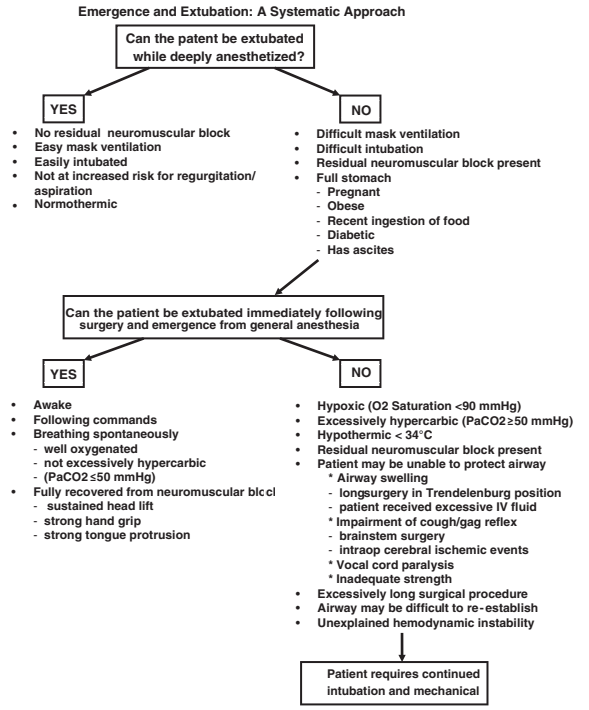
4) **Extubation criteria (Table 6-2)**
   a) **Ensure that all the equipment to reintubate is available prior to extubating!**
   b) **Assess the potential for airway obstruction.**
      i) Especially in a patient who has received large amounts of fluids and/or was positioned head down or prone, significant airway edema can develop and cause airway closure once the ETT is removed, which can result in a difficult or impossible reintubation.
      ii) Clinical inspection, possibly including direct laryngoscopy under GA to assess the feasibility of reintubation if needed, to evaluate the degree of edema. Clinical judgment should be used to decide whether extubation should be delayed.
      iii) A leak test can be performed by deflating the ETT cuff (after pharyngeal suctioning) and assessing the presence of a leak around the ETT, indicating the absence of significant airway edema; the predictive value of this test is unclear.
      iv) If there is concern for airway edema or a potentially difficult intubation, an airway exchange catheter, such as a Cook catheter, can be inserted through the ETT. Some catheters have an adapter that permits oxygen delivery if necessary.
   c) After suctioning the pharynx, deflate the ETT, and extubate immediately following inhalation such that the first action after extubation is exhalation, preventing inhalation of secretions onto vocal cords and laryngospasm. Continue O_2 administration via nasal cannula or other appropriate device (e.g., face mask if nasal packing following sinus surgery).
   d) **Reassess consciousness, ventilation, and comfort (pain, shivering).**



Ensure that all the equipment to reintubate is available prior to extubating.

5) **Deep extubation (Fig. 6-1)**
   a) **Indications:** Desire to avoid coughing, "bucking," straining, cardiovascular response to the ETT, or bronchospasm in a patient at low risk for aspiration of gastric contents
   b) **Contraindications**
      i) Full stomach, severe GERD, other situations where aspiration of gastric contents is a concern.
      ii) Difficult intubation or difficult ventilation.
      iii) Obesity, as the redundant pharyngeal tissue may make it very difficult to maintain a patent airway until muscle tone has returned. This is a relative contraindication.
   c) **Uses:** Can be used in cases such as an unclipped intracranial aneurysm, reactive airway disease, open-globe eye surgery
   d) **Technique**
      i) The goal is to extubate the patient during Stage III (surgical anesthesia), not during Stage II.
      ii) Face mask

FIGURE 6-1 A systematic approach to extubation.



Adapted from Lien et al. A&A 1997; 85:1177.

(1) The patient should not respond to stimulation and should be breathing spontaneously with a regular pattern.
(2) After oropharyngeal suctioning, the ETT cuff is deflated and the tube is removed.
(3) Ventilation is assisted as necessary via a facemask until the patient regains consciousness.
(4) An oral or nasal airway can be placed.

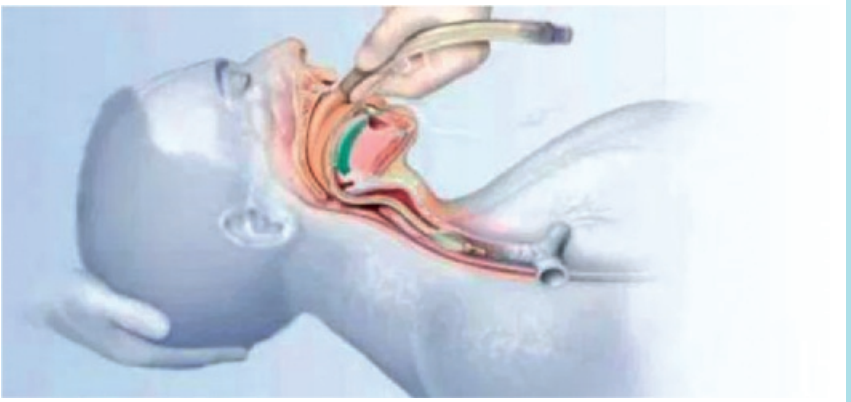
iii) Laryngeal mask: "Bailey maneuver"[1] (**Fig. 6-2**).
(1) Following oropharyngeal suctioning, a laryngeal mask device is inserted behind the ETT and the cuff inflated.
(2) The ETT cuff is deflated and the tube removed. The laryngeal mask is left in place until the patient regains consciousness.



When performing a deep extubation technique, the goal is to extubate the patient during Stage III (surgical anesthesia), NEVER during Stage II.

6) **Transport to PACU**
a) **General considerations**
i) Prior to transport, ensure that patient's vitals are stable and that emergency medications are available. If the patient is kept intubated, ensure that ventilator is available and set up in PACU or ICU.
ii) Ensure adequate fluid level in IV bag and emergency medications available in case medications have to be administered en route.

FIGURE 6-2 Bailey Maneuver



Insertion of a laryngeal mask device in Stage III while the ETT is in place, prior to extubating the trachea, in order to maintain airway patency, yet avoid the stimulation caused by the presence of an ETT in the trachea during emergence.
Adapted from Airway Challenge #1. LMA North America, www.lmana.com/docs/challenge1.pdf

iii) Transport the patient with supplemental O_2 via face mask or nasal cannula and portable pulse oximeter. Check that O_2 tank is full prior to transfer. Further monitoring of other vitals including BP and ECG may be performed if indicated.
iv) Have a bag-valve-mask system available if there is concern for adequate ventilation.
v) During transport, the anesthesia provider should be vigilant and remain at the head of the stretcher (to monitor and maintain the airway).

b) **Arrival to the PACU**
i) Connect the patient to the monitor and check vital signs.
ii) Evaluate for pain or nausea.
iii) Assess for bleeding at surgical site.
iv) Give structured sign-out to PACU nurse or ICU physician. Ensure that they are comfortable taking over care of the patient and that the patient is stable prior to leaving the area.
v) Have BiPAP, CPAP, or ventilator available prior to arrival if needed.

7) **Postoperative considerations**
a) **Respiratory failure**
i) Defined as the need to reintubate the trachea after extubation or the need for continued mechanical ventilation beyond 48 hours after the end of surgery.
ii) Can occur in as many as 3%-20% of extubated patients, with reintubated patients having a higher morbidity, mortality, and hospital length of stay.[2]
iii) The most common causes are as follows:
(1) Hypoventilation due to surgical swelling or hematoma causing airway obstruction, laryngospasm, bronchospasm, central apnea (opioid overdose, stroke), residual neuromuscular blockade.
(2) V/Q mismatch (aspiration of gastric contents, pulmonary edema, PE, atelectasis).

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iv) Prophylactic noninvasive ventilation should be considered and is beneficial with respect to reintubation, especially for patients with conditions such as sleep apnea or COPD.

b) **Delayed emergence**
i) Residual drug effects: hypnotic or opioid overdose, reduced metabolism, potentiation by other drugs, residual neuromuscular blockade

ii) Respiratory failure with significant hypercarbia
iii) Metabolic derangements: hypoglycemia, severe hyperglycemia, electrolyte imbalance, hypothermia, central anticholinergic syndrome
iv) Neurological complications: cerebral hypoxia, CVA (hemorrhage, embolism, or thrombosis)
v) Temperature derangements: Hypothermia can impair consciousness and potentiate the CNS-depressant effects of anesthetics. As little as a 2°C decrease in body temperature can slow the metabolism of anesthetics and NMBDs. Hyperthermia can also result in mental status changes.

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Crisis management: delayed emergence, Chapter 144

c) **Agitation and emergence delirium (ED)**
i) Agitation upon awakening is not uncommon in children and young patients. ED is a dissociated state of consciousness characterized by irritability, inconsolability, thrashing, and incoherence.[3] It is usually short-lived and resolves spontaneously. Incidence varies between 10% and 80%.
ii) During an episode, children exhibit nonpurposeful movements and fail to establish eye contact or to interact normally with their caregiver.
iii) Risk factors in the pediatric population include preoperative anxiety (not only in the patient but in parents as well), younger age (2-6 years of age), surgical procedure (highest incidence with ophthalmological and ENT cases), and use of volatile anesthetics. Agitation related to pain is not considered ED. Duration and depth of anesthesia do not correlate with risk of developing ED as it is as likely to occur after short procedures (eg, myringotomy and tubes).
iv) Can result in increased risk of self-harm (ie, removal of IV catheters, drains, or dressings), parental dissatisfaction, delayed discharge, and even manifestation of maladaptive behaviors not present preoperatively (such as general anxiety, nighttime crying, enuresis, and temper tantrums up to 2 weeks after surgery).
v) It is important to rule out other potential causes that can present as ED. These include inadequate analgesia, hypoxia, hypotension, hypo-/hypercarbia, hypoglycemia, hypothermia, or increased intracranial pressure.
vi) The most effective preventive approach is to avoid volatile anesthetics and administer total intravenous anesthesia (TIVA). Agents that decrease incidence of ED include propofol, opioids (particularly fentanyl), ketamine, and alpha-2 agonists such as clonidine and dexmedetomidine.



Delayed emergence can be due to residual drug effects, respiratory failure, metabolic derangements, and/or neurological complications.

d) **Inadequate postoperative analgesia**
i) Pain is a subjective experience and should be appropriately and adequately treated in the postoperative patient. Multimodal analgesia involves use of pharmacological agents from different classes that serve to target different pain receptors and pathways.
ii) Optimize recovery using ERAS protocols.[4] Opioid-sparing approaches should be utilized when possible to decrease risk of side effects including nausea, vomiting, pruritus, respiratory depression, constipation, bowel ileus, urinary retention, confusion, and tolerance.
iii) Nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen, aspirin, ketorolac, and diclofenac can help to decrease postoperative pain. They should be used cautiously as they carry increased risks such as bleeding, decreased gastric mucus production and potential worsening of ulcerations, and kidney injury.
iv) Acetaminophen is recommended by enhanced recovery after surgery (ERAS) Society guidelines, on a regular schedule, 15 mg/kg, up to 1 g four times daily in patients without contraindications, either orally or IV.

TABLE 6-3 Risk Factors for PONV in Adults

- Female sex
- History of PONV or motion sickness
- Nonsmoking
- Younger age
- General vs regional anesthesia
- Use of volatile anesthetics and nitrous oxide
- Postoperative opioids
- Duration of anesthesia
- Type of surgery (cholecystectomy, laparoscopic, gynecological)

v) IV lidocaine has growing evidence for use in certain cases including hysterectomy and laparoscopic colorectal or rectal surgery.
vi) Regional anesthesia involving peripheral or neuraxial blocks is an effective method for analgesia.
vii) Patient-controlled analgesia (PCA) delivery systems should be used when possible to optimize patient comfort.



Shivering can significantly increase oxygen consumption, leading to myocardial ischemia in susceptible patients.

e) **Hypothermia and shivering**
i) Postoperative hypothermia is best prevented by intraoperative active forced-air warming.
ii) Hypothermia may lead to increased blood loss and transfusion requirements (because of platelet dysfunction), poor wound healing and increased incidence of wound infection, slower metabolism of administered medications, and possibly dysrhythmias.
iii) Shivering is a physiological response to hypothermia, using striated muscle contraction to produce heat. However, shivering is uncomfortable for the patient and can significantly increase oxygen consumption, leading to myocardial ischemia in susceptible patients.
iv) Shivering can be treated with meperidine, 12.5-25 mg IV, and forced-air warming to correct hypothermia.

f) **Postoperative nausea and vomiting (PONV)**
i) Prophylaxis should be used, depending on the patient's risk factors (**Table 6-3**). The most likely causes are volatile anesthetics, nitrous oxide, and postoperative opioids.
ii) Emptying the stomach with a gastric tube at the end of surgery, especially following ENT or oral surgery, where blood can be present in the stomach, also helps decrease the risk of PONV.
iii) Recommended treatment options include 5-hydroxytryptamine (5-HT3) receptor antagonists (ondansetron), neurokinin-1 (NK-1) receptor antagonists (aprepitant, casopitant, and rolapitant), corticosteroids (dexamethasone and methylprednisolone), dopamine receptor antagonists (metoclopramide, prochlorperazine, droperidol), butyrophenones (droperidol and haloperidol), antihistamines (dimenhydrinate and meclizine), and anticholinergics (transdermal scopolamine).[5]

READ MORE
Postoperative nausea and vomiting, Chapter 129

g) **Low urine output and acute kidney injury (AKI)**
i) AKI is generally defined as a urine output <0.5 mL/kg/h for >6 hours. Three different classification systems (RIFLE, AKIN, and KDIGO) have been developed to help determine the severity of kidney injury.
ii) Acute kidney injury is routinely underdiagnosed and carries considerable morbidity and mortality.
iii) Studies report a risk of 5%-10% of developing AKI in mixed surgical populations.[6]

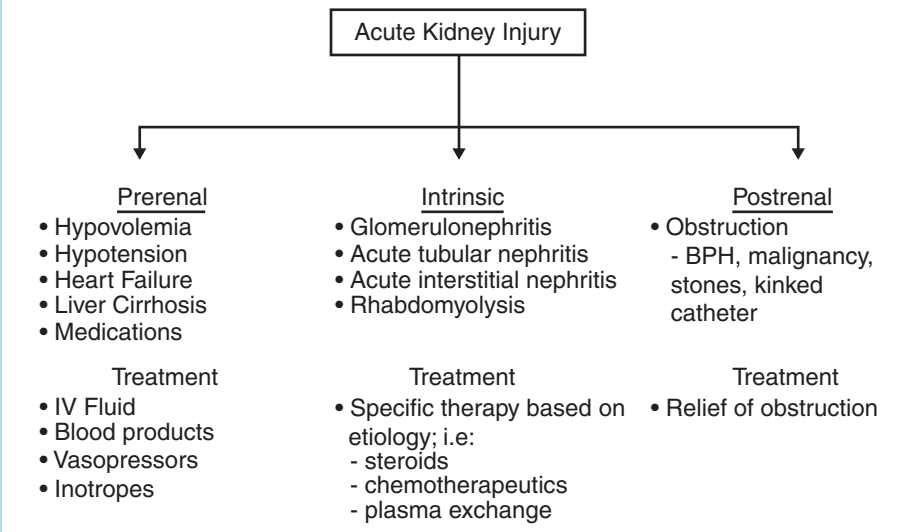
TABLE 6-4 Risk Factors for Postoperative AKI

Preoperative	• Underlying renal dysfunction • Female gender • Increasing age • Hypertension • Diabetes mellitus • Cardiovascular disease • Liver disease • COPD • ASA PS 4 or 5 • Anemia
Intraoperative	• Emergency surgery • Intraperitoneal surgery (especially exploratory laparotomy and small bowel resection) • Major vascular surgery • Cardiac surgery (especially involving cardiopulmonary bypass) • Prolonged hypotension • Hypovolemia • RBC transfusion • Inotrope use • Aortic cross-clamp time
Postoperative	• RBC transfusion • Vasopressor use • Diuretic use • Antiarrhythmic drug use

iv) There are multiple risk factors for the development of postoperative kidney injury (**Table 6-4**). However, besides the avoidance of hypovolemia and hypotension, none of them are alterable.

v) The management of AKI depends on the etiology of renal injury (**Fig. 6-3**).

FIGURE 6-3 Management of acute kidney injury.



CHAPTER SUMMARY FOR EMERGENCE AND POSTOPERATIVE ISSUES IN ANESTHESIA

Definition	Emergence is the return of consciousness, neuromuscular conduction, and airway protective reflexes.
Preparing for Emergence	Prior to extubation, ensure equipment to reintubate is available. Decrease doses of inhaled anesthetics and/or intravenous agents. Give neuromuscular blockade reversal if appropriate. Titrate opioids to analgesia. Consider a 5-HT3 antagonist for PONV prophylaxis. Administer 100% FiO_2. Typically return patient supine/backup position.
Technique of Routine Extubation	Confirm the patient conscious and responding to simple commands ("open your eyes, squeeze my hand"). Patient is hemodynamically stable, normothermic, and not having received massive amounts of fluids leading to airway edema. Adequate spontaneous ventilation with tidal volumes >8 mL/kg, rate >8. Adequate reversal of neuromuscular blockade, sustained head lift or sustained tetanus (painful) for 5 s.
Technique of Deep Extubation	Used to avoid coughing, bucking, and straining on extubation. Prior to extubation, ensure equipment to reintubate is available. Goal is to extubate the patient during Stage III of anesthesia.
Postoperative Complications	Delayed emergence is most commonly due to residual drug effects, respiratory failure, metabolic derangements, or neurologic complications.

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7 Positioning of the Surgical Patient§

Michael Akerman • Jamie Murphy

Proper positioning of the surgical patient is critical. Positioning injuries range in severity from minor skin abrasions and bruising to peripheral neuropathies, nonhealing pressure ulcers, and blindness. These injuries are generally thought to be preventable but can still occur despite diligence and preventive measures. It is important for the anesthesiologist to be aware of the multiple ways that these injuries can present and to distinguish them from other perioperative complications. When diagnosed and managed properly, many positioning injuries can completely resolve.

1) **Overview**
   a) Proper positioning of the surgical patient requires careful balance between optimal surgical field exposure and safe patient positioning.
   b) Each surgical position (supine, prone, lithotomy, and lateral) presents its own unique risks and challenges.
   c) Knowledge of potential positioning complications and their timely recognition and management are critically important for a successful outcome.
   d) A thorough history and physical exam targeting the presence of preexisting nerve injury are vital to intraoperative nerve injury prevention.[1]



A thorough history and physical exam targeting the presence of preexisting nerve injury are vital to intraoperative nerve injury prevention.

2) **Factors affecting positioning (Table 7-1)**
   a) Complications arising from patient malpositioning during surgery depend on both nonpatient and patient factors.
   b) While most of these factors (eg, type of procedure, patient age, or gender) cannot be modified, recognition of patients who are at increased risk for positioning complications should alert the anesthesia provider to take extra precautions.
   c) Specific factors that increase risk for positioning injuries may include older age; morbidly obese (BMI of >40), thin, small stature; poor preoperative nutritional status; preexisting neurologic damage; cigarette smoking;[1] or a history of diabetes or vascular disease.[2]



Perioperative cigarette smoking is associated with an increased incidence of postoperative neuropathies, presumably because of the vasoconstrictive effects of nicotine.

3) **General positioning guidelines**[1-5]
   a) Patient positioning is a major responsibility of the entire operating room team, including the anesthesiologist.
   b) Proper patient positioning balances the need for adequate exposure of the surgical field and prevention of positioning injuries.
   c) Optimal positioning is achieved when:
      i) Adequate arterial supply and venous outflow are maintained to all body parts.
      ii) Nerves are protected from undue pressure or stretching.
      iii) Bony prominences are padded.
      iv) Circulatory and respiratory systems are minimally compromised.
      v) Maximum surgical field exposure is achieved.



Patient positioning is a major responsibility of the entire operating room team, including the anesthesiologist.[5]

TABLE 7-1 Risks Factors Affecting Positioning of Patient

Non-patient Factors	Patient Factors
Type of procedure	Patient's age
Length of procedure	Patient's height
Type of anesthesia	Patient's weight
Equipment affecting patient's position	• Nutritional status • Deconditioning • Other preexisting conditions

4) **Positioning guidelines**

a) **Supine**

i) Used for abdominal, pelvic, open-heart, head and neck, and most extremity surgery.

ii) Padding should be applied to the heels, elbows, knees, spinal column, and occiput. For procedures in steep Trendelenburg, the shoulders should also be padded. Padding may also be necessary to protect the patient's upper body, head, and neck from surgical equipment that is placed on the surgical field drapes.

iii) To avoid pressure alopecia, periodically rotate the patient's head, especially during prolonged cases.

iv) To avoid brachial plexus injury, upper extremities should be abducted to <90 degrees with hands and forearms in a supinated or neutral position.

v) Shoulder braces are not recommended due to considerable risk of compression injury to the brachial plexus.

vi) IV tubing, clamps, and stopcocks abutting the skin should be padded.

vii) For the Trendelenburg position, a nonsliding mattress is recommended to prevent patients from sliding cephalad.

(1) The Trendelenburg position may cause cardiovascular and/or respiratory compromise.

(2) Patients may develop significant edema of the face and upper airway.



Beware of surgical equipment resting on the surgical drapes that cover the patient. Equipment may cause compression injuries, mechanical injuries, scratches, cuts, or other patient injuries if not properly addressed with padding and prompt removal of instruments and equipment.

b) **Prone (Fig. 7-1)**

i) Used for posterior spine, skull, buttocks, perirectal, and lower extremity surgery.

ii) The patient is typically placed in this position by a logroller after being anesthetized on a stretcher.

iii) Proper patient body alignment should be maintained while turning from supine to prone with special attention to the breasts, male genitalia, arms, legs, and cervical spine.

iv) Consider using a wire-reinforced endotracheal tube to avoid kinking and obstruction as it exits the patient's mouth while in the prone position.

v) The arms are usually kept <90 degrees at the shoulder and elbow joints.

vi) The eyes should be free of pressure and checked frequently.

vii) To avoid stress on the cervical spine and to permit venous draining from the brain, there should be at least two fingerbreadths between the chin and the chest.



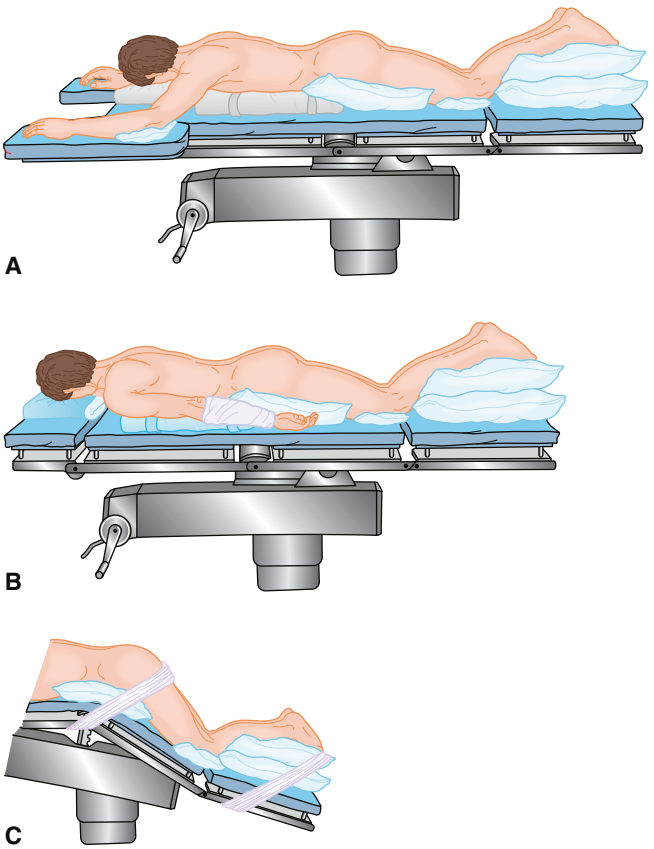
In the prone position, the eyes should be free of pressure and checked frequently.

c) **Lithotomy (Fig. 7-2)**

i) Used for gynecologic, rectal, and urologic surgery.

ii) When placing the patient into and out of this position, both legs are moved simultaneously to prevent torsion of the lumbar spine.

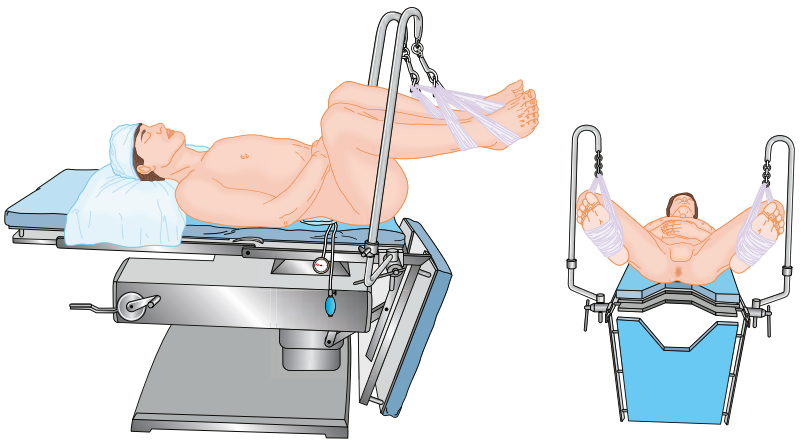
FIGURE 7-1 The Classic Prone Position



A. Flat table with relaxed arms extended alongside the patient's head. Parallel chest rolls extended from just caudad of clavicle to just beyond inguinal area, with pillow over pelvic end. Elbows and knees are padded, and legs are bent at the knees. The head is turned onto a C-shaped foam sponge that frees the downside eye and ear from compression. **B.** Same posture with arms snugly retained alongside torso. **C.** Table flexed to reduce lumbar lordosis; subgluteal area straps placed after the legs are lowered to provide cephalad thrust and prevent caudad slippage. Reproduced with permission from Warner MA, Johnson R. Patient positioning and potential injuries. In: Barash PG, ed. *Clinical Anesthesia*. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2017:809-825.

iii) The hips are flexed 80-100 degrees from the trunk, and the legs are abducted 30-45 degrees from the midline.
iv) Lower extremities are padded to prevent compression against the stirrups and injury to the common peroneal nerve.
v) Hands and arms should be positioned away from the bed to prevent crush injury during manipulation of the foot section of the OR table.
vi) The lithotomy position can increase intra-abdominal pressure resulting in impeded venous return to the heart.

FIGURE 7-2 Standard Lithotomy Position With "Candy Cane" Extremity Support



Thighs are flexed ~90 degrees on the abdomen; knees are flexed enough to bring the lower legs grossly parallel to the torso section of the tabletop. Arms are retained on boards, crossed on the abdomen, or snugged at the sides of patient. Reprinted with permission from Warner MA, Johnson R. Patient positioning and potential injuries. In: Barash PG, ed. *Clinical Anesthesia*. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2017:809-825.

vii) When the legs are elevated, venous return increases, causing a transient increase in cardiac output and, to a lesser extent, cerebral venous and intracranial pressure in otherwise healthy patients.

d) **Lateral (Fig. 7-3)**

i) Used for thoracic, retroperitoneal, elbow, and hip surgery.

ii) The patient is typically placed in this position after being anesthetized in the supine position.

iii) The patient's head and neck should be maintained in neutral position to prevent injury to the cervical spine and brachial plexus, avoiding excessive lateral rotation.

iv) To prevent injury to the brachial plexus, an "axillary roll" is placed just caudad to the dependent axilla with the dependent arm perpendicular to the torso on a padded arm board. The axillary roll should never be placed in the axilla.[3]



The axillary roll should be placed just caudad to the axilla on the chest in order to avoid compression of the axillary vasculature and the brachial plexus.

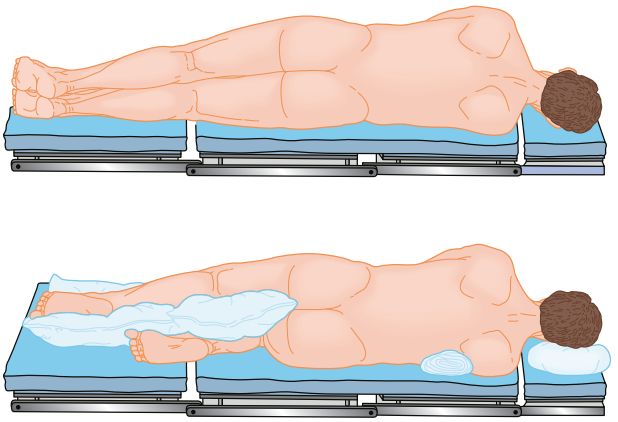
v) The nondependent arm is placed on a pillow or a padded armrest. Both arms should not be abducted more than 90 degrees.

vi) The patient's lower leg should be flexed with the upper leg straight and a pillow placed between them.

vii) The patient's head and neck should be in a neutral position with additional padding placed as needed.

viii) Before attempting extubation in the lateral position, the advantages and disadvantages must be considered. Benefits may include less gravitational pull of the tongue and saliva to obstruct the airway or induce laryngospasm and perhaps ease of transfer to stretcher for transport. Risks include difficulty with oxygenation and reintubation in emergency after extubation, if necessary.

FIGURE 7-3 The Standard Lateral Decubitus Position



Proper head support, axillary roll, and leg pillow arrangement are shown on lower figure. Downside leg is flexed at hip and knee to stabilize torso. Retaining straps and pad for downside peroneal nerve are not shown. Reprinted with permission from Warner MA, Johnson R. Patient positioning and potential injuries. In: Barash PG, ed. *Clinical Anesthesia*. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2017:809-825.

5) **Complications**[1-4]
   a) **Peripheral neuropathy**
      i) Sensory/motor neuropathy
         (1) Typically transient, especially if sensory only.
         (2) Patient follow-up is important to determine improving/worsening symptoms.
         (3) Consider neurology consultation for serial exams and if symptoms persist beyond 5 days.
         (4) A baseline EMG can help determine the presence of a preexisting nerve injury. Changes in EMG activity do not appear until 2-3 weeks after a new nerve injury.
         (5) Exclude any correctable causes: for example, vascular injury, hematoma, and cast compression.
         (6) Consider physical therapy early for the best outcome.
         (7) Patients with symptoms for 6-8 weeks or progressive deficits may need a neurosurgical consult.
      ii) Other etiologies
         (1) Other neurologic disorders such as stroke, spinal injury, disc herniation
         (2) Musculoskeletal injury
         (3) Autoimmune neurologic disorder
   b) **Common upper extremity injuries**
      i) Ulnar nerve
         (1) Most common nerve injury in anesthetized patient.[3]
         (2) The cause of ulnar nerve palsy is multifactorial and not always preventable.



In the lateral position, care should be taken that the neck remain in the neutral position as well as the nondependent arm to avoid injury to the brachial plexus. Care should also be taken to avoid external pressure on the lower eye.



For patients with postoperative peripheral neuropathy, first exclude any correctable causes such as vascular injury, hematoma, and cast compression.

(3) Presents with loss of sensation of lateral portion of hand and inability to abduct or oppose the fifth finger.
(4) The Anesthesia Closed Claims Project demonstrated that ulnar nerve injury most commonly presents in men, in an older population, and with delayed onset (3 days).[6]


Ulnar nerve injury is the most common nerve injury in an anesthetized patient.

ii) Brachial plexus
(1) Occurs when the brachial plexus is stretched or compressed between the clavicle and first rib.
(2) Manifestations depend on which nerves are injured
(a) Median nerve: inability to oppose thumb
(b) Axillary nerve: inability to abduct the arm
(c) Ulnar nerve: see above
(d) Musculocutaneous nerve: inability to flex the forearm
(e) Radial nerve: wrist drop
(3) Associated with the use of shoulder braces in patients undergoing surgery in Trendelenburg position.

c) **Common lower extremity injuries**
i) Common peroneal nerve[3]
(1) Most common injury in lithotomy position when lateral aspect of knee is compressed against a stirrup
(2) Results in foot drop and a loss of dorsal extension of toes
ii) Sciatic nerve
(1) Injured from excessive flexion of hips
(2) Results in weakness in all muscles below the knee and foot drop
iii) Femoral nerve
(1) Injured generally from lower abdominal procedures involving excessive retraction
(2) Results in decreased knee jerk, loss of flexion of hip, and extension of knee
iv) Saphenous nerve
(1) Injured when the medial tibial condyle is compressed by leg support in lithotomy position
(2) Paresthesia along medial and anteriomedial aspect of the calf
v) Anterior tibial nerve
(1) Injured when feet are plantar flexed for extended periods of time (sitting or prone)
(2) Manifests as foot drop
vi) Obturator nerve
(1) Damaged during excessive flexion of the thigh to the groin or during difficult forceps delivery
(2) Results in inability to adduct the leg and diminished sensation along medial aspect of the thigh

6) **Eye injury**[3,7,8]
a) **Corneal abrasion**
i) Most common type of eye injury
ii) Eyes should be protected and frequently checked during anesthesia.
iii) Patients are also at risk in the PACU.
iv) Treated with antibiotic ointment, patching; usually resolves without any sequelae in several days.
v) Consult ophthalmologist if persistent eye discharge or "foreign body" sensation.


Postoperative blindness requires immediate consultation by ophthalmologist.

b) **Postoperative blindness**
   i) Risk factors for blindness include intraoperative hypotension, prone position, and anemia.
   ii) Requires immediate consultation by ophthalmologist.
   iii) Etiology includes ischemic optic neuropathy and/or retinal vessel occlusion.
      (1) Ischemic optic neuropathy
         (a) Bilateral blindness
         (b) Anterior optic nerve ischemia can result in
            (i) Initial optic disc edema
            (ii) Occasional improvements in vision
         (c) Posterior optic nerve ischemia can result in
            (i) Initially normal funduscopic exam
            (ii) Delayed optic disc edema
            (iii) Vision, which seldom improves
      (2) Retinal artery or vein occlusion
         (a) Usually unilateral vision loss.
         (b) Cherry red spot with retinal edema is observed on examination.
         (c) Associated with carotid disease and atherosclerosis.
         (d) Vision occasionally improves.
      (3) Cortical blindness
         (a) Total or partial loss of vision in one or both eyes with normal funduscopic and papillary reflex
         (b) Associated with ischemia to the occipital visual cortex
         (c) Vision frequently improves
      (4) Acute glaucoma[8]
         (a) Usually occurs spontaneously and is more common in women and the elderly.
         (b) The diagnosis should be suspected in a patient with a painful red eye and cloudy or blurred vision.
         (c) It is distinguished from corneal abrasion, which also produces pain but without papillary signs, increased IOP, subjective visual loss, and headache.
         (d) Acute glaucoma is an emergency, and ophthalmologic consultation should be obtained immediately.

7) **Summary of perioperative neuropathy task force consensus**[3]
   a) **Perioperative assessment**
      i) When judged appropriate, it is helpful to ascertain that patients can comfortably tolerate the anticipated operative position.
      ii) Perform a thorough history and physical documenting any preexisting neurologic injury.
   b) **Upper extremity positioning**
      i) Arm abduction should be limited to 90 degrees in supine patients.
         (1) Patients who are positioned prone may comfortably tolerate arm abduction >90 degrees.
      ii) Arms should be positioned to decrease pressure on the postcondylar groove of the humerus (ulnar groove).
         (1) When arms are tucked at the side, a neutral forearm position is recommended.
         (2) When arms are abducted on arm boards, either supination or a neutral forearm position is acceptable.
         (3) Prolonged pressure on the radial nerve in the spiral groove of the humerus should be avoided.
         (4) Extension of the elbow beyond a comfortable range may stretch the median nerve.

c) **Lower extremity positioning**
   i) Lithotomy positions that stretch the hamstring muscle group beyond a comfortable range may stretch the sciatic nerve.
   ii) Prolonged pressure on the peroneal nerve at the fibular head should be avoided.
   iii) Neither extension nor flexion of the hip increases the risk of femoral neuropathy.



A simple postoperative assessment of extremity nerve function may lead to early recognition of peripheral neuropathies.

d) **Protective padding**
   i) Padded arm boards may decrease the risk of upper extremity neuropathies.
   ii) The use of chest rolls in laterally positioned patients may decrease the risk of upper extremity neuropathies.
   iii) Padding at the elbow and at the fibular head may decrease the risk of upper and lower extremity neuropathies, respectively.
   iv) Avoid the use of excessive padding (ie, too tight), which may increase the risk of perioperative neuropathy.

e) **Equipment**[3]
   i) Properly functioning automated blood pressure cuffs on the upper arms do not affect the risk of upper extremity neuropathies.
   ii) Shoulder braces in steep head-down positions may increase the risk of brachial plexus neuropathies.

f) **Postoperative assessment**
   i) A simple postoperative assessment of extremity nerve function may lead to early recognition of peripheral neuropathies.



Avoid improper placement of blood pressure cuffs (ie, placed below the antecubital fossa) to reduce the risk of nerve injury.

g) **Documentation**
   i) Charting specific positioning actions during the care of patients may result in improvements of care by helping practitioners focus attention on relevant aspects of patient positioning.
   ii) Providing information that continues improvement processes can lead to refinement in patient care.

CHAPTER SUMMARY FOR POSITIONING OF THE SURGICAL PATIENT

General Considerations	Proper patient positioning balances the need for adequate exposure of the surgical field and the prevention of positioning injuries. It is the responsibility of the entire OR team to ensure proper patient positioning.
Specific Considerations: Eyes	Eyes should be frequently checked to make sure that they are free from pressure, especially in the prone position. Corneal abrasion is the most common type of eye injury and is typically treated with antibiotic ointment. An ophthalmologist should immediately evaluate patients with postoperative blindness.
Upper Extremity	Arms should be abducted <90 degrees in the supine position. Avoid pressure on the ulnar groove by the elbow.
Lower Extremity	Avoid overstretching the hamstring, which can cause sciatic nerve injury. The common peroneal nerve can be compressed against the fibula below the knee in the lithotomy position.

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8 Hazards of the Operating Room

Nilda E. Salaman • Albert Ju

The complexity of the operating room (OR) environment subjects the anesthesia provider with potential hazards during the performance of clinical duties, which include occupational exposure to inhaled waste anesthetic gases, electrical hazards, potential for substance use disorder, falls/slips, exposure to blood-borne pathogens, and needlestick/sharps injury. This environment presents the health care provider with challenges that can have a significant impact on patient care and personal health.

1) **Occupational exposure to waste anesthetic gases (WAGs)**
   a) Anesthetic gases and vapors that leak into the surrounding room are considered WAGs. Anesthesiologists are exposed to trace amounts of WAGs while working in the operating room.[1]
      i) Exposure can be caused by inhalational inductions, poorly fitting masks, uncuffed endotracheal tube cuffs, supraglottic airways, leakage of anesthetic when filling the vaporizer, and malfunctioning scavenging systems and during hookup and disconnection of the anesthesia circuit.
      ii) Retrospective studies suggest an association between exposure to WAG and risk of spontaneous abortion, cancer, and congenital abnormalities. Review of the literature suggests that evidence for adverse reproductive or health effects of nitrous oxide and volatile anesthetics on exposed personnel is scarce and inconsistent.[2]
      iii) The induction of oxidative stress and oxidative DNA damage that are dependent on the magnitude of exposure has been proposed as a mechanism of injury.[3] However, no studies to date have demonstrated that trace concentrations of WAGs adversely affect personnel in operating rooms where scavenging systems are in use.



Occupational exposure to WAGs can be caused by inhalational inductions, poorly fitting masks, uncuffed endotracheal tube cuffs, supraglottic airways, leakage of anesthetic when filling the vaporizer, and malfunctioning scavenging systems and during hookup and disconnection of the anesthesia circuit.

   b) The American Society of Anesthesiologists (ASA) recommends that anesthetic gases be scavenged; however, there is insufficient evidence for recommending routine monitoring of WAG levels or routine surveillance of exposed personnel.[4]
      i) Effective scavenging of waste gases is essential in controlling the concentration of exposure to WAGs.[5]
      ii) The ASA considers proper maintenance and inspection of anesthesia machines to be an effective strategy to minimize WAGs.
      iii) ORs require efficient ventilation systems to reduce WAGs. The American Institute of Architects require 15-21 air exchanges hourly with three of those supplying outside air.
      iv) The National Institute of Occupational Safety and Health (NIOSH) recommends exposure limits for waste anesthetic gases, given in parts per million (ppm), measured as a time-weight average during the administration of an anesthetic.[1]
         (1) Nitrous oxide: 25 ppm
         (2) Halogenated anesthetics: 2 ppm
         (3) Halogenated plus nitrous oxide: 0.5 ppm

v) Thresholds in other countries are higher (up to 100 ppm nitrous and 50 ppm volatile agent).

2) **Electrical OR safety**[6,7]

a) Electricity powers up equipment, and it can be used for diagnostic (nerve stimulators) and therapeutic (pacemakers, defibrillators and electroconvulsive shock for depression) purposes. Electricity can also be a major source of injury to patients and OR personnel.

i) The OR is considered a "wet location," and it contains a multitude of electrical equipment and electronic devices that can subject the patient and OR personnel to the risks of electric shock.

(1) A ground is a physical electrical connection to the earth.

(2) Electrical power in the OR is ungrounded; however, the devices are grounded; thus faulty equipment and completion of a circuit can lead to varying amounts of shock.

ii) Contact with a large electrical current can lead to tissue damage, seizures, burns, ventricular fibrillation, and even death.

Scavenging of WAGs is essential in controlling the concentration of exposure. Proper maintenance and inspection of anesthesia machines, efficient ventilation systems, and work practices such as running low flows are considered effective measures to reduce occupational exposure to WAGs.

b) Electrical current must have a complete circuit (hot wire and neutral wire) and a source of voltage to drive flow through an impedance (ie, equipment, patient, OR personnel, anesthesia provider).

i) The power companies in the United States supply electrical energy in the form of alternating current (AC) of 120 V at 60 Hertz (Hz).

(1) The hot wire (high voltage) conducts the electricity to a source (equipment), for example an OR table, and the neutral (low voltage) ground wire returns the current back to the power source, thus completing the circuit.

(2) Touching a hot wire can lead to a shock, since current can flow from the hot wire through the victim into the ground, which then flows back into the neutral wire, completing a circuit.

See http://vam.anest.ufl.edu/simulations/ores.html for simulation on electrical power in the operating room.[8]

c) **Macroshock/Microshock**

i) The severity of an electrical shock is determined by the location, duration, frequency, and magnitude. See **Table 8-1**.

(1) Macroshock refers to large amounts of current flowing through a person, which can cause harm or death.

TABLE 8-1 Effects of Shock at Increasing Current

Current 1-s contact	Effect
Macroshock	
1 mA	Minimum perceptible
5 mA	Maximum "harmless" current
10-20 mA	"Let go" current before sustained muscle contraction
50 mA	Pain and possible mechanical injury
100-300 mA	Ventricular fibrillation
Microshock	
20-100 µA	Ventricular fibrillation
10 µA	Recommended maximum leakage current

mA, milliamperes; µA, microamperes

(2) Microshock refers to small amounts of current delivery to the myocardium in "susceptible" patients, for example, those with cardiovascular implantable electronic devices (CIEDs) or central lines.
   (a) Devices in direct contact with the heart, including central venous pressure catheters and transcutaneous pacing leads, can increase the risk of microshock.
   (b) It is important to not touch electrical equipment and objects in contact with the heart at the same time. Small amounts of charge (leakage) on the casing can cause microshock.
   (c) Temporary pacemakers should only be powered by battery to avoid connections to the ground that can create a circuit.[7]



Electrical shocks occur when a person becomes part of an electrical circuit.

Contact with a large electrical current can lead to cellular damage, seizures, burns, ventricular fibrillation, and even death.

ii) To decrease the risk of a shock, modern OR power is electrically isolated from the ground by using a **line isolation transformer**. It prevents the neutral lead from being grounded.
iii) Even if a person touches a hot wire, there is no completed circuit. Thus, no shock occurs.

d) **Line isolation systems** (**isolation transformer + line isolation monitor**) protect against electrocution by turning a normal "grounded system" (that exists outside the operating room) which only needs a single fault to cause electrocution into a "protected" system in which two faults are required to deliver a shock.
   i) Line isolation monitors (LIMs) are designed to continually detect whether the electrical power system is isolated from the ground (**Fig. 8-1**).
      (1) It provides both visual and audible alarm signals to alert personnel of the presence of a line-to-ground fault on a connected equipment.

FIGURE 8-1 Line Isolation Monitor Hazard Current Reads 3.0 mA, Which Is Within the Alarm Limits.



(2) The alarm is typically set to detect at least 2-5 mA (milliamperes) hazard current flowing between the neutral and ground leads.
(a) If the hot wire short circuits with a device, the system is no longer isolated from the ground, as all devices have a ground plug.
(b) Touching a device with a short circuit of the hot wire alone will not deliver a shock. However, the system is less safe. If the neutral wire becomes grounded, a completed circuit exists, risking shock when the device is touched.

ii) Failures of grounding can occur from faulty OR devices or faulty wiring.
(1) To determine if an OR device is the problem, unplug each device, starting with the most recently plugged in and working backwards.
(2) If the alarm stops after unplugging a device, then the device may be the cause of the problem and should be replaced as soon as possible.
(a) However, if the device cannot be interrupted without compromising patient safety, then it may be acceptable to continue using the device, as a second fault is required to create a shock hazard.
(3) If the LIM continues to alarm after all devices have been unplugged, then the problem may lie in the OR wiring.



If the LIM alarms, unplug each device in the order they were most recently plugged in.

The faulty device should be replaced as soon as possible.

e) **Ground-fault circuit interrupters (GFCI)**
i) The GFCI monitors both sides of the circuit for the equality of current flow; if a current difference is detected (5 mA), the power is immediately shut off.
ii) Compared to using an LIM, the GFCI provides better shock protection as the device creating the shock risk is turned off quickly before a second fault can occur.
iii) The disadvantage is that the GFCI will automatically switch off power to any device, including those whose function cannot be interrupted without risk to the patient.
(1) To troubleshoot the GFCI, first reset the GFCI as temporary current surges can activate the GFCI in the absence of an increased shock risk.
(2) If the GFCI still triggers, then unplug each device one at a time, resetting the GFCI after each device is unplugged.
(3) If the GFCI stops triggering after the device is unplugged, then replace the device.
iv) When used in the OR, only one outlet should be protected by each GFCI.

3) **Substance use disorder (SUD)**[9–11]
a) SUD is recognized as an occupational hazard within the practice of medicine. It is a serious and continuing problem in anesthesiology. Patient safety and well-being of the provider are of the utmost importance. Adverse outcomes such as death are not uncommon.
i) Currently, physicians are reported to have a higher prevalence of SUD than the general population.
(1) It has not been established whether the incidence of SUD is higher in anesthesiologists compared with other physician specialties.
ii) Risk factors associated with the development of SUD are male gender, comorbid psychiatric illness, family history, prior substance abuse, work-related stress, burnout, depression, and anxiety.
iii) Death is substantially increased in anesthesia residents who develop SUD.[10]
(1) Other adverse outcomes include failure to complete residency and obtain board certification, risk of relapse after the initial episode, and adverse medical licensure actions after completing training.
b) Signs of SUD include signing out inappropriate quantities of narcotics for a given case, volunteering for extra work, frequent bathroom breaks, refusing relief for lunch or

coffee breaks, being slow to answer pages, and changes in behavior such as increased episodes of anger, irritability, and hostility and withdrawal from family, friends, and leisure activities.

i) Changes in behavior may be associated or coexist with burnout, fatigue, or depression.
ii) Often, the first sign of drug addiction is sudden death.

c) Anyone with drug use should be reported to supervisors and the proper authorities.
i) Confrontation with an impaired colleague should be done carefully and by professionals. There is potential for self-harm.
ii) If there is substantial evidence of SUD, a well-organized intervention should be conducted.
iii) Treatment includes both inpatient and outpatient therapies with medically supervised detoxification, monitored abstinence, education, and participation in group therapy.
iv) Recovery is possible, though relapse is high after the initial episode.
(1) Increased risk of relapse in physicians who use opioids or have coexisting psychiatric illness
v) Return to professional duties is considered on a case to case basis.
vi) The Americans with Disability Act provides some protection for patients recovering from addiction, although it does not protect active drug users. The ASA Executive Office provides a hotline that provides information for state programs that serve impaired physicians (847-825-5586).



SUD is recognized as an occupational hazard. It is a serious and continuing problem in anesthesiology. The risk of death is substantially increased during residency training and adverse medical licensure actions after training. Often the first sign of addiction is sudden death.



The ASA Executive Office provides a hotline that provides information for state programs that serve impaired physicians (847-825-5586). Ongoing education on SUD is essential to prevent, recognize, and react when SUD is identified in oneself or a colleague. Treatment and recovery is possible.

d) Ongoing education on SUD is essential to prevent, recognize, and react when SUD is identified in oneself or a colleague.[12]

4) **Falls/Slips**[13]

a) **An anesthetized patient falling from the OR table is a rare occurrence but can be a catastrophic event.**
i) Falls can occur during transfer to and from the OR table.
ii) It is important to make sure that the patient is properly secured with a safety strap.
iii) Vigilance should be maintained especially during periods of altered consciousness.
iv) Factors considered to contribute to the risks of patient falls include taller, wider, and more obese patients.
(1) Modern OR tables are capable of quickly placing patients in extreme positions (ie, side tilt, Trendelenburg, and reverse Trendelenburg), and specialized tables can rotate the patient along the long axis from prone to supine (ie, Jackson table).
v) Preventing falls is considered a shared responsibility of the OR staff.
(1) Familiarity with the bed controls, OR bed weight limitations, and patient positioning is important.



A patient falling from an OR table is rare but can be a catastrophic event.

b) **Fall risks among OR personnel are inherent hazards of the OR environment.**
i) Equipment, electrical cords, and wet floors, from irrigation, blood, and secretions increase the risk of slipping.
ii) Taking appropriate precautions such as wearing slip-resistant footwear, maintaining a trash-free environment, reporting spills of fluids, and organized cord management is essential to minimizing risk of injury.

FIGURE 8-2 Sample Wet Floor Sign Signage Is Useful as a Visual Alert When the OR Floor Is Wet From Spills or Between Case Cleaning.



iii) "CAUTION—WET FLOOR" sign is useful as a visual alert when the OR floor is washed between cases (**Fig. 8-2**).

5) **Blood-borne Pathogen Exposure**

a) Blood-borne pathogens in human blood that can cause disease include, but are not limited to, hepatitis B (HBV), hepatitis C (HCV), and human immunodeficiency virus (HIV). Needlesticks and other sharps-related injuries (percutaneous) represent the highest risk of exposure to all operating room personnel with anesthesia residents incurring the highest incidence in one multicenter study.[14]

b) **Mechanism of injury**

i) The two most common devices involved in needlestick injury (NSI) include disposable syringes (21%) and suture needles (24%).[16] These sharp devices are used for blood draw, administering injections, or suturing.

ii) The most common cause of NSI among anesthesia personnel occur from insertion of intravascular catheters, injection of intradermal local anesthesia, and suturing of intravascular catheters.[14]

iii) Injury with a large hollow-bore needle contaminated with infected blood is the most likely to cause seroconversion.

iv) Risk for NSI during catheter insertion and suturing may be increased when personnel are transitioning to a new practice location, utilizing new equipment, or learning a new skill. In addition, working in tandem or in close proximity with others during procedures may increase the risk of NSI for both individuals.

v) Contact of other potentially infectious body fluids (vaginal secretions, semen, cerebrospinal, etc.) with mucous membranes or nonintact skin present as other possible modes of transmission.



Maintain vigilance especially during periods of altered consciousness.

Take steps to safely transfer the patient to and from the OR table and secure the patient with safety straps.

Take appropriate precautions to minimize slipping and falling.



Blood contains the greatest load of pathogen and is the most critical transmission vehicle in the health care setting.[15]

Anesthesia personnel should consider all body fluids to be infectious in situations where it is difficult to differentiate between bloody fluids.

(1) Contact with saliva, nasal secretions, vomitus, or urine were not considered at risk unless blood was present.
(2) HBV, HCV, and HIV do not spontaneously penetrate intact skin, and airborne transmission of these viruses does not occur.

c) **Pathophysiology**
i) The most common transmittable diseases by NSI among health care workers are HBV, HCV, and HIV.
(1) Seroconversion.[15,17] See **Table 8-2**.
(a) Type of exposure and efficiency of transmission for that exposure (ie, exposure via percutaneous, mucosal, or non-intact skin)
(b) Prevalence of the infectious agent in the patient population being served
(c) Infectivity of the virus (viral load)

d) **Treatment and management**
i) Immediate treatment
(1) Wash site of contamination with soap and water.
(2) Irrigate exposed mucous membrane with water or sterile saline solution.
(3) Alcohol is virucidal to HIV, HBV, and HCV. Antiseptics, such as iodophors and chlorhexidine (CHG), also inactivate HIV.
(a) The efficacy of these agents in preventing HIV transmission is unknown.

e) **Determine risk associated with exposure**
i) Helps to determine an appropriate postexposure treatment plan
(1) Type of fluid (blood, blood contaminated fluid, or other contaminated fluid).
(2) Mode of exposure (NSI, nonintact skin, mucosal, or human bite).
(3) The date and time of the exposure.
(4) Describe the injury (depth of wound, sharp used (solid vs hollow needle).

f) **Report the injury**
i) Report the incident to your supervisor or the person in your practice responsible for managing exposures.
(1) Needlestick injuries by health care workers are underreported.
(2) Occupational health/emergency department (after hours) or agent responsible for managing the reported occupational exposure.



Health care workers (HCWs) who received hepatitis B vaccine and developed immunity to the virus are at virtually no risk for infection.

For an unvaccinated person, the risk from a single needlestick or a cut exposure to HBV-infected blood ranges from 6% to 30%.



HBV is highly infectious and can be transmitted in the absence of visible blood.

Blood contaminated with HBV remains viable on environmental surfaces for at least 7 days.

HCV has also been demonstrated to remain infectious on inanimate surfaces at room temperatures for prolonged periods of time.[18]

TABLE 8-2 Infectious Agent: Risk and Mode of Infection

Transmissible Agent	Incidence (%) Seroconversion	Mode
HBV	6-30	Percutaneous exposure The risk of acquiring depends upon the hepatitis B surface antigen (HBsAg) and hepatitis B e antigen (HBeAg) status of the patient
HCV	1.8	Percutaneous exposure
HIV	0.3 0.09	Percutaneous exposure Mucous membrane

ii) Every health care facility is required to comply with federal regulations by Occupational Safety and Health Administration (OSHA).

g) **Test the patient or source of exposure**
   i) Follow state and federal laws regarding patient confidentiality.
      (1) Informed consent may be required.
      (2) Review medical record for information about disease status of known source.
      (3) For unknown source, risk is based on prevalence of disease in the patient population.



High-risk exposure: large hollow-bore needle, deep penetration, visible blood contamination, and needle/sharp used to enter blood vessel.

h) **Therapy and follow-up should be based on the current CDC recommendations**[19,20]
   i) HBV—hepatitis B immunoglobulin and vaccination are given to individuals who are unvaccinated or with incomplete vaccination.
      (1) Ideally given within 24 hours, up to 1 week postexposure
      (2) 3-6 months protection
   ii) HCV—no postexposure prophylaxis (PEP) is currently available.
      (1) If the source (patient) is HCV positive or if the status of the source is unknown.
         (a) HCP should have baseline testing for anti-HCV within 48 hours of the exposure.
         (b) Repeat testing for anti-HCV is recommended at 3 weeks postexposure.
         (c) HCPs who test positive for HCV RNA should be referred to a specialist.
            (i) Treatment is associated with an excellent cure rate.
   iii) HIV[21]
      (1) Rapid HIV testing of source patient.
         (a) It is sensitive and specific and can be used to determine whether to offer PEP.
      (2) Expert consultation is recommended for any occupational exposures to HIV.
      (3) An HIV antibody test for the exposed HCP should be done, and then repeated 6 month postexposure.
      (4) Antiretroviral PEP is most effective when started within hours of exposure.
         (a) PEP antiretroviral regimens consist of three drugs for 28 days.
            (i) If the source patient is later determined to be HIV negative, PEP could be discontinued, and no further HIV follow-up testing is indicated for the exposed provider.
            (ii) Potential adverse effect can include myalgias, fatigue, nausea, peripheral neuropathy, neutropenia, and pancreatitis.
            (iii) Early reevaluation within 72 hours after exposure is strongly recommended.
      (5) Risk reduction strategies should be practiced by the HCP to prevent secondary transmission until the follow-up HIV serology (6- to 12-week postexposure) is negative.[22]
         (a) Sexual abstinence or use of condoms.
         (b) Refrain from blood/tissue donation.
         (c) Women with childbearing potential will require special counseling on PEP.
            (i) Discontinuation of breast-feeding, though not a contraindication to PEP, should be discussed.



The source and type of exposure are important in determining an HIV postexposure regimen.

Clinical Consultation PEPline (888) 448-4911 provides up-to-date expert advice on exposure management. Website: http://www.nccc.ucsf.edu/

i) **Risk reduction**
   i) Vaccination
      (1) Health care workers who received hepatitis B vaccine and developed immunity to the virus are at virtually no risk for infection with HBV.

ii) Standard precautions established by CDC, previously "universal" precautions CDC, recommend
  (1) The minimum infection prevention practice applied to all patient care, regardless of the health care setting or whether a patient is known or suspected to carry disease
    (a) Treat all blood and other potentially infectious materials with appropriate precautions.
    (b) Wash hands before and after patient contact.
    (c) Use barrier devices such as personal protective equipment (PPE) gloves, masks, eye protection or face shield, and gowns if blood or exposure to other potentially infectious materials is anticipated.
    (d) Use engineering and work practice controls to limit exposure.

j) **Management of sharps**
  i) Containers for contaminated sharps must be puncture-resistant, leakproof, and appropriately labeled as biohazardous waste to warn everyone that the contents are hazardous; it should contain a closable lid and be positioned upright.
  ii) All needles and sharp objects should be properly disposed of into the sharps container (**Fig. 8-3**).
  iii) Avoid two-handed recapping of needles.
    (1) If unavoidable, sharps should be passed between personnel in a "neutral zone" such as emesis basin or Mayo stand.
  iv) Never open or reach into sharps container to remove anything.



Hypodermic injections using disposable syringes and needles are the most frequent cause of NSIs.

If you accidentally drop something into a sharps container, NEVER reach inside.

k) **Safety devices** reduce NSI by 43%[23]
  i) Use engineering controls, such as autodisposable syringes, needleless devices, and retractable or sheathed needles.

FIGURE 8-3 Sharps Container



ii) Multiple needle and sharps safety devices are available, and familiarity and proper use are paramount in decreasing sharps injuries.
iii) Experts agree that safety devices and work practices alone will not prevent all sharps injuries.
    (1) Significant declines in sharps injuries also require
        (a) Education
        (b) A culture of safety



Multiple needle and sharps safety devices are available. The use of safety devices, education, and culture of safety all contribute to a decrease in NSI.

CHAPTER SUMMARY FOR HAZARDS OF THE OR

Types of OR Hazards	Occupational exposure to inhaled waste anesthetic gases, electrical hazards, potential for substance use disorder (SUD), falls/slips, exposure to blood-borne pathogens, and needlestick/sharps injury (NSI).
Waste Anesthetic Gases (WAGs)	WAGs can be caused by inhalational inductions, poorly fitting masks, uncuffed endotracheal tube cuffs, and leakage of anesthetic during filling vaporizers. Scavenging of WAGs is essential in controlling the concentration of exposure. Anesthesiologists need to be aware of the effect of WAG on the environment.
Electrical Safety	Electrical shocks occur when a person becomes part of an electrical circuit with contact leading to cellular damage, seizures, burns, ventricular fibrillation, and even death. Macroshock refers to large amounts of current flowing through a person, which can cause harm or death. Microshock refers to small amounts of current delivery to the myocardium in "susceptible" patients. To decrease the risk of a shock, modern OR power is electrically isolated from the ground by use of a line isolation transformer. If the line isolation monitor alarms, unplug each device in the order they were most recently plugged in.
Substance Use Disorder	SUD is a serious and ongoing problem in anesthesiology. Risk of death is substantially increased during residency training and adverse medical licensure actions after training. Often the first sign of addiction is sudden death. Treatment is available; relapse is high. The ASA executive office provides a hotline for information for state programs that serve impaired physicians (847-825-5586).
Falls/Slips	An anesthetized patient falling from the OR table is a rare occurrence but can be a catastrophic event. It is important to make sure that the patient is properly secured with a safety strap. Fall risks among OR personnel are inherent hazards of the OR environment. Equipment, electrical cords, and wet floors are unavoidable in the OR; vigilance and reporting minimize risks.
Blood-borne Pathogens	Occupational exposures to the most common infectious agents are HBV, HCV, and HIV. Of the blood-borne diseases, HBV is the most transmissible infection and remains viable on environmental surfaces for at least 7 days.
Needlestick/Sharps Injury	High risk of exposure with large hollow-bore needle, deep penetration, visible blood contamination, and needle/sharp used to enter blood vessel. Immediately wash site and report incident to proper authorities. Test source and follow CDC guidelines for postexposure prophylaxis.
Risk Reduction	Health care workers should receive hepatitis B vaccine and practice standard precautions as established by CDC, inclusive of hand hygiene and personal protective equipment. All sharp objects should be properly disposed.

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9 Standard ASA Monitors

Trent Bryson

In 1986 the American Society of Anesthesiologists (ASA) approved the first edition of *Standards for Basic Anesthetic Monitoring*. Periodically these standards are updated according to advances in technology and practice, the most recent being in 2015.[1] The standards are to be considered a minimum during **all general, regional, and monitored anesthetic care** and may be exceeded at any time as deemed necessary. These measures are to only be suspended when interfering with emergent life supportive measures or in the rare instance when they are impractical or failing to detect clinical developments.

1) **Overview of required elements—**Published standards address broad elements instead of specific devices and are broken down into two required standards.[2]
   a) **Standard I**
      i) The continuous presence of a qualified anesthesia provider is required throughout the duration of anesthetic care due to the rapidity with which physiologic derangements may occur during surgical intervention.
   b) **Standard II**
      i) Continual monitoring of oxygenation, ventilation, circulation, and temperature.
      ii) Frequency of mandatory monitoring varies between each category but never exceeds 5 minutes.
         (1) If not used, a reason should be recorded on the patient record.
      iii) The following are all specifically mandated:
         (1) Oxygen analyzer with a low inspired concentration limit alarm during general anesthesia.
         (2) Quantitative assessment of blood oxygenation.
         (3) Ensuring adequate ventilation during all anesthetic care including verification of expired oxygen (when possible), quantitative measurement of tidal volume, and capnography in all moderate or deep sedation as well as all general anesthetics.
         (4) Qualitative evaluation of ventilation is required during all other care.
         (5) Ensure correct placement of endotracheal tube or laryngeal mask airway via expired carbon dioxide (CO_2).
         (6) Alarms for disconnects when a mechanical ventilator is used.
         (7) Continuous display of ECG.
         (8) Determination of arterial BP and heart rate.
         (9) Adequacy of circulation is to be determined by quality of pulse electronically, through palpation, or through auscultation.
         (10) The means to determine temperature must be available and should be employed when changes in temperature are anticipated or intended.
2) **Oxygen analyzer**
   a) Most modern anesthesia machines monitor both inspired and expired concentrations of O_2.



Continuous presence of a qualified anesthesia provider is a standard set by the ASA.

b) This is essential during anesthesia because it is possible to deliver a hypoxic gas mixture when mixing O_2, air, nitrous oxide, and/or volatile anesthetic agents.



An audible alarm should be set to notify the anesthesia provider of a hypoxic gas mixture.

3) **Pulse oximetry**
   a) Provides quantitative analysis of the patient's saturation of hemoglobin with O_2
4) **Carbon dioxide (CO_2)**
   a) Inspired and expired CO_2 should be monitored.
   b) Expired CO_2 is frequently displayed through capnography with a displayed value correlating to the peak expired CO_2 of each breath.
   c) **Capnography**
      i) Provides qualitative and quantitative information regarding expired CO_2.
      ii) Quantitatively, this is useful to ensure that the endotracheal tube is within the respiratory tract as well as to ensure adequate cardiac output.

READ MORE
Pulse oximetry, Chapter 10
Capnography, Chapter 11

   d) Inspired CO_2
      i) Monitored to ensure that the CO_2 absorber of the anesthesia machine is adequately removing all CO_2 from the circuit.
      ii) If inspired CO_2 is >0, consider changing the absorbent. The absorbent turns blue when its capacity is exhausted.
5) **Multiple expired gas analysis**
   a) While not an essential monitor according to the ASA, this allows determination of the percent inspired and expired of the volatile agents and nitrous oxide.
   b) This allows the ability to better determine the delivery of an adequate anesthetic without over- or underdose.
6) **ECG**
   a) A minimum of three leads is to be used, although five leads are used for most adults.[3]
   b) Consideration must be taken for the surgical field and patient positioning.
      i) Lead placement is commonly altered for cases involving the chest, shoulders, back, and neck.
   c) **Five-lead ECG**
      i) Includes the right arm (RA), left arm (LA), right leg (RL), left leg (LL), and V.
      ii) The five-lead arrangement can be used to display I, II, III, aVR, aVL, aVF, and/or V (**Fig. 9-1**).

FIGURE 9-1 Normal Five Lead ECG Lead Placement

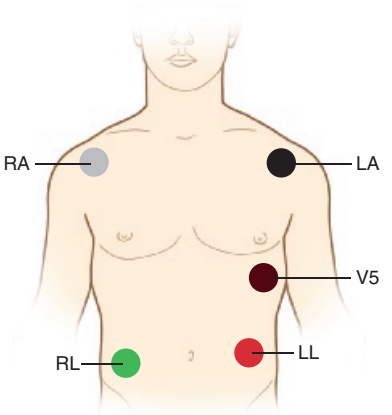
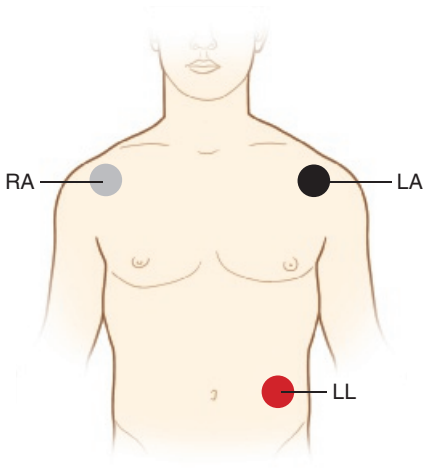


FIGURE 9-2 Normal Configuration of a Three-Lead EKG



d) **Three-lead ECG**
   i) Includes the RA, LA, and LL leads and can be used to display leads I, II, and/or III (**Fig. 9-2**).
      (1) A three-lead ECG can be modified to display V5 by moving the LA lead to the V5 position in the fifth intercostal space at the anterior axillary line (**Fig. 9-3**).

e) **The most commonly monitored leads are II and V5.**
   i) II is best used to monitor rhythm because it provides the best visibility of the P wave.
   ii) V5 monitors for anterior and lateral ischemic events.
   iii) Most anesthesia machines will allow you to change how many and which leads are displayed.
      (1) Some will show all leads simultaneously, although this often removes all other monitors from the display while viewing all leads.
      (2) If an arrhythmia or ischemic event appears to be present, temporarily viewing all leads simultaneously may be helpful for diagnostic purposes.

7) **Arterial blood pressure (BP)**
   a) **BP can be monitored invasively or noninvasively.**
   b) **Noninvasive methods**
      i) Include palpation, auscultation, Doppler probe, oscillometric cuff, or tonometry.
      ii) Palpation, auscultation, Doppler, and tonometry are rarely employed and are not discussed here.
   c) **Automatic oscillometric**
      i) The cuff is able to sense oscillations in cuff pressure which correlate with arterial pulsation.

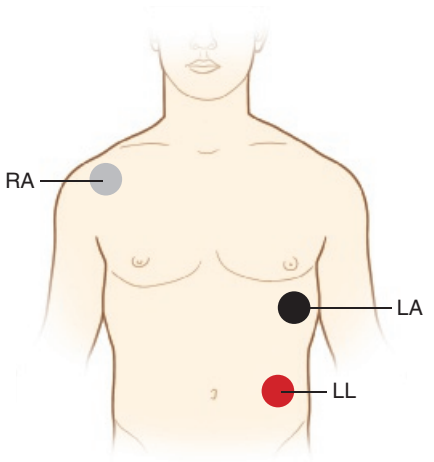


EKG leads are color-coded to assist placement. RA and LA leads are easily remembered: imagine the LA becomes suntanned (black) driving a car with the window open, while the RA remains untanned (white) inside the car.



For most patients, printing the ECG tracing at the beginning of the case allows for later comparison should an intraoperative event occur.

FIGURE 9-3 Modification of Three-Lead ECG



Modification of the three-lead ECG allows display of both leads II and V5. Lead V5 is monitored through the display of lead I in this configuration.

ii) The cuff is inflated beyond the point at which oscillation ceases and then is slowly deflated.
iii) The oscillation in cuff pressure begins at systolic pressure, peaks at mean arterial pressure, and disappears once again at diastolic pressure.
iv) Due to the speed at which the cuff deflates, all three values are mathematically derived, with MAP being the most accurate (**Fig. 9-4**).
v) Placement
(1) Each cuff is labeled with an arrow pointing to where arterial pulsation is felt best.
(2) The cuff is then placed on the arm over the brachial artery, forearm over the radial artery, or thigh/calf over the popliteal artery.
vi) Patient positioning
(1) When monitoring noninvasive pressure, consideration must be taken of patient position.
vii) Invasive BP monitoring

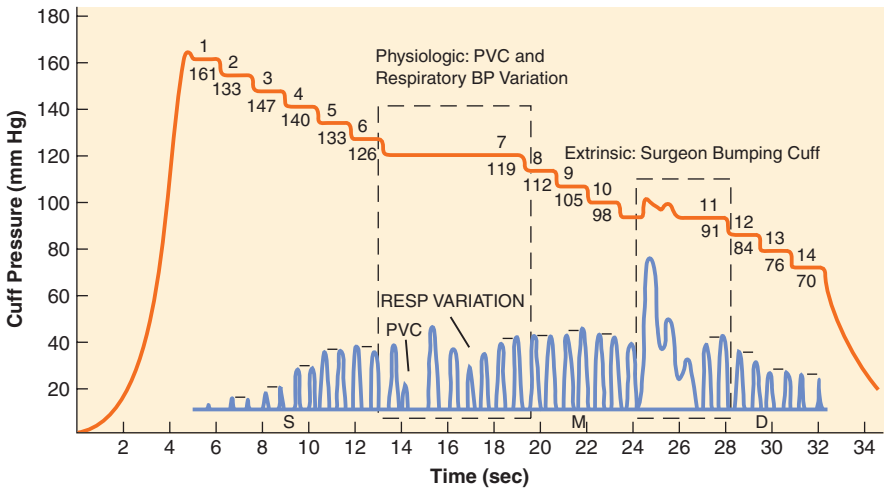


Patients in the beach chair position are especially prone to cerebral ischemia even with "normal" brachial BPs.

8) **Temperature**
a) Temperature changes should be anticipated and expected under any general anesthetic and therefore any general anesthetic requires temperature measurement.
i) Very brief procedures may be an exception, but the availability of temperature monitoring should be recorded.
b) The temperature may be measured from many locations including skin, nasopharynx, esophageal, bladder, rectal, or a pulmonary arterial catheter.
c) Core temperatures obtained from a pulmonary catheter, esophageal stethoscope, or rectal probe are preferable sources.

READ MORE
Invasive arterial blood pressure monitoring, Chapter 12

FIGURE 9-4 Illustration of Oscillometric Cuff Pressure Measurement Methodology With Corresponding Cuff Measurements With Various Common Sources of Artifact



Reproduced with permission from Greenberg SB, Murphy GS, Vender JS. Standard monitoring techniques. In: Barash PG, Cullen BF, Stoelting RK, et al., eds. *Clinical Anesthesia*. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009:702.

CHAPTER SUMMARY FOR STANDARD MONITORS

Definition	ASA standard monitors are to be considered a minimum during **all general, regional, and monitored anesthetic care** and may be exceeded at any time as deemed necessary. *These measures are to only be suspended when interfering with emergent life supportive measures or in the rare instance when they are impractical or failing to detect clinical developments.*		
Function	**Physical Signs**	**Monitor**	**Equipment Alarms and Monitoring**
Oxygenation	Color of skin and mucous membranes	Pulse oximeter, inhaled oxygen concentration	Low limit alarms
Ventilation	Breath sounds, chest excursion	Ventilator, end-tidal CO_2 analysis	Disconnect alarm, tidal volume measurement, $ETCO_2$ alarms
Circulation	Pulse palpation, auscultation of heart sounds	ECG BP Heart rate	ECG, BP cuff, arterial line, pulse oximeter
Temperature	Skin	Temperature probe	Low-temperature alarm

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10 Pulse Oximetry

Alison Brainard

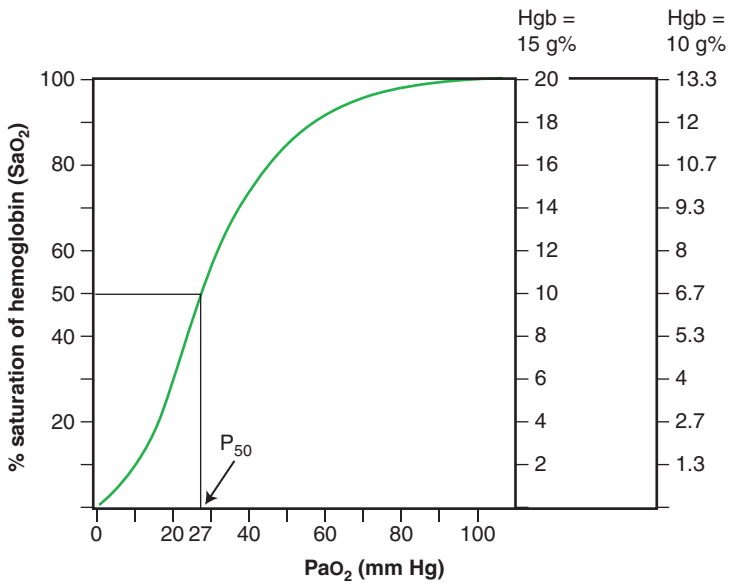
Pulse oximetry is one of the most commonly employed monitoring modalities in anesthesia. It is a noninvasive way to monitor the oxygenation of a patient's hemoglobin. A sensor with both red and infrared wavelengths is placed on the patient. Absorption of these wavelengths by the blood is measured and oxygen saturation (SpO_2) can be calculated.

1) **Basic concepts**
   a) Pulse oximetry measures the amount of oxyhemoglobin using the Lambert-Beer law.
      i) The Lambert-Beer law is a mathematical means of expressing how light is absorbed by matter.[1]
   b) **There are two main types of oximetry:** fractional oximetry and functional oximetry.[2]
      i) Fractional oximetry
         (1) Fractional oximetry measures the arterial oxygen saturation (SaO_2).
         (2) Oxyhemoglobin/(oxyhemoglobin + deoxyhemoglobin + methemoglobin + carboxyhemoglobin) = SaO_2.
         (3) It can only be measured by an arterial blood sample.
      ii) Functional oximetry
         (1) Functional oximetry expresses the SpO_2.
         (2) Oxyhemoglobin/(oxyhemoglobin + deoxyhemoglobin) = SpO_2.
         (3) It can be measured noninvasively by a standard pulse oximeter.
2) **How pulse oximetry works**[2]
   a) A pulse oximeter emits two wavelengths of light: red (660 nm) and infrared (940 nm).
      i) Deoxyhemoglobin absorbs more light in the red band.
      ii) Oxyhemoglobin absorbs more light in the infrared band.
   b) Sensors in the oximeter detect the amount of red and infrared light absorbed by the blood.
   c) Photoplethysmography is then used to identify pulsatile arterial flow (alternating current [AC]) and nonpulsatile flow (direct current [DC]).
   d) The ratio of AC/DC at both 660 and 940 nm is measured using the equation $(AC/DC)_{660}/(AC/DC)_{940}$.
   e) The pulse oximeter calculates the SpO_2 by taking the above equation and using an algorithm built into the software to derive the SpO_2.
      i) The calibration to derive SpO_2 from the $(AC/DC)_{660}/(AC/DC)_{940}$ ratio was made from studies of healthy volunteers.
3) **Accuracy of the pulse oximeter**[2]
   a) If the SpO_2 is between 70% and 100%, the pulse oximeter is accurate to within 5%.
   b) SaO_2 measurement remains the gold standard in critically ill patients and neonates.
   c) The oxygen dissociation curve flattens above 95%, which can create dangerous hyperoxic states in newborns putting them at higher risk of ROP (retinopathy of prematurity).
   d) It is not accurate below 70% because calibration of the pulse oximeter involved healthy volunteers whose SpO_2 did not routinely reach levels <70%.



SpO_2 measurements are not accurate below 70%.

FIGURE 10-1 The Oxygen-Hemoglobin Dissociation Curve



The oxygen dissociation curve showing the relationship between SpO_2 and PaO_2. P_{50} is the PaO_2 at which hemoglobin is 50% saturated with oxygen. The normal value is 27 mm Hg. Adapted with permission from Martin L. *All You Really Need to Know to Interpret Arterial Blood Gases*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 1999.

i) For the relationship between SaO_2 and PaO_2 (see **Fig. 10-1**).[3]
ii) It is also not as accurate below 70% because there is more deoxyhemoglobin present.[3]
(1) The absorption spectrum of deoxygenated hemoglobin is very steep at 600 nm in the red range so small changes in the amount of deoxyhemoglobin can cause very wide variances in SpO_2 (**Fig. 10-2**)

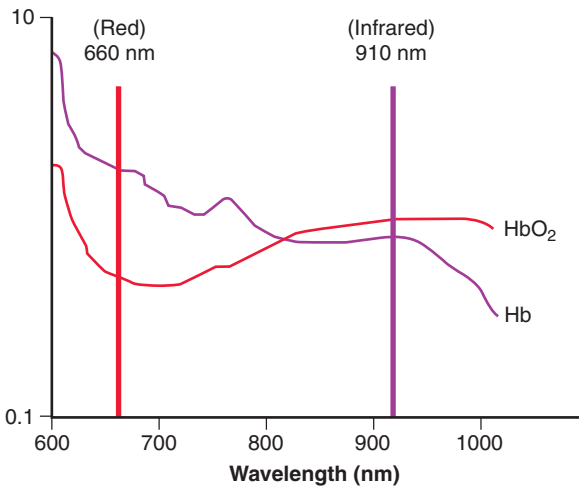


SpO_2 of 90% correlates to a PaO_2 of 60 mm Hg.

e) Ear probes may be more accurate than finger probes because the probe is closer to the heart.[4]
f) Pulse oximetry is not as accurate in low amplitude states (**Table 10-1**).
i) Low perfusion makes it difficult for the pulse oximeter to distinguish a true signal from background noise.

4) **Oximetry paradoxus**[5]
a) **Pulsus paradoxus** is the exaggerated decline in systolic blood pressure during a spontaneous inspiration ≥10 mm Hg or during mechanical ventilation exhalation >10 mm Hg.
i) This can easily be measured using invasive arterial blood pressure monitoring and comparing peak systolic blood pressures during different phases of ventilation.
b) Oximetry paradoxus uses pulse oximetry to estimate blood flow variation during inhalation and exhalation. If maximal amplitude of the pulse oximetry curve varies by more than 10%, this can indicate pulsus paradoxus.

FIGURE 10-2 Light Absorption With Oxygenated and Deoxygenated Hemoglobin



Oxygenated hemoglobin (*red line*) absorbs more infrared light and allows more red light (*vertical red line*) to pass through. Deoxygenated hemoglobin (*purple line*) absorbs more red light and allows more infrared light (*vertical purple line*) to pass through. Adapted from Red and infrared light absorption. http://www.oximeter.org/pulseox/principles.htm. Accessed March 8, 2010.

i) This can indicate cardiac tamponade, right heart failure, obstructive lung disease, and severe hypovolemic states.
ii) If severe paradoxus presents, it is important to decipher the cause using echocardiography, trial of fluid bolus, auscultation, etc.

5) **Dyshemoglobinemias**[3]
   a) Pulse oximetry only accurately measures oxyhemoglobin and deoxyhemoglobin—all other forms of hemoglobin are not accurately measured.
      i) Carboxyhemoglobin is measured as 90% oxyhemoglobin and 10% deoxyhemoglobin.
         (1) Thus, when there are high amounts of carboxyhemoglobin, it will overestimate the SpO_2.
         (2) This is an important consideration in patients exposed to smoke or fires.
      ii) Methemoglobin absorbs equal amounts of red and infrared light, so the SpO_2 will read 85%.

TABLE 10-1 Low Amplitude States

- Hypovolemia
- Hypothermia
- Cardiac arrest
- Arrhythmias
- Cardiac bypass
- Vasoconstriction
- Tourniquet
- BP cuff inflation

TABLE 10-2 Causes of Methemoglobinemia

- Nitrates/Nitrites
- Local anesthetics (eg, benzocaine or Hurricaine Spray)
- Chlorates
- Antimalarials
- Antineoplastics
- Sulfonamides
- Dapsone
- Metoclopramide

(1) Methemoglobin is formed when iron goes from its +2 ferrous form to the +3 ferric state
(2) The ferric state of iron displays a left shift on the oxygen dissociation **curve** and releases oxygen less easily.
(3) Methemoglobinemia can be caused by many drugs (**Table 10-2**).

iii) Patients with sickle cell anemia presenting in a vasoocclusive crisis can have an inaccurate SpO_2 reading.
iv) High levels of bilirubin do not alter SpO_2 readings.

6) **Other limitations**[3]
   a) **IV dyes**
      i) Methylene blue, indocyanine green, and indigo carmine all cause transient decreases in the SpO_2 lasting 30 seconds to up to 20 minutes.
   b) **Dark skin pigmentation:** melanin may inhibit passage of light through tissue.
   c) **Motion artifact:** the pulse oximeter becomes unable to measure peaks and troughs of the waveform correctly.
   d) **Fluorescent light** emits wavelengths in the 660 nm region, which interferes with the red band of the pulse oximeter.
   e) **Nail polish** inhibits passage of light through nail.

CHAPTER SUMMARY FOR PULSE OXIMETRY

Definition	Pulse oximetry is noninvasive method to monitor oxygenation of a patient's hemoglobin using light.
How It Works	Sensors in the pulse oximeter probe measure the ratio of red (660 nm) and infrared (940 nm) light absorbed during pulsatile and nonpulsatile flow and use software to derive an SpO_2 value based on data from healthy volunteers.
Limitations	Not accurate when SpO_2 is below 70% Not accurate in low amplitude states Does not accurately measure dyshemoglobinemias IV dyes, motion artifacts, dark skin pigmentation, and nail polish can interfere with measurement.

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11 Capnography

Jacob Elliott Schaff • Jessica Spellman

Monitoring of CO_2 through capnometry and capnography can be a valuable tool for detecting acute alterations in ventilation, metabolism, and circulation. Continuous monitoring of CO_2 is an American Society of Anesthesiology (ASA) standard for monitoring in all patients receiving moderate to deep sedation and general anesthesia (unless invalidated by the nature of the patient, procedure, or equipment) and for confirming proper endotracheal tube and supraglottic airway placement.[1]

1) **Definitions**[2]
   a) **Capnometry**
      i) Measurement and numeric representation of CO_2 concentration or partial pressure in respiratory gases during inspiration and expiration
   b) **Capnogram**
      i) Concentration-time display of CO_2 concentration sampled at a patient's airway
   c) **Capnography**
      i) Continuous monitoring of a patient's capnogram during the respiratory cycle



Capnography is continuous monitoring of a patient's capnogram during the respiratory cycle.

2) **Methods of capnometry**[3]
   a) **Sidestream capnometers**
      i) Sample tubing connected to the airway that continually aspirates respiratory gases at a rate of 50-400 mL/min to a measurement chamber
      ii) Advantages
         (1) Allows for accurate capnography when attached as close as possible to the patient
         (2) Lightweight
         (3) Can be used to measure CO_2 and anesthetic gases
      iii) Disadvantages
         (1) Condensation of humidified gases may cause frequent clogging of the tubing (water traps and filters must be used to prevent condensation from reaching the measuring chamber).
         (2) Delay in measurement response time depending on the size and length of the tubing.
   b) **Mainstream capnometers**
      i) Light absorption chamber directly attached to the airway
      ii) Advantages
         (1) Direct measurement allows for fast response time
      iii) Disadvantages
         (1) Bulky equipment placed directly in endotracheal tube
         (2) Does not measure anesthetic gases
3) **Techniques of CO_2 measurement**[4]
   a) **Infrared spectrography**
      i) Employed by most operating rooms
      ii) In the measuring chamber, gases are exposed to an infrared light beam

iii) Each molecule (CO_2, anesthetic gases) has a specific absorbance spectrum of the infrared beam that can be quantified

b) **Mass spectrography**
   i) Measurement of CO_2 and other gas concentrations on the basis of differing molecular weights

c) **Raman spectrography**
   i) The use of spectral analysis of scattered light energy that results from an argon laser light source exposed to a gas mixture

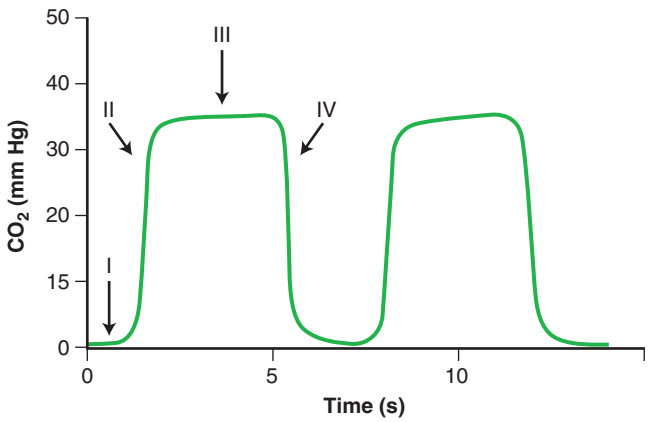
d) **Colorimetric CO_2 analysis**[5]
   i) pH-sensitive test paper
   ii) When in the presence of CO_2, color change occurs
   iii) Can be used to confirm CO_2 and endotracheal intubation outside of the operating room



Colorimetric CO_2 analysis is often used to confirm CO_2 and endotracheal intubation outside of the operating room.

4) **Normal capnogram**[6] **(Fig. 11-1)**
   a) **Phase I**
      i) Initiation of expiration
      ii) CO_2-free gas from anatomic dead space
   b) **Phase II**
      i) Expiration of mixture of dead space and alveolar gas
   c) **Phase III**
      i) Alveolar plateau
      ii) CO_2-rich gas from alveoli
   d) **Phase IV or 0**[7]
      i) Inspiration
5) **Clinical uses of capnography**[4]
   a) **Confirmation of endotracheal intubation**
   b) **Monitoring** adequacy of ventilation in controlled or spontaneously ventilating patients

FIGURE 11-1 Phases of a Normal Capnogram



Reproduced with permission from Eisenkraft JB, Leibowitz AB. Hypoxia and equipment failure. In: Yao FS, ed. *Yao & Artusio's Anesthesiology: Problem-Oriented Patient Management*. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008:1185.

c) **Noninvasive estimate of $PaCO_2$**
   i) Assumes the normal 2-5 mm Hg difference between expired ($PETCO_2$) and arterial ($PaCO_2$) that exists in the awake state is present.
   ii) The gradient between $PETCO_2$ and $PaCO_2$ may be increased with age, pulmonary disease, pulmonary embolus, low cardiac output, and hypovolemia.



Capnography can be used to monitor the adequacy of ventilation in controlled or spontaneously ventilating patients.

d) **Detection of patient disease (Table 11-1)**
   i) **Table 11-1** outlines the abnormalities of CO_2 production in various patient disease states.
   ii) Airway obstruction may be detected due to abnormalities in the capnography tracing
      (1) Bronchospasm
      (2) COPD
      (3) Mechanical obstruction/kinked endotracheal tube



Utilize capnography to detect esophageal intubation! Watch out for end-tidal CO_2 that quickly goes away with subsequent attempts at ventilation **(Fig. 11-3)**.

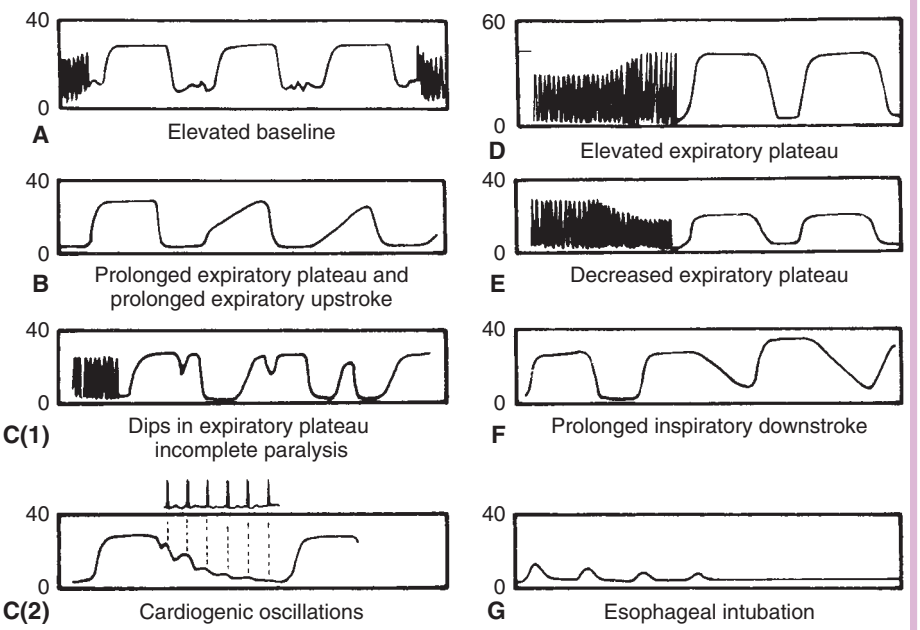
e) **Detection of problems with the anesthetic breathing system**
   i) Rebreathing
   ii) Incompetent valves
   iii) Circuit disconnects
   iv) Circuit leak

6) **Interpretation of abnormal capnograms**[3,4,6] **(Fig. 11-2)**
   a) **Rebreathing of CO_2**
      i) Elevation in baseline CO_2 and phase I
      ii) Can eliminate by increasing fresh gas flow or changing CO_2 absorber
   b) **Obstruction to expiratory gas flow**
      i) Prolonged phase II and steeper phase III slope
      ii) Occurs with bronchospasm, COPD, and kinked endotracheal tube
   c) **"Curare cleft"**
      i) Dip in phase III
      ii) Indicates return of spontaneous respiratory efforts
   d) **Cardiogenic oscillations**
      i) Oscillations of small gas movements during phases III and IV (or 0)
      ii) Produced by aortic and cardiac pulsations
   e) **Increased CO_2**
      i) Elevated plateau height
      ii) Indicates increased CO_2 production states (see above), other sources of CO_2 (as in laparoscopic surgery), or inadequate minute ventilation
   f) **Decreased measured CO_2**
      i) Decreased plateau height
      ii) May indicated decreased CO_2 production state (see above) or increased minute ventilation

TABLE 11-1 Abnormalities in CO_2 Production

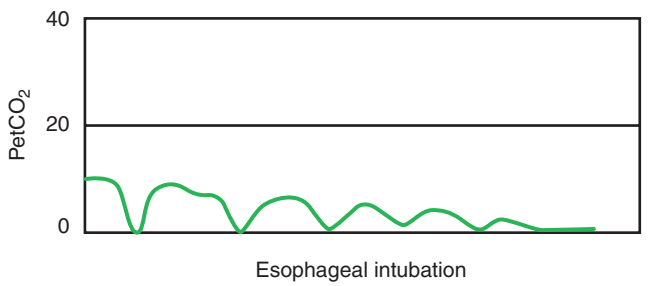
Causes of Increased CO_2 Production	Causes of Decreased CO_2 Production
Fever	Decreased cardiac output
Sepsis	Hypovolemia
Malignant hyperthermia	Pulmonary Embolism
Shivering	Hypothermia
Hyperthyroidism	Hyperventilation
Exogenous (insufflation)	

FIGURE 11-2 Abnormal Capnograms



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FIGURE 11-3 Esophageal Intubation Capnogram



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g) **Incompetent inspiratory valve**
   i) Prolonged phase III with elevation of baseline CO_2 and plateau height
   ii) Results in rebreathing
   iii) May be difficult to detect without simultaneous analysis of flow waveforms[7]
h) **Esophageal intubation (Fig. 11-3)**
   i) Initial presence of CO_2 followed by no CO_2 detection

CHAPTER SUMMARY FOR CAPNOGRAPHY

Definition	Capnography is the continuous monitoring of a patient's CO_2 during the respiratory cycle.
Intraoperative Use	Basic standard for ASA monitoring in moderate to deep sedation and general anesthesia. Also utilized to confirm endotracheal intubation
Techniques for CO_2 Measurement	Most operating rooms utilize sidestream capnometers and infrared spectrography to sample and measure CO_2.
Clinical Applications	Continuous monitoring for alterations in the CO_2 waveform may be useful for detecting esophageal intubation, rebreathing, hypermetabolic or low cardiac output states, obstruction, and return of spontaneous respiratory efforts.

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12 Invasive Arterial Blood Pressure Monitoring§

Sarah J. Cano • Alyssa B. Brzenski

Invasive arterial blood pressure (BP) monitoring allows for continuous, beat-to-beat monitoring of arterial BP displayed as a waveform and can provide reliable access for frequent arterial blood sampling.

1) **Indications**
   a) Patients requiring instantaneous analysis of BP including, but not limited to, those with end-organ disease, prone to wide and rapid changes in BP, and patients or procedures requiring special anesthetic techniques, such as trauma, cardiac, vascular, thoracic, spine, obstetric hemorrhage, craniotomies, and monitored hypotension.
   b) Procedures with anticipated fluid shifts where multiple blood samples for arterial blood gas analysis may be needed
   c) Long procedures to reduce risk of noninvasive blood pressure–related nerve damage
2) **Mechanics**
   a) Arterial lines utilize saline-filled tubing, which is connected directly into the artery via a catheter, to transmit pressure waves to a transducer.
   b) Transducers consist of a diaphragm in the base of a plastic dome that converts pressure changes into the arterial pressure tracing.
      i) As the diaphragm moves, a silicon crystal is stretched and its resistance changes.
      ii) Changes in resistance are interpreted as voltage changes, which are amplified and filtered into a tracing.
      iii) This is based upon the strain gauge principle, where stretching a wire or silicon crystal changes its resistance.[1]
   c) In the supine patient, the transducer should be placed at the midaxillary line, corresponding to the level of the right atrium, and must be readjusted with patient-level adjustments.
   d) When patients are in the sitting position, cerebral pressure can also be determined by placing the transducer at the level of the external auditory meatus, which approximates the level of the circle of Willis.
   e) The transducer must be zeroed to create a reference point, most commonly to atmospheric pressure. This is accomplished by opening the line and transducer to room air via a stopcock.
   f) Due to hydrostatic pressure, every 1-cm change in height of the transducer correlates with a 0.75-mm Hg pressure difference. Thus, raising a zeroed transducer 20 cm from the right atrium will lower the systolic and diastolic pressure reading by 15 mm Hg.
   g) The fluid column between the catheter and transducer has no effect on the pressure reading. Thus, raising or lowering a patient's arm with a radial catheter will not affect the reading from a stationary transducer.



Raising or lowering the transducer will alter the BP reading, but changes in position of the patient's arm where the arterial catheter is located will not change the reading.

3) **Arterial access sites**

a) **Radial artery**

i) Commonly cannulated due to its superficial location and good collateral flow, using a 20- or 22-gauge catheter (22- to 24-gauge for children and infants)

ii) While 5% of individuals have an incomplete palmar arch, far fewer patients develop complications from vascular compromise following arterial cannulation.

iii) The Allen test, Doppler probe, and pulse oximetry are all proposed methods to evaluate collateral flow, although none have been clinically proven to improve outcomes or prevent complications.

The ulnar artery can be cannulated; however, it is technically more difficult and should be avoided once attempts on the radial artery have been made.

b) **Brachial artery**

i) The brachial artery is large and easily identifiable.

ii) The artery's proximity to aorta reduces waveform distortion.

iii) Brachial artery cannulation was traditionally avoided because hematoma formation or injury could potentially threaten the vascular supply to the entire forearm and risk injury to the adjacent median nerve.

iv) Evidence suggests that the complication rate following brachial arterial cannulation is lower than previously reported, making this site an acceptable alternative for cannulation.[2,3]

v) Ultrasound use during placement may reduce the incidence of injury to adjacent structures.

c) **Axillary artery**

i) Generally avoided because it is adjacent to the axillary plexus, making nerve injury a concern.

ii) The artery is also much closer to the cerebral circulation, potentially increasing the risk of cerebral embolism during flushing.

d) **Femoral artery**

i) The femoral artery provides rapid and accurate access using a long 18- or 20-gauge catheter.

ii) Prone to pseudoaneurysm and atheroma formation.

iii) Aseptic necrosis of femoral head in children is a rare but reported complication.

iv) Infection rates are generally higher than radial cannulation, although some evidence suggests comparable rates of infection.[4-6]

READ MORE
Atlas of anesthesia procedures, Radial artery catheterization, Chapter 167

e) **Other**

i) Dorsalis pedis and posterior tibial arteries form an anastomotic arch in the foot analogous to the hand.

ii) They are appropriate alternatives in situations of difficult upper extremity access such as trauma or neurosurgery.

iii) Theses arteries are far from the aorta and therefore prone to more distortion and systolic pressure exaggeration.[1]

Increasing use, familiarity, and availability make ultrasound a viable option when difficulty during placement is either encountered or anticipated.

f) **Cannulation technique**

i) Sterile precautions must be followed during placement of an arterial line. Multiple techniques for placement exist and are largely determined by practitioner familiarity.

ii) In general, the catheter can be advanced directly off the needle or using the "through and through" artery technique. Either technique can be performed using a Seldinger technique by first threading a wire.

FIGURE 12-1 Pulse pressure variation (PPV) equation

$$PPV = \frac{PPmax - PPmin}{PPmean} \qquad SVV = \frac{SVmax - SVmin}{SVmean}$$

4) **Interpretation**
   a) **Waveform**
      i) The rate of upstroke generally correlates with contractility, and downstroke rate indicates peripheral vascular resistance.
      ii) As the arterial lumen decreases in size, velocity increases, leading to a peaked but shorter wave.
      iii) The systolic BP and pulse pressures are falsely elevated, while the diastolic pressure and mean pressures are lower when the distal arterial system is cannulated. This is often clinically evident by a lack of congruency between NIBP and the arterial line measurements. A transducer in the aortic root theoretically provides the most accurate measurement.[7]
      iv) Systolic pressure variation during the respiratory cycle >8 mm correlates strongly with hypovolemia. This is a particularly useful tool during mechanical ventilation when variables that affect systolic pressure variation including tidal volume, frequency, and positive end-expiratory pressure (PEEP) can be held constant.[8]
   b) **Advanced waveform analysis**
      i) Pulse pressure variation (PPV) is the ratio of the maximal minus the minimal values to the mean values, averaged over three or more breaths (**Fig. 12-1**).
      ii) PPV is integrated into most modern anesthesia workstation monitors. It can also be roughly estimated by visually analyzing the standard arterial blood pressure tracing during inhalation and exhalation.
      iii) Stroke volume variation (SVV) is determined by commercially available, minimally invasive monitoring devices. These devices are typical used in place in conjunction with standard arterial line transducer.
      iv) Both PPV and SVV can be used as a tool to predict a patient's need for and responsiveness to fluid resuscitation.[9]
      v) In theory, patients with PPV/SVV >10%-15% may be considered hypovolemic and could respond positively to fluid challenge.
      vi) Calculation of an accurate PPV or SVV is dependent on several clinical/physiological criteria (**Table 12-1**).



An individual patient's overall clinical picture should always be considered in conjunction with PPV/SVV when considering a fluid challenge and/or resuscitation. PPV/SVV values >10%-15% are not representative of hypovolemia in all patients. A so-called gray zone has been suggested to exist among these values.[10]

TABLE 12-1 Requirements for Accurate PPV/SVV Calculation

- Mechanical ventilation without spontaneous ventilations
- Normal sinus rhythm
- Normal to high tidal volume (≥8 mL/kg)

Adapted by permission from Springer: Yang X, Du B. Does pulse pressure variation predict fluid responsiveness in critically ill patients? A systematic review and meta-analysis. *Crit Care*. 2014;18(6):650.

c) **Dampening of the arterial tracing**

i) Underdampening

(1) Refers to a system that resonates when the frequency of the pressure waveform approaches the frequency of the system

(2) This can be thought of as a diaphragm within the transducer that vibrates for a prolonged period of time in relation to a single pressure wave.

(3) Underdampened systems overestimate systolic pressure by 15-30 mm Hg and amplify catheter artifact or catheter whip. This occurs with very small tubing (<1.5 mm diameter) or long connection lines (>1.5 m), stiff tubing, or large catheters (18 G).[7]

ii) Overdampening

(1) Refers to a transducer diaphragm or measuring system that is too rigid

(2) Overdampened systems underestimate systolic pressure and occur with high viscosity, soft tubing, air bubbles, blood clots, and kinked catheters.[7] Mean pressures are typically less affected.

iii) Optimization

(1) The system is optimized by minimizing tubing length (<120 cm), eliminating stopcocks and air bubbles, using low compliance tubing with an internal diameter of 1.5-3.0 mm, and using continuous flush with heparinized saline of 1-3 mL/h (1 unit of heparin per mL saline).

iv) Assessment of dampening

(1) The engineering and *in vitro* laboratory gold standard for assessing the accuracy of the damping coefficient is the square wave test.

(2) The fast flush test is a clinical equivalent of this method, where the system is flushed and the subsequent oscillations from the system are observed.[11]

(3) Several oscillations following a flush occur in underdampened systems, whereas fewer than 1.5 oscillations following a flush will occur in overdampened systems.

(4) The amplitude ratio between any two oscillating peaks can also be measured, which can be extrapolated graphically to determine dampening coefficient.[1,7] As natural frequency increases, broader ranges of dampening coefficients become allowable (**Fig. 12-2**).

5) **Complications of arterial cannulation (Table 12-2)**

a) Risks are minimized with small catheters, heparinized saline is continuously infused slowly (2-3 mL/h), flushing is minimized, aseptic technique is utilized, and pulse oximetry is utilized to continuously assess perfusion.



A common cause of BP exaggeration is when extra tubing is in use, such as for a dorsalis pedis arterial line. A common cause of BP underestimation is when an arm is tucked and the catheter is kinked.



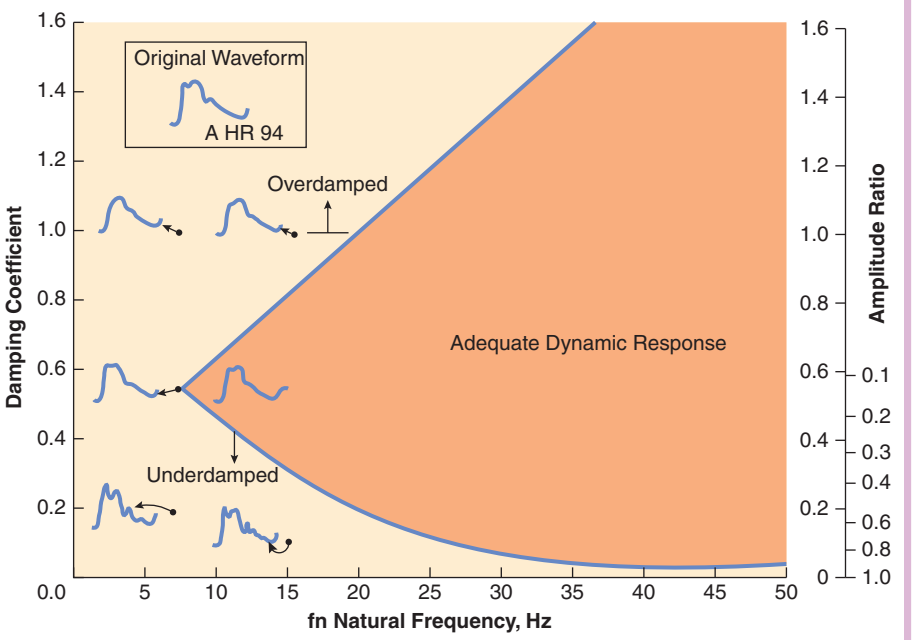
Place padding between the stopcock and the patient's arm when tucking to avoid bruising or skin excoriation.



Unintentional intra-arterial injection of medication can have devastating and permanent consequences. Medications via arterial access has led to clinically important sequelae, including paresthesias, severe pain, motor dysfunction, compartment syndrome, gangrene, and limb loss.[12]

ALL vascular access sites should be labeled appropriately and clearly. A thorough handoff should **ALWAYS** be given to **ALL** medical personnel caring for a patient.

FIGURE 12-2 Parameters That Enable a System to Have an Adequate Dynamic Response



As natural frequency increases, the range of allowable damping coefficients increases. The right-hand side of the figure enables a quick determination of damping coefficient based upon amplitude ratio that is determined from the fast flush technique. Reproduced with permission from Greenberg SB, Murphy GS, Vender JS. Standard monitoring techniques. In: Barash PG, Cullen BF, Stoelting RK, et al., eds. *Clinical Anesthesia*. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009:704.

TABLE 12-2 Complications From Arterial Cannulation

- Vascular insufficiency and vasospasm
- Hematoma
- Blood loss
- Arterial thrombosis
- Air or thrombotic embolic
- Nerve damage
- Infection
- Intra-arterial drug injection

From Spiess BD, Gomez MN. Hemodynamic monitoring. In: Longnecker DE, Tinker JH, Morgan GE, eds. *Principles and Practice of Anesthesiology*. 2nd ed. St. Louis, MO: Mosby-Year Book Inc.; 1998:802-825; Greenberg SB, Murphy GS, Vender JS. Standard monitoring techniques. In: Barash PG, Cullen BF, Stoelting RK, et al., eds. *Clinical Anesthesia*. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009:704.

CHAPTER SUMMARY FOR ARTERIAL BP MONITORING	
Indications	Need for continuous BP reading, patients at high risk for adverse outcomes with BP changes, surgical procedure with anticipated large fluid shifts, and need for frequent blood gas analyses
Mechanics	Based on pressure in fluid column; therefore the transducer must be zeroed after placed at midaxillary line or at level of the external auditory meatus in neurologic procedures to monitor cerebral perfusion pressure
Waveform Interpretation and Analysis	Overdampened system underestimates systolic pressure and vice versa for underdampened system(see square wave testing 4b). PPV and SVV are values derived from the arterial line waveform that can be used to predict a patient's fluid status and guide treatment.
Access Site	Radial artery is the most common, but many alternatives including brachial are possible. Consider use of ultrasound for difficult placements.
Complications	See Table 12-2

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13 Central Venous Monitoring/ Pulmonary Artery Catheter

Cassandra L. Bailey • Maggie W. Mechlin

Central venous catheterization involves placement of a catheter into a large central vein allowing for intravenous access as well as hemodynamic monitoring. The central venous pressure (CVP) is the pressure in the superior vena cava. Due to its limitations, the CVP should be used in combination with other hemodynamic parameters when evaluating intravascular volume. A larger bore catheter (an "introducer") can be placed in a central vein for use as an access point for a pulmonary artery catheter (PAC). The PAC will travel through the right heart into the pulmonary artery. The PAC can measure CVP, pulmonary artery pressures, and pulmonary occlusion pressure (PAOP, or "wedge") as well as provide additional hemodynamic parameters such as cardiac output and mixed venous oxygen saturation. The PAC should only be used by those skilled in placement and interpretation.

Central Venous Catheters

1) **Indications**
   a) Intravenous access
   b) Delivery of medications
      i) Some inotropes and vasopressors, parenteral nutrition, or other potentially caustic medications should be delivered to a central vein.
      ii) Large bore catheters can be used for rapid fluid or blood administration.
   c) CVP monitoring
   d) Access for:
      i) PAC
      ii) Transvenous pacer
      iii) Hemodialysis
   e) Aspiration for air embolism
2) **Technique**
   a) Patient position
      i) Trendelenburg position increases the diameter of both the internal jugular (IJ) and subclavian (SC) veins.
      ii) The head should be turned away from the cannulation site for an upper body central line.
   b) Sterile technique
      i) Hand washing; caps, masks, sterile gown, gloves and drape should be used.
      ii) Chlorhexidine should be used for skin preparation.
   c) Sites
      i) IJ vein
         (1) The right IJ has a straighter course to the SVC.
         (2) The left IJ lies next to the thoracic duct, which can be injured during cannulation.
      ii) SC vein
         (1) The SC catheter may be more comfortable for the patient.
      iii) Femoral vein

(1) Femoral catheters have more infectious and thrombotic complications, and upper body sites are preferred.

iv) Antecubital veins

(1) Peripherally inserted central catheter (PICC)

(2) Uses

(a) Long-term IV access such as a several week course of antibiotics, parenteral nutrition, or continuously infused medications (such as a pulmonary vasodilator in patients with pulmonary hypertension)

(b) For IV access when peripheral access is difficult

(c) Not suitable for rapid fluid administration due to high resistance

d) Ultrasound guidance for placement

i) Allows for direct visualization during needle placement

ii) High-frequency, linear probe is used for high resolution but short depth of image.

iii) Reduces the risk of complications and increases the chances of successful cannulation.[1]

iv) Allows for visualization of the wire in the vessel (**Fig. 13-1**).

v) Due to widespread availability of USG, this is now a standard of care

e) Method

i) Seldinger technique

(1) Catheter over wire method.

(2) In awake patients, local anesthetic is used to infiltrate the site.

(3) The vein is punctured with the needle, and a guidewire is advanced through the needle. The needle is then removed.

(4) A small incision is made with a scalpel, and a dilator is advanced. The catheter is then advanced over the guidewire, and the guidewire is removed.

(5) The ports are checked by first aspirating blood back and then flushing with saline.

ii) Modified Seldinger technique—catheter over needle—a wire is then placed through the catheter and procedure continues as above.



The wire should be visualized with ultrasound in the vein after the needle is removed and prior to dilation of the vessel.



The scalpel only needs to be advanced 1/3 or ½ of its depth into the skin. By keeping the incision small, the risk of bleeding around the line insertion site is less.



The dilator should only be inserted far enough to dilate the wall the vessel (not the full length of the dilator). Inserting the dilator too deep can lead to vascular injury and even cardiac perforation.

FIGURE 13-1 Visualization of Cannulation Wire in the Internal Jugular Vein

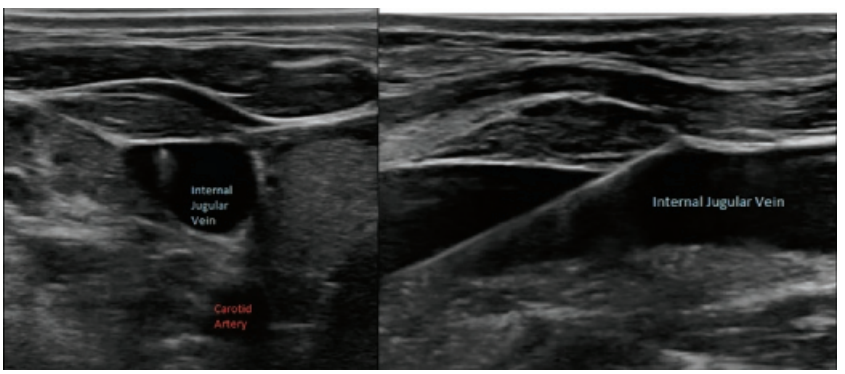
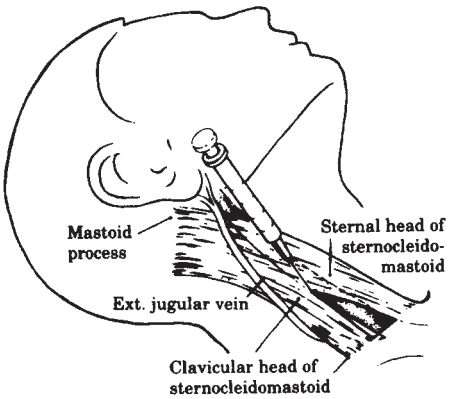


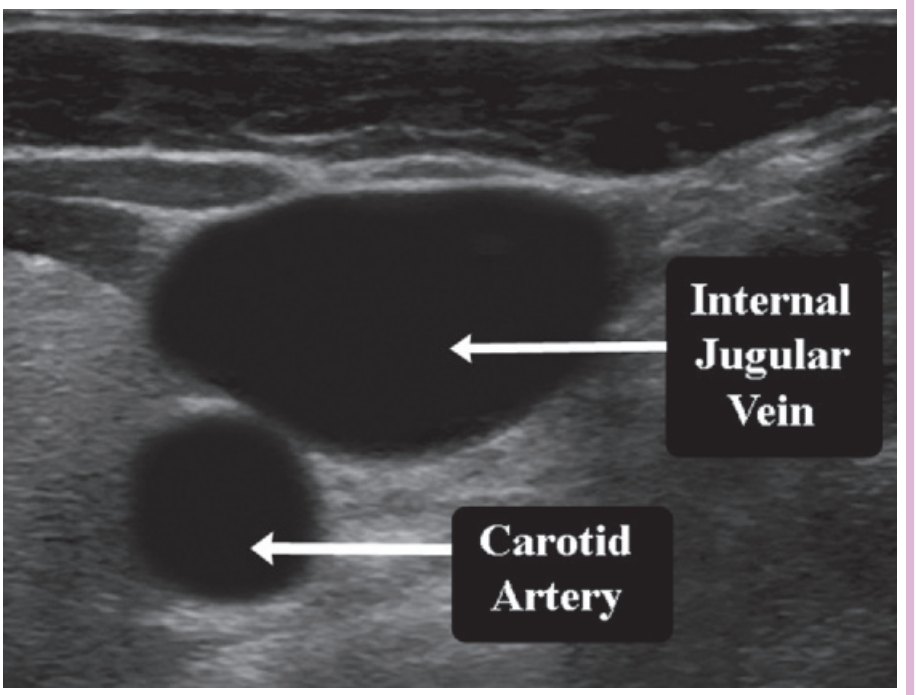
FIGURE 13-2 Anatomy for a Right-Sided Internal Jugular Vein Puncture



Note the triangle formed by the two heads of the sternocleidomastoid muscle indicating the typical site of access. Reproduced with permission from Chatburn J, Sandberg WS. Monitoring. In: Levine WC, ed. *Handbook of Clinical Anesthesia Procedures of the Massachusetts General Hospital.* 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010:136.

f) Landmarks
   i) IJ approach
      (1) The needle insertion should be between the medial and lateral heads of the sternocleidomastoid. The vein is lateral to the carotid artery. Ultrasound should be used when available (**Figs. 13-2** and **13-3**).
   ii) SC approach
      (1) The needle insertion should be 1 cm lateral to the middle third of the clavicle. The needle should be advanced underneath the clavicle at as flat of an angle as possible and aimed horizontally towards the sternal notch.
      (2) There are now well-described techniques for utilizing ultrasound for the SC approach.
   iii) Femoral approach
      (1) The vein is located medial to the artery. Insertion of the needle should be below the level of the inguinal ligament. Can be placed using ultrasound or palpating the femoral artery (then puncturing medial to the pulse).
g) Verification of correct site prior to dilation
   i) Correct guidewire placement should be confirmed prior to dilation of the vessel in order to avoid inadvertent arterial dilation.
   ii) Ultrasound is useful to visualize the wire in the correct vessel prior to dilation (**Fig. 13-1**).
      (1) Cross-sectional
      (2) Long axis
   iii) Fabian test.
      (1) Connect IV tubing to a small (peripheral IV sized) catheter placed in vessel. Allow blood to fill the tubing, and hold tubing upright to see the column of blood fall. Rising column of blood indicates arterial placement. Stagnant column of blood could indicate arterial placement or venous placement in the setting of high CVP.

FIGURE 13-3 Ultrasound Image Demonstrating Typical Relationship of Right Internal Jugular Vein and Carotid Artery



iv) Transduce the system during cannulation.
(1) Place a small IV catheter over the guidewire, remove the guidewire, and connect the small IV catheter to a pressure transducer and monitor.
(2) Inspect the waveform and pressure to determine if arterial or venous.
v) Blood gas sampling.
vi) Transesophegeal ECHO can visualize the wire in the SVC.

h) Checklist (**Fig. 13-4**): the use of a checklist is associated with reduced infection rates.[2,3]
i) Chest X-ray (CXR) should be obtained for upper body central lines to confirm location of the catheter tip and evaluate for pneumothorax.
(1) For upper body cannulation, the catheter tip should lie at the junction of the superior vena cava and right atrium (**Fig. 13-4**).

General rules of thumb for depth of insertion (varies with patient body habitus): RIJ or SC should be roughly 15-16 cm at the skin and LIJ or LSC 18-20 cm; femoral lines should always be "hubbed" at the skin; recommend use of at least a 24-cm catheter.

3) **Complications**
a) Infection
i) Prevention
(1) Sterile technique including a full body drape.
(2) Antibiotic or silver impregnated catheters are available.

FIGURE 13-4 Central Venous Line Insertion Checklist

Attach Patient Label Here

CENTRAL LINE INSERTION CHECKLIST

Date/Time: _____

Access Device :

- ☐ PICC Line
- ☐ Triple Lumen
- ☐ Swan Ganz
- ☐ Dialysis
- ☐ Tunneled
- ☐ Other_____

Vessel Location:

- ☐ Basilic
- ☐ Cephalic
- ☐ Brachial
- ☐ Jugular
- ☐ Subclavian
- ☐ Fomoral-Not recommonded (Reason) _____

Status of Line Placement:

- ☐ Elective
- ☐ Emergent
- ☐ Re-positioned
- ☐ Other_____

1. Perform a time out using the informed consent		
2. Perform hand washing		
3. Proceduralist- Place on gown/ gloves,cap,mask and eye protection a. Others entering the room during this procedure must wear cap and mask		
4. Assistant-if enters sterile field, must use sterile gown/gloves, cap, mask, eye protection until sterile dressing in place		
5. Prep site with chlorhexidine 30 sec (fem site 120 sec) a. Allow site to dry		
6. Drape patient from head to toe using sterile technique		
7. Prepare catheter by pre-flushing and clamping all lumens not in use a. Strongly consider the use of ultrasound		
8. Place patient in Trendelenburg position unless contraindicated (eg, increase ICP) or if femoral/PICC (Place supine and flat)		
9. Maintain sterile field		
10. Ensure grasp on guide wie is maintained throughout procedure and remove post procedure.		
11. Aspirate blood from all tumens, flush and apply sterile caps		
12. Confirm tip placement with appropriate verification (e.g. xray)		
13. Clean site with chlorhexidine and apply sterile dressing (CHG or Biopatch)		

Proceduralist: _____ (Name)

Assistant: _____ (Name)

(3) Avoid femoral placement if upper body site is available.
(4) Place catheter away from and skin infection or burn.
(5) Bio-occlusive dressings.

b) Pneumothorax
   i) More common with SC approach.[4]
   ii) Evaluate for lung slide on pleural ultrasound following placement. Absence of lung slide is consistent with pneumothorax.[5]
   iii) CXR should be obtained after all IJ and SC line placements and is current standard of care.

c) Malposition
   i) IJ malposition is typically inappropriate depth.
   ii) SC malposition can include inappropriate depth, as well as course.
      (1) Catheter may go into the SCV and up the ipsilateral or contralateral IJ.
      (2) Catheter may go "straight across" from one SCV into the contralateral SCV.



Confirmation of the guidewire into the vein should be done prior to dilating the vessel in order to avoid inadvertent arterial dilation.

d) Arterial puncture
   i) Confirmation of IJ wire placement should be done prior to dilation in order to avoid arterial dilation.
   ii) Arterial placement of dilator or catheter may require surgical repair. Vascular surgery should be consulted prior to removal of the catheter.
   iii) Arterial puncture can lead to further complications (**Table 13-1**).
e) Bleeding and hematoma formation
f) Retained wire
g) Air embolism
h) Thoracic duct injury
   i) Most common with left-sided IJ placement.
   ii) Leads to chylothorax
i) Arrhythmia
   i) Can occur during wire placement when it reaches the right ventricle, wire should be promptly withdrawn a few centimeters
j) Thrombosis
   i) Most common with femoral catheters.[2]
   ii) For patients with DVTs related to a central venous catheter, the catheter does not need to be removed if it is functioning and there is an ongoing need for the catheter.[6]



For more information, consult the Practice Guidelines for Central Venous Access. *Anesthesiology* 2012 March; 116:539.

4) **Transducing CVP**
   a) Transducer setup
      i) Saline-filled pressure tubing connected to a transducer
      ii) Transducer zeroed at the level of the right atrium
5) **CVP interpretation (Fig. 13-5)**
   a) Waveform
      i) "a" wave—atrial contraction, end of ventricular diastole
         (1) Cannon A wave—occurs when the right atrium contracts against a closed tricuspid valve during complete heart block or AV dissociation
         (2) Large A wave—occurs when the right atrial pressure is increased as in pulmonary hypertension and tricuspid stenosis
      ii) "c" wave—tricuspid valve bulging into the RA, early ventricular systole
      iii) "x" descent—atrial relaxation, midventricular systole

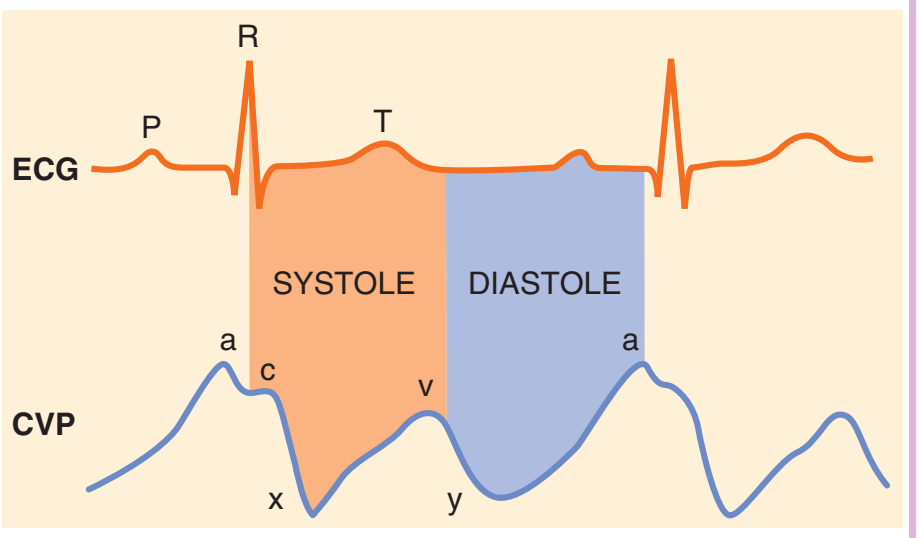


There will be an absence of an "a" wave in atrial fibrillation due to absence of atrial contraction.

TABLE 13-1 Complications of Arterial Dilation or Catheter Placement

- Hemorrhage
- Hematoma—potential airway compromise
- Pseudo aneurysm
- Arterial venous malformation
- Stroke—with carotid artery cannulation

FIGURE 13-5 Diagram of Normal CVP Waveforms Along with Corresponding ECG Tracing



Reproduced with permission from Greenberg SB, Murphy GS, Vender JS. Standard monitoring techniques. In: Barash PG, Cullen BF, Stoelting RK, et al., eds. *Clinical Anesthesia*. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009:705.

iv) "v" wave—atrial filling, late ventricular systole
  (1) Large "v" wave seen in tricuspid regurgitation
v) "y" descent—blood leaving the atrium to the ventricle, tricuspid valve opening and early ventricular systole

b) Volume status
  i) Theory behind CVP monitoring is that CVP correlates with, and predicts, central venous volume.
  ii) Multiple studies have demonstrated that this is not necessarily true.[7]
    (1) There may be benefit to trending a CVP over time in a specific patient
      (a) For example, it is more meaningful to know that the CVP is now 12, but 2 hours ago it was 4, rather than just knowing that the CVP is 12.
    (2) CVP (and trends) are valuable with considerations for right heart failure (new LVAD, heart transplant, patient with pulmonary HTN, or RHF).
      (a) If CVP increases in these clinical settings, it can be a sign that the RV can no longer pump blood forward effectively. Particularly in the instance of having simultaneous PA numbers, CVP > mPAP is a very worrisome finding.
    (3) Mechanical ventilation and changes in intrathoracic pressure may alter CVP.



CVP is an unreliable marker for volume status.

c) Central venous saturation ($ScvO_2$)
  i) Expected to be about 5% higher than a true MV O_2 (or SvO_2, these terms are used interchangeably) since MV O_2 includes blood drained from the coronary sinus and the heart has a high O_2 extraction ratio



Sternal retraction during cardiac surgery can cause advancement of the PAC and PA rupture. For this reason, the PAC should be pulled proximal to the wedged position prior to sternal retraction.

TABLE 13-2 Indications for Pulmonary Artery Catheter

Indications for PAC
• Assessment of volume status and complex fluid management • Assessment of pulmonary arterial pressure and management of pulmonary hypertension • Assessment of cardiac index and management of cardiac failure and shock • Use during cardiac surgery for patients with reduced ejection fraction or severe valve disease

Pulmonary Artery Catheters

1) The use of the PAC is controversial and should be only used by those skilled in placing the catheter as well as interpreting the data it provides. For indications to PAC, see **Table 13-2**.
   a) Measured values
      i) Pulmonary artery pressures (systolic, diastolic, and mean)
      ii) PA occlusion pressure ("wedge" pressure)
         (1) The physiologic principle behind measuring PA occlusion pressure is that in the absence of anatomic pathology, the pressure in an occluded pulmonary artery is equal to the pressure in the left atrium and that at the end of ventricular diastole, while the mitral valve is open, the left atrial pressure is equal to the left ventricular end-diastolic pressure and that these pressures are reflective of the left ventricular end-diastolic pressure (LVEDP).
            (a) This will not hold true with mitral valvulopathy, irregular arrhythmias such as atrial fibrillation, or pulmonary vein stenosis.
            (b) LVEDP may be overestimated in aortic insufficiency and pulmonic insufficiency.
      iii) Cardiac output and cardiac index
         (1) Cardiac index = cardiac output/body surface area.
            (a) Allows for interpretation of cardiac output relative to what a patient is expected to need based on body habitus
         (2) CO can be calculated using the Fick principle or by using thermodilution technique.
            (a) Thermodilution involves injecting a known volume of room temperature fluid into the RA and the change in temperature is measured in the pulmonary artery.
               (i) Usually the average of three measurements is used to determine the cardiac output.
               (ii) Thermodilution will not be accurate in the presence of tricuspid regurgitation, pulmonic regurgitation, or intracardiac shunts.
      iv) Mixed venous oxygen saturation (SvO_2 or MV O_2)
         (1) SvO_2 measures oxygen saturation in blood leaving the right ventricle. Clinically, SvO_2 is a reflection of total body tissue oxygen utilization.
         (2) Some PACs have a fiber optic tip that can measure SvO_2 (without having to draw a blood gas off of the PA port).
         (3) Interpretation and utilization are based on the Fick principle which is represented as $SvO_2 = SaO_2 - VO_2/1.34(Hb)(CO)$ (**Table 13-3**).
      v) Temperature
   b) Calculation of hemodynamic values (**Table 13-4**)



In normal healthy patients, PCWP = Left atrial pressure = LVEDP = LVEDV.



Hypoxia, hypercarbia, and acidosis will cause an increase in PVR.



Alternative monitoring should be considered including CVC, TEE, arterial line with pulse wave analysis based on indication for monitoring, and user experience with each of these methods.



PAC use contraindicated in LBBB, mechanical valves, bacteremia, and hypercoagulable state



Use closed-loop communication when floating a PAC to decrease the risk of complication.

TABLE 13-3 Treatment of Decreased Mixed Venous Oxygen Saturation[8]

Causes of Low Mixed Venous Oxygen Saturation	Management
Low cardiac output	Inotropes
Anemia	Transfusion
Hypoxia	Increase FiO_2, PEEP
High oxygen utilization/extraction	Treat/correct the underlying cause (including conditions such as shivering, seizures, and agitation, among others)

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2) Technique—The PAC is a long multiport catheter that is inserted in the pulmonary artery via a central vein introducer following a path through the right atrium and right ventricle (**Fig. 13-6**).
   a) Sterile technique should be used to attach the PAC to pressure transducers.
   b) Flush the line and check balloon inflation prior to insertion.
   c) Monitor BP, ECG, and pulse oximetry during placement.
   d) Insert catheter with balloon deflated into the introducer.
   e) Advance PAC until past the end of the introducer.
   f) Assistant inflates the balloon, and the catheter is advanced slowly watching hemodynamic waveforms (**Fig. 13-7**).
      i) Only advance the catheter with the balloon up, and only retract with the balloon down. Advancing with the balloon down can lead to the catheter being too deep and increase the risk of PA rupture when the balloon is inflated. Retracting with the balloon up could lead to the balloon and catheter tip getting caught in subvalvular apparatus for the PV or TV and injury.
   g) The PA waveform shows a diastolic rise or "step-up".
   h) Note the depth of where the catheter leaves the hub (**Table 13-5**).
   i) Obtain a CXR to evaluate the location of tip of the catheter (regardless of whether it is floated through the IJ, SC, or femoral vein).
3) **Complications**
   a) Complications associated with central line placement
   b) PA rupture and hemorrhage
      i) Increased risk in patients with pulmonary hypertension and coagulopathy.



Arrhythmias normally occur due to the catheter irritating the RV. Prior to beginning to float, establish with the person assisting you if you will continue to advance through the PVCs (generally going another few cm gets the catheter tip past the irritable focus and the PVCs stop) or if you will ask them to deflate the balloon and withdraw the catheter until the PVCs stop.



Equalization of PA pressures, Wedge, and CVP means cardiac tamponade until proven otherwise.

TABLE 13-4 Important Equations for Cardiac Monitoring

Important Equations	
Fick equation for cardiac output	$VO_2/(CaO_2 - CvO_2)$
SVR	$MAP - CVP/CO \times 80$
PVR	$Mean\ PAP - PAOP/CO \times 80$
Stroke volume	LVEDV – LVESV
Oxygen content of blood	$Hb(g/dL) \times 1.34 \times O_2\ saturation + 0.0032 \times PO_2$
Mixed venous Hb saturation	$SaO_2 - (VO_2/CO \times 1.36 \times Hb)$

CaO_2, oxygen content of arterial blood; CvO_2, oxygen content of venous blood; SVR, systemic vascular resistance; PVR, pulmonary vascular resistance; MAP, mean arterial pressure; CVP, central venous pressure; CO, cardiac output; PAP, pulmonary artery pressure; PAOP, pulmonary artery occlusion pressure; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; Hb, hemoglobin; SaO_2, arterial oxygen saturation; VO_2, oxygen consumption.

FIGURE 13-6 Typical Oximetric PAC

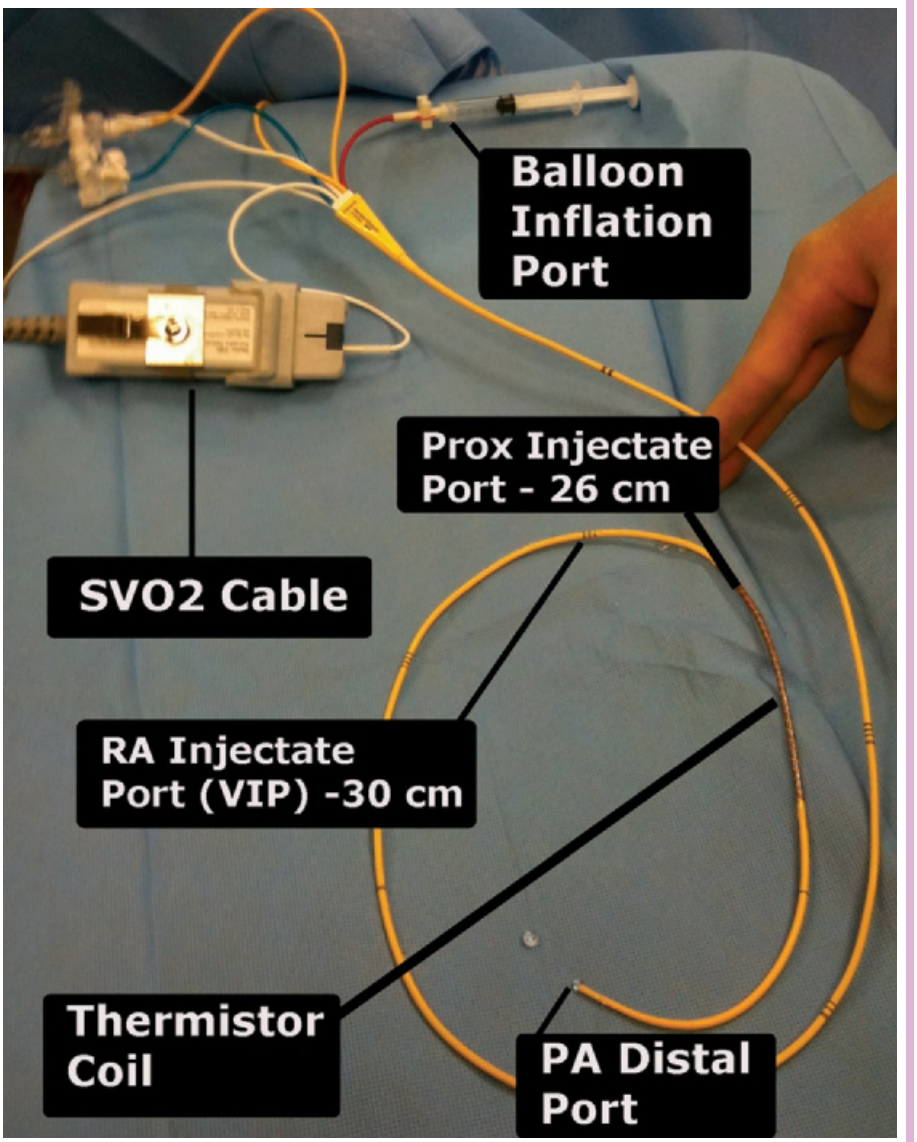
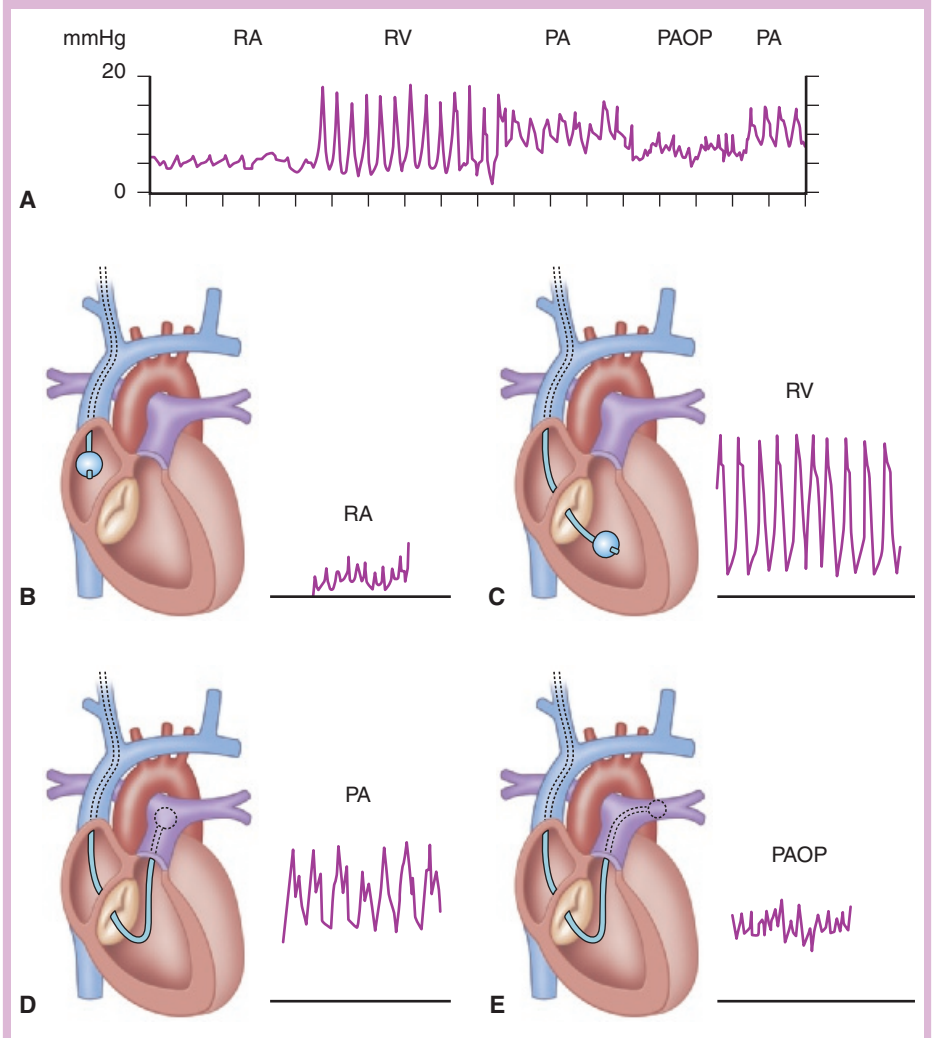


FIGURE 13-7 Typical PAC Waveform Pressure Tracing



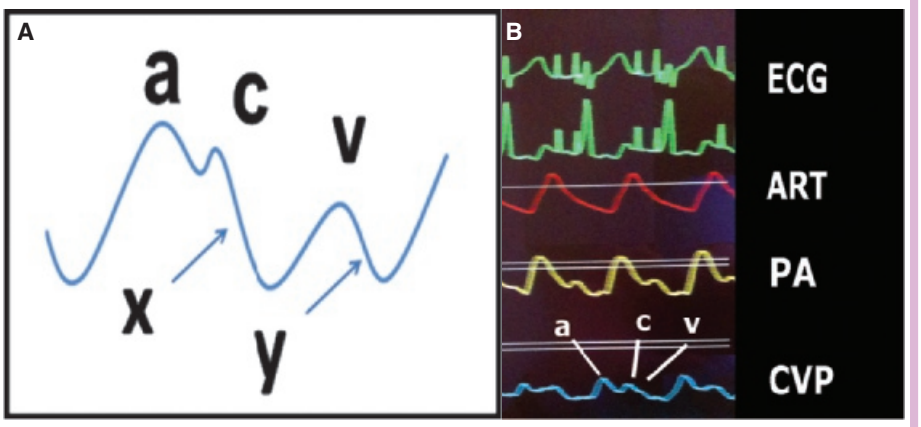
As the PA catheter is advanced from the right atrium to the pulmonary capillary wedge. **A.** Entire PAC Series. **B–E.** Sequential tracings expected as the catheter is advanced from the right atrium through to the PA wedge. Reproduced with permission from Mihm FG, Rosenthal MH. Pulmonary artery catheterization. In: Benumof JL, ed. *Clinical Procedures in Anesthesia and Intensive Care*. Philadelphia, PA: Lippincott Williams & Wilkins, 1992:416.

TABLE 13-5 Depth of PA Catheters in Different Anatomic Locations

Approximate Depth of PAC When Reaching Location (From RIJ Position)[a]	
Right ventricle	24.6 cm
Pulmonary artery	36 cm
Wedge position	42.8 cm

[a]Reprinted from Tempe DK, Ghandi A, Datt V, et al. Length of insertion for pulmonary artery catheters to locate different cardiac chambers in patients undergoing cardiac surgery. *Br J Anaesth.* 2006;97(2):147-149, with permission from Elsevier.

FIGURE 13-8 PA Waveform Diagram



A. Depicts the Waveform Expected when the PAC is in the Wedge Position. **B** is a screenshot of a typical hemodynamic series. Note the CVP tracing has similar waveform characteristics of the PCWP in **(A)**. The PA tracing below is a PA tracing, not the PCWP tracing. Also note that the waveform timing is slightly delayed from the CVP compared to the PA due to timing of flow from the right and left heart.

ii) Balloon should only be inflated with the 1.5 cc syringe that comes in the PAC kit to prevent over inflation and PA rupture.
iii) Advancing with the balloon down can lead to the catheter being too deep and increase the risk of PA rupture when the balloon is inflated.

c) Arrhythmias
i) PVCs during placement
ii) Ventricular tachycardia
iii) Ventricular fibrillation
iv) Right bundle branch block—can lead to complete heart block in patients with preexisting left bundle branch block

d) Thromboembolism
e) Infection and endocarditis
f) Endocardial damage

4) **PAC Interpretation (Fig. 13-8)**
a) Normal values: see **Table 13-6**.[9]
b) CVP is preload for the RV, and PA is afterload for the RV
c) PAOP is preload for the LV, and systemic BP is afterload for the LV
d) The PAC waveform is similar to CVP waveform with "a" wave representing atrial contraction, "c" wave representing mitral valve closure (often not visualized on PAC tracings), x descent representing atrial relaxation, v wave representing atrial filling, and y descent representing mitral valve opening (**Table 13-7**).



Based on the Fick Principle, low SvO_2 can be increased with transfusion of red blood cells, increasing cardiac output, or increasing a low arterial oxygen saturation.



A high SvO_2 can be seen in settings with end-organ inability to extract oxygen, such as the early phase of sepsis, or cyanide toxicity.



The wedge pressure should be obtained at end expiration.



Several studies have shown that many providers do not know how to correctly identify the PAOP on a PA waveform.

TABLE 13-6 Normal Values From Pulmonary Artery Catheter

CVP	2-6 mm Hg
PAP systolic/diastolic	15-30/8-15 mm Hg
Mean PAP	9-18 mm Hg
PAOP (wedge pressure)	8-12 mm Hg
Cardiac output (CO)	4-8 L/min
Cardiac index (CI)	2.5-4 L/min/m^2
SvO_2	65-70%
SVR	900-1300 (dynes sec)/cm^5
PVR	40-150 (dynes sec)/cm^5
SV	50-100 mL s

TABLE 13-7 Interpretations of Abnormalities in PA Waveform

Abnormality	Potential diagnosis
Large A wave	Mitral/tricuspid stenosis, heart failure, heart block
Large V wave	Mitral/tricuspid regurgitation, heart failure, VSD, increased PVR

CHAPTER SUMMARY CENTRAL VENOUS CATHETERIZATION AND PRESSURE WAVE INTERPRETATION

	Indications	Contraindications

Indications and Contraindications	• Intravenous access
	• Delivery of medications
	• CVP monitoring
	• Access for:
	  • PAC
	  • Transvenous pacer
	  • Hemodialysis
	• Aspiration for air embolism
		• Inexperience, unsupervised operator
		• Local infection
		• Distorted local anatomy
		• Coagulopathy
		• Previous radiation therapy
		• Suspected proximal vascular injury

Technique	• Right internal jugular vein is typically approached using the Seldinger technique with ultrasound guidance.	
Complications	• Complications such as infections, pneumothorax, malposition of the catheter, and arterial puncture can occur. Other rare complications exist.	
Pressure Wave Interpretation	• "a" wave—atrial contraction, end of ventricular diastole • "c" wave—tricuspid valve bulging into the RA, early ventricular systole • "x" descent—atrial relaxation, midventricular systole • "v" wave—atrial filling, late ventricular systole • "y" descent—blood leaving the atrium to the ventricle, tricuspid valve opening and early ventricular systole	

CHAPTER SUMMARY PULMONARY ARTERY CATHETERIZATION AND PRESSURE WAVE INTERPRETATION

	Indications	Contraindications
Indications and contraindications	• To assess volume status and complex fluid management • To measure pulmonary artery pressure and manage pulmonary hypertension • Assess cardiac index and manage cardiac shock • Guide intraoperative management of patients with severe reduced ejection fraction or value disease	• Inexperience, unsupervised operator • Local infection • Distorted local anatomy • Coagulopathy • Previous radiation therapy • Suspected proximal vascular injury
Technique	• The PAC is a long multiport catheter that is inserted in the pulmonary artery via a central vein introducer following a path through the right atrium and right ventricle.	
Complications	• Complications associated with pulmonary artery catheterization include rupture of the artery, arrhythmias, thromboembolism, infection or endocarditis, and endocardial damage.	
Pressure Wave Interpretation	• The PAC waveform is similar to CVP waveform with "a" wave representing atrial contraction, "c" wave representing mitral valve closure (often not visualized on PAC tracings), x descent representing atrial relaxation, v wave representing atrial filling, and y descent representing mitral valve opening.	

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14 Focused Transthoracic Echocardiography

Clemens M. Ortner

With the availability of mobile, portable, and pocket-sized ultrasound machines, transthoracic echocardiography is available as a bedside tool to serve in medical emergencies for timely assessment of the hemodynamically unstable patient. While many acronyms have been used to describe techniques such as bedside echocardiography, point-of-care echocardiography, handheld echocardiography, or goal-directed echocardiography, national and international scientific bodies agreed on the term of focused cardiac ultrasound (FoCUS).[1,2]

This chapter provides an overview on the definition, techniques, diagnostic aims, clinical applications, and training requirement of a FoCUS examination.

1) **FoCUS Definition**
   a) **An ultrasound examination that has the following characteristics:**
      i) Goal directed and problem oriented
      ii) Simplified and limited
      iii) Usually performed by a physician at the point of care
      iv) Qualitative or semiquantitative
      v) Time sensitive and repeatable
   b) **FoCUS is potentially suitable for all clinical settings as a targeted diagnostic test performed by the appropriately trained clinician treating the patient.**
   c) A FoCUS examination answers questions in a binary fashion (*yes or no*?) either qualitatively (eg, is the right ventricle dilated or nondilated?) or semiquantitatively (eg, left ventricle is hyperkinetic, normokinetic, hypokinetic, or severely hypokinetic).
   d) Providing a detailed grading of cardiac size, function, or valve pathology is NOT goal of a Focused TTE examination (**Table 14.1**).



It is important to acknowledge the limitations of the FoCUS and refer patients for comprehensive echocardiographic examination when appropriate.

2) **Limitations: Patients in whom there is suspicion of undetected cardiac pathology or with abnormalities detected beyond the scope of the examination should be referred for comprehensive echocardiographic evaluation.**
3) **Equipment**
   a) **FoCUS requires two-dimensional (2D) imaging.** Current portable US machines, hand carried and pocket sized, or light cart devices are suitable equipment.

TABLE 14.1 Targets of the Focused TTE Examination

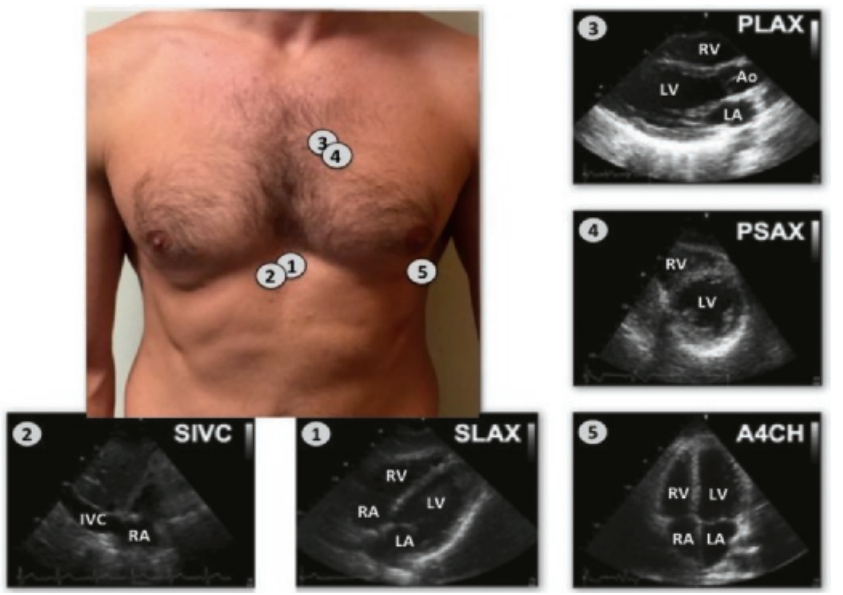
Defined diagnostic targets of the Focused TTE examination are assessment of the following:

- Left ventricular (LV) dimensions
- LV systolic function
- Right ventricular (RV) systolic function
- Volume status
- Presence pericardial effusion and potential tamponade physiology
- Gross signs of chronic cardiac disease
- Gross valvular abnormalities
- Gross intracardiac masses

i) Machine modalities include:
   (1) 2D-imaging: required
   (2) Image archiving capability: required
   (3) M-mode imaging: not required but may be useful
   (4) Doppler function: not required but may be useful
   (5) ECG synchronization and acquisition: not required
   (6) Phased array transducer: suggested, alternative probes not precluded

4) **Technique**
   a) **Recommended views for FoCUS examination include the following** (**Fig. 14-1**):
      i) Subcostal long axis (SLA) view
      ii) Subcostal inferior vena cava (SIVC) view
      iii) Parasternal long axis (PLA) view
      iv) Parasternal short axis (PSA) view
      v) Apical 4 chamber (A4Ch) view
   b) **Completing all or most views listed in a systematic and standardized way increases a FoCUS screening capability.**
   c) **A limited number of views can suffice to assess diagnostic targets.**
   d) **Ideally, each target structure should be visualized in at least two different views to confirm the findings, provided the patient's condition allows it.**
   e) Whenever FoCUS is performed, the findings of the examination should be appropriately documented. This can be accomplished by the use of simplified standardized report forms, with box checking and minimal free text (**Fig. 14-2**).

FIGURE 14-1 Recommended Views for FoCUS Examination



(*1*) Subcostal long axis view (SLAX); (*2*) subcostal inferior vena cava view (SIVC); (*3*) parasternal long axis view (PLAX); (*4*) parasternal short axis view (PSAX); (*5*) apical 4 chamber view (A4CH). Courtesy of Dr. Clemens Ortner.

FIGURE 14-2 FoCUS Cardiac Echo Report Form

FoCUS CARDIAC ECHO REPORT FORM

Patient Name: Date of Birth:
Operator: Exam Date:
Indication: PEA Shock / Hypotension Resp. failure Oliguria Screening Other
History:
Drugs / Treatment:
Ventilation: Spontaneous MV passive MV active CPAP PEEP / Vt =...../.....
Image Acquisition: Easy Difficult Very Difficult Impossible

CLINICAL QUESTION (check all boxes that apply):

Hemodynamic instability LV Evaluation RV Evaluation
Pericardium Volume Status Valvulopathy
Monitoring Training Cardiac Arrest
Other

LEFT CHAMBERS

LA Size: Non dilated Dilated
LV Size: Small Normal Dilated Markedly Dilated
Systolic Function: Normal Mild Dysfunction Moderate Dysfunction
Severe Dysfunction Asystole Hyperdynamic
Wall Thickness Normal Hypertrophied Markedly hypertrophied
Wall Motion Abnormalities: Yes No
Septal Shift: Yes No

RIGHT CHAMBERS

RV Size: Normal Dilated Markedly Dilated Cannot assess
RV Function: Normal Decreased Cardiac Standstill Cannot assess
TAPSE:_____ **RVSP:**_____ **Septum:** Normal Flattened Paradoxical motion

IVC

Visualized? Yes No
Diameter (cm): ≤ 10 mm 10-15 mm 15-20mm 20 - 25mm > 25mm
Respiratory Variation collapse > 50% reduction < 50% reduction absent

PERICARDIUM ☐ Normal ☐ Mild Effusion ☐ Large circumf Effusion ☐ RA syst collapse ☐ RV diast collapse

VALVES

Tricuspid: ☐ normal ☐ regurgitation ☐ marked regurgitation
☐ stenosis ☐ very stenotic ☐ prosthetic
Mitral: ☐ normal ☐ regurgitation ☐ marked regurgitation
☐ stenosis ☐ very stenotic ☐ prosthetic
Aortic: ☐ normal ☐ regurgitation ☐ marked regurgitation
☐ stenosis ☐ very stenotic ☐ prosthetic

5) **Clinical integration**
   a) FoCUS is complementary to the physical examination for assessing LV systolic function and for detecting valvular disease. In the following clinical conditions, FoCUS has been shown to provide useful information for patient management and is recommended for clinical integration:
      i) Initial assessment of patients with cardiopulmonary instability
      ii) Evaluation of systolic function
      iii) Narrow differential diagnosis in undifferentiated shock state
      iv) Risk stratification in pericardial effusion
      v) Identification of a reversible cause in pulseless electrical activity (PEA)
      vi) Identification of patients with hypovolemia
      vii) Triage in the prehospital setting



FoCUS is NOT recommended for assessment of diastolic function and detection of raised left ventricular end diastolic pressures (LVEDP)!

6) **Clinical outcomes**
   a) **FoCUS changes medical management and improves diagnostic accuracy and outcome in cardiac arrest, assessment of hemodynamic instability, and in patients with penetrating cardiac injury.**
   b) **FoCUS for screening cardiovascular disease**
      i) FoCUS has shown significant promises in detecting unexpected relevant cardiac abnormalities and can therefore be considered as a screening tool during preanesthesia evaluation and assessment.
      ii) FoCUS accurately detects following echocardiographic markers for cardiovascular pathology, including:
         (1) Left atrial enlargement
         (2) Left ventricular (LV) hypertrophy
         (3) LV dilatation
         (4) LV systolic dysfunction



FoCUS has shown significant promises in detecting unexpected relevant cardiac abnormalities and can therefore be considered as a screening tool during preanesthesia evaluation and assessment.

   c) Depending on the clinical scenario, the observed ultrasound abnormalities help the clinician in identifying the underlying pathology (**Fig. 14-3**).[3]
      i) FoCUS is NOT reliable in screening for coronary artery disease or rheumatic valve disease.



FoCUS is NOT reliable in screening for coronary artery disease or rheumatic valve disease.

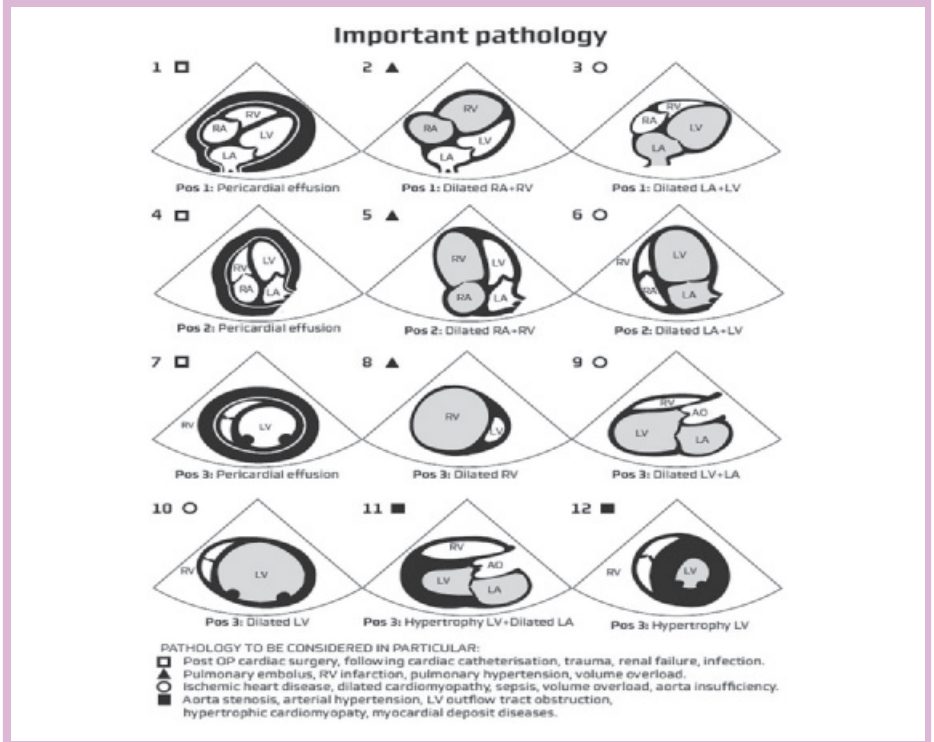
   d) **FoCUS and cardiac arrest**
      i) In the setting of cardiac arrest, FoCUS can differentiate electromechanical dissociation from organized myocardial activity.
      ii) Differentiating between a true PEA and a "Pseudo PEA" has significant impact on guiding clinical management and predicting outcome.
      iii) States of severe hypovolemia, LV dysfunction, or RV decompensation can appear clinically pulseless but are amendable to treatment and potentially reversible.
      iv) True PEA has significantly worse outcomes, and cardiac standstill on FoCUS during cardiopulmonary resuscitation predicts death with 97%-100% probability.[4,5]
      v) In cardiac arrest, FoCUS
         (1) Changes management in 89% of patients[6]
         (2) Is more accurate than ECG for determining mechanical cardiac function
         (3) Is more accurate than physical examination for diagnosing the cause of cardiac arrest and for assessing cardiac function
         (4) Helps clinicians to predict outcome



In cardiac arrest, the differentiation between electromechanical dissociation or organized myocardial function is of greatest clinical relevance and well answered by FoCUS examination.

   e) **Penetrating cardiac injury**
      i) In patients with suspected penetrating cardiac injury, FoCUS has the following characteristics[7]:

FIGURE 14-3 Perioperative Use of FoCUS-Assessed Transthoracic Ultrasound. Important pathologies as seen on TTE, including pericardial effusion, hypertrophy, and/or dilatation of right atrium (RA), right ventricle (RV), left atrium (LA), and left ventricle (LV)



Reproduced with permission from Holm JH, Frederiksen CA, Juhl-Olsen P, Sloth E. Perioperative use of focus assessed transthoracic echocardiography (FATE). *Anesth Analg*. 2012;115:1029-1032.

(1) Sensitivity of 100%, specificity of 97%, and accuracy of 97% for diagnosing cardiac injury and effusion (▶ Video 14-1)
(2) As accurate as comprehensive standard echocardiography for identifying a pericardial collection
(3) Superior in diagnosis rather than relying on clinical signs and symptoms
(4) Associated with decreased mortality

f) **Shock and hemodynamic instability**

i) The FoCUS examination allows clinicians to correctly diagnose the etiology of shock in 80% of patients, as compared to 50% when ultrasound was not used (8), and leads to changes in patient management in ~66% of patients in shock (6).

ii) In the setting of shock and hemodynamic instability, FoCUS
(1) Should be part of initial assessment of hemodynamically unstable patient
(2) Narrows the differential diagnosis
(3) Changes management and improves outcome
(4) Accurately assesses global LV systolic function
(▶ Video 14-2 demonstrating normal vs decreased LV systolic function)



National and international expert panels strongly recommend FoCUS to become standard of care during initial assessment of all hemodynamically unstable patients.

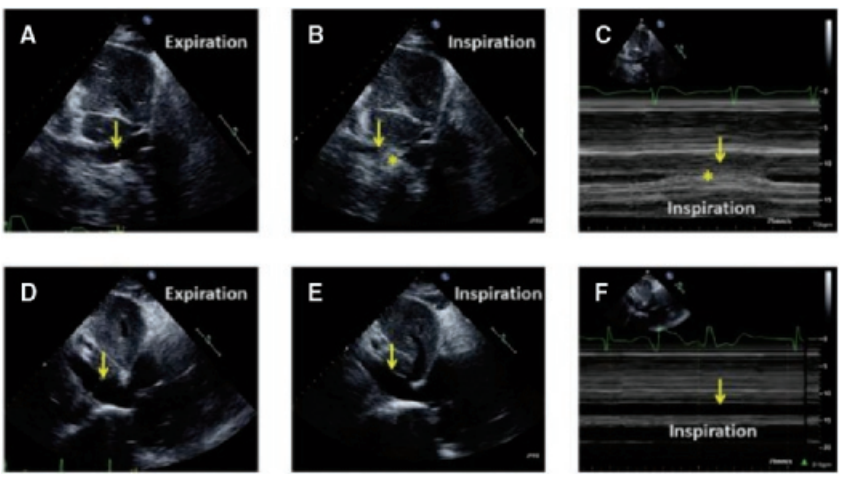
g) **Estimating central venous pressure, diagnosing hypovolemia, and predicting fluid responsiveness**
   i) A hypovolemic FoCUS pattern—the presence of small hyperdynamic ventricles and a small inferior vena cave (IVC) at end expiration—is accurate for the diagnosis of clinically relevant hypovolemia.
   ii) Assessing IVC diameter over the respiratory cycle can estimate central venous pressure (CVP), diagnose hypovolemia, and predict fluid responsiveness.
      (1) Small IVC size with spontaneous ventilation can identify patients with low CVP and rule out high CVP (**Figs. 14-4** and **14-5**, **Table 14-2**).
      (2) Low CVP does NOT correlate with fluid responsiveness.
      (3) In ventilated patients, ultrasound assessment of IVC size CANNOT accurately assess high CVP.



Fluid responsiveness can be accurately predicted by measuring changes in stroke volume with passive leg raising (PLR), but this requires pulsed-wave Doppler flow measurements, which is beyond the scope of FoCUS.

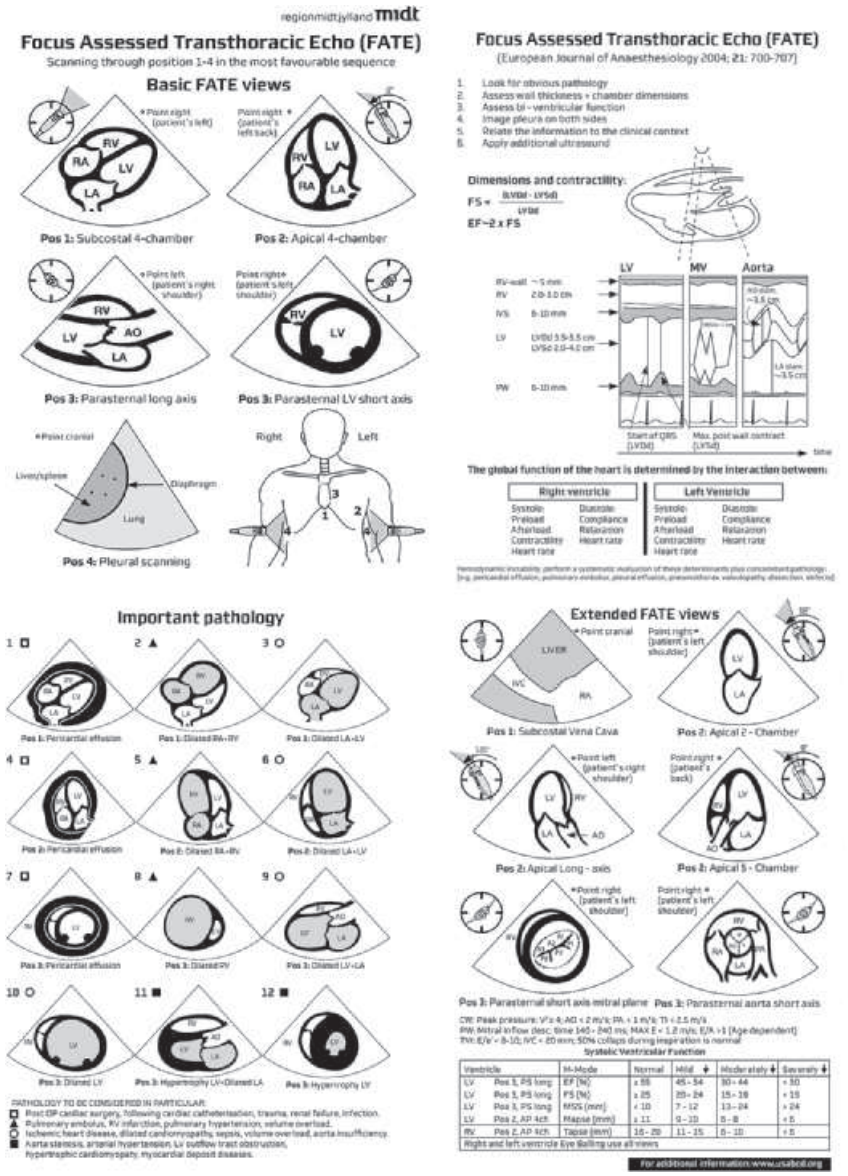
7) **Risks**
   a) **The main risks of FoCUS are inadequate skill and competence in the practitioner and use beyond its intrinsic limitations.**
   b) **Avoid potential risk by:**
      i) Close adherence to image acquisition techniques
      ii) Careful integration of sonographic findings into clinical decision-making
      iii) Awareness of boundaries between a comprehensive standard TTE and FoCUS

FIGURE 14-4 Echocardiographic Evaluation of Right Atrial Pressure (RAP) Using Inferior Vena Cava (IVC) Dimension and Collapsibility. Subcostal view of the IVC during expiration **(A)** and inspiration **(B)**, and M-mode echocardiography **(C)** demonstrating good inspiratory collapse (*asterisk*) of the IVC (*arrow*) in a patient with normal RAP and 2D-echo during expiration **(D)** and inspiration **(E)** and M-mode echocardiography **(F)** demonstrating no inspiratory collapse of the IVC in a patient with elevated RAP



Reproduced with permission from Beigel R, Cercek B, Luo H, Siegel RJ. Noninvasive evaluation of right atrial pressure. *J Am Soc Echocardiogr*. 2013;26:1033-1042.

FIGURE 14-5 Focus Assessed Transthoracic Echo (FATE) Card



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TABLE 14-2 Estimation of Right Atrial Pressure (RAP) Using IVC Parameters

ASE 2010 Recommendations[8]		Brennan et al.[9]	
IVC Diameter (cm) and Collapse (%)	RAP Range in mm Hg (Mean)	IVC Diameter (cm) and Collapse (%)	RAP Range in mm Hg
Normal: ≤2.1 cm and >50%	0-5 (3)	≤2.1 cm and >55%	<5
Indeterminate[a]	5-10 (8)	≤2.1 cm and 35-55% or >2.1 cm and >55%	0-10
High: >2.1 cm and <50%	1-20 (15)	>2.1 cm and 35%-55% >2.1 cm and <35% ≤2.1 cm and <35%	10-15 10-20 Undetermined

[a]In cases in which the IVC diameter and collapse do not fit the normal or high criteria, secondary signs of elevated RAP should be considered. These include a dilated IVC with diminished respiratory collapse, a dilated right atrium, or a bulging interatrial septum to the left atrium.

8) **Training**

Currently there is no defined process to obtain certification in FoCUS. The American Society of Critical Care Medicine (SCCM), European Society of Intensive Care Medicine (ESCIM), and 11 other critical care societies worldwide have outlined following framework for organizing training in basic and advanced critical care echocardiography:

a) **Competence in basic critical care echocardiography:**
   i) Minimum theoretical program: 10 hours of standard lectures or Internet-based learning.
   ii) Required minimum number of examinations is 30 fully supervised TTEs defined as reasonable target to teach image acquisition, but to be defined on an individual basis.
   iii) Logbook:
      (1) Each study performed and/or interpreted by trainee is to be reported and documented by the trainee.
      (2) Each report is to be cosigned by trainee and supervisor.
   iv) Minimal number of cases of each clinical syndrome
      (1) No consensus
      (2) Comprehensive panel of important abnormal images must be part of regular didactical training and image review.

b) **Training in transesophageal echocardiography (TEE) is optional.**

c) **No certification required.**



The biohazard associated with ultrasound exposure during diagnostic ultrasound and FoCUS is minimal and negligible.

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15 Point-of-Care Ultrasound

Sofia Ivana Diaz • Neil S. Kalariya • Ranjit Deshpande

Advances in ultrasound technology have resulted in expanded use of real-time bedside ultrasonography.[1–3] Point-of-care ultrasound (POCUS), or real-time bedside ultrasonography, is performed to answer a specific diagnostic question and monitor response to management. For an Anesthesiologist, POCUS functions as an additional perioperative tool for assessment of hemodynamic and pulmonary changes, aids in vascular access, and can facilitate successful endotracheal intubation.[3,4] The purpose of this chapter is to review the different noncardiac uses of POCUS in the perioperative setting. The use in cardiac assessment, vascular access, and regional and neuraxial anesthesia is outside the scope of this chapter.

1) **Overview (Table 15-1):**
   a) Ultrasound (US) is defined as a frequency above human hearing or 20 000 Hz. The frequency for diagnostic US is in the millions (MHz).[2]
   b) Low-frequency US probes improve penetration (depth) at the expense of resolution; higher-frequency probes provide a higher resolution at the cost of penetration (difficult to visualize deeper structures).[2]
   c) A portable ultrasound device is necessary to perform POCUS. Typically, three types of probes are utilized depending on quality of image and penetration required for examination.[5]
      i) Linear (5-13 MHz): High frequency, better for imaging superficial structures, vessels, and pleural sliding
      ii) Curvilinear (1-8 MHz): Wider footprint, low-frequency probe used for abdominal and lung imaging
      iii) Phased array (1-5 MHz): Routinely used for cardiac imaging due to its small footprint and deeper penetration. This is useful for imaging between ribs and the heart and lung. A phased array probe may be used for lung ultrasound if a curvilinear probe is unavailable.



Low-frequency US probes improve penetration (depth) at the expense of resolution; higher-frequency probes provide a higher resolution at the cost of penetration (difficult to visualize deeper structures)



Lung ultrasound is highly sensitive and specific in differentiating dyspnea of noncardiac vs cardiac origin.[7]

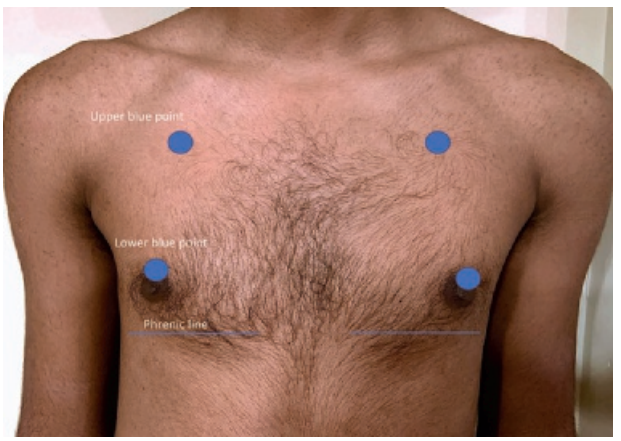
2) **Pulmonary: lung ultrasound (LUS)**
   a) **Background**
      i) Can aid in lung reaeration and recruitment, differentiate cardiac vs noncardiac dyspnea, and may be better than auscultation or chest radiography for the detection of pleural effusion, atelectasis, consolidation, and alveolar interstitial syndrome[1,3,6]
   b) **How to perform basic examination:**
      i) Probe: A curvilinear probe should be used for a lung ultrasound, but a phased array probe can be used if a curvilinear probe is unavailable.
      ii) Position: With the patient supine, the probe is used to obtain sagittal views across the lungs with the indicator cephalad.
      iii) Points for assessment: surface anatomy for lung ultrasound (**Fig. 15-1**)
         (1) To identify the BLUE (Bedside Lung Ultrasound in Emergency) protocol points, place both hands together flat on the patient's chest with little finger against the inferior border of the clavicle and longest fingers at the sternum.[8]

TABLE 15-1 Conditions That Can Be Identified and Managed With POCUS

	Diagnostic	Therapeutic
Cardiac	• Pericardial tamponade • Pericardial effusion • Valvular issues • Preload or contractility issues	• Pericardiocentesis
Pulmonary	• Pneumothorax • Pulmonary edema • Pulmonary effusions • Atelectasis	• Thoracentesis • Chest tube placement
Vascular	• Deep vein thrombosis • Pulmonary embolism	• Venous access • Arterial access
Abdominal and pelvic	• Nephrolithiasis/hydronephrosis • Hepatic • Gallbladder stones • Bladder/urinary retention • Free fluid (FAST)	• Suprapubic catheter insertion • Paracentesis
Airway	• Esophageal intubation • Airway edema	• ETT confirmation • Percutaneous tracheostomy

(2) Upper BLUE point: The point is at the base of the middle and ring finger of the upper hand. This correlates with the upper lobe of the lung.
(3) Lower BLUE point: The point is in the middle of the lower palm. This correlates with the lower lobe of the lung or the lingula.
(4) Posterolateral alveolar and/or pleural syndrome (PLAPS) point: This point is at the posterior axillary line with the probe in the same plane as the lower BLUE point. At this point, the diaphragm appears as a concave structure moving with respiration. The liver or spleen should be seen immediately below the diaphragm with the vertebral bodies at the bottom of the visual field primarily in the abdomen.

FIGURE 15-1 Surface Anatomy for Lung Ultrasound



Courtesy of the Yale Anesthesia Media Lab.

(5) At the upper or lower point, there will be ridges indicating the ribs. Below the ribs, there will be significant acoustic shadowing, and assessment is focused on the area between the ribs.



The presence of lung sliding rules out a pneumothorax. However, the absence of lung sliding is not diagnostic of pneumothorax.

c) **Findings**[1,3,6,9,10]

i) Lung sliding: the movement of parietal pleural over the visceral pleura during normal breathing (**Fig. 15-2**)

ii) Lung pulse: the movement of the pleura with every heartbeat (**Fig. 15-3**)

iii) Motion mode (M-mode) findings

(1) Seashore sign: The soft tissue above the pleural line remains still while the normal lung tissue moves below the bright pleural line. Structures above the pleural line appear as lines or waves while below appears as speckled sand.

(2) Stratosphere or barcode sign: The soft tissue above the pleural line and lung tissue below the pleural line both remain still forming parallel lines as in a barcode.

iv) Lung point: Point at which the partially inflated lung comes in contact with the chest wall (**Fig. 15-4**)

v) Positive PLAPS point[11]: At the PLAPS point, a pleural effusion appears as a hypoechoic wedge above the spleen or liver and can have enhanced visualization of the vertebral bodes in the thorax (**Fig. 15-5**).

vi) Artifacts

(1) A line

(a) Horizontal linear reverberation artifacts that are the reflection of the parietal pleura

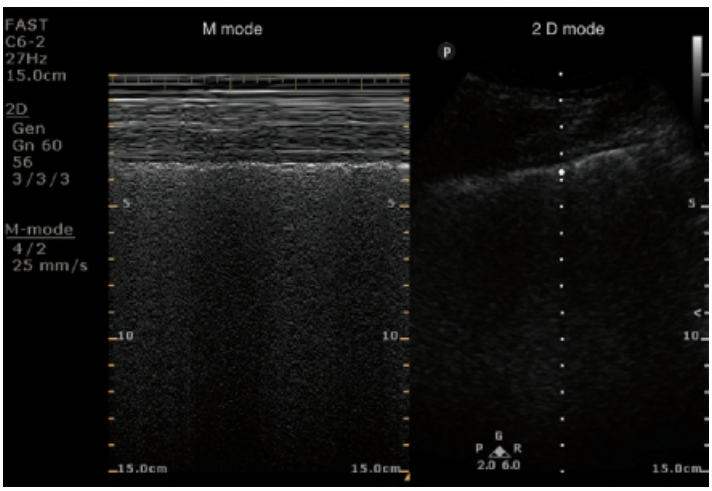
(b) Multiple A lines indicate a good-quality image in normal lung tissue.

(c) This pattern is often lost with any pathology.

(2) B lines

(a) Vertical comet-tail reverberation artifacts at the air-fluid interface.

FIGURE 15-2 Lung Sliding



Courtesy of the Yale Anesthesia Media Lab.

FIGURE 15-3 Lung Pulse



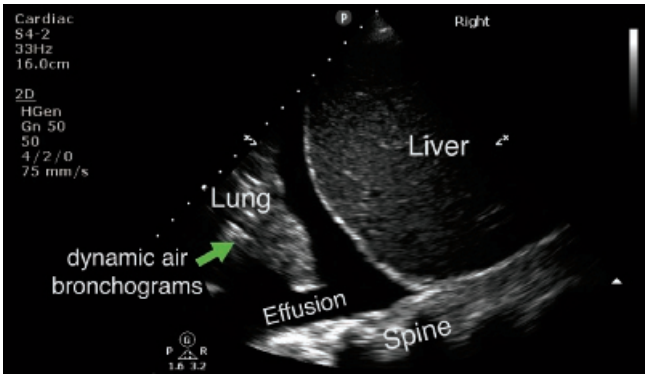
Courtesy of the Yale Anesthesia Media Lab.

FIGURE 15-4 Lung Point



Courtesy of the Yale Anesthesia Media Lab.

FIGURE 15-5 PLAPS/Pleural Effusion



Courtesy of the Yale Anesthesia Media Lab.

(b) All comet-tail artifacts are not B-lines. In order to call a comet-tail artifact a B-line, it must start from the pleura and extend to the end of image.

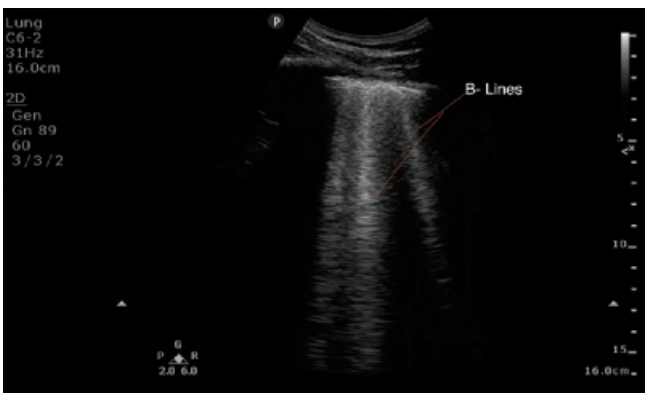
(c) May represent normal dependent lung tissue but may also be related with alveolar interstitial syndrome

d) **Assessment via BLUE protocol**[8]

i) Lung sliding present

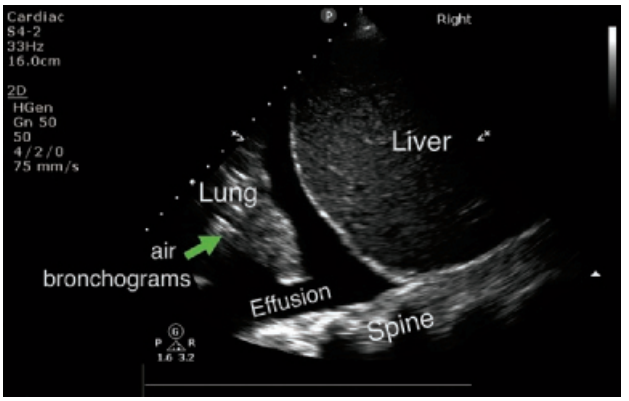
(1) B-lines indicate pulmonary edema (**Fig. 15-6**). Multiple, close B-lines in a nondependent lung field indicates severe pulmonary edema.

FIGURE 15-6 Pulmonary Edema



Courtesy of the Yale Anesthesia Media Lab.

FIGURE 15-7 Consolidation/Pneumonia



Courtesy of the Yale Anesthesia Media Lab.

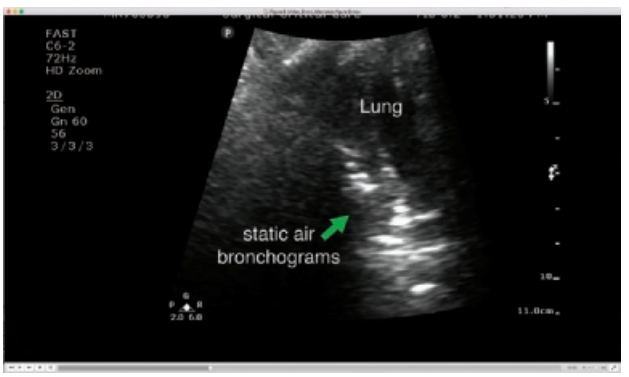
(2) A lines with thrombosed veins indicate pulmonary embolism.
(3) A lines and positive PLAPS indicate pneumonia.
(4) A lines and negative PLAPS indicate COPD or asthma.

ii) Absent lung sliding
(1) B lines indicate consolidation.
(a) On examination of the PLAPS point, lung parenchyma can be better visualized. There may be hepatization, a loss of aeration and conversion to a tissue-like pattern, with air bronchograms.
(i) Dynamic air bronchograms move with respirations and are more often associated with pneumonia (**Fig. 15-7**).
(ii) Static air bronchograms indicate atelectasis (**Fig. 15-8**).



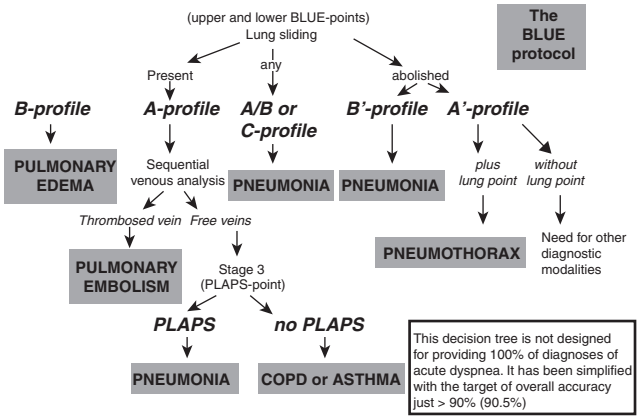
Multiple, close B lines in a nondependent lung field indicates severe pulmonary edema.

FIGURE 15-8 Atelectasis



Courtesy of the Yale Anesthesia Media Lab.

FIGURE 15-9 BLUE Protocol.



Reprinted from Lichtenstein DA, Mezière GA. Relevance of lung ultrasound in the diagnosis of acute respiratory failure: the BLUE protocol. *Chest*. 2008;134(1):117-125, with permission from Elsevier.

(2) A lines with lung point highly specific for pneumothorax. B lines should not be present (**Fig. 15-9**)[8].

iii) Pneumonia may have either A or B lines.

3) **Pulmonary: diaphragmatic ultrasound**[12]

a) **Background**

i) Diaphragmatic excursion (DE) and thickening fraction (DTF) can be used to wean mechanical ventilation. They are used together to assess for diaphragm dysfunction, a key contributor to failed weaning.

b) **How to perform basic diaphragmatic examination:**

i) Probe: Curvilinear or phased array probe

ii) Position: The patient is supine. The probe is placed at the posterior axillary line to visualize the diaphragm through a full respiratory cycle in M-mode. Patient should be spontaneously breathing.

c) **For diaphragmatic excursion, the difference between the highest and lowest points in a respiratory cycle is measured.**

i) Although there is no definitive measure for weaning failure, a difference of <15 mm indicates high probability of diaphragm dysfunction.[13]

d) **For diaphragm thickening fraction, the thickness of a point on the diaphragm is measured at end expiration (EE) and end inspiration (EI).** These are used in the following formula to calculate the fraction:

i) (EI thickness – EE thickness)/EE thickness

ii) A fraction <0.2 or 20% is associated with diaphragm dysfunction.[13]

4) **Abdominal and pelvic: gastric ultrasound**[14]

a) **Background**

i) Can be used to confirm fasting status or evaluate risk for aspiration

ii) Either quantitative measurements are made to calculate gastric volume or qualitative assessments to confirm fasting status.

b) **How to perform basic examination:**

i) Probe: A curvilinear or phased array probe can be utilized.

ii) Position: Measurements should initially be taken in the supine or semirecumbent position and then taken in the right later decubitus (RLD) position. The probe is positioned for a sagittal view immediately below the costal margin, left of midline (epigastric area).
iii) The left lobe of the liver is a homogeneous triangular structure immediately below subcutaneous tissue. This is used as a landmark to identify the gastric antrum, which is located inferior to the liver.

c) **The type of gastric content present can be differentiated based on sonographic appearance.**
i) Clear fluids such as gastric contents, juice, or water will appear homogenous and anechoic to hypoechoic. Volumes exceeding 1.5 mL/kg would be ingested fluids and not gastric secretions.
ii) Thick fluids appear hyperechoic and homogenous.
iii) Solid foods appear as "frosted glass" due to air trapping. Due to the gaseous content, visualization of the posterior wall of the antrum is difficult.



A gastric cross-sectional area $\geq$340 mm^2 is considered high risk for aspiration. This correlates to gastric volume >1.5 mL/kg.

d) **Quantitative assessment: The cross-sectional area (CSA) of the gastric antrum can be measured to provide an estimate of gastric volume**.
i) Supine position may lead to an underestimate of contents.
ii) Calculation for CSA: Anteroposterior (AP) and craniocaudal (CC) diameters measured
(1) $CSA = (AP \times CC \times \pi)/4$

e) **Qualitative assessment**:
i) Grade 0: No fluid visible in the antrum in either supine or RLD position. Antrum is small, collapsed, and bull's-eye in appearance.
ii) Grade 1: Fluid visible in the antrum only in the RLD position (representing <100 cc)
iii) Grade 2: Fluid visible in the antrum in both positions (representing >100 cc)

5) **Abdominal and pelvic: focused assessment with sonography for trauma (FAST) (Figs. 15-10 and 15-11)**

a) **Background**[3,6]
i) Designed to evaluate for intra-abdominal fluid extravasation, also can include a focused cardiac echocardiogram
ii) Can detect 250-600 cc of free fluid
iii) May be used to assess decompensating patients in the postanesthesia care unit or intensive care unit after abdominal or pelvic procedures[15]

b) **How to perform basic examination:**
i) Probe: Use a curvilinear probe.
ii) Position: The patient should be supine. The probe is positioned in the bilateral flanks to provide a view of the diaphragm and structures immediately inferior such as the liver or spleen. The probe also is placed above the pubic symphysis for a sagittal view of the pelvis, including the bladder.

c) A hyperechoic strip in Morrison pouch or perisplenic space likely represents intra-abdominal fluid.

6) **Airway**

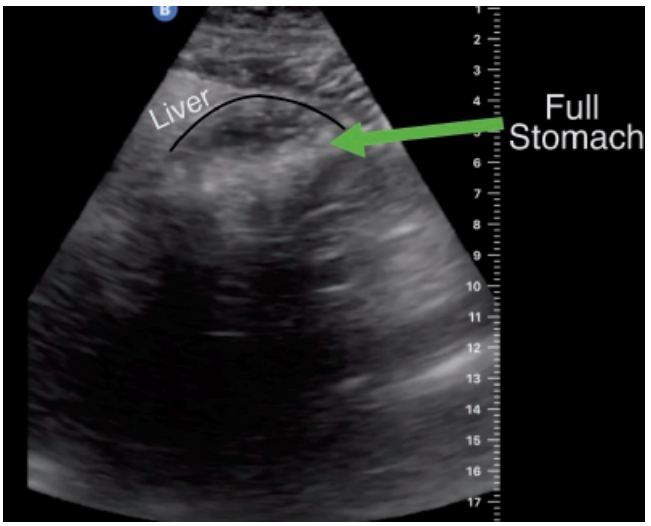
a) **Background**
i) Can be used for identifying surgical airway access point, confirming intubation, selecting appropriate endotracheal tube size, evaluating vocal cord function, and identifying potential difficult intubations (**Fig. 15-12**)



Airway ultrasound can be used to locate the cricothyroid membrane, predict difficult airway, and look for vocal cord mobility.

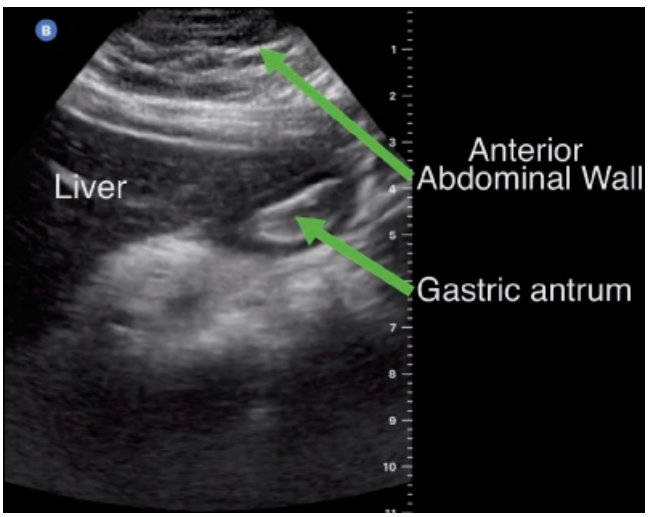
b) **How to perform basic examination:**
i) Probe: Typically, a linear probe is used, but a curvilinear probe should be used to visualize anatomy in obese patients, deeper structures, or the submandibular region.[16]

FIGURE 15-10 Full Stomach



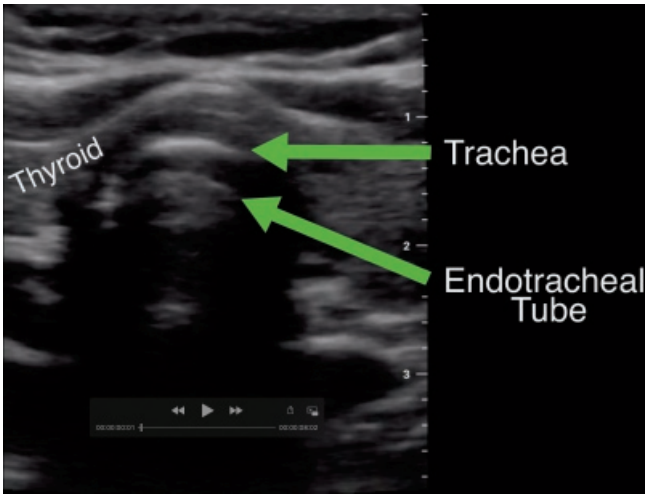
Courtesy of the Yale Anesthesia Media Lab.

FIGURE 15-11 Empty Stomach



Courtesy of the Yale Anesthesia Media Lab.

FIGURE 15-12 Endotracheal Intubation

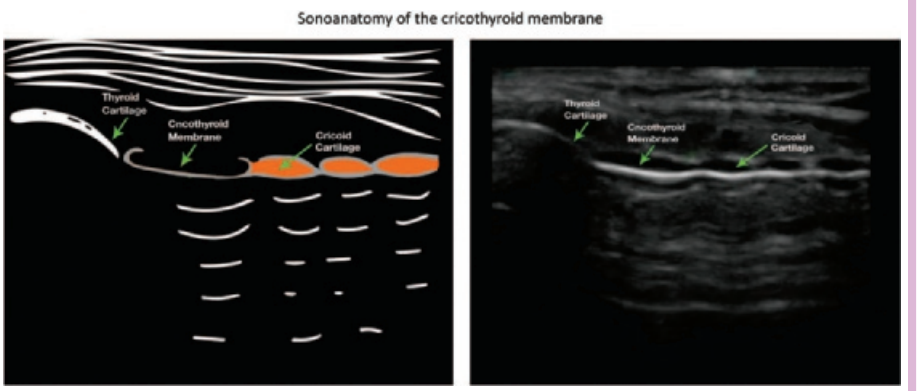


Courtesy of the Yale Anesthesia Media Lab.

ii) Position: Supine patient in sniffing position
iii) A combination of sagittal/parasagittal and transverse views are used to identify structures.

c) **The cricoid cartilage and cricothyroid membrane are identified to confirm endotracheal intubation or facilitate surgical airway (Fig. 15-13).**
i) In a sagittal or parasagittal view, the probe is placed with the marker cephalad at the sternal notch. The tracheal rings should be identified as a hyperechoic

FIGURE 15-13 Cricothyroid Membrane



Courtesy of the Yale Anesthesia Media Lab.

band resembling a "string of beads." The transducer is moved cephalad until a large hump is identified. This represents the cricoid cartilage, and the cricothyroid membrane is a thin hyperechoic band that connects the large hump, cricoid cartilage, with another large structure, the thyroid cartilage, more cephalad.

ii) In a transverse view, the probe again is placed at the sternal notch and moves cephalad. Round structures representing the tracheal rings will be immediately visible. These are followed up until a larger U-shaped structure with an echogenic homogenous band, the thyroid, immediately above it. The cricothyroid membrane is cephalad to this and is a thin band with reverberation artifact below it. Further cephalad will be a V-shaped structure that represents the thyroid cartilage.

(1) Measuring the diameter of a tracheal ring at the level of the sternoclavicular joint in the transverse view correlates with the diameter of the left bronchus. This can be used for selecting an appropriate size for a double lumen endobronchial tube or tracheostomy.

(2) An esophageal intubation is identified by an echogenic oval outside the trachea, typically to left (**Fig. 15-14**).

d) **The hyoid bone is a marker in several measurements to identify difficult intubation:**

i) The hyoid bone is a horseshoe-shaped structure above the level of the thyroid cartilage. Can be identified via a transverse view or a sublingual view.

ii) Difficult intubation can be predicted by any of the following:

(1) Inability to visualize the hyoid bone using sublingual ultrasound[17]

(2) A hyoid to mandibular mentum distance ratio of 1-1.05[18]

(3) Higher amount of pretracheal soft tissue in morbidly obese patients[19]

e) **Evaluation of vocal cord mobility can help diagnose postoperative etiology of stridor.**

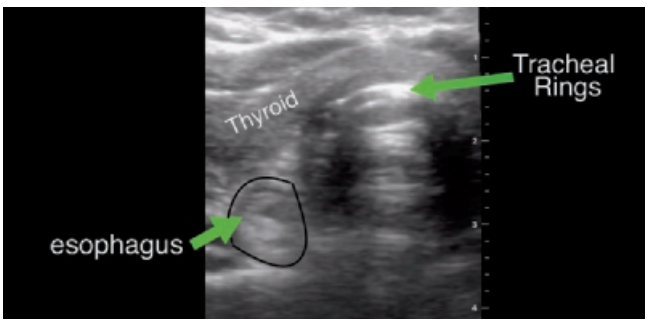
i) Best evaluated in a transverse view at the level of the thyroid cartilage

ii) Visualization decreases in older males and those with calcified thyroid cartilage.

iii) The true vocal cords are seen as two hypoechoic triangles, outlined medially by hyperechoic vocal ligaments. The true vocal cords will oscillate with phonation.

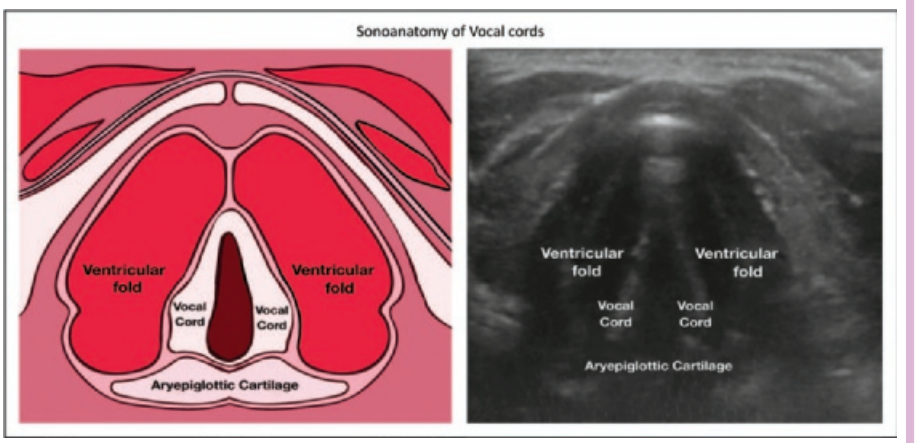
iv) The false vocal cords are cephalad and parallel to the true vocal cords (**Fig. 15-15**).

FIGURE 15-14 Esophagus



Courtesy of the Yale Anesthesia Media Lab.

FIGURE 15-15 Vocal Cords



Courtesy of the Yale Anesthesia Media Lab.

7) **Vascular: deep venous thrombosis (DVT) ultrasound of lower extremity**[20]
   a) **Background**
      i) A complete venous US should be performed if the pretest probability assessed by a risk assessment tool like the Wells test indicates high risk of DVT.
   b) **How to perform basic examination:**
      i) Probe: A linear or curvilinear probe can be used.
      ii) Position: Supine with study leg slightly bent at knee and internally rotated.
      iii) Scanning should be done from the inguinal ligament to the ankle, including the posterior tibial and peroneal veins with compression at every 2 cm.
      iv) Doppler waveforms should be obtained from the common femoral veins and the popliteal vein.[20]
   c) **Extended US:** From inguinal ligament to the confluence of the popliteal vein with the calf veins
   d) **Two-point US (more commonly performed):** Limited to compression of the femoral and popliteal regions
   e) Acute thrombi are soft and compressible with pressure in a large vein. Chronic thrombi are rigid and not compressible.

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16 Neurophysiologic Monitoring§

Shilpa Rao

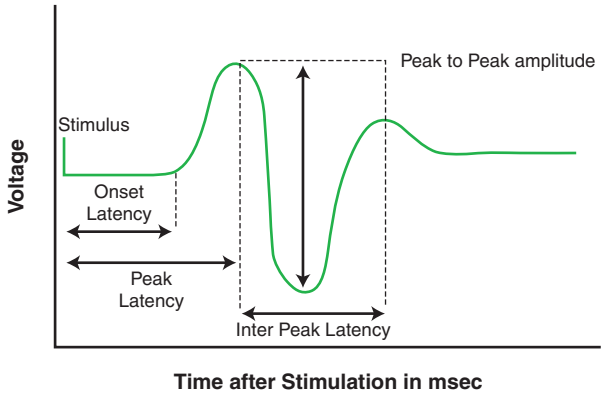
Neurophysiologic monitoring or neuromonitoring allows early detection of critical intraoperative events that may increase postoperative neurological morbidity. The aim of monitoring is to identify changes in brain, spinal cord, and peripheral nerve perfusion and function prior to irreversible damage. Neuromonitoring is also useful in identifying important anatomical structures to monitor intraoperative depth of anesthesia (eg, processed EEG) and as an effect-site monitor to aid in the titration of anesthetic drugs and may be useful in minimizing the incidence of intraoperative awareness and undesirable recall.

1) **Overview of neurophysiologic monitoring in anesthesia**
   a) Neurophysiologic monitoring is quickly becoming the standard of care in all cases where the potential exists for damage to the brain and spinal cord due to surgical manipulation of the tissue or its blood supply. Due to its widespread use, it is important to understand the basic concepts, techniques, and anesthetic implications including minimizing intraoperative awareness.
   b) **Basic concepts**
      i) Evoked potentials (responses recorded from the nervous system following electrical stimulation) are described in terms of latency and amplitude (**Fig. 16-1**).
      ii) Intraoperative changes in either parameter may result from surgical injury or ischemia of the specific neural pathway, or they may be due to nonspecific physiologic or pharmacologic influences.
      iii) Physiologic factors that influence evoked potentials include temperature, BP, hematocrit, acid-base balance, and O_2 and CO_2 tensions.
      iv) Anesthetic drugs are the most common pharmacologic causes of nonspecific changes in evoked potentials.[1]
2) **Techniques**
   a) **Electromyography (EMG)**
      i) EMG is the recording of electrical activity of a single muscle/group of muscles and therefore an indirect indicator of function of the innervating peripheral nerve.
      ii) This technique is also used to identify and verify the integrity of a peripheral nerve, including cranial nerves as well as pedicle screw testing during spine surgery.
      iii) EMG is sensitive to neuromuscular blocking agents.
      Some of the examples of EMG monitoring include facial nerve monitoring during cerebellopontine angle tumors/parotid surgeries, laryngeal innervation monitoring during certain intracranial procedures and thyroid surgeries, etc.
   b) **Somatosensory evoked potentials (SSEPs)**
      i) SSEPs are recordings, usually at the cerebral cortex, of responses from electrically stimulated peripheral afferent nerves.
         (1) The most commonly used peripheral nerves are median, ulnar, posterior tibial, and common peroneal nerves.



Physiologic factors that influence evoked potentials include temperature, BP, hematocrit, acid-base balance, and O_2 and CO_2 tensions.

FIGURE 16-1 Schematic of Evoked Potential in Terms of Latency and Amplitude



Reproduced with permission from Banoub M, Tetzlaff JE, Schubert A. Pharmacologic and physiologic influences affecting sensory evoked potentials: Implications for perioperative monitoring. *Anesthesiology*. 2003;99(3):717.

ii) Significant changes in SSEP are as follows:
   (1) A decrease in amplitude of ≥50%
   (2) An increase in latency of ≥10%
   (3) Both of the above reflect loss of integrity of a neural pathway, provided these changes are not caused by physiologic or pharmacologic factors.[2]

iii) SSEPs are sensitive to volatile anesthetics including nitrous oxide in a dose-dependent manner, which cause a profoundly decreased amplitude at higher doses.[3]
   (1) Etomidate and ketamine increase SSEP amplitude.[4]
   (2) SSEPs are not affected by neuromuscular blockade agents or opioids.
   (3) A balanced anesthetic technique for maintenance is suggested, using a combination of intravenous anesthetics (propofol/ketamine/dexmedetomidine) along with narcotics and neuromuscular blocking agents as needed.
   (4) Inhalational agents <0.5 MAC can also be added if required at the judgment of clinician.

c) **Brainstem auditory evoked potentials (BAEPs)**

i) BAEPs are recordings of brainstem responses to auditory stimuli.

ii) Other acronyms include brainstem auditory evoked responses (BAER) and auditory brainstem responses.

iii) BAEP monitors the function of the entire auditory pathway along the acoustic nerve, through the brainstem to the cerebral cortex.

iv) BAEP is insensitive to both volatile anesthetics and neuromuscular blocking agents.[5] Hence most of the commonly used anesthetic techniques can be safely used if BAEPs are being monitored.

v) A BAER test can help to diagnose hearing loss and nervous system disorders, especially in newborns and very young children under anesthesia, who may not be able to participate in a standard hearing test.

d) **Motor evoked potentials (MEP)**
   i) MEP is the recording obtained from electrical stimulation of the motor cortex, which elicits potentials in the spinal cord or (myogenic) potentials from the innervated muscle.
   ii) Monitors motor pathway function
   iii) MEP is sensitive to both volatile anesthetics and neuromuscular blocking agents.
   iv) MEP is almost always performed in conjunction with SSEP. These techniques function as complementary tests.[6]
   When MEPs are being measured, TIVA with narcotics, propofol, and/or ketamine is recommended and neuromuscular blockade must be withheld after appropriate induction dose (if required). In some cases, inhalational agents at <0.5 MAC can be safely used if MEPs remain intact.

e) **Electroencephalography (EEG)**
   i) EEG monitoring can be a useful supplement to surgery when:
      (1) Seizure foci need to be identified
      (2) The general state of cerebral metabolism needs monitoring
      (3) Cerebral ischemia can occur
   ii) EEG is a standard of care in many institutions for carotid endarterectomy.
   iii) EEG is the recording of brain electrical activity and is highly dependent on anesthetic depth.
      (1) Alpha waves are rhythmically regular waves of 8-12 Hz seen in a lightly anesthetized patient.
      (2) A faster, disorganized beta (>12 Hz) rhythm is seen upon awakening.
      (3) Slower theta waves (4-8 Hz) are seen with deep inhalational or moderate dose narcotic anesthesia.
      (4) Slow delta waves (<4 Hz) indicate deep anesthesia or ischemia if the amplitude is low.
      (5) Burst suppression represents bursts of EEG activity with alternating periods of inactivity.
      (6) Under special conditions, "spike and wave" seizure activity identifies seizure foci for resection.
      (7) EEG also allows for monitoring depth of anesthesia using mathematical processing of the EEG (processed EEG).
   iv) EEGs are moderately sensitive to volatile anesthetic and insensitive to neuromuscular blockade. Hence, a balanced maintenance regime using inhalational agents at <0.5 MAC, with opioids and neuromuscular blockade as needed, can be used.
   v) Dexmedetomidine infusion elicits an EEG pattern similar to that of stage II sleep and does not affect the quality and interpretation of EEG signals and hence can be useful as an adjunct.[7]
   vi) As always, when a combination of above monitoring techniques are being used, constant vigilance and communication is essential between the involved team members, namely, anesthesiologists, neuromonitoring personnel, and surgeon to maximize the benefit and facilitate safe surgery.



Avoid NMB when EMG or MEP is being monitored because neuromuscular transmission is inhibited.

Dexmedetomidine infusion elicits an EEG pattern similar to that of stage II sleep and does not affect the quality and interpretation of EEG signals and hence can be useful as an adjunct.

3) **Intraoperative awareness**
   a) **Symptoms**
      i) The most common symptoms reported by patients suggesting awareness with recall are auditory perceptions such as voices or noises, followed by loss of motor function (inability to move, sensation of weakness, or paralysis), pain, and feelings of helplessness, anxiety, panic, impending death, or catastrophe.

ii) Awareness with recall can lead to anxiety, sleep difficulties, insomnia, irritability, nightmares, and posttraumatic stress disorder.

b) **Incidence and risk factors for intraoperative awareness**

i) The incidence of awareness with recall varies among studies, countries, anesthetic techniques, patient characteristics, and types of surgery.

ii) The most commonly cited rate of intraoperative awareness is 0.13%.[8] This data was obtained from a multicenter study of 19 575 patients, and the rate of awareness was 1-2 cases per 1000 patients at each site.[8] This figure is thought to reflect the incidence in routine cases but not including cardiac or obstetric surgeries. When further stratified, awareness occurs in ~1.14%-1.5% of cardiac surgery cases, 0.4% of obstetric cases, and 11%-43% of trauma surgeries.[9] Awareness with recall associated with pain is estimated to occur in 0.01%-0.03% of cases.

iii) Other risk factors include "light" anesthesia (eg, delivering a low level of inhaled anesthetic minimum alveolar concentration either due to operator error or improper use of anesthesia delivery systems), history of intraoperative awareness, younger age, chronic use of central nervous system depressants, and obesity.



Awareness with recall can lead to anxiety, sleep difficulties, insomnia, irritability, nightmares, and posttraumatic stress disorder.

Targeting a range of BIS values between 40 and 60 is marketed to help prevent anesthesia awareness while allowing for minimizing the anesthetic dose to the patient.

c) **Detecting episodes of intraoperative awareness**

i) Often it is difficult to know for sure that intraoperative awareness with recall occurred. For example, if the patient is not asked specifically about it, they may not report it voluntarily. Or, the patient may recollect hearing sounds during surgery, when in fact they are remembering something that occurred in the recovery room.

ii) One accepted method to assess intraoperative awareness with recall is to conduct three structured interviews with open-ended questions at intervals of 24 hours, between 24 and 72 hours, and at 30 days after surgery (awareness may not arise until days to weeks postoperatively).

iii) It is important to note that bispectral index (BIS) monitoring (see below) is a probability distribution where a measure of 40 does not provide a 100% guarantee of no awareness.

4) **Prevention or vigilance for detecting intraoperative awareness**

a) **Monitor delivered volatile anesthetic levels**

The unintended inadequate delivery of volatile anesthetic agents ("light anesthesia") during maintenance of anesthesia may be avoided by the addition of a low alarm limit to end-tidal gas monitoring settings, as well as use of a "near empty" alarm in anesthetic vaporizers.

b) **Monitor processed EEG signals**

Depth-of-anesthesia monitoring, via the processed EEG, has proved useful in reducing the amount of anesthetic drugs, optimizing extubation times, and in some studies reducing awareness with recall. Although most anesthesiologists in the UK, USA, and Australia accept that clinical signs are unreliable indicators of awareness, few believe that monitors of anesthetic depths should be used for all routine cases.[10]

i) Depth-of-anesthesia monitors

Several brain-function monitors based on the processed electroencephalogram (EEG) or evoked potentials have been developed to assess anesthetic depth.

(1) BIS (Aspect Medical Systems) The most widely used monitor is the BIS monitor. This device integrates several parameters of an EEG into a calculated, dimensionless variable (0-100). The BIS calculations are derived from large patient databases that incorporate recorded EEG waveforms as they relate

to anesthetic drug concentration data, memory, and awareness.[11] The term bispectral applies because it incorporates both power and phase spectrums of an EEG into the calculated 0-100 value. BIS values between 40 and 60 purportedly indicate adequate general anesthesia for surgery, and values below 40 indicate a deep hypnotic state. Targeting a range of BIS values between 40 and 60 is marketed to help prevent anesthesia awareness while allowing for minimization the anesthetic dose.

(2) M-Entropy Module (GE Healthcare): A mathematical approach that quantifies EEG using non-linear dynamics. This mode measures spectral entropy and applies it to the power spectrum of EEGs. Two variables, state and response entropy, which measure EEG and combined EEG/EMG activity, respectively, are displayed on the awareness monitor as a dimensionless unit (0-100).[12]

ii) Measurement errors and artifacts associated with depth-of-anesthesia monitors

(1) Depth-of-anesthesia monitors have built-in mechanisms that detect external artifacts (electrocautery, pacemakers, ECG monitoring), increases in beta EEG activity from anesthetics, and burst suppression patterns.

(2) Clinical situations that falsely increase depth-of-anesthesia monitoring include excessive facial muscle tone, electrocautery, pacemaker, 12-lead ECG monitoring, movement, surgical drilling, and agents that may produce anesthesia without a reduction in the awareness monitor index (nitrous oxide, xenon, ketamine).

(3) A subdural hematoma—via reduced voltage at the scalp—may falsely decrease depth-of-anesthesia recordings.[13]

iii) Using processed EEG as a depth-of-anesthesia monitor

(1) Depth-of-anesthesia monitoring may be especially useful as an effect-site monitor during total intravenous anesthetic (TIVA) and especially in patients who are paralyzed during TIVA. Unlike volatile anesthetics in which there is a real-time metric to assess end-tidal concentrations of the anesthetic, TIVA procedures are limited to ED50 values that do not translate as well to depth of anesthesia.

(2) Furthermore, if a patient's IV is not under direct visualization, it is difficult to assess if the IV has dislodged. In such a scenario, the IV pump will not alarm, and the anesthesia provider may believe that the patient is receiving the anesthetic. This can be especially troubling if the patient received neuromuscular blockade. Use of a depth-of-anesthesia monitor may help prevent such a scenario.

(3) Titration of an anesthetic to an adequate depth based on hemodynamic parameters and depth-of-anesthesia monitors can also help limit the negative impact of excessive anesthesia. In addition, in the ICU, depth-of-anesthesia monitoring may help reduce total ventilation time as well as hypotension related to chronic sedation.

iv) Postoperative outcomes

While processed EEG monitoring provides a more accurate way to gauge of depth of anesthesia than hemodynamic monitoring alone, multiple studies have found correlations between deep hypnotic states as recorded by BIS and increased postoperative mortality.[14]

(1) For example, Monk et al. speculated that the reason for the association might be an inherently toxic effect of anesthetics, perhaps through a proinflammatory mechanism. These effects may not only increase mortality but also impair cognitive function as volatile anesthetics have been associated with neuronal apoptosis in rodents and cognitive decline in humans.[15,16]

(2) Further research studies to explore these relationships are ongoing.

CHAPTER SUMMARY FOR NEUROPHYSIOLOGIC MONITORING

Definition	Neuromonitoring during anesthesia measures electrical activity in various parts of the nervous system, including the brain, spinal cord, and peripheral nerves.
Goals	The goal of neuromonitoring is to identify changes in neurologic function during surgery in order to prevent irreversible damage.
Physiologic Effects	Neuromonitoring affected by temperature, BP, hematocrit, acid-base balance, and O_2 and CO_2 tensions
Volatile Effects	↓ EEG, ↓ SSEP, ↓↓ MEP, ↔ BAER
NMB	↓↓ MEP, ↔ EEG, ↔ BAER, ↔ SSEP

BAER, brainstem auditory evoked response; EEG, electroencephalogram; MEP, motor evoked potential; NMB, neuromuscular blockade; SSEP, somatosensory evoked potential.

CHAPTER SUMMARY PROCESSED EEG AND AWARENESS MONITORING

Awareness	Intraoperative awareness with recall involves explicit recall of sensory perceptions during general anesthesia including aspects of the surgical environment, procedure, and even pain related to the intervention.
Incidence	The most cited rate of intraoperative awareness is 0.13%.
Goals	Targeting a BIS range of 40-60 is marketed to help prevent anesthesia awareness while allowing for minimization of anesthetic dose.
False ↑ DOA	Excessive facial muscle tone, electrocautery, pacemaker, ECG monitoring, movement, surgical drilling, agents that may produce anesthesia without a reduction in the awareness index (N_2O, xenon, ketamine)
False ↓ DOA	Subdural hematoma, which causes reduced voltage at the scalp
Other Uses	Titration using DOA monitors may help limit the negative effects of excessive anesthesia and detect inadvertent light anesthesia during TIVA.

BIS, bispectral index; DOA, depth of anesthesia; ECG, electrocardiogram; N_2O, nitrous oxide; TIVA, total intravenous anesthesia.

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17 The Anesthesia Machine

Chad Pritts • Samsun (Sem) Lampotang

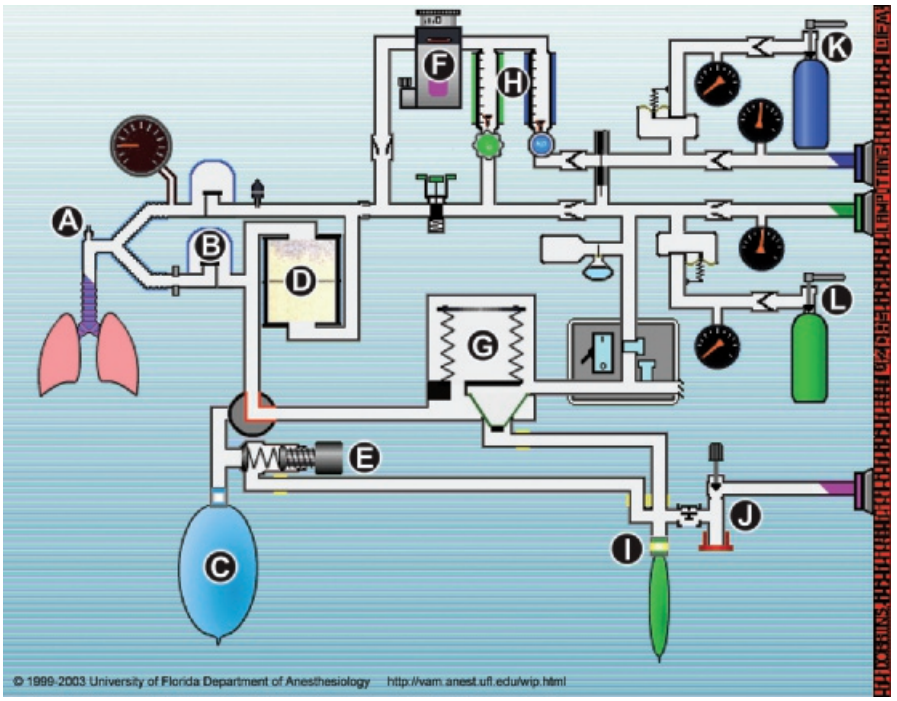
The anesthesia machine is a vital component to the safe and reliable delivery of anesthesia and provides oxygenation, ventilation, and anesthetic gases to the patient. Anesthesia machines have become increasingly complex; however, they all employ the same fundamental functions.

1) **Overview of anesthesia machine elements.** In order to understand the anesthesia machine, it helps to divide it into four component subsystems (**Fig. 17-1**).
   a) **The high-pressure system**
      i) Receives connections from the hospital pipeline gas supply and includes the cylinder gas supplies
   b) **The low-pressure system**
      i) Blends oxygen and anesthetic gases according to the control settings
   c) **Breathing system**
      i) Delivers the fresh gas mixture from the low-pressure system to the patient and provides ventilation
   d) **Scavenging system**
      i) Collects excess gas from the breathing system and delivers it to the waste gas evacuation system
2) **The high-pressure system**
   a) **Gas sources**
      i) Central or pipeline supply
         (1) The primary gas source for the anesthesia machine.
         (2) There is a central piping system in most hospitals that delivers medical gases (oxygen, nitrous oxide, and air) to the operating room.
         (3) For the anesthesia machine to function properly, the correct gas must be delivered to its corresponding inlet port at an appropriate pressure.
         (4) Inadequate oxygen pressure is the most commonly reported problem.
         (5) The most devastating problem is accidental delivery of hypoxic gases from the oxygen pipeline, which has resulted in hypoxic deaths (e.g., crossover of oxygen and nitrous oxide).
            (a) If pipeline crossover is suspected, two steps must be taken.
               (i) The backup oxygen cylinder should be turned on.
               (ii) The pipeline supply must be disconnected.
                  1. This step is essential because the machine uses the inappropriate pipeline gas at 45-55 psi instead of the lower-pressure 40-45 psi oxygen cylinder.
         (6) The pipeline inlet fittings are gas specific.
            (a) The Diameter Index Safety System provides threaded body fittings that are noninterchangeable to minimize the risk of misconnection



For the anesthesia machine to work properly, the correct gas must be delivered at an appropriate pressure into the corresponding inlet port of the anesthesia machine.

FIGURE 17-1 A Typical Layout for a Bellows Anesthesia Machine with the Four Component Subsystems Depicted



(A) Anesthesia breathing circuit, **(B)** unidirectional flow valve, **(C)** reservoir bag, **(D)** CO_2 absorber, **(E)** APL "pop-off" valve, **(F)** anesthetic vaporizer, **(G)** ventilator, **(H)** flowmeters, **(I)** scavenging bag, **(J)** scavenging manifold, **(K)** nitrous oxide cylinder, **(L)** oxygen cylinder. From the virtual anesthesia machine simulation—http://vam.anest.ufl.edu/wip.html, with permission.

ii) The cylinder supply
   (1) Reserve E cylinders are for use if a pipeline source is not available or if the pipeline fails.
   (2) Color-coded cylinders are attached to the anesthesia machine through a hanger yoke assembly.
      (a) In the United States, the color-coding is green for oxygen, blue for nitrous oxide, and yellow for air.
      (b) The international color code is white for oxygen, blue for nitrous oxide, and white and black for air.
   (3) Hanger yoke assembly
      (a) Orients and supports the cylinder and provides a gas tight seal to the machine.
      (b) It is equipped with a Pin Index Safety System, which is a safeguard to eliminate accidental cylinder misconnection.
         (i) Each gas has a specific pin arrangement.
   (4) Check valve located downstream
      (a) Minimizes gas transfer from a cylinder with high pressure to one with low pressure.



If pipeline crossover is suspected, the backup oxygen cylinder should be turned on and the oxygen pipeline supply must be disconnected.

(b) It allows an empty cylinder to be exchanged for a full one while gas flow continues into the machine.

(5) The cylinder gauge indicating supply pressure is located downstream or upstream from the check valve.

(a) Newer machines have an electronic pressure sensor instead of a gauge, and the measured pressure is displayed on the display panel.

(6) Each cylinder has a pressure regulator that reduces the high and variable storage pressure to a more constant pressure.

(a) The oxygen cylinder pressure regulator reduces the cylinder pressure from a high of 2,200 to 40-45 psi.

(b) The nitrous oxide cylinder pressure regulator reduces the cylinder pressure from a high of 745 to 40-45 psi.

(7) If both the cylinder and the pipeline are open and connected, gas flows preferentially from the pipeline because the pressure is slightly higher than the cylinder's regulated pressure.

(8) Cylinders should be turned off except during the preoperative machine check.

(a) They can become depleted if open and the pressure in the pipeline falls below 45 psi.

b) **Pneumatic and electronic alarm devices**

i) Many older machines have a pneumatic alarm device that sounds a warning when the oxygen supply pressure falls below a threshold value.

(1) A commonly used mechanism employs a pressurized canister that is filled with oxygen.

(2) When the machine is turned on, a stream of oxygen passes through a whistle.

(3) If the oxygen pressure drops below a certain value, the canister will empty, and a reverse stream of oxygen will flow through the whistle.

ii) Electronic alarms are used to meet this requirement on newer machines.

c) **Oxygen flush valve**

i) It provides a high flow directly to the common gas outlet.

ii) The oxygen flush can be used even when the machine is not turned on.

iii) It bypasses the flowmeters and vaporizers.

iv) The flow is at 35-75 L/min.

(1) Because of this, there is a real potential for barotrauma.

v) If the oxygen flush valve is used during the expiratory phase of mechanical ventilation, the bellows will fill to their maximum capacity.

(1) After the maximum capacity is reached, any excess will be vented through the ventilator pressure relief valve.

vi) During inspiration, the ventilator pressure release valve is closed, so there is no outlet for the excess gas.

(1) If the oxygen flush valve is pressed during mechanical inspiration, dangerously high pressures can be transmitted to the lungs, if the high pressure limit is improperly set.

d) **Oxygen pressure failure devices**

i) Fail-safe system

(1) As oxygen supply pressure decreases below a preset level, the supply of gases other than oxygen is shut off and an alarm goes off.

(2) This prevents the delivery of a hypoxic gas mixture.

(3) An oxygen analyzer in the inspiratory arm is mandated to ensure that a hypoxic mixture is detected.



In older anesthesia machines, if the oxygen flush valve is pressed during mechanical inspiration, barotrauma to the lungs can result from high pressures transmitted to the lungs.

3) **Low-pressure system**
   a) **The flowmeter assembly**
      i) Precisely controls and measures the gas flow to the common gas outlet.
      ii) Gas flows continuously from the anesthesia machine to the breathing circuit.
      iii) The gas flow rate is determined by the flow control valves and is measured by the flowmeters.
      iv) Many anesthesia machines have a mandatory minimum oxygen flow rate of 200 mL/min when the machine is turned on.
         (1) In these machines, the bobbin in the oxygen flowmeter will not completely return to zero.
   b) **Flow control valves**
      i) The high-pressure system is separated from the low-pressure system by the flow control valves.
      ii) There is a substantial pressure drop as gas passes through the flow control valve.
      iii) The flow control valves are variable-orifice needle valves.
         (1) When the knob of the flow control valve is turned counterclockwise, a pin is disengaged, allowing gas to flow through the valve.
      iv) The knobs are touch and color coded to reduce the likelihood of turning on or off the wrong gas.
   c) **Flowmeters**
      i) There are two types of flowmeters, constant pressure and variable orifice.
      ii) With the constant pressure flowmeter, the pressure decrease remains constant across the float at all positions of the tube.
      iii) The variable orifice flowmeter has a tapered tube with the largest diameter at the top. These tapered, transparent, glass tubes are also known as *Thorpe tubes*.
      iv) The bobbin floats at a point of equilibrium, where the downward force of gravity is equal to the upward force generated by gas flow.
      v) Flow is read at the top of plumb bob and at the center of ball type floats.
      vi) Flowmeters are calibrated to a specific gas density and viscosity and can't be interchanged.
      vii) Many newer machines have conventional control knobs and flow control valves but have digital flowmeters (see **Fig. 17-3**).
      viii) The oxygen flowmeters should be positioned downstream from the other medical gas flowmeters to lessen the risk of delivering a hypoxic gas mixture.



Flow control valves are touch and color coded to reduce the likelihood of turning on or off the wrong gas.

   d) **Vaporizers**
      i) Volatile anesthetics must be vaporized before being delivered to the patient.
      ii) Modern vaporizers are agent specific and capable of delivering a constant concentration of agent even at different temperatures and flows.
      iii) Agent-specific vaporizers are also known as variable bypass vaporizers; some of the entering gas is never exposed to (bypasses) the anesthetic liquid.
      iv) Variable bypass vaporizers should be located outside of the circle system, between the flowmeters and the common gas outlet.
      v) Desflurane requires a special vaporizer called the Tec 6.
         (1) Desflurane's vapor pressure is so high that it almost boils at room temperature, and it has a potency that is only one-fifth that of the other volatile agents.
      vi) Machines have interlock devices that prevent the use of more than one vaporizer at a time.

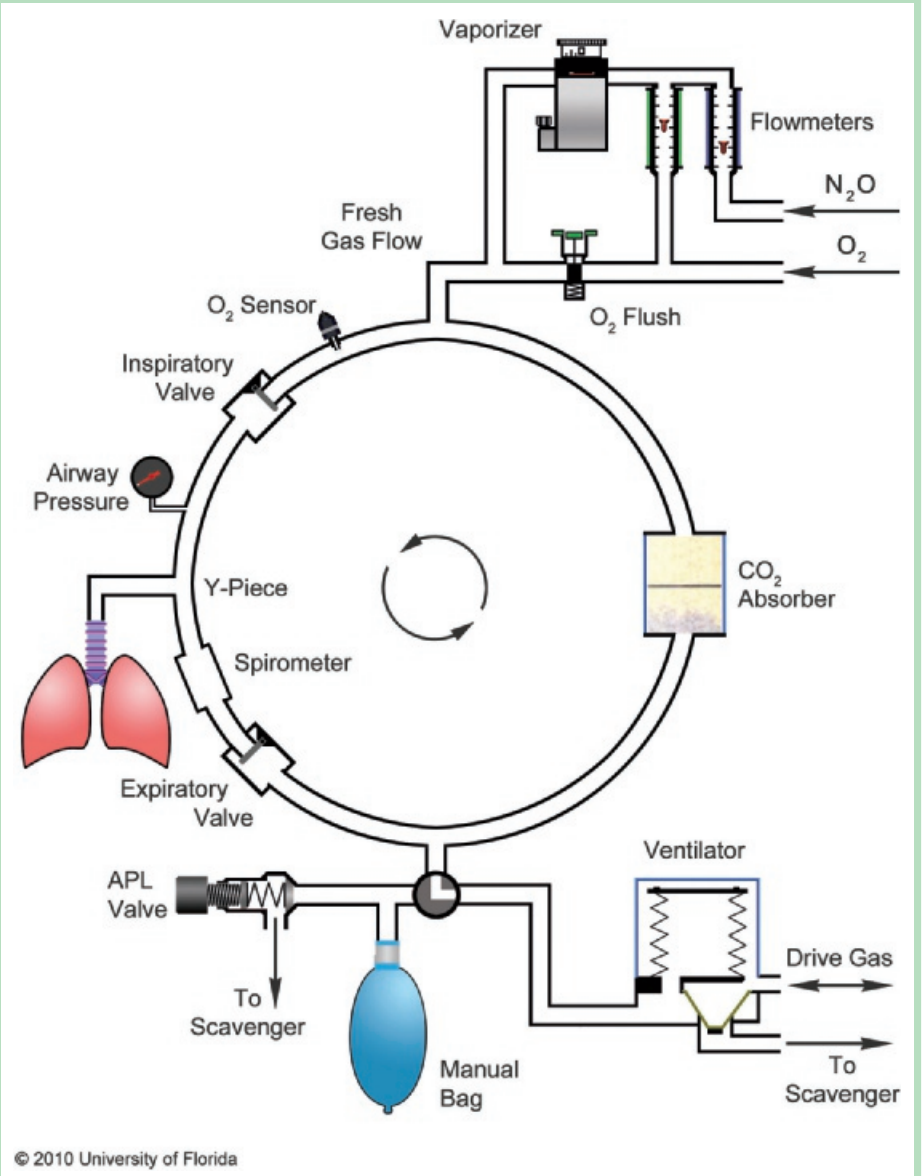
4) **Breathing system**
   a) **Traditional circle breathing system**
      i) Circle system prevents rebreathing of carbon dioxide by use of CO_2 absorbents but allows partial rebreathing of other exhaled gases.
      ii) Unidirectional valves are used to ensure that gas flows in the proper direction.
         (1) The inspiratory valve prevents backflow through the inspiratory limb during expiration, and the expiratory valve prevents backflow through the expiratory limb during inspiration.
      iii) Types of circle systems
         (1) Semiopen
            (a) No rebreathing and requires a very high flow of fresh gas
         (2) Semiclosed
            (a) Associated with rebreathing of gases and is the most commonly used system in the United States
         (3) Closed
            (a) The inflow gas exactly matches that being consumed by the patient.
            (b) There is complete rebreathing of exhaled gases after the absorption of CO_2.
            (c) No excess gas flows into the scavenging system.



The circle system prevents rebreathing of CO_2 but allows partial rebreathing of other exhaled gases.

      iv) The circle system consists of seven components (**Fig. 17-2**):
         (1) Fresh gas inflow source
         (2) Inspiratory and expiratory unidirectional valves
         (3) Inspiratory and expiratory corrugated tubing
         (4) Y-piece connector
         (5) Overflow or pop-off valve; (APL—adjustable pressure limiting) valve
         (6) Reservoir bag
         (7) Canister with CO_2 absorbent
5) **Scavenging system**
   a) **Classification of scavenging systems**
      i) Active scavenging systems
         (1) Vacuum source, vacuum hose, and vacuum control
         (2) The negative pressure vacuum source actively pulls the excess gas from the anesthesia machine, transports it through the hospital's gas disposal system, and typically releases it through the roof.
      ii) Passive scavenging system
         (1) Waste gas evacuation hose
         (2) Does not require a vacuum source
         (3) Relies on a slight positive pressure difference to push waste gas through the evacuation hose
6) **Preanesthesia machine checkout**
   a) In 1993, a preanesthesia checkout was developed and widely accepted.
   b) Anesthesia delivery systems have evolved (most notably with the addition of piston ventilators—**Fig. 17-3**) and the 1993 PreAnesthesia Checkout is not applicable to all machines on the market.
   c) In 2008, the ASA Committee on Equipment and Facilities made recommendations for new checkout procedures.
      i) Their document was meant to be a template for developing checkout procedures for individual machines and practice settings.

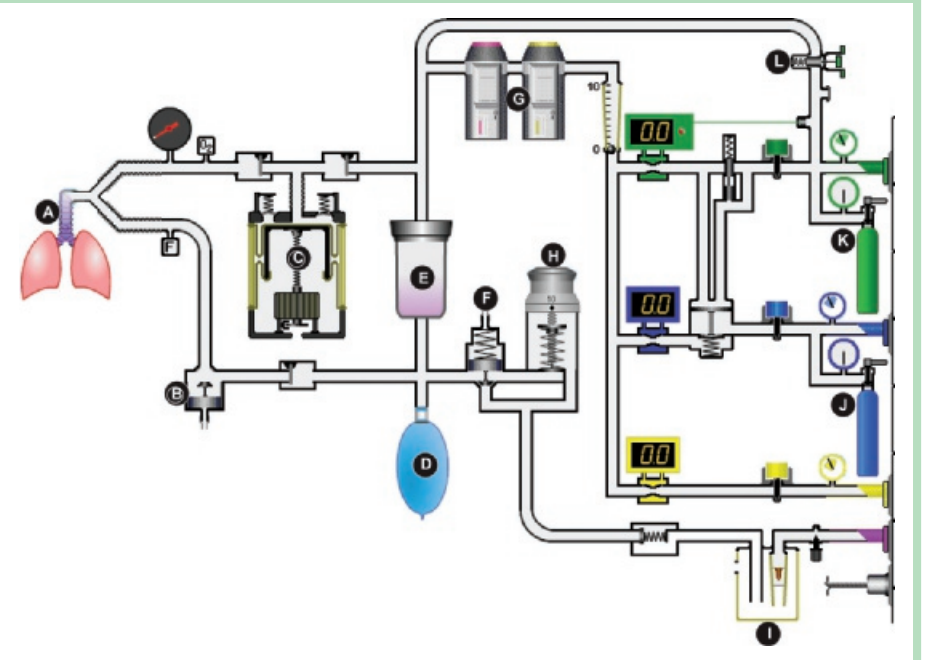
FIGURE 17-2 The Circle System with Relevant Parts Labeled



The scavenging system is not shown. With permission from Dr. Lampotang and Dr. Lizdas. Copyright © 2010 University of Florida.

d) **Checkout steps**
   i) Verify auxiliary oxygen cylinder and self-inflating manual ventilation device are available and functioning.
   ii) Verify patient suction is adequate to clear the airway.
   iii) Turn on the anesthesia delivery system and confirm that AC power is available.

FIGURE 17-3 A Typical Layout for an Anesthesia Workstation With a Piston Ventilator



(A) Anesthesia breathing circuit, **(B)** active exhalation/peep valve, **(C)** piston ventilator, **(D)** reservoir bag, **(E)** CO_2 absorber, **(F)** manual/automatic ventilation selection actuator, **(G)** anesthetic vaporizers, **(H)** APL valve, **(I)** bagless active scavenger system, **(J)** nitrous oxide cylinder, **(K)** oxygen cylinder, **(L)** oxygen flush valve. Adapted with permission from the virtual Fabius GS simulation—https://simulation.health.ufl.edu/

iv) Verify availability of required monitors and check alarms.
v) Verify that pressure is adequate on the spare oxygen cylinder mounted on the anesthesia machine.
vi) Verify that piped gas pressures are ≥50 psi.
vii) Verify that vaporizers are adequately filled and that the filler ports are tightly closed.
viii) Verify that there are no leaks in the gas supply lines between the flowmeters and the common gas outlet.
ix) Test scavenging system function.
x) Calibrate or verify calibration of the oxygen monitor and check the operation of the low oxygen alarm.
xi) Verify CO_2 absorbent is not exhausted.
xii) Breathing system pressure and leak testing
xiii) Verify that gas flows properly through the breathing circuit during both inspiration and exhalation.
xiv) Document completion of checkout procedures.
xv) Confirm ventilator settings, and evaluate readiness to deliver anesthesia care (anesthesia time out).

CHAPTER SUMMARY FOR ANESTHESIA MACHINES

Definition	The anesthesia machine is a vital component to the safe and reliable delivery of anesthesia and provides oxygenation, ventilation, and anesthetic gases to the patient. The anesthesia machine can be divided into four component subsystems.
High-Pressure System	Receives connections from the hospital pipeline gas supply and includes the cylinder gas supplies
Low-Pressure System	Blends oxygen and anesthetic gases according to the control settings
Breathing System	Delivers the fresh gas mixture from the low-pressure system to the patient and provides ventilation
Scavenging System	Collects excess gas from the breathing system and delivers it to the waste gas evacuation system
Anesthesia Machine Checkout	The ASA Committee on Equipment and Facilities made new recommendations for anesthesia machine check out practices prior to administration of anesthesia. These should be reviewed and adopted to individual machines and practice settings.

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18 Anesthesia Breathing Circuits

Vivekanand Kulkarni • Romy Yun

The anesthesia breathing circuit connects a system of tubes from the anesthesia machine to the respiratory system of the patient. This creates the environment within which respiratory gas exchange occurs. The circuit is vital for ventilation of the patient undergoing anesthesia and provides a conduit through which anesthetic gases can be administered.

1) **Overview**
   a) Most modern circuits incorporate 22 mm corrugated breathing tubes with one end (elbow) linked to the patient's airway via a mask/supraglottic airway (SGA)/endotracheal tube (ETT) and a fresh gas inlet (FGI) at the other.
   b) A spring-loaded pop-off valve/adjustable pressure-limiting (APL) valve and a reservoir breathing bag (RBB) are also included.
   c) With most circuits, there is an increase in *dead space*, with V_d/V_t increasing from 0.33 to 0.46 with an ETT and to 0.66 with a mask.
   d) The *resistance* to breathing depends on the presence of valves and the diameter of the tubing.
   e) The positions of the elements of these circuits determine their functional characteristics.
   f) When there is no CO_2 absorber present, they constitute nonrebreathing systems, which were classified by Mapleson into A, B, C, D, E, and F.
   g) When unidirectional valves and a CO_2 absorber are included, the resistance to breathing is greater, and these circuits are rebreathing systems.



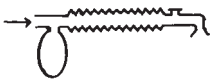
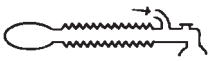
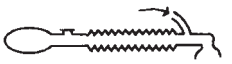
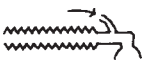
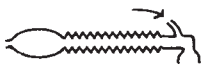
Incorporation of unidirectional valves and a CO_2 absorber into an anesthesia circuit allows rebreathing of some exhaled gases and are rebreathing systems.

2) **Nonrebreathing systems—Mapleson circuits (Fig. 18-1)**
   a) Nonrebreathing systems work by using fresh gas flow (FGF) to push the patient's exhaled gas down the expiratory limb of the circuit.
      i) The exhaled gas collects in the RBB, and some escapes through an expiratory (APL) valve.
         (1) The remaining exhaled gas is diluted by FGF.
      ii) The patient draws his or her next breath through the expiratory limb and RBB.
         (1) Some rebreathing of the previous breath may occur depending upon the degree of dilution (from FGF rate, patient's tidal volume [TV], and length of expiratory pause).
   b) Mapleson in 1954 classified the circuits into five groups depending on the position of the FGI, RBB, and APL in the circuit.
   c) The F system, as described by Willis, was later added to the original group of five.
   d) Gas is expelled through the APL.
      i) In the A, B, and C classes, expired gas leaves the circuit close to the patient.
      ii) The D, E, and F are T-piece systems, and expired gas leaves the circuit away from the patient.
   e) Since there is no CO_2 absorber, there is always potential for rebreathing some amount of exhaled gases using Mapleson circuits.
      i) The amount of rebreathing depends on the following:



Circuits classified as nonrebreathing systems are not intended to allow rebreathing of exhaled gases, but rebreathing can still occur.

FIGURE 18-1 Mapleson Circuits

	Spontaneous	Controlled
A 	1 × MV	>3 × MV
B 	2 × MV	2.5 × MV
C 	2 × MV	2.5 × MV
D 	2–3 × MV	1–2 × MV
E 	2–3 × MV	3 × MV
F 	2–3 × MV	3 × MV

The FGF necessary to prevent rebreathing is represented as a function of the patient's minute ventilation. MV, minute ventilation. Adapted with permission from from Dorsch JA, Dorsch SE. *Understanding Anesthesia Equipment.* 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008.

(1) The circuit elements and their position
(2) Minute ventilation (MV)
(3) The pattern of breathing (TV, respiratory rate, peak inspiratory flow rate, expiratory pause duration, and I:E ratio)
(4) The FGF
(a) The FGF needed to prevent significant rebreathing depends also on the mode of ventilation—spontaneous or controlled.

ii) The efficiency of each circuit with respect to FGF needed to prevent rebreathing is as follows:
(1) During spontaneous ventilation A > D,F,E > C,B
(2) During controlled ventilation D,F,E > B,C > A (**Table 18-1**)

f) **Mapleson circuits**

i) Mapleson A—Magill circuit
(1) It has a single breathing tube with the FGI and RBB close to the machine, while the APL is close to the patient connector or elbow.
(2) A drawback of Mapleson A is that waste gas is released close to the patient, which makes scavenging difficult.
(3) Spontaneous ventilation (**Fig. 18-2**).
(a) During expiration, the corrugated breathing tube is filled first by dead space gas, which does not contain CO_2, followed by alveolar gas from the patient end of the circuit and simultaneously by FGF from the FGI.

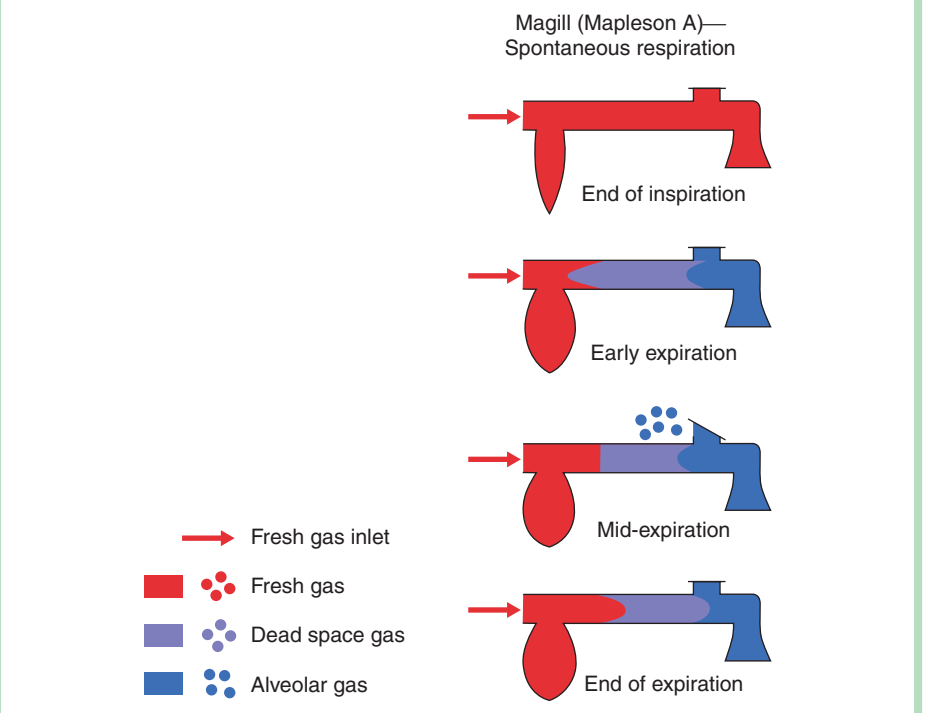
TABLE 18-1 The Advantages and Disadvantages of Mapleson Circuits

Advantages of Mapleson Circuits	• Inexpensive; lightweight; simple to use, disassemble, and sterilize. • Rugged, no moving parts except APL, and low resistance to flow. • Changes in FGF result in rapid changes in inspiratory gas concentrations. • Since there is no absorber, there are no toxic products of agent breakdown. • In coaxial systems, inspiratory gas is heated by the warm exhaled gas.
Disadvantages of Mapleson Circuits	• Higher flows are needed to prevent CO_2 rebreathing, hence more expensive. • Because of higher flows, there is loss of heat and humidity. • Scavenging is more difficult in some circuits, and environmental pollution is greater. • In some circuits, the APL is close to the patient and more difficult to access. • Assembly errors can cause significant CO_2 rebreathing.

FGF, fresh gas flow; APL, adjustable pressure-limiting valve.

(b) The flow from the FGI continues during the expiratory pause.
(c) If the FGF is high enough, pressure in the circuit rises, causing the APL to open and expel expired alveolar gas, which contains CO_2.
(d) During the next inspiration, the FGF and some dead space gas will be inhaled.

FIGURE 18-2 Mapleson A (Magill Circuit) During Spontaneous Ventilation MV



Courtesy of Dr. John Brock-Utne.

(e) When the FGF is above 70% of MV during spontaneous ventilation, rebreathing is prevented as long as breathing is regular.

(4) Controlled ventilation

(a) The inspiratory force is provided by the anesthesiologist who squeezes the RBB after partially closing the APL.

(b) During inspiration, therefore, some fresh gas is vented via the APL.

(c) During expiration, expired gas fills the RBB. This is mixed with the FGF during the expiratory pause, so that rebreathing CO_2-containing gas is inevitable.

(d) Now the system is inefficient because the FGF needs to be more than three times MV to prevent CO_2 buildup from rebreathing.

(e) The Miller modification of the Magill system keeps the APL closed during inspiration thus decreasing FGF requirements.

ii) Lack system (coaxial Mapleson A)

(1) The Lack coaxial system has two tubes.

(a) The outer tube is similar to that of the Mapleson A with FGI and RBB close to the machine end.

(b) The expired gases from the patient are, however, conducted by the inner tube to the machine end so that the APL is closer to the machine end.

(2) Functionally similar to the Mapleson A, the arrangement allows for easier scavenging and manipulation of the APL.

iii) Mapleson B and C systems

(1) In these systems, the APL and FGI are close to the patient connector.

(2) The RBB and corrugated tubing when present (Mapleson B) form a blind end where mixed expired and fresh gases collect.

(3) A FGF $> 2 \times$ MV is needed to prevent rebreathing in both spontaneous and controlled modes.

(4) The C system is also called Waters to and fro system without absorber.

(5) These systems are rarely used today.

iv) Mapleson D system

(1) This system is basically a T-tube system with the FGI close to the patient, forming one limb of the T-tube.

(2) Expired gases flow through the other limb of the T, which is a long corrugated tube with a reservoir bag and a pop-off valve.

(3) Spontaneous ventilation

(a) In expiration, the dead space gas, alveolar gas, and FGF mix in the corrugated tubing and the RBB.

(b) When the bag fills, the pressure in the system rises and the APL opens to vent the mixture.

(c) In the expiratory pause, the FGF continues to push the expired Alveolar gas toward the RBB and APL.

(d) During spontaneous breathing, $2 \times$ MV of FGF is needed to prevent rebreathing.

(e) It is less efficient than Mapleson A but more efficient than Mapleson B and C.

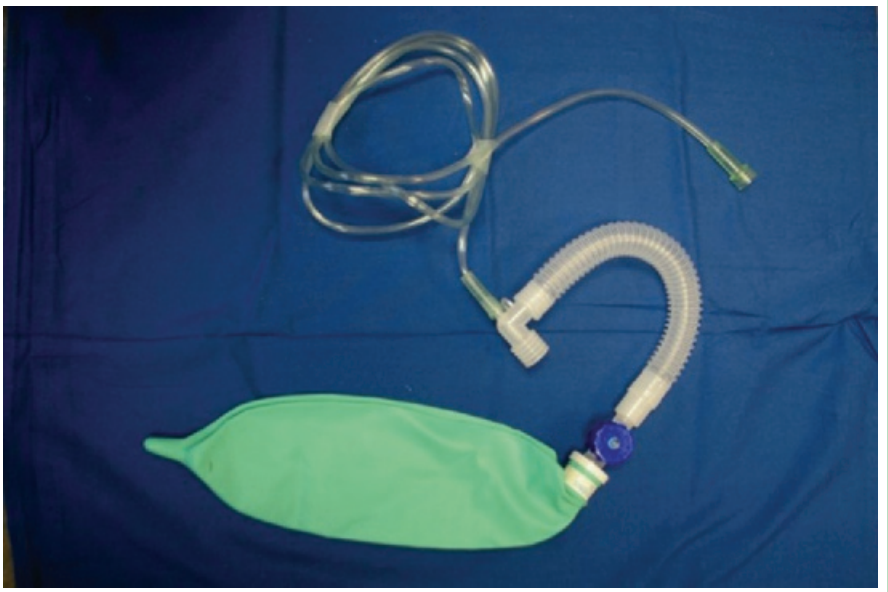
(4) Controlled ventilation

(a) On expiration, the corrugated tubing and RBB fill up with a mixture of FGF, dead space, and alveolar gas.

(b) In the expiratory pause, the FGF pushes the expired gas toward the RBB and APL.

(c) Compressing the RBB manually allows FGF in the corrugated tubing to enter the lungs, whereas the higher pressure opens the APL to vent the FGF + expired gas mixture from the RBB.

FIGURE 18-3 Mapleson D Used in Transporting Intubated Patients

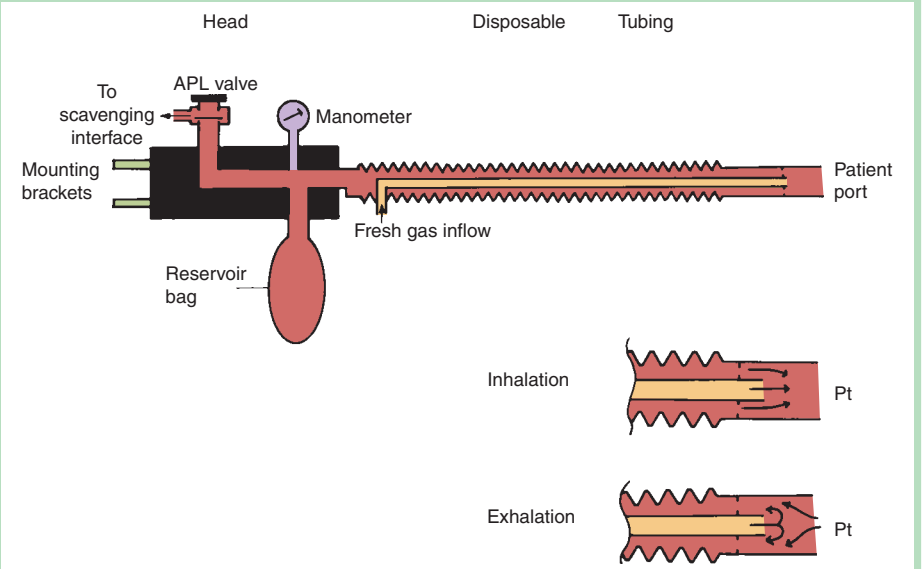


Courtesy of Dr. John Brock-Utne.

(d) During controlled ventilation, 1-2 × MV of FGF is needed to prevent rebreathing.
(e) The circuit is often used for manual ventilation during transport of patients (**Fig. 18-3**).

v) Bain system (coaxial Mapleson D)
(1) Fresh gas enters the inner small-bore tube and is delivered to the patient connection.
(2) Expired gases enter into the outer corrugated tubing to the RBB and APL close to the machine end (**Fig. 18-4**). They warm the fresh gas in the inner tube, thus conserving heat.
(a) Spontaneous ventilation
(i) During expiration, dead space and alveolar gas enter the outer corrugated tubing and RBB.
(ii) During the expiratory pause, FGF from the inner tube fills the outer tube, pushing the expired gas to the RBB and APL.
(iii) Keeping the FGF adequate (2.5-3 × MV) prevents rebreathing.
(b) Controlled ventilation
(i) During controlled ventilation, 70 mL/kg/min or 1-2 × MV prevents rebreathing.
(ii) For controlled ventilation, the reservoir bag can be removed for ventilator attachment.
(3) Pethick test
(a) If the inner tube that delivers FGF gets disconnected or kinked at the proximal end, the entire corrugated tubing becomes additional dead space, which is difficult to compensate for, leading to respiratory acidosis.

FIGURE 18-4 Bain Circuit



Adapted with permission from Dorsch JA, Dorsch SE. *Understanding Anesthesia Equipment*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008.

(i) Prevention by checking for the patency of the inner tube connection uses the Pethick test.
1. Occlude the patient end and close the APL valve.
2. Fill the circuit with the oxygen flush button, distending the RBB.
3. Release the patient end.
4. The resulting Venturi effect flattens the RBB confirming inner tube connection patency.

(4) CPAP system
(a) Occasionally, during one-lung ventilation, a modified Mapleson D system can be attached to the nonventilated lung and a secondary O_2 source.
(b) This provides CPAP to the nonventilated lung and decreases the shunt fraction, thus improving oxygenation.

vi) Mapleson E and F systems
(1) These are valveless T-piece systems, which have low resistance.
(2) Ayre's original T-piece system with additional expiratory limb of open corrugated tube (which acts as a reservoir) forms the E-system.
(3) Loss of moisture because of the high FGF can be significant.
(4) Partial occlusion of the expiratory limb opening (Mapleson E) or the RBB (Mapleson F) during manual ventilation can lead to high pressures.
(a) A pressure relief mechanism is necessary to prevent barotrauma.



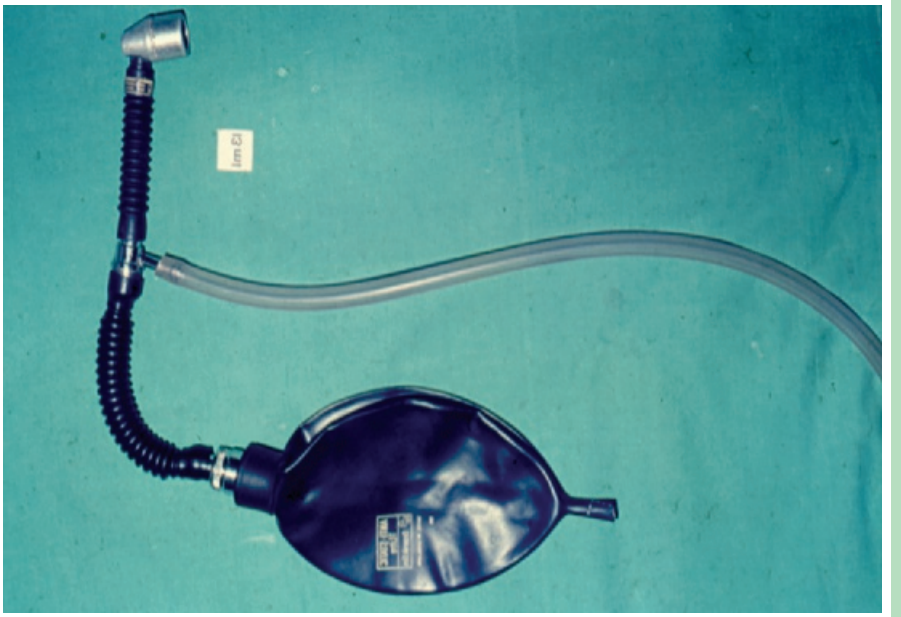
Efficiency, as measured by lowest flow rate required to avoid rebreathing of carbon dioxide, of Mapleson circuits varies during spontaneous vs controlled respirations according to the following:

Spontaneous respiration: A > D,F,E > C,B

Controlled respiration: D,F,E > B,C > A

This mnemonic for remembering would be helpful: Spont—A Dog Can Bite; Controlled—Dog Bites Can Ache

FIGURE 18-5 Mapleson F: The Jackson-Rees Circuit



Courtesy of Dr. John Brock-Utne.

(5) Spontaneous ventilation
  (a) The Mapleson E is similar to the D system.
  (b) During inspiration, no rebreathing occurs if there is no expiratory limb.
    (i) With an expiratory limb, however, rebreathing is avoided if the FGF is adequate.
      1. If the volume of the expiratory limb is less than the TV of the patient, air dilution can occur.
    (ii) A FGF of 2-3 × MV prevents rebreathing (**Fig. 18-5**).
(6) The Mapleson F
  (a) The Jackson-Rees modification has a RBB with an open end attached to the expiratory limb of the Mapleson E.
  (b) The addition of a bag acts as a reservoir to prevent dilution with air and allows manual ventilation when necessary.
  (c) FGF requirements are similar to that of the Mapleson E.

3) **Rebreathing systems**
  a) **Circle system**
    i) This is currently the most popular system in the United States (**Table 18-2**).
    ii) The system was first described by Brian Sword in 1926.
      (1) It allows low FGF.
      (2) It conserves heat and humidity.
      (3) It decreases pollution by allowing efficient scavenging.
    iii) The earlier problem of predicting inspired gas composition is no longer an issue because gas concentrations are sampled for measurement at the patient yoke (Y-connector).

TABLE 18-2 The Advantages and Disadvantages of Circle Systems

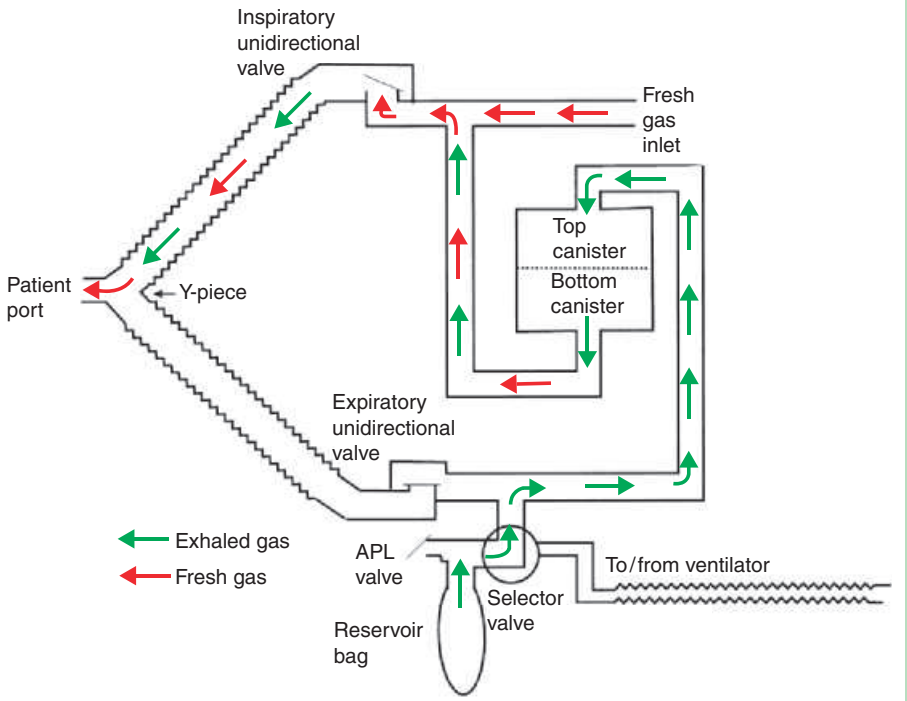
Advantages of circle systems	a. Lower gas flows are economical. b. Reduced environmental pollution. c. Conservation of heat and humidity. d. More stable anesthetic gas concentrations and less chance of barotrauma due to sudden rise in circuit pressure.
Disadvantages of circle systems	a. Greater attention required to prevent hypercarbia from exhaustion of the CO_2 absorber. b. Decreased ability to alter anesthetic concentrations rapidly with low flows. c. Accumulation of small quantities of undesirable compounds from breakdown by desiccated absorbent.



The circle system is the most popular system in the United States. It allows low FGF, conserves heat and humidity, and allows efficient scavenging.

iv) There is no further increase in dead space compared to other systems since mechanical dead space ends at the patient yoke where inspired and expired gases meet and where gas can be sampled.
v) The resistance in the circle is <3 cm of water.
vi) Rebreathing of CO_2 is prevented by absorption of CO_2 from the exhaled gas using the carbon dioxide absorber.
   (1) The position of components of the circle is also important in this respect.
vii) Classical circuit configuration (**Fig. 18-6**)
   (1) Vaporizer is outside the circle.
   (2) Unidirectional valves lie between the patient yoke and the RBB in both inspiratory and expiratory limbs.
   (3) Two additional requirements are that the FGI, APL, and RBB should all be in between the two valves and not on the patient side of the circle.
   (4) Spontaneous ventilation
      (a) Inspiration
         (i) The unidirectional expiratory valve closes.
         (ii) Gas flows to the patient from the RBB (via the absorber) and the FGI through the inspiratory limb and the open unidirectional inspiratory valve.
      (b) Expiration
         (i) The unidirectional inspiratory valve closes.
         (ii) Expired gas flows through the open unidirectional expiratory valve in the expiratory limb to the RBB and to the CO_2 absorber.
         (iii) The APL is positioned just before the CO_2 absorber in the expiratory limb.
         (iv) When flows are high, CO_2-rich expired gas is the first to spill out of the circle thus conserving the CO_2 absorber.
   (5) Manual ventilation
      (a) Inspiration
         (i) Excess gases are vented through the partially open APL valve during inspiration.
         (ii) The gas flowing through the absorber to the patient will be a mixture of FGF and expired gases.
         (iii) The composition of the mixture depends on the FGF.
      (b) Expiration
         (i) Exhaled gases flow into the RBB.
         (ii) There may be some retrograde flow of fresh gas through the absorber depending on FGF rate.

FIGURE 18-6 Classic Circle System



Reused with permission from Dorsch JA, Dorsch SE. *Understanding Anesthesia Equipment.* 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008:250.

c) Mechanical ventilation
  i) Inspiration
    (1) During inspiration, gas flows from the ventilator through the absorber and unidirectional inspiratory valve to the patient.
    (2) The gas in the bellows of the ventilator would be a mix of FGF and exhaled gas that has been scrubbed of CO_2.
  ii) Expiration
    (1) During expiration, exhaled gases flow into the bellows where they will mix with FGF flowing retrograde through the absorber.
    (2) Excess gases spill through the APL in the latter part of expiration.

viii) Circle system function
  (1) During the initial 5-10 minutes of use, large amounts of inhalation agent and nitrous oxide are taken up by the patient.
  (2) The circuit also contains nitrogen from the lungs of the patient.
    (a) Dilution of the anesthetic agents is decreased by using high flows during this period.
    (b) When high flows are used, the concentration of inspired agent is very close to vaporizer settings.
    (c) The use of high flows for prolonged periods is uneconomical, increases environmental pollution, and does not conserve heat and humidity.



The use of high gas flows for prolonged periods is uneconomical and does not conserve airway heat and humidity.

ix) Use of low flows in the circle system
  (1) When the circle system has stabilized, the FGF can be decreased to 1.5-2 L/min.
  (2) The inspired gases are then made up of the FGF and the expired gas cleared of CO_2 by the absorber, but also with a lower O_2 concentration.
    (a) Because of dilution, higher FGF O_2 concentrations are needed to keep inspired O_2 concentrations above 30%.
    (b) Changes of anesthetic concentration when desired will need higher flows to achieve a more rapid change.
      (i) Advantages of low flows include economy, decreased environmental pollution, and conservation of heat and humidity.
      (ii) Disadvantages include greater use of the absorber and possible accumulation of undesired breakdown products from the absorber.



The use of high flow for a prolonged period in a circle system does not conserve heat and humidity and leads to unnecessary environmental pollution.

x) Closed circle system
  (1) In this system, the APL is completely closed, and the minimum FGF flow should be equivalent to the oxygen consumption (3.5 mL/kg/min for adults).

xi) CO_2 absorber
  (1) Soda lime and Baralyme are the most commonly used absorbers.
    (a) Small amounts of silica or kieselguhr are added for hardening to reduce dust formation.
    (b) The absorptive efficiency is inversely related to hardness.
    (c) Absorptive surface area depends on granule size.
      (i) Smaller granules increase the absorptive surface but also increase resistance to flow of gas, while larger granules decrease the absorptive surface and decrease resistance leading to channeling of gas flow.
      (ii) The usual mesh size is therefore 4-8 mesh (1/4-1/8 in).
    (d) Up to 26 L of CO_2 can be absorbed by 100 g of absorbent.
    (e) Composition of soda lime: NaOH 4%; KOH 1%; H_2O 15%; 80% $Ca(OH)_2$.
    (f) Composition of Baralyme: $Ba(OH)_2$ 20%; $Ca(OH)_2$ 80%; plus KOH indicator.
    (g) The moisture or water content of the absorbent forms carbonic acid with CO_2, which reacts with alkali forming carbonate and generating heat.
    (h) $H_2O + CO_2 \Leftrightarrow H_2CO_3 \Leftrightarrow H^+ + HCO_3^-$
    $NaOH + H_2CO_3 \Leftrightarrow NaHCO_3 + H_2O$
    $2NaHCO_3 + Ca(OH)_2 \Leftrightarrow 2\ NaOH + CaCO_3 + H_2O$
  (2) Amsorb is another absorber. It contains $Ca(OH)_2$ and $CaCl_2$. The hardening agents are $CaSO_4$ and polyvinylpyrrolidine.
    (a) Since it does not contain NaOH or KOH, there is no CO or Compound A production.
    (b) It is more expensive, and the CO_2 absorption capacity is less.
  (3) Indicators
    a) These are pH sensitive and added to the granules to show when they are exhausted.
      (i) Phenolphthalein: White when fresh and pink when exhausted
      (ii) Ethyl violet: White when fresh and purple when exhausted
      (iii) Clayton yellow: Red when fresh and yellow when exhausted
  (4) Interactions with anesthetic agents
    (a) Carbon monoxide (CO)
      (i) When the absorbents become dry and desiccated, they can react with anesthetic agents containing the CHF_2-O functional group resulting in carbon monoxide production.

1. CO production can be significant with soda lime and Baralyme leading to carboxyhemoglobin concentrations of up to 30%.
2. CO production is increased by dehydrated absorbent, higher temperatures, lower FGF, and higher anesthetic agent concentration.
3. CO production is also dependent on anesthetic agent.
   a. Desflurane > isoflurane > halothane = sevoflurane
4. CO production is dependent on absorbent.
   a. Baralyme > soda lime

(ii) The production of CO can be decreased by decreasing the drying of absorbent by shutting off gas flow after cases, changing the absorbent if gas flow had not been shut off, rehydrating the absorbent, and using absorbents such as Amsorb that do not contain NaOH and KOH.



Desiccated absorbents, such as may occur with soda lime/Baralyme if the FGF is not turned off overnight, may lead to carbon monoxide production.

(b) Other Breakdown compounds

(i) Compound A

1. Sevoflurane reacts with dry absorbent to produce an olefin compound called Compound A (fluoromethyl-2–2-difluoro-1-trifluoromethyl vinyl ether).
   a. The production is worse in dehydrated Baralyme with higher concentrations of sevoflurane, low FGF, and higher absorbent temperatures.
   b. The concentration is low and is not known to cause toxic effects in humans.

(ii) Other compounds produced in small quantities include acetone, methane, hydrogen, and ethanol.



CO production is increased by dehydrated absorbent, higher temperatures, lower FGF, and higher anesthetic agent concentration.

(c) Fire

(i) There is a small risk of fire within the absorber and breathing circuit with high temperatures and dry absorbents when sevoflurane is used.

CHAPTER SUMMARY FOR ANESTHESIA BREATHING CIRCUITS

Overview	The anesthesia breathing circuit creates a local environment surrounding the patient within which respiratory gas exchange occurs.
Nonrebreathing Systems	Nonrebreathing systems do not have the ability to absorb exhaled CO_2 gas There are 6 main types of Mapleson circuits: A, B, C, D, E, and F. Each differs based on the position of the FGI, RBB, and APL valve in the circuit The Bain circuit is a modification of the Mapleson D.
Rebreathing Systems	Rebreathing systems incorporate unidirectional valves and CO_2 absorbent to allow rebreathing of some exhaled gases. The circle system is currently the most popular rebreathing system used in the United States. It has methods for both spontaneous and manual ventilation.

RBB, reservoir breathing bag; APL, adjustable pressure-limiting valve; FGI, fresh gas inflow.

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19 Mechanical Ventilation

Scott M. Ahlbrand

Mechanical ventilation is considered by some to be one of the most influential discoveries of modern medicine. Defined as a method to mechanically assist or replace spontaneous breathing by the application of positive airway pressure, mechanical ventilation allows critically ill patients to survive surgeries of greater complexity. This artificial means of lung ventilation has come a long way since its inception in 1550. Modern mechanical ventilators are subtly tuned to each patient's ventilatory needs.

1) **Indications for mechanical ventilation**
   a) **Respiratory failure or inability of the patient to maintain spontaneous ventilation (SV)**
   b) Hypoventilation
      i) Respiratory: Reduced alveolar ventilation whereby $PaCO_2 > 50$ mm Hg is produced
      ii) Neurologic: Central causes from the brain, such as the following:
         (1) Oversedation
         (2) Narcosis secondary to elevated PCO_2 levels
         (3) Ischemic or hemorrhagic stroke
         (4) Any other general cause of brain injury
         (5) Diseases of spine or spinal cord
            (a) Any injury to the spinal cord near levels C3, C4, or C5 is associated with the potential loss of diaphragmatic function.
            (b) An injury to the thoracic spine associated with loss of intercostal muscle function and associated weakness.
            (c) Injury to peripheral nerves can also be associated with ventilatory failure as is the case when phrenic nerve loss occurs following certain surgical procedures.
            (d) Additionally, any lower motor neuron disease such as Guillain-Barre, poliomyelitis, or amyotrophic lateral sclerosis can contribute to muscle weakness and impending ventilatory failure.
      iii) Muscular causes of ventilatory failure are less common but no less life threatening.
         (1) Autoimmune diseases such as myasthenia gravis can create a state of increasing muscle weakness resulting in inability to expire CO_2.
         (2) Other entities such as steroid-induced myopathy or protein malnutrition, more commonly seen in critically ill patients, are all associated with an increased likelihood of ventilatory failure.
      iv) Anatomic causes of ventilatory failure
         (1) Can occur almost anywhere throughout the chest or the thorax
         (2) Damage to structures of the chest wall such as rib fractures or flail chest
         (3) Any clinical situation that increases transthoracic pressure
         (4) Severe obesity, abdominal hypertension, or the presence of a restrictive dressing (abdominal binders)
      v) Disorders of the pleura
         (1) Pleural effusions, pneumothoraces, and hemothoraces impair inspiration, thereby decreasing minute ventilation (MV).

vi) Upper airway obstruction—Laryngeal edema, bronchospasm, laryngospasm, or the presence of a foreign body can cause hypercapnia putting patients at increased risk for ventilatory failure.

vii) Gas exchange abnormalities can cause significant ventilatory failure.

(1) Most commonly caused by ventilation/perfusion (V/Q) mismatch.

(2) "*V/Q*" mismatch results from inequality between the amount of ventilation and the amount of perfusion in a specific part of the lung.

(a) This can result from many clinical situations including an increase in alveolar dead space where a decrease in the functional residual capacity (FRC) is produced.

(b) Any kind of injury to the lung as would occur from hyperinflation, pulmonary contusion, acute lung injury (ALI), or a pulmonary embolism.

viii) It is important to realize that ventilatory failure can occur in the presence of normal oxygenation. Therefore, adequate ventilation cannot be assessed solely by pulse oximetry.



Ventilatory failure can occur in the presence of normal oxygenation and cannot be assessed solely by pulse oximetry.

c) **Failure to oxygenate (hypoxic respiratory failure)**

i) Defined as any impairment in the ability to oxygenate, resulting in $PO_2 < 60$ mm Hg.

ii) The two main causes of hypoxic respiratory failure are diffusion defects and V/Q mismatching.

(1) Diffusion defects

(a) Any process that thickens the alveolar membrane where gas exchange occurs.

(b) Thickened alveolar membranes can be the result of pulmonary fibrosis, where an abnormal and excessive amount of fibrotic tissue is deposited in the pulmonary interstitium.

(c) Thickened alveolar membranes can also be the result of the accumulation of fluid in the area of gas exchange, as in the case of pulmonary edema.

(2) Ventilation/perfusion (V/Q) mismatching

(a) Can result in hypercapnic or hypoxic respiratory failure.

(b) One can think of *V/Q* mismatch as involving a spectrum of gas exchange abnormalities.

(c) At one end is pure dead space ventilation, where alveoli are ventilated but not perfused.



Dead space ventilation occurs when alveoli are ventilated but not perfused.

(d) Dead space ventilation can be divided into anatomic or physiologic.

(i) Increased anatomic dead space

1. An example is rapid, shallow breathing.
2. Since the conducting airways typically occupy 150 mL or 1-2 mL/kg, when one takes breaths of 150-200 mL, these small tidal volumes (TVs) do not reach the alveoli. This leads to decreased gas exchange and results in hypercapnia and hypoxia.

(ii) Pathologic dead space

1. An example would be a pulmonary embolus (PE).
2. In the case of a PE, there iss no blood flow to an area or normal ventilated lung.



Shunting occurs when alveoli are normally perfused but are not ventilated.

d) At the other end of the spectrum is shunt.

i) An example is alveoli that are normally perfused, but are not ventilated.

ii) Shunt results in the admixture of well-oxygenated blood (that which went around the shunt) with deoxygenated blood (that which passed through the shunt).
(1) Dilutes the resultant partial pressure of oxygen and creating hypoxic blood
iii) Examples of shunt include acute respiratory distress syndrome (ARDS)/ALI, airway collapse, pneumonia, or pulmonary contusion.

2) **There are no absolute contraindications to mechanical ventilation; however, relative contraindications do exist.**
3) **Modes of ventilation (Fig. 19-1)**
a) **There are two primary modes of noninvasive positive-pressure ventilation (NPPV) and three primary modes of invasive positive-pressure ventilation (IPPV) (Table X-1).**
b) **Noninvasive positive-pressure ventilation**
i) The application of positive airway pressure using a mechanical ventilator, without an endotracheal tube
ii) Modes of NPPV
(1) Bilevel positive airway pressure (BiPAP)
(a) A noninvasive form of positive-pressure ventilation that is used to increase oxygenation by providing high-flow positive airway pressure
(b) Cycles between high positive pressure and a lower positive airway pressure depending on its timing in the respiratory cycle

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Pos-top ventilation of the surgical Patient, Chapter 131

FIGURE 19-1 Modes of Ventilation.

Mode	TV	RR	PIP	PEEP
PSV	*varies*	*varies*	*varies*	set
VCV	set	set	*varies*	set
PCV	*varies*	set	set	set
APRV	*varies*	set	set	set
PRVC	set	set	*varies*	set

The mode of ventilation will dictate what parameters are set, and what are variable. These settings will be specific to their clinical indication. Many different modes of ventilation can be utilized to achieve the same goal.

(2) Continuous positive airway pressure (CPAP)
(a) A noninvasive ventilation strategy to overcome airway obstruction and stimulate normal respirations.
(b) Continuous flow pressure is applied throughout the respiratory cycle.
(c) Commonly used as a treatment for obstructive sleep apnea.



Consider NPPV in cases of reversible respiratory failure, such as a COPD exacerbation and cardiogenic pulmonary edema.

iii) Indications for NPPV[1,2]
(1) Any condition where reversible respiratory failure is present and there is concern for alveolar collapse
(2) Examples of clinical situations include
(a) An exacerbation of congestive heart failure
(b) Obstructive sleep apnea
(c) Asthma
(d) COPD exacerbation

iv) Advantages of NPPV[1]
(1) Reduced cost
(2) Avoidance of intubation-related trauma
(3) Shorter duration of invasive mechanical ventilation
(4) Enhanced patient comfort
(5) Shorter hospital stay
(6) Decreased incidence of nosocomial pneumonias



Invasive positive-pressure ventilation (IPPV) applies positive airway pressure through an endotracheal tube using a mechanical ventilator and is indicated when mechanical ventilation is required and NPPV cannot be used.

v) Disadvantages of NPPV[1]
(1) Difficult to tolerate
(a) 20% intolerance rate in patients who are started on NPPV
(b) May be due to a preexisting anxiety disorder or an uncomfortable fit on the face
(2) Unsuitable for patients with neuromuscular disease
(a) These patients may lack the strength and endurance to expire against the positive pressure.

c) **Invasive positive-pressure ventilation (IPPV)**
i) The application of positive airway pressure using a mechanical ventilator, with an endotracheal tube
ii) Indications for IPPV
(1) When mechanical ventilation is required, but NPPV is not tolerated, indicated, or adequate for the patient.
iii) Modes of IPPV
(1) Volume-cycled ventilation (VCV)
(a) The ventilator delivers a set TV irrespective of the pressure that it generates.
(b) Can be divided into assisted, controlled, assist-controlled, and intermittent mechanical ventilation (IMV).
(c) Assisted VCV
(i) Mode of ventilation where the ventilator is triggered by an initiated breath from the patient
(ii) The ventilator does not generate a breath without the patient's effort.
(d) Controlled VCV
(i) Requires the operator to assign a set MV by determining an appropriate TV and respiratory rate (RR)
(ii) This mode generates breaths whether the patient is spontaneously breathing or not.
(e) Assist-controlled volume (ACV)-cycled ventilation
(i) Combination of ACV and VCV

(ii) The ventilator will generate breaths whenever triggered by the patient's spontaneous breathing.
(iii) Will also deliver set TVs to make sure the predetermined MV is achieved.

(f) Intermittent Mandatory Ventilation (IMV)
(i) Essentially ACV + SV
(ii) The ventilator will allow SV, assist with spontaneous breaths, and generate its own breaths.
(iii) Advantage of IMV
1. When SV occurs, muscle atrophy is reduced.
2. Risk of lung hyperinflation and the generation of intrinsic positive end-expiratory pressure (PEEP) is minimized.
(iv) Disadvantage of IMV
1. Spontaneously breathing through a breathing circuit and mechanical ventilator is difficult.
2. A patient typically requires a pressure support of 10 cm H_2O to overcome that resistance.
3. Due to the pressure support required, IMV becomes much more similar to ACV, as there is no longer any truly SV occurring.

(2) Pressure-cycled ventilation (PCV)
(a) Indicates that the ventilator generates a constant pressure that is applied to inflate the lungs
(i) This principle is important to remember anytime someone is on PCV, as one can see variability in the inflation volumes depending on the patient's lung compliance.



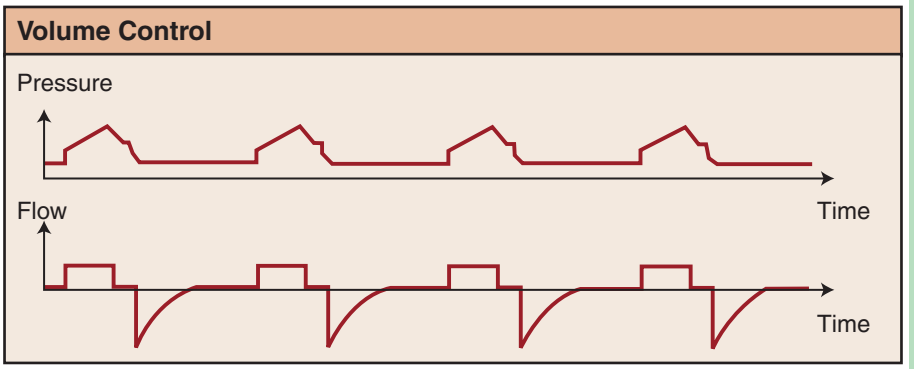
PCV generates a constant pressure to inflate the lungs, but the delivered tidal volume varies depending upon lung mechanics.

(b) Advantages of PCV
(i) Generates a decelerating inspiratory flow pattern
1. This effectively decreases the peak inspiratory pressure (PIP) and can thereby improve gas exchange.
(c) Disadvantages of PCV
(i) Changes in lung mechanics will change the resultant inflation volumes
1. Therefore, it is imperative that the delivered TVs are monitored to prevent the occurrence of volutrauma.

(3) Pressure-regulated volume control ventilation (PRVC) (**Fig. 19-2**)
(a) Combines the benefits of VCV and PCV.
(b) Ventilator estimates volume:pressure relationship per breath.
(c) Adjusts level of pressure to deliver "targeted" volume.
(i) Constant pressure during breath
(ii) Variable pressure from breath to breath

(4) Pressure support ventilation (PSV) (**Fig. 19-3**)
(a) The spontaneously ventilating patient has each TV "supported" with a set pressure.
(b) When the patient initiates a spontaneous breath, the ventilator is triggered to apply a positive pressure and augment each TV.
(c) Pressure support settings
(i) Typically range from 5 to 20 cm H_2O depending on the patient's needs.
(d) Common indications for PSV
(i) Weaning a patient from ventilatory support can be particularly beneficial when a patient has a minimal oxygen requirement but may be deconditioned and require additional pressure support for adequate ventilation.

FIGURE 19-2 Pressure-regulated volume control ventilation (PRVC) is a Form of Dual-controlled Ventilation in Which the Ventilator Attempts to Achieve an Appropriate Tidal Volume Target at the Lowest Possible Airway Pressure



(ii) The amount of pressure support can be reduced on a regular basis until the patient is able to ventilate without the need for pressure support and is possibly ready for discontinuation of mechanical ventilation.

(iii) Contraindication to PSV

1. A patient receiving neuromuscular blocking agents
   a. Since PSV has no backup set RR, ventilating a paralyzed patient with this mode would be catastrophic.

(5) Airway pressure release ventilation (APRV) (**Fig. 19-4**)

(a) CPAP with intermittent release phase

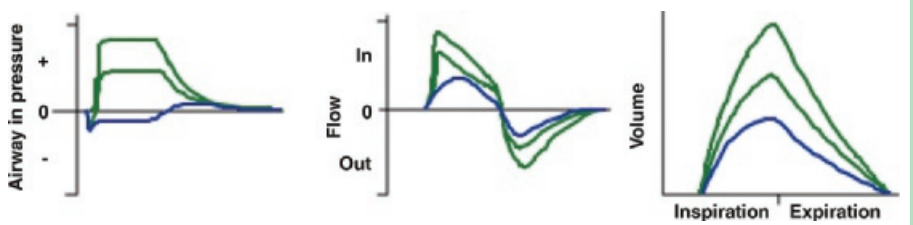
(b) Integrated SV

(c) It is essentially pressure control ventilation if the patient is apneic.

(d) Associated with:

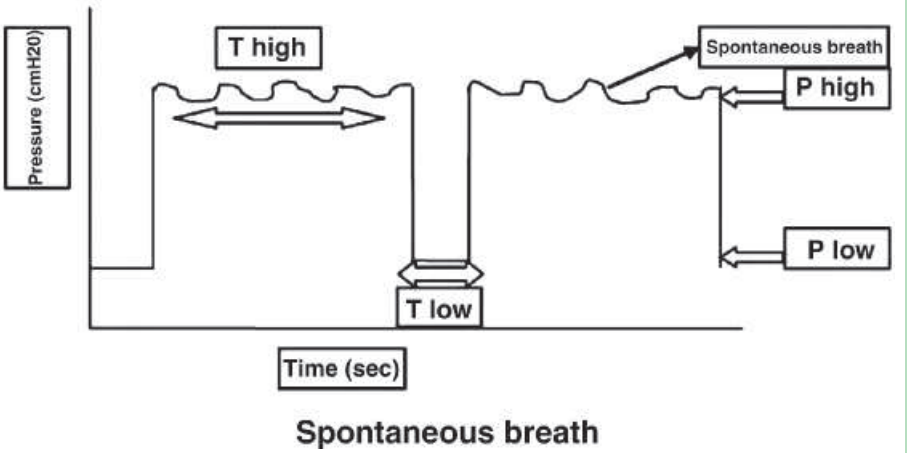
(i) Improved oxygenation

FIGURE 19-3 Pressure Support Ventilation.



Once the patient generates a negative pressure, or trigger, the ventilator provides a sufficient flow to reach and maintain a set airway pressure for the duration of the patient's spontaneous respiratory effort.
Data from MacIntyre NR. Pressure support ventilation: effects on ventilatory reflexes and ventilatory-muscle workloads. *Respir Care*. 1987;32:447-457.

FIGURE 19-4 Pressure-Time Curve for Airway Pressure Release Ventilation. P high, high CPAP; P low, low CPAP; T high, duration of P high; T low, release period or the duration of P low. ***APRV is a*** form of bilevel mechanical ventilation where spontaneous breathing occurs at an elevated CPAP level (P high) with intermittent timed pressure releases (P low).



Reprinted with permission from Daoud E. Airway pressure release ventilation. *Ann Thorac Med*. 2007;2(4):176-179.

(ii) Increased cardiac output
(iii) Less dead space when compared to conventional mechanical ventilation

4) **Ventilator settings**
   a) **Ventilators typically include a combination of settings, which may vary based on the patient's condition, lung compliance, and reason for mechanical ventilation.**
      i) The fraction of inspired oxygen (FiO_2)
      ii) Tidal volume
      iii) Peak airway pressure
      iv) Respiratory rate
      v) Inspiratory to expiratory ratio
      vi) Positive end-expiratory pressure
   b) **Fraction of inspired oxygen (FiO2)**
      i) The concentration of oxygen in the gas mixture with which the patient is ventilated.
      ii) Immediately following an endotracheal intubation and the initiation of mechanical ventilation, most patients should be placed on 100% inspired oxygen.
      iii) A FiO_2 of 1.0 should be used until adequate arterial oxygenation is documented
      iv) A short period of ventilation with a FiO_2 of 1.0 is not dangerous to the patient and offers some clinical advantages.[5]
         (1) Protects the patient against hypoxemia if unrecognized problems occur as a result of the endotracheal intubation.



PSV cannot be used to ventilate a paralyzed patient because the patient must trigger a breath. There is no backup set respiratory rate.

(2) Using the PaO_2 measured with a FiO_2 of 1.0, a clinician can more easily estimate the shunt fraction and calculate the next desired FiO_2.[6]
   (a) The degree of shunt while receiving a FiO_2 of 1.0 can be estimated by applying the rule "700 minus PaO_2."
   (b) For each difference of 100 mm Hg, the shunt fraction is 5%.
      (i) For example, if the PaO_2 is 500 mm Hg while receiving a FiO_2 of 1.0, then the shunt fraction is estimated at 10%.
      (ii) A shunt fraction of 25% should remind the clinician to consider the use of PEEP.
      (iii) A low PaO_2, despite a FiO_2 of 1.0, should prompt a search for complications related to endotracheal intubation and positive-pressure ventilation.
   (c) Etiology of hypoxemia (low PaO_2)
      (i) Mainstem bronchial intubation
      (ii) Pneumothorax
      (iii) Atelectasis
      (iv) Pneumonia
      (v) ARDS
      (vi) Congestive heart failure
      (vii) Hemorrhage



A shunt fraction of 25% or more should remind the clinician to consider the use of PEEP.

c) **Tidal volume**
   i) The volume of gas the ventilator will deliver to the patient with each breath.
   ii) For a patient without preexisting lung disease, 6-8 mL/kg is a commonly used TV for the assist-control mode.[7]
   iii) In the setting of ARDS, it is suggested that the lungs may function best, and volutrauma may be minimized, with a lower TV of 6 mL/kg in the assist-control mode.[8]
      (1) These lower volumes may lead to slight hypercarbia.
      (2) However, this elevated PCO_2 is typically recognized and accepted without correction, leading to the term permissive hypercapnia.



The use of a lung-protective ventilation strategy in intermediate-risk and high-risk patients undergoing major abdominal surgery is associated with improved clinical outcomes and reduced health care utilization.[7]

d) **Peak airway pressure**
   i) The pressure measured by the ventilator in the major airways—Strongly reflects airway resistance
   ii) As the TV increases, so too does the pressure required to drive that volume into the lung.
   iii) Persistent peak pressures higher than 45 cm H_2O are a risk factor for barotraumas and should be avoided.
      (1) Therefore, tidal volumes suggested by the above rules are only estimates.[7]
      (2) May need to be decreased in some patients to keep peak airways pressures <45 cm H_2O



Early application of and adherence to low tidal volume ventilation improves mortality, as well as other clinically important outcomes in patients with ARDS.[9]

e) **Plateau pressures**
   i) Pressures measured at the end of the inspiratory phase of a volume-cycled TV.
   ii) Most modern ventilators are programmed not to allow expiratory airflow for a set time, typically a half of a second.
   iii) The pressure measured to maintain this lack of expiratory airflow is the plateau pressure.
   iv) The plateau pressure should be monitored to prevent barotrauma in patients with ARDS.



Keep plateau pressures <30 cm H_2O to minimize barotraumas to the lungs.

(1) Barotrauma can be minimized when the plateau pressure is maintained <30 cm H_2O.[5]

f) **Respiratory rate**
   i) The frequency of breaths delivered per minute during mechanical ventilation.
   ii) Minute ventilation (MV), by definition, is set by the tidal volume (TV) and respiratory rate (RR) by an equation.
      (1) MV = RR × TV
   iii) Typical respiratory rates
      (1) Range from 8 to 16 breaths/min but really depend on the desired PCO_2.
         (a) Higher RR decreases PCO_2
         (b) Lower RR increases PCO_2
      (2) A situation where a higher-than-normal PCO_2 may be desired is in the case of weaning a patient from mechanical ventilation.
         (a) The higher PCO_2 is required for the patient's drive to breathe.
      (3) A situation when one may desire a lower-than-normal PCO_2 is in the case of elevated intracranial pressure.
         (a) The low PCO_2 causes cerebral vasoconstriction and a decrease in cerebral blood flow and a theoretical decrease in intracranial pressure.
         (b) If the respiratory rate is too high, the risks include:
            (i) Inadequate expiratory time
            (ii) Breath stacking, in which inadequate time is allotted for exhalation such that subsequent inhaled breaths are additive to the pulmonary volume and risk barotrauma.
            (iii) Development of intrinsic PEEP
            (iv) In order to minimize this risk, the I:E ratio can be adjusted to allow more expiratory time and completion of the expiratory phase.

g) **I:E ratio**
   i) Defined as inspiratory time + inspiratory pause time:expiratory time.
   ii) This ratio is usually set to 1:2 or 1:2.5 in an attempt to mimic the usual pattern of spontaneous breathing.
   iii) A higher I:E ratio is the equivalent to a longer inspiratory time.
      (1) This adjustment may improve oxygenation by increasing the mean airway pressure and allowing redistribution of gas from more compliant alveoli to less compliant alveoli.
      (2) However, this also increases the risk of breath stacking (leading to intrinsic PEEP) and barotrauma by reducing the expiratory time.[7]
      (3) It is important to keep in mind, however, that a higher *I:E* ratio is generally less well tolerated by the patient and typically necessitates a deeper level of sedation.
   iv) A lower I:E ratio is the equivalent to a longer expiratory time.
      (1) This can be particularly useful when obstructive pulmonary disease is present such as asthma or COPD.
      (2) It is also useful in settings where high minute ventilation is required, necessitating a higher-than-normal RR.
         (a) The risk of breath stacking must be minimized by optimizing the I:E ratio, often by reducing the ratio or increasing the "E" time.

h) **Positive end-expiratory pressure (PEEP)**
   i) Positive pressure applied at the end of the expiratory cycle
   ii) PEEP is most effective when used in patients with diffuse lung disease that results in a decrease in FRC.
   iii) PEEP works by increasing end-expired lung volume and preventing airspace closure at the end of expiration.

iv) Most patients who require mechanical ventilation may benefit from the application of 5 cm H_2O of PEEP.
   (1) This limits the atelectasis that frequently accompanies endotracheal intubation and mechanical ventilation in the supine position.
v) More importantly, PEEP can be utilized to permit lower levels of FiO_2, while preserving adequate oxygenation.
   (1) This is important in limiting lung injury that may result from prolonged exposure to high inspired oxygen concentrations ($FiO_2 > 0.6$).
   (2) Higher levels of PEEP, in the range of 5-15 cm H_2O, have been shown to improve oxygenation in more severe alveolar filling disorders such as cardiogenic pulmonary edema in ARDS.[8,10-12]
      (a) The mechanism behind this is likely redistribution of fluid from the alveoli to the interstitium and expanding collapsed alveoli.
vi) PEEP is not a benign mode of therapy and should have a well-defined indication.
   (1) In the hypovolemic patient, PEEP increases intrathoracic pressure and can impede venous return. This may result in hypotension and hemodynamic instability.
   (2) PEEP also increases intra-alveolar pressures and thus places patients at increased risk for barotrauma.
   (3) The addition of PEEP is typically justified when a PaO_2 of 60 mm Hg cannot be achieved with a FiO_2 of 0.6 or the estimated initial shunt fraction is >25%.[10,11]

5) **Sedation for mechanical ventilation (Fig. 19-5)**
   a) **Sedation is defined as depression of a patient's awareness to the environment and reduction of his or her responsiveness to external stimulation.**

FIGURE 19-5 Continuum of Depth of Sedation: Definition of General Anesthesia and Levels of Sedation/Analgesia.

	Minimal Sedation Anxiolysis	*Moderate Sedation/ Analgesia ("Conscious Sedation")*	*Deep Sedation/ Analgesia*	*General Anesthesia*
Responsiveness	Normal response to verbal stimulation	Purposeful** response to verbal or tactile stimulation	Purposeful** response following repeated or painful stimulation	Unarousable even with painful stimulus
Airway	Unaffected	No intervention required	Intervention may be required	Intervention often required
Spontaneous Ventilation	Unaffected	Adequate	May be inadequate	Frequently inadequate
Cardiovascular Function	Unaffected	Usually maintained	Usually maintained	May be impaired

Continuum of Depth of Sedation: Definition of General Anesthesia and Levels of Sedation/Analgesia; https://www.asahq.org/standards-and-guidelines/continuum-of-depth-of-sedation-definition-of-general-anesthesia-and-levels-of-sedationanalgesia, is reprinted with permission of the American Society of Anesthesiologists, 1061 American Lane, Schaumburg, Illinois, 60173-4973. Ref.[13]

b) The level of sedation required by a patient receiving mechanical ventilation depends not only on the mode of ventilation but also on other variables such as:
   i) Respiratory status
   ii) Hemodynamic profile
   iii) Drug sensitivities
c) Various levels of sedation have been defined to allow clinicians to describe clinical states and patient needs.
d) **Minimal sedation/anxiolysis**
   i) A drug-induced state during which patients respond normally to verbal commands
      (1) Cognitive function and physical coordination may be impaired.
      (2) Airway reflexes and ventilatory and cardiovascular functions are unaffected.
e) **Moderate sedation/analgesia (conscious sedation)**
   i) A drug-induced depression of consciousness during which patients respond purposefully to verbal commands, either alone or accompanied by light tactile stimulation
      (1) No interventions are required to maintain a patent airway, and SV is adequate.
      (2) Cardiovascular function is usually maintained.
f) **Deep sedation/analgesia**
   i) A drug-induced depression of consciousness during which patients cannot be easily aroused but respond purposefully following repeated or painful stimulation
      (1) The ability to independently maintain ventilatory function may be impaired.
      (2) Patients may require assistance in maintaining a patent airway, and SV may be inadequate.
      (3) Cardiovascular function is usually maintained.
g) **General anesthesia**
   i) A drug-induced loss of consciousness during which patients are not arousable, even by painful stimulation
      (1) The ability to independently maintain ventilatory function is often impaired.
      (2) Patients often require assistance in maintaining a patent airway.
      (3) Positive-pressure ventilation may be required because of drug-induced depression of SV or neuromuscular function.
      (4) Cardiovascular function may be impaired.
h) **Dissociation**
   i) A drug-induced disconnect between the thalamoneocortical system and the limbic systems where airway reflexes, SV, and cardiovascular function are all maintained

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Opioid agonists and antagonists, Chapter 28, Opioids, Chapter 215

6) **Medications used for patients requiring mechanical ventilation**
a) Typically fall into two broad categories: analgesics and sedatives.
b) The most frequently used analgesic medications to augment sedation are opioids.
c) **Opioids**
   i) Induce systemic analgesia, some anxiolysis, and mild sedation by binding to specific opioid receptors in the central nervous system
   ii) Attempts being made to curtail the widespread use of opioids given current opioid abuse crisis.
      (1) Alternative classes of analgesics are often used in their place both in the operating room and intensive care unit.
   iii) Commonly used opioids include morphine, hydromorphone, fentanyl, and remifentanil
d) **Although analgesics are commonly used as adjuvants, sedative hypnotics, as a class, are the mainstay of sedation regimens.**

i) Propofol
   (1) The most widely used agent for sedation of mechanically ventilated patients.
   (2) Propofol achieves its effects by modulating the GABA receptor.
      (a) Ideal for patients who are expected to have a short-term need for mechanical ventilation.
   (3) Advantages of propofol
      (a) Short onset
      (b) Short duration of action
   (4) Disadvantages of propofol
      (a) Respiratory depression
      (b) Cardiovascular depression
      (c) Relative contraindication to patients with allergies to eggs
      (d) Pancreatitis
      (e) Propofol infusion syndrome
         (i) Characterized by acute refractory bradycardia leading to asystole, in the presence of one or more of the following:
            1. Metabolic acidosis
            2. Rhabdomyolysis
            3. hyperlipidemia
            4. Enlarged or fatty liver

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Intravenous induction agents, Chapter 30

ii) Benzodiazepines
   (1) Benzodiazepines are the most widely used class of drugs for sedation.
      (a) Commonly used benzodiazepines include diazepam, lorazepam, and midazolam.

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Benzodiazepines, Chapter 27

iii) Dexmedetomidine[15]
   (1) Selective alpha 2 adrenergic agonist
   (2) Hypnotic and analgesic
   (3) No significant suppression of respiratory drive
   (4) Adverse effects: bradycardia and hypotension



Perioperative dexmedetomidine is associated with a decrease in postoperative delirium and overall mortality in patients undergoing cardiac surgery.[14]

iv) Ketamine
   (1) NMDA antagonist
   (2) Preserves airway reflexes
   (3) Minimal respiratory depression
   (4) Associated with:
      (a) Increased CMRO, CBF, sympathetic outflow, PA pressures
      (b) Bronchodilation
      (c) Increased oral secretions

CHAPTER SUMMARY FOR MECHANICAL VENTILATION

Indications for Mechanical Ventilation	Failure to oxygenate or failure to ventilate
Modes of Mechanical Ventilation	Various different modes can be used to achieve the same goal but have unique advantages and disadvantages Noninvasive positive-pressure ventilation includes biphasic positive airway pressure and continuous positive airway pressure Various modes of intermittent positive-pressure ventilation include volume-cycled, pressure-cycled, pressure-regulated volume control, airway pressure release, and pressure support ventilation

Ventilator Settings	Proper ventilator settings provide safety while also minimizing risk Certain modes of ventilation cannot be used in certain patients, such as pressure support ventilation in the paralyzed patient
Sedation	Always ensure adequate and appropriate sedation for the patient undergoing mechanical ventilation Inappropriate use of sedation can frequently lead to unnecessary tests, monitoring, and interventions

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20 Endotracheal Intubation

Eric Brumberger • Arthur Hertling

Intubation of the trachea using a cuffed endotracheal tube (ETT) is the gold standard to ensure airway patency and protection. Endotracheal intubation allows for the provision of prolonged positive-pressure ventilation to patients receiving general anesthesia or experiencing respiratory failure. This chapter discusses principles, technique, and complications of elective endotracheal intubation in adults.

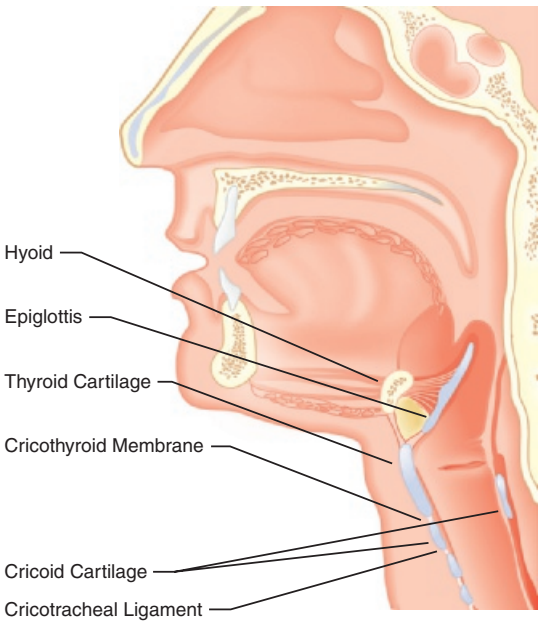
1) **Overview**
   a) **Endotracheal intubation consists of inserting a tube into a patient's trachea in order to ensure airway patency and protection and to provide prolonged positive-pressure ventilation.**
   b) Magill developed this technique after World War I because of the difficulty of administering chloroform by mask to patients with facial injuries.
   c) It is typically performed using direct laryngoscopy (DL), but alternative techniques such as video laryngoscopy and flexible fiberoptic intubation are increasingly used both electively and in specific difficult or emergent situations.
   d) **Indications**
      i) To ensure airway patency in an unconscious patient
      ii) To protect the lungs from the aspiration of gastric contents
      iii) To provide prolonged positive-pressure ventilation in the setting of respiratory failure or general anesthesia because endotracheal intubation allows for generation of higher airway pressures than what is attainable with other invasive (supraglottic airways) and noninvasive devices
   e) **Alternatives to DL for endotracheal intubation might be considered in certain circumstances such as**:
      i) Predictably difficult DL due to abnormal anatomy and trauma to the airway or in patients with a known history of difficult intubation with DL
      ii) Patients with cervical spine precautions due to trauma or disease where a preference for in-line neck stabilization may exist

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   f) **Complications**
      i) Inability to intubate the trachea in a patient who cannot ventilate spontaneously (failed intubation)
      ii) Esophageal or endobronchial intubation
      iii) Trauma to the airway during ETT insertion
      iv) Aspiration of gastric contents prior to intubation
2) **Anatomy**
   a) **Airway anatomy (Fig. 20-1)**
   b) **Anatomical considerations for endotracheal intubation**
      i) The ETT will have to pass through the oral or the nasal conduits through the pharynx, under the epiglottis, through the larynx, the vocal cords, and finally into the trachea.
      ii) The oral and nasal axes are not aligned with the laryngeal axis (**Fig. 20-1**). Therefore, careful positioning and instrumentation are needed to allow direct visualization of the glottis, the uppermost laryngeal opening.

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FIGURE 20-1 The Major Landmarks of the Upper Airway.



Note that the cricoid cartilage is <1 cm in height in its anterior aspect but may be 2 cm in height posteriorly. Reproduced with permission from Rosenblatt WH, Sukhupragarn W. Airway management. In: Barash PG, Cullen BF, Stoelting RK, et al., eds. *Clinical Anesthesia*. 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2013:763.

3) **Preoperative airway assessment (Tables 20-1 and 20-2, Fig. 20-1)**
   a) The anesthesiologist should be familiar with evaluation of the airway to assess the likelihood of difficulty with intubation (**Fig. 20-2**). A detailed discussion of predictors for difficult airway can be found in Chapter 21, Difficult Airway Management.
4) **Equipment for endotracheal intubation**
   a) **Endotracheal tubes**
      i) An ETT is a tube inserted into a patient's trachea that allows positive-pressure (in addition to spontaneous) ventilation and



An airway assessment should be performed on every patient, even if endotracheal intubation is not planned for the procedure.

TABLE 20-1 Techniques of Common Airway Indexes Measurement

Examination	
Thyromental distance	Measured from tip of mentum to thyroid notch in neck-extended position
Mouth opening	Interincisor distance (or interalveolar distance when edentulous) with the mouth maximally opened
Mallampati score	(See Fig. 20-2)
Head and neck mobility	Range of motion from full flexion to full extension
Ability to prognath	Capacity to bring lower incisors in front of upper incisors

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TABLE 20-2 Simplified Risk Score for Difficult Intubation

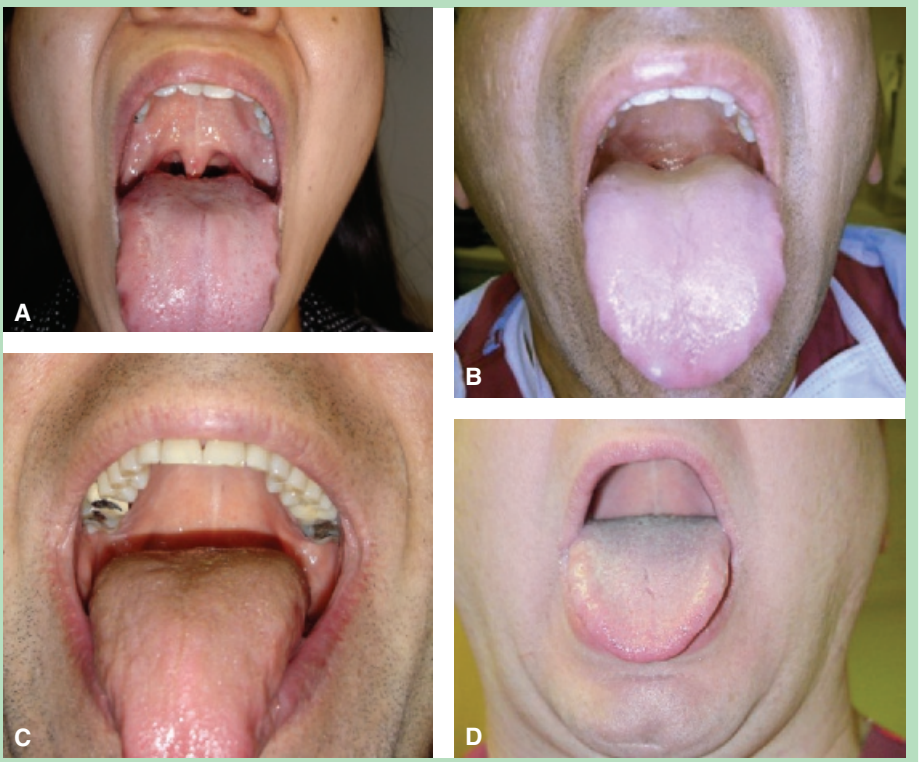
Number of Risk Factors[a]	Incidence of Difficult Intubation
0	0
1	2%
2	4%
3	8%
4 or 5	17%

[a]Presence of upper front teeth, history of a difficult intubation, Mallampati >1, Mallampati of 4, mouth opening <4 cm.
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typically isolates the airway from the oroesophageal tract by means of an inflatable cuff, thus preventing aspiration of gastric contents. The components of a typical ETT are as follows:

(1) A clear curved PVC tube, which allows for visualization of moisture condensation and secretions; ETT sizes are based on the internal diameter (ID) in mm
(2) A standard 15-mm ISO connector that adapts to the breathing circuit at the proximal end of the tube

FIGURE 20-2 Mallampati Classification.



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FIGURE 20-3 Types of ETTs.



Standard **(A)**, Reinforced or "anode" **(B)**, Oral RAE **(C)**, Nasal RAE **(D)**, Laser ETT **(E)**.

(3) An inflatable cuff (for ETT above 3 mm ID) at the distal end of the tube. Modern ETTs have high-volume, low-pressure cuffs to prevent tracheal wall injury from pressure-related mucosal ischemia.

(4) The tip of the ETT is beveled to facilitate passage into the trachea and has an additional small side opening known as the *Murphy eye* whose purpose is to provide an alternate ventilation port should the distal opening become obstructed.

ii) Types of endotracheal tubes **(Fig. 20-3)**. ETTs are available in many forms and materials to accommodate a wide range of anesthetic and surgical situations.

(1) Standard (A)

(2) Reinforced or "anode" (B)

(a) Used in situations where a standard tube would be likely to kink

(3) Oral RAE, taped to the lower lip (C)

(a) Used in ENT surgery to provide full surgical access to the face

(4) Nasal RAE (D)

(a) Used in oral surgery

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The *Murphy eye* is an additional side opening on an ETT whose purpose is to provide an alternate ventilation port should the distal opening become obstructed.

FIGURE 20-4 Laryngoscope Blades.



Macintosh 3 **(A)**, Macintosh 4 **(B)**, Miller 2 **(C)**, Miller 3 **(D)**.

(5) Laser ETT (E)
(a) Covered with nonflammable material; some laser ETTs are made out of metal

b) Laryngoscope blades (**Fig. 20-4**)

i) A laryngoscope blade is a metallic curved or straight blade, fitted with a light source, which is used to lift the tongue and allow direct visualization of the vocal cords through the patient's mouth.
ii) Several types of laryngoscope blades are used for endotracheal intubation. They differ in shape and design, and a number of modified blades have been designed to facilitate DL.
iii) The Macintosh blade is inserted into the vallecula/glossoepiglottic fold. The flange is used to hold the tongue to the left side, out of the field of vision.
iv) A lifting motion in the axis of the handle elevates the epiglottis and reveals the vocal cords.
v) The Miller blade is used to lift the epiglottis.

c) **Other equipment**

i) A means to provide mask ventilation in order to maintain oxygenation after apnea has been induced while awaiting the optimal intubating conditions provided by neuromuscular blockade
(1) This can be an anesthesia machine reservoir bag or a bag-valve-mask device.
ii) An oxygen source
iii) Oral and nasal airways should be available in case of difficult mask ventilation.
iv) Magill forceps are occasionally used to maneuver the ETT, especially during nasotracheal intubation.
v) A functioning suction catheter with a Yankauer-type tip to clear secretions or gastric contents

vi) A malleable stylet to facilitate guiding the tube; the tip of the stylet should not extend beyond the Murphy eye of the ETT to avoid airway trauma
vii) ETTs of various sizes should be available.
viii) An empty syringe for cuff inflation
ix) Tape or a commercially available securing device for the ETT
x) Lubricant and mucosal local anesthetic can be used if desired.
xi) Gloves and a face-shield should be worn to minimize exposure to body fluids.



During intubation, oral and nasal airways should be available in case of difficult mask ventilation.

5) **Principles of DL**
a) DL consists of visualizing the glottic opening directly by means of positioning the patient's head and using a laryngoscope to lift the tongue and the epiglottis.
b) It is performed in order to insert the ETT into the trachea under direct vision and avoid both esophageal intubation and airway trauma that could be caused by blind ETT insertion.

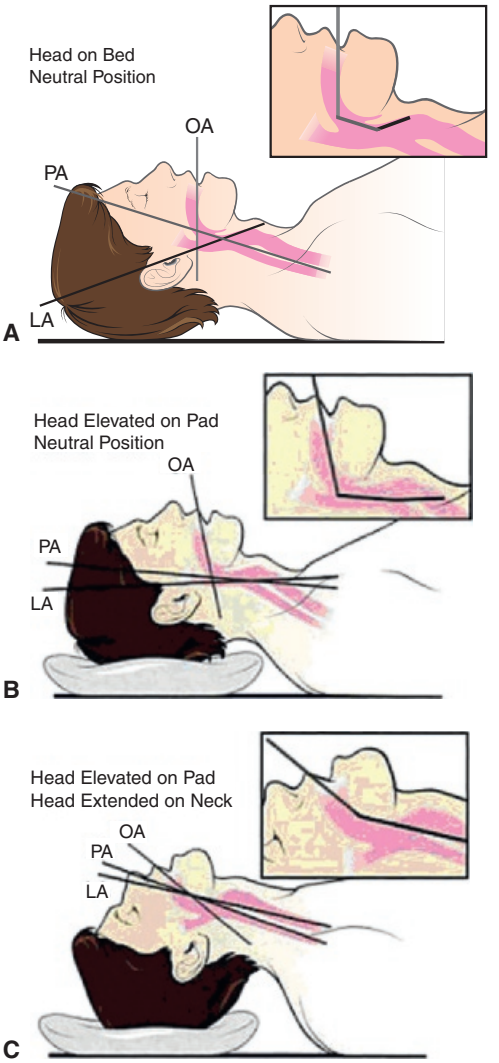
DL is performed in order to insert the ETT into the trachea under direct vision and avoid airway trauma that could be caused by blind ETT insertion.

c) **Positioning for DL**
i) Appropriate elevation
(1) The table's height should be adjusted so that the patient's face is at the level of the practitioner's xiphoid.
ii) Sniffing position
(1) The sniffing position consists of flexing the cervical spine over the trunk by means of a small pillow placed under the head and extending the head over the neck (**Fig. 20-5C**).
iii) Three-axes theory
(1) The three-axes theory postulates that, in order to facilitate DL, the oral, pharyngeal, and laryngeal axes should be aligned through patient positioning.
(2) Adaptability and flexibility are important. A one-size-fits-all approach for successful intubation does not exist; in 11% of the patients, the sniffing position actually worsens the laryngoscopy view.[1]
iv) Head-elevated laryngoscopy position (HELP)[2]
(1) HELP is a position used to facilitate DL in morbidly obese patients.
(2) These patients typically have a "buffalo hump," and positioning them flat on a bed will force the head backward, hyperextending the neck and making DL very difficult.
(3) In addition, morbidly obese patients have a large chest that renders laryngoscope blade insertion difficult (a short-handle laryngoscope can facilitate insertion).
(4) Position the patient using pillows such that the patient's sternum is level with the external auditory meatus (**Figs. 20-6 and 20-7**).
(5) Blankets or a commercially available "ramp" placed under the upper back, neck, and occiput can facilitate achieving optimal positioning for obese patients and other suspected or known difficult intubations.
v) Manual in-line stabilization for cervical spine trauma
(1) In cases of cervical spine trauma or disease with suspected or confirmed cervical spine instability, head hyperextension is contraindicated as it could lead to spinal cord compression.
(2) Two operators are needed to perform this technique.
(3) One operator holds the head and stabilizes it, minimizing cervical spine motion.



Patient positioning is paramount for optimizing laryngoscopic view but what works for one patient may not work for another; adaptability and flexibility are critical skills for successful airway management.

FIGURE 20-5 Understanding Aligning the Oral Axis, Pharyngeal Axis, and Laryngeal Axis.



A. Anatomical neutral position. The oral axis (OA), pharyngeal axis (PA), and laryngeal axis (LA) are at greater angles to one another. **B.** The head, still in neutral position, has been lifted by a pillow flexing the lower cervical spine and aligning the PA and the LA. **C.** The head has been extended on the cervical spine, aligning OA with the PA and the LA creating the optimum sniffing position for intubation. Reprinted with permission from Brown CA, Sakles JC, Mick NW. *The Walls Manual of Emergency Airway Management*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2017.

FIGURE 20-6 The Patient Positioned Flat on the OR Bed.



The head and neck are hyperextended, making DL challenging. Photo Courtesy of Dr. Jay Brodsky.

FIGURE 20-7 The Patient Positioned in the Head-Elevated Laryngoscopy Position (HELP), Flexing the Neck and Allowing Head Extension.



Photo courtesy of Dr. Jay Brodsky.

(4) The other operator performs DL without extending the head.
(5) While this technique allows vocal cord visualization and tracheal intubation in most cases, having a secondary indirect airway device (eg, a video laryngoscope) as backup is recommended.



In case of cervical spine trauma or disease with suspected or confirmed cervical spine instability, head hyperextension is contraindicated as it could lead to spinal cord compression.

6) **Performing DL for orotracheal intubation**
   a) **In most patients, DL for orotracheal intubation requires general anesthesia to minimize patient discomfort, optimize vocal cord view, and avoid unwanted physiologic perturbations/reflexes such as hypertension, tachycardia, or laryngospasm.**
      i) Induction of anesthesia should be performed after a thorough preoperative assessment and preoxygenation, using either normal tidal ventilation for about 3 minutes or 4 forced vital capacity breaths; adequate denitrogenation is achieved when the expired FiO_2 is above 0.8.
      ii) An appropriate dose of an induction agent and, when appropriate, a muscle relaxant should be administered.
      iii) Once apnea is induced, ventilation should be assisted until adequate paralysis is achieved, unless positive-pressure ventilation is contraindicated.
      iv) Once optimal intubation conditions have been achieved, DL may be attempted.
   b) **Place patient in proper "sniffing" or "HELP" (head-elevated laryngoscopy position) positions (obese patients).**
   c) Open the patient's mouth, typically using a scissoring motion of the thumb and the index finger of the right hand.
   d) Insert the laryngoscope blade into the right corner of the mouth, and "load" the tongue on the flange using a sweeping motion toward the midline, as the blade is inserted deeper to the base of the tongue. Before the blade is fully inserted, take advantage of opportunities to sweep the upper and lower lips away in order to prevent them from being caught between the blade and the teeth.
   e) **Once the epiglottis is visualized, the laryngoscope should be lifted in the axis of the handle.**
      i) Do not use a lever motion, as this might lead to dental trauma.
      ii) This will uncover the glottic opening.
   f) **When using a straight blade, insert the blade under the epiglottis prior to lifting (Fig. 20-8).**
   g) **Insert the ETT, held in the right hand, through the vocal cords 1-2 cm beyond the passage of the cuff through the cords.**
   h) **Confirm endotracheal position by waveform capnography (CO_2 detection) for at least three breaths.**
      i) Laryngoscopic views are graded according to Cormack and Lehane (Fig. 20-9).
      ii) If visualization of the glottis is suboptimal, a BURP maneuver (backward-upward-rightward pressure on the neck at the level of the cricoid cartilage) may improve the view; this is one version of optimal laryngeal manipulation (OLM), external techniques that are used to improve alignment of the three axes.



Preoxygenation before the induction of general anesthesia, also known as denitrogenation, is a vital part of airway management; by replacing the functional residual capacity with near 100% oxygen, additional time is afforded to create a plan should the "cannot intubate, cannot ventilate" scenario be encountered.

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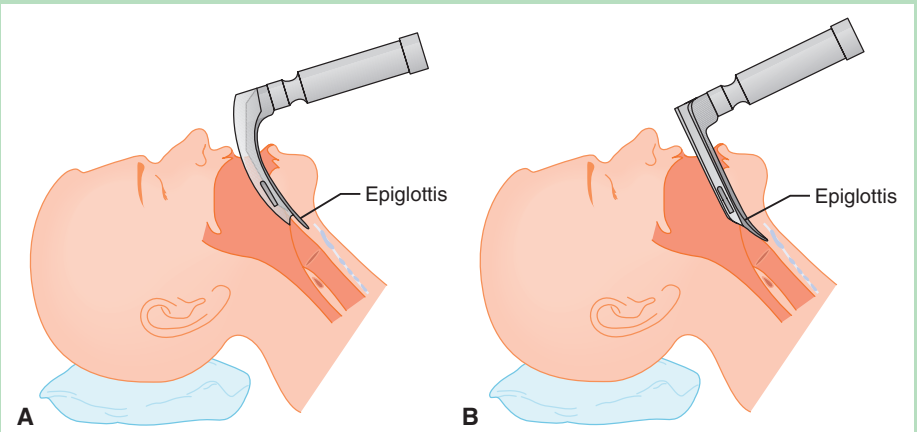


Do not use a lever motion with a laryngoscope—this motion could lead to dental trauma.



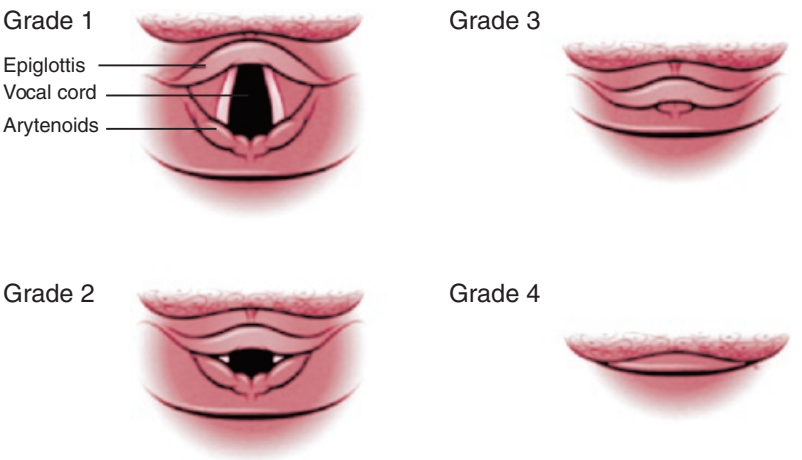
If the ETT will not advance beyond the level of the vocal cords, consider either withdrawing the stylet while advancing the tube to achieve maximum flexibility at the distal end or rotating the ETT 90 degrees clockwise in order to bring the most distal tip posterior into the trachea and off the laryngeal/tracheal cartilages.

FIGURE 20-8 Use of a Mac vs Miller Blade.



When a curved laryngoscope blade is used, the tip of the blade is placed in the vallecula, the space between the base of the tongue, and the pharyngeal surface of the epiglottis **(A)**. The tip of a straight blade is advanced beneath the epiglottis **(B)**. Reproduced with permission from Rosenblatt WH, Sukhupragarn W. Airway management. In: Barash PG, Cullen BF, Stoelting RK, et al., eds. *Clinical Anesthesia*. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009:776.

FIGURE 20-9 Cormack and Lehane Laryngoscopic Views.



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iii) If the vocal cords are not seen, it is almost never correct to attempt to insert the ETT blindly. Instead, make changes in positioning, equipment, or personnel, calling for help, and in general, acting within the guidelines of the difficult airway algorithm.



The BURP (backward-upward-rightward pressure) maneuver is the most popular version of optimal laryngeal manipulation: external manipulation of the larynx to improve glottic view with the right hand of the laryngoscopist or a secondary practitioner.

7) **Technique for nasotracheal intubation**

a) **Indications**

i) Oral surgery, to provide full access for the surgeon

ii) Anticipated or unanticipated difficult oral approach, as might be expected with limited mouth opening (eg, temporomandibular joint stiffness)

b) **Contraindications**

i) Increased risk of bleeding: nasal polyps, coagulation disorder

ii) Severely limited mouth opening; a fiberoptic technique should be employed

iii) Likelihood of prolonged intubation, as a nasal ETT can lead to sinus obstruction and infection within a few days

iv) Presence of a basilar skull fracture

c) **Prepare both nostrils with two puffs of a decongestant,** for example, oxymetazoline (Afrin), in the holding area prior to bringing the patient to the OR, and two more puffs once in the OR.

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After ETT placement, confirm placement in the trachea by waveform capnography for at least three breaths.

d) **Assess which nostril is larger; consider septum deviation.**

e) Preoxygenate

f) Induce GA and administer muscle relaxation.

g) **Insert a soft nasal trumpet lubricated with lidocaine ointment to ensure easy passage of the ETT.**

h) **The trumpet/ETT should be inserted perpendicular to the face.**

i) Remove nasal trumpet; sequentially upsize with larger sizes if needed.

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When managing a patient's airway, avoid fixation error, which is the practice of continuing the same ineffectual practices with the hopes of achieving unlikely success.

i) **Insert nasal RAE ETT until the tip reaches the oropharynx.**

i) Do not force if resistance is felt!

j) **Perform DL;** visualize vocal cords and ETT.

k) **Advance ETT into the trachea.**

i) Use Magill forceps if needed.

ii) Do not use the forceps to grasp the ETT by the cuff, which can tear.

(1) An assistant can push or pull the ETT as directed, as one hand holds the laryngoscope and the other the Magill forceps.

iii) If the ETT passes through the vocal cords but does not advance into the trachea, it is likely abutting the anterior wall of the larynx.

iv) Removing the laryngoscope and flexing the head while maintaining a mild pressure on the ETT usually allows the ETT to go down the trachea. Alternatively, simultaneously rotating the ETT

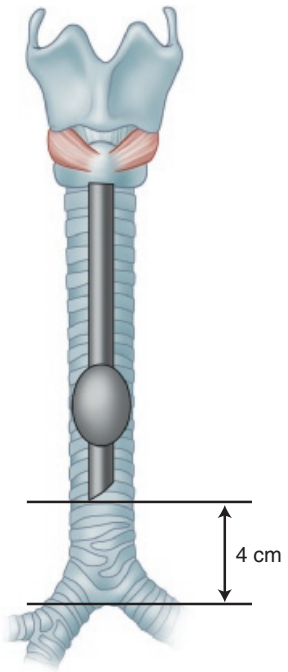


Nasotracheal intubation is often used for oral surgery and/or patients with limited mouth opening.



Do not force nasotracheal tubes when resistance is felt to avoid injury and bleeding.

FIGURE 20-10 Position of the ETT in the Trachea and Distance of the Tip of the ETT From the Carina.



Position the tip of the ETT above the carina. It should ideally be 4 cm.

tube 90 degrees with the Magill forceps distally, as well as the proximal end of the tube, while applying mild pressure, should also facilitate passage down the trachea.

l) **Tape ETT to the nose.**
   i) Ensure that the taping is not so tight that the nostril blanches, as this might lead to local ischemia and necrosis.

8) **ETT positioning**
   a) **The tip of the ETT should be placed below the vocal cords, ideally 4 cm (and at least 3 cm) above the carina (Fig. 20-10).**
      i) However, this is difficult to assess without a chest radiograph or fiberoptic bronchoscopy.
      ii) In most patients, a position of 21 cm at the lips for women and 23 cm for men provides correct positioning.
   b) **Auscultation should confirm bilateral breath sounds and the absence of epigastric sounds.**
   c) **The cuff of the ETT can be palpated in most patients in the suprasternal notch.**



Avoid grabbing the ETT at the cuff with the Magill forceps as the forceps can tear the cuff.



The tip of the ETT should be placed below the vocal cords, ideally 4 cm (and at least 3 cm) above the carina.

d) **The ETT position in the trachea will change with head motion and should be verified after repositioning the patient.**



Palpation of the cuff by "ballottement" in the sternal notch may confirm proper placement of the ETT.

9) **Videolaryngoscopy**

a) Videolaryngoscopy is an indirect way of viewing the glottic opening. Videolaryngoscopy can be performed using a rigid scope such as the GlideScope or a flexible fiberoptic scope and may offer an improved view of the vocal cords in the following cases:

i) Limited mouth opening

ii) Inability to manipulate the neck

iii) Anterior larynx

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10) **Complications**

a) **Dental injury**

i) DL can lead to dental injury, especially of the upper incisors, as it is very easy to apply force on those teeth when lifting the blade to improve glottic visualization.

ii) Teeth can be chipped or dislodged, occasionally leading to aspiration of dental fragments; it is very important to ascertain preoperatively which teeth are loose or in such poor state that they may be easily dislodged.

(1) Preexisting chipping, missing teeth, etc. should be documented preoperatively.



As good as an indirect view of the glottic opening may appear, it may not portend success with actual passage of the ETT.

iii) In case of dental injury, ensure to recover all tooth fragments and obtain dental consult as soon as possible. Dislodged teeth can be reinserted.

b) **Esophageal intubation**

i) Insertion of the ETT into the esophagus instead of the trachea

ii) Always confirm endotracheal placement with end-tidal CO_2 monitoring (gold standard) in addition to auscultating lung sounds, visualizing the ETT passing through the vocal cords, observing adequate chest excursion, seeing cyclic condensation of moisture inside the ETT, and recognizing the typical resistance/feel of the reservoir bag.



Videolaryngoscopes can offer an improved view of the vocal cords for patients with limited mouth opening, an inability to extend the neck, or an anterior larynx.

iii) Undetected esophageal intubation will prevent effective gas exchange and lead to rapid hypoxemia, with a potential for cardiac arrest and hypoxic brain injury unless recognized and treated.

iv) In the following scenarios where CO_2 may not be detectable by an ETT correctly placed in the trachea, fiberoptic bronchoscopy can be a useful method to confirm successful endotracheal intubation if sustained capnography cannot be demonstrated:

(1) Cardiovascular collapse or cardiac arrest

(2) Severe bronchospasm

(3) Massive pulmonary embolus

(4) Equipment malfunction



Sustained carbon dioxide (CO_2) as demonstrated by capnography is the gold standard by which successful endotracheal intubation is measured.

c) **Aspiration of gastric contents**

i) Passive regurgitation of gastric contents with subsequent aspiration into the lungs can lead to severe chemical pneumonitis (pH of gastric contents is ~2.5) even in a patient who was NPO for an appropriate duration.

ii) Consider administering a nonparticulate antacid (Bicitra) or an H_2 blocker preoperatively to patients at risk for aspiration of gastric contents.

iii) A working suction should always be available and ready to use.

iv) Cricoid pressure is controversial and is probably of limited efficacy to limit passive regurgitation.[3]

(1) Patients at risk for regurgitation probably benefit more from mild reverse Trendelenburg (head-up) positioning.

v) In case of aspiration:

(1) Intubate trachea

(2) Suction trachea vigorously prior to applying PPV, which may push aspirated gastric contents into the bronchi.

vi) In case of vomiting rather than passive regurgitation:

(1) Turn patient to the side and head down (Trendelenburg).

(2) Suction pharynx as thoroughly as possible.

(3) Return to supine position once vomiting has stopped.

(4) Intubate trachea.

(5) Suction trachea vigorously prior to applying PPV.



Undetected esophageal intubation can lead to rapid hypoxemia with a potential for cardiac arrest and hypoxic brain injury unless rapidly recognized and treated.



During intubation, a working suction should always be available and ready to use.

d) **Endobronchial intubation**

i) Typically in the right mainstem bronchus, leading to substantial shunt and arterial hypoxemia

ii) This is detected by auscultation, with decreased breath sounds on the left. If auscultation is inconclusive, fiberoptic bronchoscopy or a portable chest radiograph/fluoroscopy can be utilized to ascertain ETT tip position.

iii) The ETT should be withdrawn into correct position.



Endotracheal tube cuffs should be inflated with the minimum volume of air required to prevent leakage; even high-volume low-pressure cuffs can cause tracheal wall injury from unnecessary overinflation. When using N_2O, the ETT cuff can expand over time; check the cuff regularly to prevent overinflation.

e) **Airway trauma**

i) The tip of the ETT can injure the larynx and trachea, especially if a rigid stylet is used.

ii) Mild trauma will lead to edema and bleeding and can be diagnosed postoperatively by indirect laryngoscopy. Severe trauma can disrupt the airway, perforate the trachea, and lead to pneumomediastinum and mediastinal infection, necessitating surgery.

iii) Trauma prevention consists of performing DL and intubation under direct vision rather than blindly, using smooth motions and avoiding excessive force, and sliding the ETT off the stylet once the tip is beyond the vocal cords, rather than inserting the more rigid ETT-stylet assembly down the trachea.

f) **Compression of the tracheal wall may be caused by overinflation of the cuff.**

i) This can lead to the following complications:

(1) Tracheitis

(2) Pressure necrosis of the tracheal wall

(3) Tracheal rupture

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Crisis management: bronchospasm, Chapter 143

ii) Patients requiring prolonged intubation are most at risk, although it can occur in any long case, especially when nitrous oxide is used.

(1) Cuff pressure can be checked periodically using an adapted device and maintained below 25 mm Hg.

g) **Bronchospasm**[4]

i) Bronchospasm is defined as a reflex narrowing of the small airways (bronchioles) through a combination of smooth muscle contraction and, secondarily, inflammation with edema.

ii) Endotracheal intubation can cause severe bronchospasm, such that no $ETCO_2$ will be detected and no breath sounds will be heard, despite the ETT being correctly positioned.
   (1) Perform DL again or fiberoptic bronchoscopy to ensure correct ETT position in the trachea.
   (2) Ensure that the ETT is not kinked or too deep, against the carina.
   (3) Continue ventilation using 100% O_2.
   (4) Deepen anesthesia using IV anesthetics, ideally bronchodilators such as propofol or ketamine.
   (5) Consider low-dose epinephrine (10 μg subcutaneously) or terbutaline (0.2 mg subcutaneously).
   (6) Inhaled beta-2 agonists and volatile anesthetics can be used as bronchodilators if the patient can be ventilated.



Endotracheal intubation can cause severe bronchospasm, such that that no $ETCO_2$ will be detected and no breath sounds will be heard, despite the ETT being correctly positioned.

CHAPTER SUMMARY FOR ENDOTRACHEAL INTUBATION

Definition	Endotracheal intubation consists of inserting a tube into a patient's trachea in order to ensure airway patency and protection and to provide prolonged positive-pressure ventilation.
Positioning for Endotracheal Intubation	The sniffing position consists of flexing the cervical spine over the trunk by means of a small pillow placed under the head and extending the head over the neck. For morbidly obese patients, the HELP position consists of positioning the patient such that the patient's sternum is level with the external auditory meatus.
Technique of Orotracheal Intubation	Preoxygenate, induce general anesthesia and muscle relaxation, mask ventilate, open mouth, insert laryngoscope blade, visualize glottic opening, insert ETT, confirm proper positioning by capnography and chest auscultation, and secure ETT.
Technique of Nasotracheal Intubation	Prepare nostrils with a decongestant; preoxygenate; induce general anesthesia and muscle relaxation; insert soft nasal trumpet lubricated with lidocaine ointment; mask ventilate; insert ETT into nostril until the tip reaches the oropharynx; open mouth; insert laryngoscope blade; visualize glottic opening; place ETT, using Magill forceps if needed; confirm proper positioning by capnography and chest auscultation; secure ETT.
ETT Positioning	The tip of the ETT should be placed 4 cm below the vocal cords and 3 cm above the carina.
Complications	Major complications are due to esophageal intubation, aspiration of gastric contents, endobronchial intubation, airway trauma, compression of the tracheal wall, and bronchospasm.

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21 Difficult Airway Management

Elizabeth Koch • Vladimir Nekhendzy

Management of the difficult airway is critical to the expertise of an anesthesiologist. Difficult tracheal intubation, unrecognized esophageal intubation, inadequate ventilation or oxygenation, and premature extubation can result in death or permanent brain damage. Each difficult airway is unique, and competent management of the difficult airway will depend on successful integration of one's knowledge, clinical judgment, and technical skills—factors that are constantly evolving throughout one's clinical career.

1) **Overview**
   a) **Definition**
      i) The American Society of Anesthesiologists (ASA) Task Force on Difficult Airway Management defines a difficult airway as "the clinical situation in which a conventionally trained anesthesiologist experiences difficulty with face mask ventilation of the upper airway, difficulty with tracheal intubation, or both."[1]
   b) **Incidence of difficult airway and management outcomes**[2–4] **(Table 21-1)**
      i) The vast majority (98%) of patients are easy to mask (EMV).
      ii) Difficult mask ventilation (DMV) may be encountered in one in 100 patients.
      iii) Impossible mask ventilation (IMV) may be expected to happen once in 700 patients.
      iv) Should mask ventilation prove to be difficult or impossible after induction of anesthesia, the incidence of difficult and failed tracheal intubation increases dramatically.
      v) Although the majority (94%) of patients in the DMV group can still be intubated conventionally, the success rate in the IMV group decreases significantly (86%). Nevertheless, the 86% success rate is still sufficiently high to suggest that, patient's condition permitting, a single attempt at direct laryngoscopy (DL) may be feasible if IMV is encountered in anesthetized patient.
      vi) The majority of patients (97%) with IMV can still be intubated with the use of alternative airway management techniques, highlighting the crucial role of the anesthesiologist's familiarity and proficiency with different difficult airway management devices.
      vii) Fortunately, the most dangerous situation, "cannot ventilate-cannot intubate" (CVCI), is extremely rare.
2) **Approach to the difficult airway**
   a) **The ASA difficult airway algorithm**
      i) Has been associated with significantly decreased airway management-related morbidity and mortality[5]



Practice difficult airway technical skills before they are emergently needed. Simulation, airway workshops, and elective use of difficult airway equipment in the operating room are opportunities to refine skills.

If DMV is encountered, immediate preparations should be made for alternative airway management.



The first attempt at direct laryngoscopy is always the best, as subsequent airway instrumentation results in progressive trauma and potential for impaired visualization from bleeding, secretions, and edema. Therefore, positioning and oxygenation should be optimized from the beginning.

TABLE 21-1 The Incidence of Difficult Airway[2–4]

	EMV	DMV	IMV
Overall incidence	98%	1.4%	0.15%
Difficult DL/intubation	8%	3-4 times higher	
Failed DL/intubation	0.5%	10-12 times higher	
Successful DL	99.5%	94%	86%
Overall success (DL + alternative techniques)	(100%)	~100%	97%
CVCI	(0%)	~0%	1:50 000

CVCI, "cannot ventilate-cannot intubate"; DL, direct laryngoscopy; DMV, difficult mask ventilation; EMV, easy mask ventilation; IMV, impossible mask ventilation.

ii) Despite some limitations (eg, primarily focusing on tracheal intubation), the updated ASA guidelines[1] continue to provide a rational and effective framework for the anesthesiologist's approach to the difficult airway (**Fig. 21-1**).

iii) As a general rule, when difficult ventilation is anticipated, it is safest to secure the patient's airway awake (**Fig. 21-1**).

(1) Almost all intubation techniques can be performed in an awake patient.

(2) For the majority of cases of anticipated difficult airway, awake flexible fiberoptic intubation (FOI) remains the gold standard of management.

b) Predicting difficult airway

i) Conducting a routine and thorough preoperative airway history and examination is always warranted to detect potential difficult ventilation and laryngeal exposure (**Fig. 21-2**).

ii) A history of previous difficult airway, neck irradiation, mediastinal masses, and acquired or congenital disease states (eg, ankylosis, subglottic stenosis, lingual thyroid or tonsillar hypertrophy, Treacher Collins, etc) should raise concern for anticipated difficult airway management.[1]

iii) Physical examination may identify risk factors for DMV and difficult intubation (**Tables 21-3 to 21-5**). However, predictive values for bedside airway exams are relatively low, and it remains challenging to accurately predict the presence of difficult airway.

c) **Face mask ventilation**

i) Complications of unsuccessful tracheal intubation can be avoided if the ability to ventilate the patient's lungs by face mask is preserved.

ii) As with the recognition of a difficult airway, predicting difficulty with face mask ventilation can be relatively easy when significant anatomical abnormalities are present (**Table 21-2**).

iii) Independent risk factors are associated with DMV and IMV (**Tables 21-3 and 21-4**).[2–4] Note shared factors (presence of beard, snoring or obstructive sleep apnea [OSA], and high Mallampati grade) between these categories, pointing to a possible easy transition from DMV to IMV in patients at risk.



When tracheal intubation is difficult, the primary focus should be ventilation of the lungs. Repeated attempts at direct laryngoscopy should be discouraged, as this is the primary reason for airway-related morbidity and rapid progression to the "cannot ventilate-cannot intubate" situation.[5]

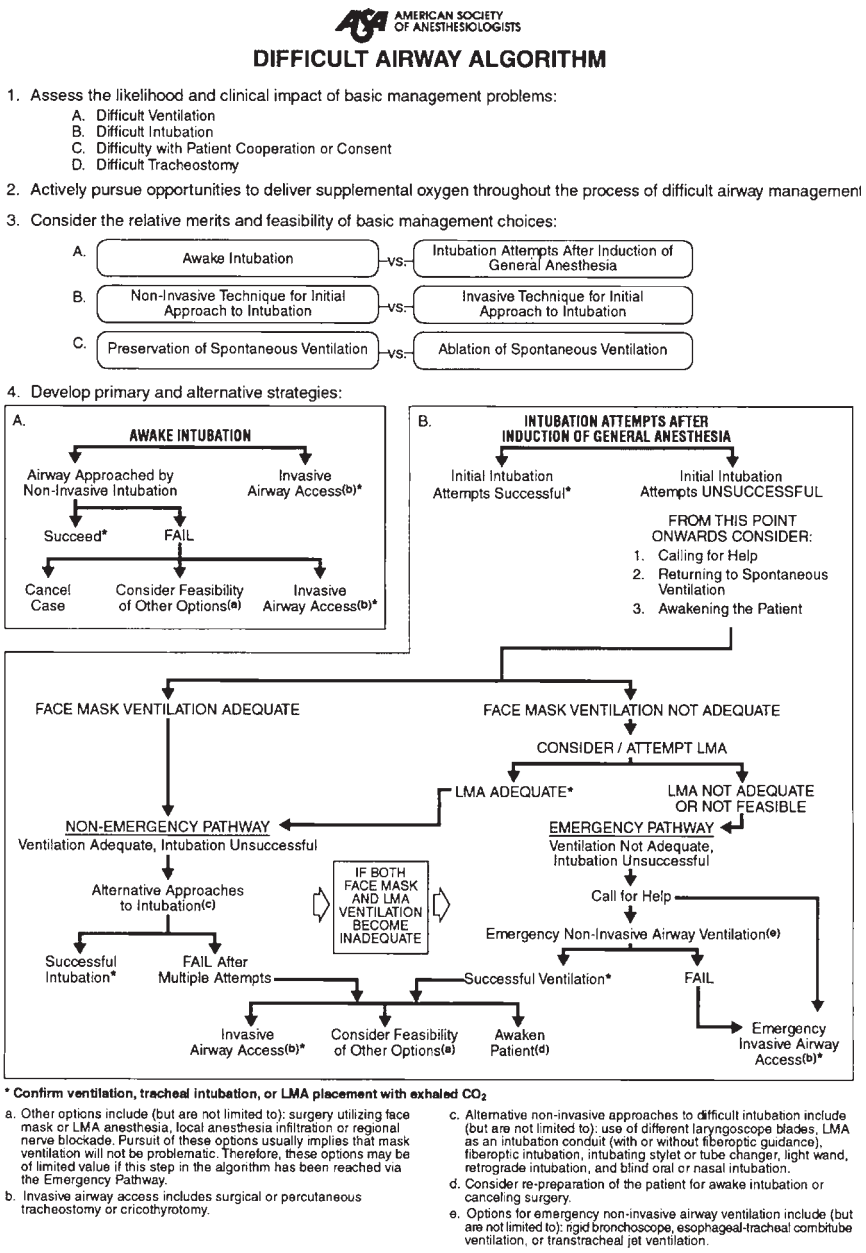


The ASA difficult airway algorithm is intended for use during administration of anesthesia care; the approaches to difficult airway may be different in nonoperative settings.



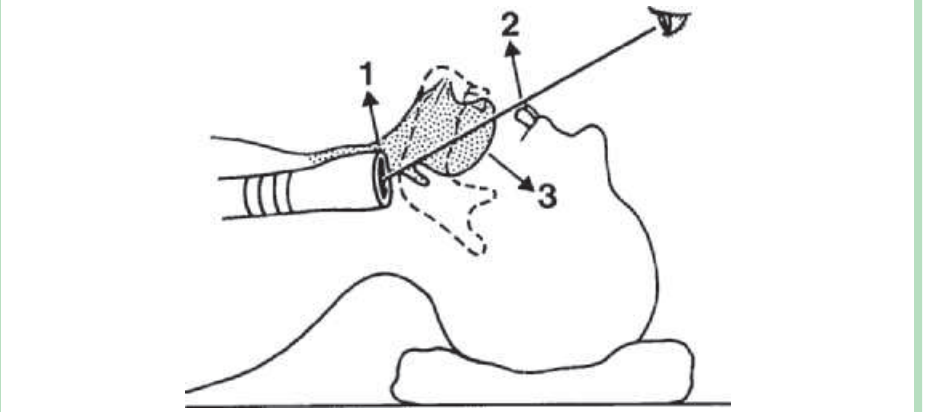
The presence of three or more abnormal airway assessment tests should raise the anesthesiologist's level of suspicion for difficult airway.

FIGURE 21-1 The ASA Difficult Airway Algorithm



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FIGURE 21-2 The Direct Line of Sight to the Vocal Cords May Be Blocked by a Relatively Anterior Larynx (*1*), Prominent Upper Incisors (*2*), and a Large and Posteriorly Located Tongue (*3*)



From Cormack RS, Lehane J. Difficult tracheal intubation in obstetrics. *Anaesthesia*. 1984;39(11):1105-1111. Reprinted by permission of John Wiley & Sons, Inc.

TABLE 21-2 Anatomic Abnormalities Predictive of Difficult or Impossible Face Mask Ventilation

Standard approach to upper airway and airway opening maneuvers not possible
Wired jaws
Facial trauma
Cervical rigidity
Halo fixation
Poor seal between the face and a mask
Facial abnormalities
Facial trauma
Airway narrowing
Laryngospasm
Upper and lower airway tumors, especially mobile, and those associated with respiratory systems
Narrowed pharyngeal space that cannot be easily modified (pharyngeal edema, copious amount of blood, pus, or secretions)
Upper and/or lower airway collapse

TABLE 21-3 Predictors of Difficult Face Mask Ventilation[2,3]

- Age >55-57 years old
- Body mass index ≥26-30 kg/m^2
- Beard
- Snoring
- Lack of teeth
- Mallampati III or IV
- Limited mandibular protrusion

TABLE 21-4 Predictors of Impossible Face Mask Ventilation[4]

- Male sex
- Beard
- OSA
- Mallampati III or IV
- Neck radiation changes

Reprinted with permission from Kheterpal S, Martin L, Shanks AM, et al. Prediction and outcomes of impossible mask ventilation: a review of 50,000 anesthetics. *Anesthesiology*. 2009;110:891-897.

TABLE 21-5 Predictors of Difficult and IMV and Difficult Intubation[4]

- Body mass index ≥30 kg/m^2
- Snoring
- OSA
- Thick/obese neck
- Limited mandibular protrusion

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(1) Decreased mandibular protrusion and the presence of neck radiation changes are the most significant factors for predicting DMV and IMV.[3,4]

(2) Moderate to severe OSA requiring either the use of CPAP/BiPAP or a history of OSA surgery (**Table 21-6**)

(3) Of particular interest are the findings predicting both DMV and IMV and difficult tracheal intubation (**Table 21-5**). The overall incidence of this event in patient population is 0.37%.[4]

(a) One may consider an inhalation induction in these patients, therefore preserving spontaneous ventilation, but this approach does not exclude possible loss of airway.

(b) Appreciate that patients with DMV and IMV are at high risk for iatrogenic complications associated with difficult airway management. Keep in mind that the transition from DMV to IMV may be sudden, especially if previous repeated attempts on DL were present.

(c) Almost 15% of patients with IMV will be difficult to intubate, which places them at highest risk for a CVCI situation.

(d) Note that limited mandibular protrusion (**Tables 21-3 and 21-5**) constitutes the single most important predictor for both DMV and difficult intubation, and therefore this test should become an essential part of preoperative airway assessment.

READ MORE
Atlas of anesthesia procedures: mask ventilation, Chapter 160



The transition from DMV to IMV may be sudden, especially if previous repeated attempts on DL were present.

READ MORE
Atlas of anesthesia procedures: awake fiberoptic intubation, Chapter 164

d) **Deciding on awake vs asleep airway management**

i) An awake intubation should be considered if difficulty with ventilation by both mask and supraglottic airway devices is anticipated.

ii) Given the very low incidence of IMV, it would be difficult to justify a conservative awake approach in all patients.[4]

iii) Risk stratification for anticipated difficult ventilation

(1) Consider the presence of at least three factors predictive of DMV, IMV (see **Tables 21-3 and 21-4**), and their combination with difficult tracheal intubation, to assure appropriately high "likelihood" (odds ratio) of encountering these events.[3,4]

(2) Anatomical features most commonly associated with unanticipated difficult intubation may include the following[6]:

(a) Anterior larynx (most common)

(b) Abnormal neck anatomy (poor neck mobility and short neck)

(c) Decreased mouth opening



Awake intubation should be strongly considered if the predictors of DMV and IMV coincide with the factors predicting difficult intubation, and difficulty with the placement of a supraglottic device (eg, LMA) to provide rescue ventilation is anticipated.

TABLE 21-6 Alternative Difficult Airway Management Devices and Techniques: Nonemergency Pathway

- Simple intubating aids (gum-elastic bougie, introducers, Trachlight, video laryngoscopy)
- Blind intubation (largely superseded by FOI)
- LMA techniques (use as a rescue aid, a guide for intubation, or as ultimate ventilatory device)
- Fiberoptic techniques (flexible and rigid)
- Transtracheal techniques (retrograde intubation and cricothyrotomy)

iv) If an asleep approach is chosen, *several* preformulated alternative airway management plans for tracheal intubation must be devised, and necessary assistance (equipment, personnel) should be in place before induction of anesthesia.

e) **Difficult tracheal intubation in the anesthetized patient**. If difficulty in tracheal intubation is encountered after induction of general anesthesia (unanticipated difficult airway), or asleep approach to the recognized difficult airway was intentionally chosen (eg, uncooperative patient), a variety of techniques can be employed (see **Table 21-6**).

i) Overview

(1) The nonemergency pathway of the algorithm implies that the patient's lungs can be adequately ventilated through face mask or supraglottic airway.

(2) If this ventilation becomes inadequate or fails, and tracheal intubation proves unsuccessful—"cannot ventilate-cannot intubate" situation—management approaches for salvaging the airway will be severely limited (emergency pathway of the algorithm) (see **Table 21-7**).

ii) Avoid direct laryngoscopy.

(1) If difficulty in tracheal intubation is anticipated, consider instituting an initial alternative airway approach, therefore avoiding DL completely. The use of the intubating laryngeal mask (ILMA), video laryngoscopy, and fiberoptic bronchoscopy are associated with a 95%-100% success rate.[7,8]

iii) Perform direct laryngoscopy.

(1) If DL is chosen as an initial approach, it is important to remember that the first attempt will always be the best. The patient's head position should be carefully optimized for laryngoscopy, and full preoxygenation should precede the induction of anesthesia.

iv) Use of neuromuscular blockade

(1) The data support use of muscle relaxants for managing anticipated difficult tracheal intubation under general anesthesia[2,4,9] and demonstrate possible preference for depolarizing agents over nondepolarizing neuromuscular blockers.[9]

3) **Specialized airway devices and techniques**

a) **Introducer stylets (bougies)**

i) Solid or hollow semimalleable stylets

(1) Solid: Portex tracheal tube guide (Smith); coude tip bougie 15 Fr × 7 cm (SunMed) (**Fig. 21-3**)

TABLE 21-7 Devices and Techniques for Managing "Cannot Ventilate-Cannot Intubate" Situation: Emergency Pathway

- Combitube
- Transtracheal jet ventilation
- Rigid bronchoscope
- Transtracheal techniques (cricothyrotomy, tracheostomy)

FIGURE 21-3 Examples of Solid Bougies, Coude Tip From SunMed (*Top*), and Portex Tracheal Tube Guide (*Bottom*)



(2) Hollow: Frova intubating introducer (Cook)
(a) Allows insufflation of oxygen and detection of CO_2
ii) Can be used to assist with tracheal intubation when the glottic opening is not adequately visualized with DL
iii) Manipulate angled segment anteriorly under the epiglottis.
iv) "Clicks" are felt as the angulated tip of the bougie slides against the tracheal rings, and the "distal holdup" sign is elicited when the angulated tip of the bougie lodges in the small bronchus.
v) ETT can be guided into the trachea over the bougie device.



"Clicks" are felt as the bougie passes under the epiglottis and into the trachea. An endotracheal tube (ETT) can then be advanced into the trachea over the bougie.

b) **Supraglottic airway devices in difficult airway management**
i) General advantages
(1) Placement of the LMA-Classic results in a 94% success rescue ventilation rate in patients with *unanticipated* difficult airway.[10]
(2) Successful placement of the LMA-Classic (and other types of the LMA) in patients with abnormal and/or difficult airway is probably independent of factors used to predict or score difficult intubation.[11]
ii) Limitations
(1) Failures of the supraglottic airway and esophageal tracheal airway (Combitube) may occur, and they cannot be considered fail-safes for the difficult airway, particularly when an infraglottic obstruction is present.[5]
(a) Predictors of supraglottic airway failure (**Table 21-8**)
(2) Some of the problems common to both these devices that may preclude their use are as follows:
(a) Inability to insert the device (eg, restricted mouth opening)
(b) Inability to properly position the device in the hypopharynx (eg, when cricoid pressure is applied or with upper airway pathology)

TABLE 21-8 Predictors of Supraglottic Airway Failure

- Restricted mouth opening
- Obstruction at or below larynx
- Disruption/distortion
- Stiff lungs (poor compliance)
- Neck irradiation
- Decreased thyromental distance
- Limited C-spine mobility
- Male sex
- Age >45 years
- Aspiration risk

(c) Inability to maintain a seal or to ventilate (eg, disrupted trachea, decreased lung compliance, or obstructing pathology either at or below the cords)

iii) Flexible FOI through the LMA-Classic
(1) Offers the advantage of direct visualization of the laryngeal structures and carries minimal risk of intubation trauma and esophageal intubation
(2) Use of the standard LMA insertion technique will maximize chances for obtaining a clear view of the vocal cords while performing the flexible FOI through the LMA.
(3) Success rate
(a) The true success rate of this technique in patients with unanticipated difficult airway is unknown. The technique, however, offers 96%-100% success rate in patients with anticipated difficult airway.[12,13]
(b) Troubleshooting
(i) Should the advancement of the ETT over the fiberoptic bronchoscope (FOB) placed inside the LMA-Classic prove to be difficult secondary to the S-bend in the FOB, or impaction of the ETT with the LMA aperture bars, the glottic inlet, or the anterior tracheal wall,[12] advanced flexible FOI techniques employing the use of the gum-elastic bougie (GEB), guidewire, or the Aintree intubation catheter can be attempted.

iv) Intubating LMA (ILMA, LMA-Fastrach)
(1) Advantages
(a) Prospectively documented 100% success rate in providing adequate ventilation and oxygenation, even during prolonged intubation attempts; however, several insertion attempts and repositioning maneuvers may be required.[6,13,14]
(2) Troubleshooting
(a) Use of the manufacturer's supplied straight silicone ETT is important to increase a success rate for the blind endotracheal intubation and to minimize the incidence of airway trauma.
(3) Success rates
(a) Use of an ILMA *in patients with different types of the difficult airway* is associated with an overall 96.5% success rate of blind intubation, and it may increase to 100% with the use of the FOB.[6,14]
(i) Several intubating attempts may be required, which predictably prolongs the intubation time.

v) The LMA-ProSeal and the LMA-Supreme
(1) Advantages
(a) Superior seal and better airway protection port for gastric access allows quick and reliable detection of malposition of the device and access to the gastrointestinal tract.[15]

(b) The GEB-assisted technique of the LMA-ProSeal placement is probably superior to the finger- and metal introducer-guided techniques and assures that the LMA will be optimally positioned against the upper esophageal sphincter.[15]

(c) Can be used for airway exchange with the aid of the Aintree catheter

vi) Contraindications to the supraglottic airway

(1) Relative contraindications include supraglottic pathology, glottic or subglottic airway obstruction, and patients with bleeding diatheses.

(2) Airway obstruction caused by collapsible airways or pathology at the glottic and subglottic level cannot be prevented with a supraglottic airway.

(3) Patients at risk for aspiration, patients with low chest wall/lung compliance, and high airway resistance constitute the categories of patients in whom the use of the LMA would most likely be contraindicated.



The risk for aspiration of gastric contents is the single most common limiting factor of the LMA use.[15]

vii) Esophageal tracheal airway (Combitube)

(1) The Combitube is a rescue supraglottic ventilatory device, which can be used to ventilate the patient's lungs in the esophageal as well as the tracheal position.

(2) Though designed to be inserted blindly, concomitant use of the laryngoscopic blade helps to minimize the incidence of upper airway trauma.

(3) In a recent retrospective study,[16] the Combitube reliably provided rescue ventilation in emergency airway management, when the GEB and LMA had failed.

(4) Advantages

(a) Rapid and easy airway control

(b) No need for patient's neck and head movement (implications for patients with neck injury)

(c) Effective protection against aspiration

(d) Special benefit in patients with limited access to the airway and massive upper airway bleeding or regurgitation



The rescue ability of the LMA or Combitube may be reduced by the effects of multiple preceding attempts at conventional intubation.[5]

If ventilation is not possible through either lumen of the Combitube, it is likely inserted too deep.

(5) Placement of Combitube device

(a) Insert Combitube into the oropharynx according to the manufacturer's recommendations.

(b) After insertion, inflate the esophageal (10-12 mL) and oropharyngeal (60-100 mL) cuffs and attach the ventilator bag to lumen No. 1.

(c) Ventilation should be possible in the vast majority of cases (esophageal position). If ventilation is impossible, switch over to lumen No. 2 (Combitube is in the trachea-tracheal position) (**Fig. 21-4**).

(d) If ventilation is not possible through either lumen, the Combitube is likely inserted too deep (**Fig. 21-5**).

(e) Switch the ventilation back to lumen No. 1 and slowly withdraw the Combitube until ventilation becomes possible.

READ MORE

Video-assisted laryngoscopy, Chapter 163

c) **Video laryngoscopic techniques**

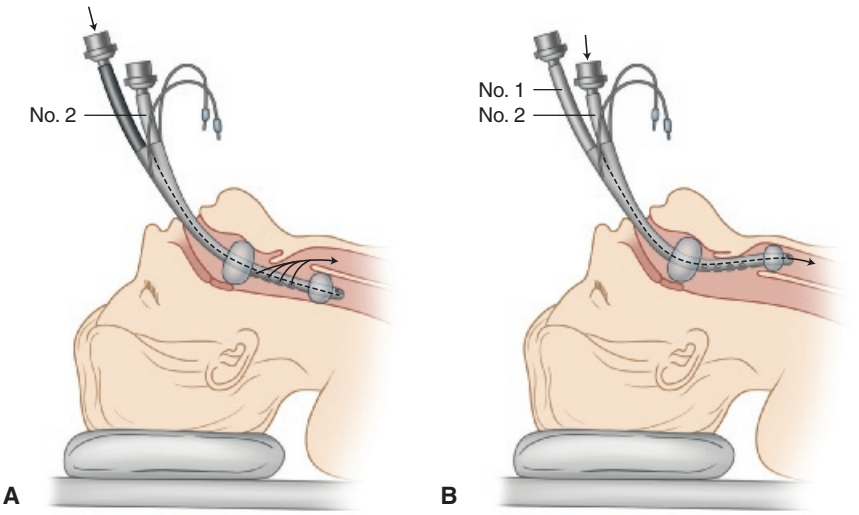
i) General overview

(1) Major technological innovation in difficult airway management

(2) Airway is visualized by high-resolution digital imager within the oropharynx.

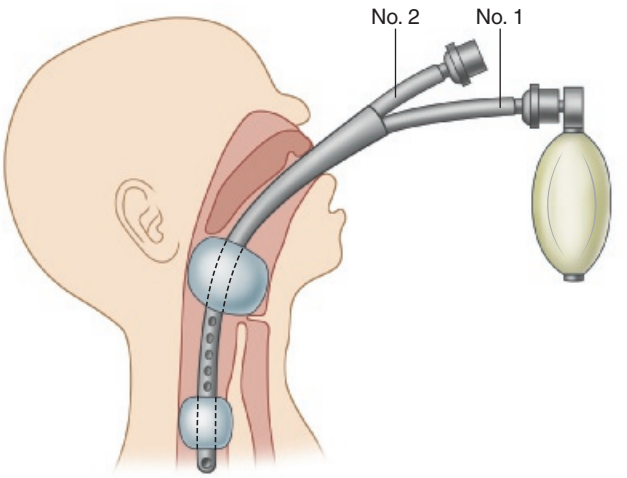
(3) Captures glottis through a wide angle of view and transmits to video monitor (**Fig. 21-6**). This improves glottic view over DL and may reduce laryngeal/airway trauma.[17]

FIGURE 21-4 Esophageal Tracheal Airway (Combitube) Positioning Esophageal (Normal) Position **(A)** and Tracheal Position **(B)**. Ventilation is possible through either lumen No. 1 **(A)** or lumen No. 2 **(B)**.



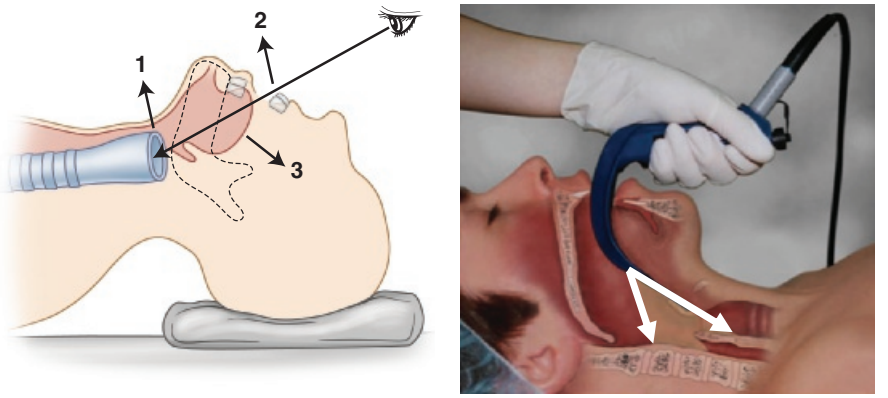
Adapted with permission from Ovassapian A, ed. *Fiberoptic Endoscopy and the Difficult Airway*. 2nd ed. Philadelphia, PA: Lippincott-Raven; 1996.

FIGURE 21-5 Combitube Malpositioned (Too Deep). Ventilation is impossible through either lumen. Ventilation should be switched over back to lumen No. 1, and the Combitube should be slowly withdrawn until the chest rise is observed.



Adapted with permission from Green KS, Beger TH. Proper use of the Combitube. *Anesthesiology*. 1994;81:513-514.

FIGURE 21-6 **Left**: Optical Axis During Direct Laryngoscopy. **Right**: Optical Axis During Video Laryngoscopy.



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(4) Unlike conventional DL, it is unnecessary to align laryngeal, pharyngeal, and oral axes to visualize the glottis.[18]
(5) Use of video laryngoscopy as compared to DL results in fewer failed intubations in cases of anticipated difficult airway.[17]

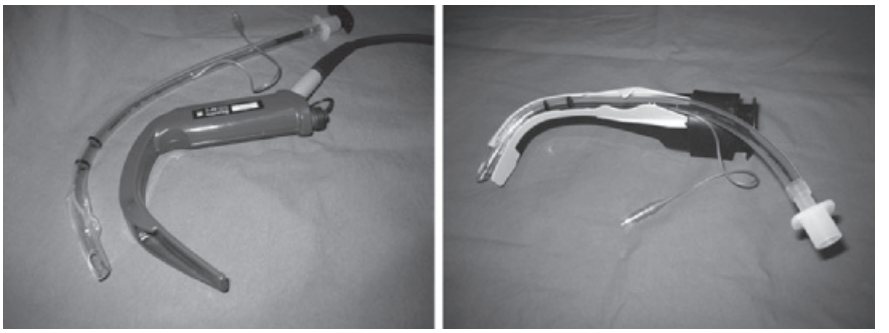
ii) Predictors of failure
(1) Video laryngoscopes are not always successful, though failure rates are low (single digits or teens, depending on if the airway difficulty was anticipated, mask ventilation was difficult or impossible).
(2) Risk factors for failure are detailed in **Table 21-9**.

iii) Types of video laryngoscopes (VLs)
(1) VLs are represented by five categories of the devices:
(a) GlideScope (GVL)
(b) VideoMac (or CMac)
(c) McGrath VLs
(d) Airway scope (AWS)

TABLE 21-9 Predictors of Video Laryngoscopy Failure

- Neck irradiation
- Anatomic asymmetry (eg, neck mass, scarred neck, contractures)
- Sniffing head position
- Decreased thyromental distance
- Limited C-spine mobility
- Mouth opening <3 cm
- Limited jaw mobility
- Obesity
- Retrognathia
- Oropharyngeal bleeding or swelling
- ENT or cardiac surgery

FIGURE 21-7 Example of Steering (GlideScope) and Channeled (Airtraq) Video Laryngoscopic Devices. **Left**: The GlideScope VL with tracheal tube and rigid stylet. **Right**: The Airtraq optical laryngoscope with tracheal tube inserted in the adjacent channel.



From Lange M, Frommer M, Redel A, et al. Comparison of the GlideScope and Airtraq optical laryngoscopes in patients undergoing direct microlaryngoscopy. *Anaesthesia*. 2009;64:323-328. Reprinted by permission of John Wiley & Sons, Inc.

(e) Airtraq (although, strictly speaking, Airtraq is an optical laryngoscope). Some of these devices are shown in **Figure 21-7**.

iv) Proper use of videolaryngoscopes

(1) Placement of VL

(a) All VLs require adequate mouth opening to accommodate the device in the patient's oropharynx.

(b) Standard cross-finger technique is used to open the mouth with the right hand, and the laryngoscope is introduced into the oropharynx in the midline axis with the left hand.

(c) The intubation should proceed in a stepwise fashion, by moving from one anatomical landmark to another.

(i) The first landmark is the uvula, and the edge of the epiglottis usually comes in the view within 2-2.5 cm upon further advancement of the blade.

(ii) The intubation sequence should alternate the operator's view between the manipulations in the patient mouth and the monitor screen: eyes in the mouth upon blade introduction—eyes on the screen immediately after the blade is in—eyes in the mouth for ETT introduction (for steering technique)—eyes on the screen.

(iii) Compared to other devices, the tip of the Pentax AWS blade is lodged under the epiglottis, as opposed to the advancement in the vallecula.

(iv) With either technique, it is important to not advance the tip of the blade too close to the vallecula or too deep under the epiglottis, in order not to obscure the wide-angle view of the glottis.



Continuously alternate attention between the video monitor/eyepiece and the styletted ETT when attempting to advance ETT through the glottic opening to avoid damaging airway soft tissues.

(2) Placement of the ETT

(a) With the steering technique, care must be taken to avoid damaging the tonsillar pillars, soft palate, or palatoglossal arch when advancing the ETT,

especially since the attention may be focused on the video screen during this time.

(b) Steering technique. With the use of the GlideScope (**Fig. 21-5**), VideoMac (CMac), and McGrath VLs, the ETT is introduced in the regular fashion—that is, independent of the laryngoscope through the patient's mouth—and typically requires the use of the ETT stylet to overcome steep angulation of the blade.

(i) Adjustment of the stylet angle or withdrawing or advancing the VL may assist with advancing the ETT through the glottic opening.



Channeled devices may be superior to the steering technique in patients with unstable c-spine injuries.

(b) Channeled technique. The AWS and Airtraq (**Fig. 21-5**) possess the integrated ETT channel guide system, where the stylet use is usually unnecessary: the ETT is advanced through the channel directly into the glottis.

(i) Advancement of the ETT through the glottic opening can sometimes be difficult even with excellent visualization of the glottis.

(ii) Advancement of a bougie through the ETT can help guide the ETT through the glottic opening.

v) Steering vs channeled VL[7,18]

(1) Irrespective of which technique is used, all VLs effectively improve the Cormack-Lehane grade by 2 (or at least 1).

(2) There is less interference of blood and secretions with visualization, and OR personnel are better able to provide airway assistance by observing the intubation procedure on the video monitor.

(3) Both steering and channeled techniques offer similar intubation success rates in anticipated difficult airway (overall success of 98%-99% steering vs 95%-99% channeled) (**Table 21-10**).

(4) Substantial differences between techniques in patients with simulated c-spine injuries have been found,[19,20] related to the speed of intubation and the movement of the C-spine, suggesting the channeled technique may be superior to the steering technique in these patients.

d) **Scalpel-bougie Tehnique for Emergency Front of Neck Access (FONA)**

i) Airway rescue procedure most often recommended by experts for the in "cannot ventilate-cannot intubate" scenario

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Scalpel-bougie technique for emergency front of neck access (FONA), Chapter 166

e) **THRIVE: transnasal humidified rapid-insufflation ventilatory exchange**

TABLE 21-10 Differences in Videolaryngoscope Techniques

	Steering	Channeled
Stylet use	Yes	No
Use of adjuncts (eg, GEB)	No	Yes
ETT maneuverability	More	Less
Intubation time vs DL	Longer	Equal or faster
Potential for upper airway trauma	More	Less
Learning curve	May be better for channeled	

GEB, gum-elastic bougie; DL, direct laryngoscopy; ETT, endotracheal tube.

i) Humidified high-flow oxygen delivered by nasal cushion at rates up to 70 L/min is used to improve oxygenation during periods of apnea.

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ENT and laser procedures, Chapter 122

ii) THRIVE is different from classic apneic oxygenation in that its high flows of fully conditioned gas actively flush oxygen in and carbon dioxide out of the patient's anatomical dead space, achieving some degree of active gas exchange. THRIVE also provides PEEP, recruiting atelectatic alveoli and reducing V/Q mismatch.

iii) Useful during difficult airway management as it prolongs time to desaturation, providing adequate saturation and stable vital signs for upwards of 30 minutes[21]

iv) Buildup of carbon dioxide and resulting respiratory acidosis is the primary limiting factor in the use of THRIVE, as gas exchange is imperfect in the absence of ventilation. Patient selection is important, since those with cardiovascular conditions such as pulmonary hypertension may not be able to tolerate the associated hypercapnia.

CHAPTER SUMMARY FOR DIFFICULT AIRWAY MANAGEMENT

Overview	Expertise in managing patients with difficult airway anatomy is critical for safe anesthesia practice. Approaches to difficult airway management are outlined in the ASA difficult airway algorithm. The anesthesiologist should be familiar with specialized airway management devices that may assist management of patients with difficult airways.
Incidence	98% patients have EMV. 1:100 have DMV. 1:700 have IMV. The majority (97%) of patients can be intubated, even if they have IMV. To achieve this high success rate, the use of alternative airway management devices and techniques may be required in at least 10% of these patients.
Approach	As a general rule, when difficult tracheal intubation is anticipated, it is the safest to secure the patient's airway awake, with FOI remaining the gold standard of care. Consider specialized equipment and alternative airway techniques where appropriate.
Specialized Equipment/ Alternative Techniques	Intubation aids such as bougie-type devices Supraglottic airway devices: LMA-Classic (Unique), ILMA, and LMA-Proseal (Supreme) for rescue ventilation, airway exchange, and the use in lieu of the ETT Video laryngoscopy devices Cricothyroidotomy (Seldinger technique) THRIVE (transnasal humidified rapid-insufflation ventilatory exchange)

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22 Supraglottic Airway Devices§

Brian J. Egan

Supraglottic airway devices sit above the glottic opening to facilitate ventilation of patients under general anesthesia. These devices, when placed properly, seal the laryngeal inlet to allow easy ventilation of the patient.

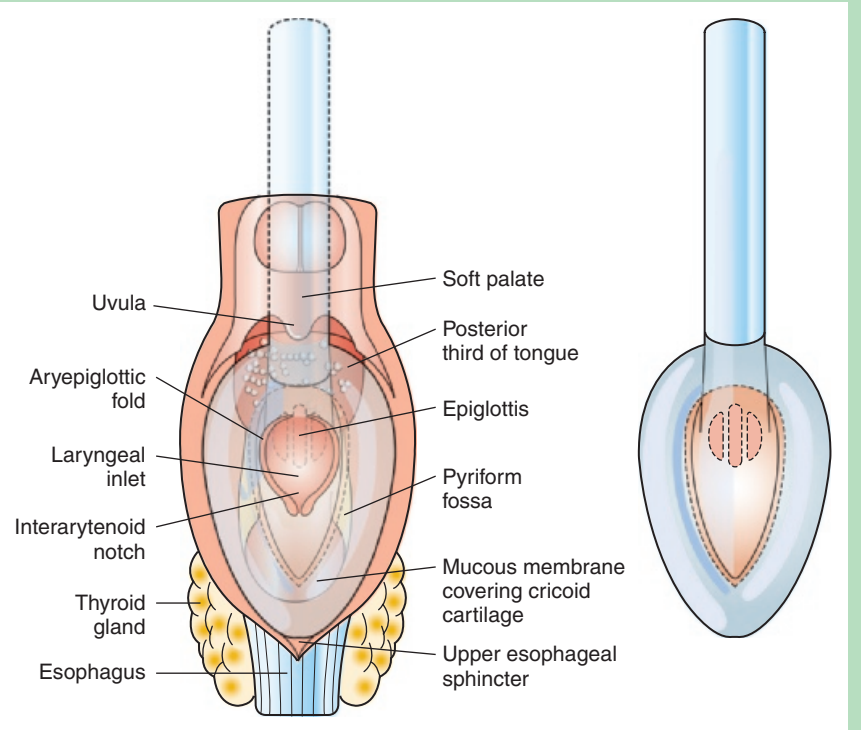
1) **Description of different types of laryngeal mask airways (LMAs)**
   a) **Classic LMA and Unique LMA**
      i) The Classic
         (1) The original LMA
         (2) Reusable for 40 uses
      ii) The Unique
         (1) The single-use version of the Classic
      iii) All versions are latex free.
      iv) Function:
         (1) The tip of the mask sits in the hypopharynx, at the level of the upper esophageal sphincter (UES), with the lateral aspects of the mask spreading into the pharynx and the superior component displacing the base of the tongue forward (**Fig. 22-1**).
         (2) The glottic opening and epiglottis sit within the "bowl" formed by this chamber; spontaneous ventilation (SV) or positive-pressure ventilation (PPV) is effectively channeled via the bowl and into the respiratory tract.[1]
      v) Designed for SV, though PPV to a maximum of 20 cm H_2O has been safely utilized without any sequelae of gastric insufflation.
      vi) Can be used as a rescue airway device for difficult intubation/ventilation scenarios.[2]



The LMA can be used as a rescue airway device for situations where difficult intubation and/or ventilation occur.

   b) **ProSeal LMA**
      i) The cuff of the ProSeal rests deeper in the hypopharynx, thus improving the seal and permitting PPV up to 30 cm H_2O.
      ii) A gastric drainage conduit was added to allow contents of the gastrointestinal tract to be safely suctioned, bypassing the mouth and respiratory tract.
         (1) A 14-French orogastric tube can be placed through the drainage tube to decompress the stomach.
   c) **Flexible LMA**
      i) Designed to optimize LMA use in shared airway cases in which surgeons operate in close proximity to the airway.
         (1) The Flexible LMA can be easily positioned out of the surgeon's working space.
      ii) Wire-reinforced, flexible breathing tube.
         (1) Allows maintenance of proper mask position in the hypopharynx while the tube is proximally displaced at extreme angles.
      iii) The tube is longer and of smaller diameter.
         (1) Increases resistance to air flow through the LMA.
   d) **Supreme LMA**

FIGURE 22-1 Dorsal View of the LMA Cuff Showing Position in Relation to Pharyngeal Anatomy.



The LMA consists of an inflatable cuff connected to a tube. This figure shows the final position of the LMA in the airway after correct placement. The tip of the mask sits in the hypopharynx at the level of the UES with the lateral aspects spreading into the pharynx and the superior component displacing the base of the tongue forward. Adapted from http://www.lmana.com/docs/LMA_Airways_Manual.pdf.

i) Like the ProSeal, a "drain tube" terminates in the tip of the mask and should sit in the UES.
ii) Has a built-in bite block, a more rigid breathing tube that facilitates nondigital placement, and a fixation-tab and dual-tube configuration that minimizes rotational displacement.

e) **Intubating LMA (Fastrach) and LMA C-trach**
i) Designed to facilitate tracheal intubation.
ii) The basic components of the Classic LMA are retained but modified to achieve both blind and videoscope-guided placement of an ETT.
iii) After successful ETT placement, they are typically removed from the airway.
(1) However, they can remain in situ when conditions make removal prohibitive.

f) **Other supraglottic airway devices**
i) Classification systems have been proposed to help categorize newer devices by common features and mechanisms of seal. However, Cook and Howes' 2011 proposal to divide supraglottic airways into first- and second-generation devices has been the most widely adopted.[3]

(1) First-generation devices, such as the Classic and Unique LMAs, are characterized as a simple airway tube.
(2) Second-generation devices, such as the ProSeal and Supreme, are defined as having features that augment safety, in particular a gastric drainage tube which may help reduce the risk of aspiration.[3]

ii) Evaluation of new devices
(1) New devices should ideally be evaluated on two parameters: efficacy and safety. Establishing efficacy (though not necessarily superiority or even equivalency to the LMA) may be achieved in studies of a small number to several hundred patients.[4]
(2) Establishing a safety profile equivalent to that demonstrated by the LMA family of airways is more challenging. Due to the relative safety of the LMA, a competing supraglottic device would require use in several hundred thousand to millions of patients to achieve the necessary power to demonstrate noninferiority, and such studies are unlikely to be undertaken.[3,5]

iii) Choosing from among competing devices
(1) Choice of the best supraglottic airway for a patient will depend on practitioner experience and preference, intended use (eg, as a primary airway vs a conduit for endotracheal intubation), and patient-related factors (eg, body habitus, concern about aspiration risk)[5] (**Table 22-1**).
(2) The following table contains details about the LMA family of airways as well as several other popular brands including the Air-Q and the I-gel and may help in decision-making.

2) **Indications for supraglottic airway device use**
a) Traditionally, LMAs were used in elective surgical procedures requiring general anesthesia of a duration longer than what could be comfortably provided with a mask general anesthetic but unlikely to require paralysis.
b) Now LMA use has expanded to include more complex surgeries, as a rescue airway device and as a conduit for endotracheal intubation. A survey in the United Kingdom found that in 2011, 56% of general anesthetics were being performed with an LMA.[6]
c) **Absolute and relative contraindications**
i) Although rates of aspiration are extremely low in large retrospective studies, those studies are subject to the selection bias introduced through proper patient selection.[7] Therefore, LMAs should be avoided in all patients with known risk factors for aspiration.
ii) Relative contraindications:
(1) Surgical procedures which require sharing of the airway
(2) Positioning the operating table with the patient's head away from and poorly accessible to the anesthesiologist
(3) Prone surgical procedures
(4) Procedures of longer duration (though no absolute maximum safe duration of use is known)
(5) Procedures requiring significant Trendelenburg positioning.
(6) Procedures requiring paralysis, particularly laparoscopic procedures, are relatively contraindicated. Although multiple studies have demonstrated the safe use of the LMA during laparoscopy in conjunction with paralysis (Maltby and Abdi), care should be taken to avoid this technique in procedures with high insufflation pressures, long (>60 minutes) duration, or nonsupine positions (eg, steep Trendelenburg).

3) **Advantages of supraglottic airway devices**
a) Minimal hemodynamic changes with placement due to decreased stimulation with LMA placement.

TABLE 22-1 Choice of Airway for Routine Use during Elective Anaesthesia

	Overall Insertion Success	Speed of Insertion	Quality of Ventilation	Airway Seal	Aspiration Protection	Avoiding Airway Trauma	Avoiding Sore Throat	Sum
Maximal score	4	3	4	3	2	3	3	22
BVM	2	3	1	0	0	3	2	11
cLMA	3	3	2	1	1	2	2	14
ILMA	4	3	2	2	1	2	1	15
PLMA[a]	3	3	3	3	2	2	2	18
i-gel[a]	3	3	3	2	2	3	3	19
SLMA[a]	3	3	3	2	2	2	2	17
LTS II[a]	2	3	2	3	2	2	2	16
Air-Q	3	3	3	3	2	2	2	18

Aspects of device performance are given a maximal score according to importance in the clinical circumstance. Each device is then scored for each aspect of performance. The sum of these scores gives an indication of the most and least suitable device.
[a]Second-generation device.
BVM, bag–mask ventilation; cLMA, classic laryngeal mask airway (LMA); ILMA, intubating LMA; LTS II, laryngeal tube suction mark II; PLMA, ProSeal LMA; SLMA, Supreme LMA.
Modified from Cook TM, Kelly FE. Time to abandon the 'vintage' laryngeal mask airway and adopt second-generation supraglottic airway devices as first choice. *Br J Anaesth*. 2015;11(4):497-499.

b) Low risk of dental/perioral trauma.
c) First-pass success rates of up to 89%, and successful use in up to 98% of patients appropriately selected for LMA use.[8]
d) Minimal airway reactivity upon placement, assuming appropriate anesthetic depth.
e) Low incidence of postoperative sore throat.
f) Compared to a mask general anesthetic, the LMA frees the anesthesiologist's hands from having to hold a mask.
g) Minimizes coughing/pharyngeal irritation on emergence.
h) Avoids tracheal intubation in special patient populations (eg, singers).

4) **Disadvantages of supraglottic airway devices**
a) **PPV is limited to peak airway pressures of 20 cm H_2O with the Classic LMA and 30 cm H_2O with the ProSeal and Supreme LMAs.** The newer devices, such as the Igel and AirQ, have been found to have similar oropharyngeal leak pressures to the LMA supreme.[9]
i) This limitation is due to the pressure at the lower esophageal sphincter (LES) which is approximately 20 cm H_2O.
ii) Ventilation with airway pressures >20 cm H_2O carries the risk of gastric insufflation.



Patients at high risk of aspiration may not be appropriate candidates for a LMA.

b) **Although the aspiration risk is reported as <0.02%, the airway is not completely protected from aspiration of gastric contents.[7]**
i) Patients at high risk of aspiration (eg, obese, pregnant, gastroesophageal reflux disease, full stomach) may not be appropriate candidates for a LMA.
c) **Adequate function of the LMA is dependent on appropriate position in the hypopharynx.**
i) It is susceptible to displacement with changes in patient position and/or manipulation of the head and neck.
d) **Reported rare, but severe, neurovascular complications due to trauma secondary to LMA use include:**
i) Lingual nerve injury[10]
ii) Recurrent laryngeal nerve injury[11]
iii) Hypoglossal nerve injury[12]
iv) Tongue cyanosis[13]

5) **Placement of an LMA—standard technique**
a) **Preparation of the LMA**
i) Completely deflate the cuff of the mask with a syringe into a flat, smooth, wedge-shaped tip to minimize down-folding of the epiglottis on placement.
ii) Place water-soluble lubricant on the posterior surface of the cuff only.
(1) Placement on the ventral surface could cause airway obstruction and aspiration of lubricant, as well as laryngospasm secondary to vocal cord irritation by lubricant.
(2) Lubricant should be placed right before LMA insertion to minimize drying.

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Atlas of clinical procedures: Laryngeal mask airway insertion, Chapter 161

b) Preparation of the patient
i) Similar to endotracheal intubation, LMA placement is facilitated by first placing the patient in a position of neck flexion and head extension (ie, the "sniffing" position).
ii) Preoxygenation commences with patient breathing 100% oxygen through a facemask resting with adequate pressure to prevent entrainment of room air. This will optimize apneic time following induction.
iii) Induction: In a manner similar to induction of a patient in whom endotracheal intubation has been planned, induction commences with hypnotic agent of choice.

(1) Propofol is the preferred agent for its ability to blunt airway reflexes.
(2) The use of premedications depends on physician preference. Many advocate the avoidance of opioid during induction in order to facilitate the rapid return of spontaneous ventilation.
(3) Generally, paralytic is unnecessary for LMA placement because of both the high success rates achieved without its use as well as the desire to rapidly return the patient to spontaneous ventilation. However, anecdotally, a small dose of succinylcholine may be utilized to facilitate placement when difficult.

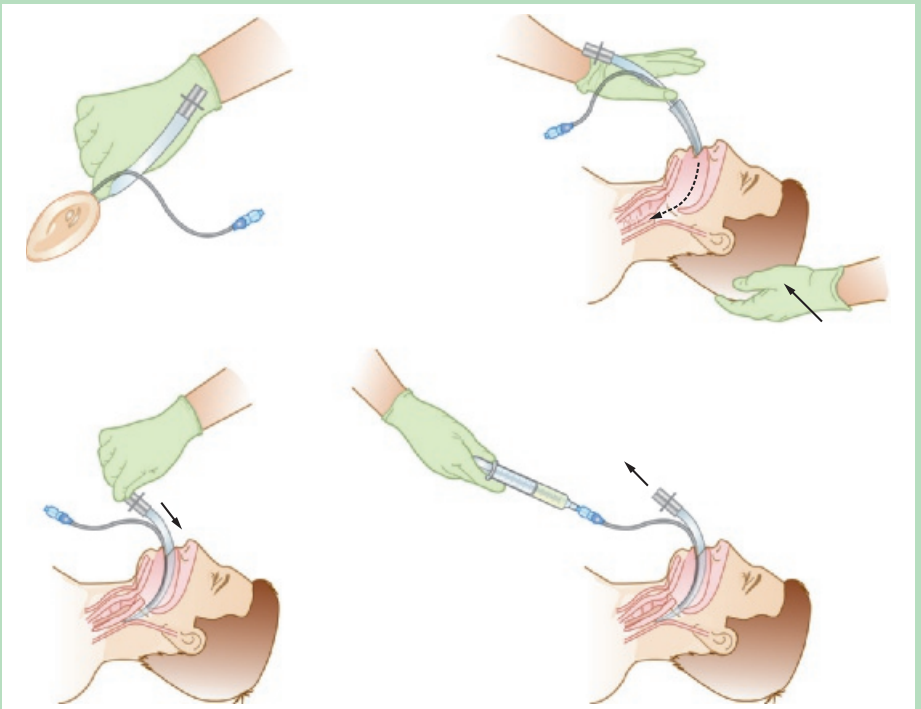


A small dose of succinylcholine may be utilized to facilitate supraglottic airway device placement when difficult.

(4) Mask ventilation prior to LMA insertion is practitioner dependent. Many argue that should mask ventilation prove difficult, the next step in the difficult airway algorithm is placement of an LMA, and therefore assessing ease of mask ventilation is an unnecessary step.

c) **Insertion (Fig. 22-2)**[14]
   i) LMA Classic, Unique, and Flexible
      (1) Grasp the tube in the dominant hand, and place the index finger in the space between the tube and the deflated cuff of the mask.

FIGURE 22-2 Technique for Insertion of LMA.



Top left: Grasp the airway tube in the dominant hand and place the index finger between the tube and the deflated cuff of the mask. **Top right:** With the nondominant hand adjusting the head into the "sniffing" position, place the tip of the mask firmly against the palate and advance the LMA along the palate and into the posterior pharynx with the initial direction of force directed toward the operator's umbilicus. **Lower left:** The nondominant hand is then used to push the LMA further into the hypopharynx until resistance is encountered at the UES. **Lower right:** Once inserted, the cuff of the LMA is insufflated with just enough air to create a seal. Adapted with permission from Walls RM, Murphy MF. *Manual of Emergency Airway Management*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008:125-127.

(2) Use the nondominant hand to place the patient's head in the "sniffing" position.
(3) With the flat side of the cuff facing the patient's head, place the tip of the mask firmly against the hard palate and advance the LMA along the palate, above the tongue, and into the posterior pharynx in a smooth continuous motion; the initial direction of force should be directed toward the operator's umbilicus.
(4) The nondominant hand is then used to push the LMA further into the hypopharynx until resistance is encountered at the UES; 7-10 cm of LMA should protrude from the oral cavity.
(a) Withdraw the index finger while stabilizing the LMA with the opposite hand.

d) **Completion**
i) Inflate the mask with just enough air to create a seal.
(1) Maximum inflation volumes (to keep the pressure <60 cm H_2O) are printed on each device.
ii) A slight rise (1-2 cm) of the LMA out of the mouth on inflation suggests that the LMA is appropriately positioned.
iii) Connect the circuit, and confirm proper placement by gentle manual ventilation.
(1) Observe for chest rise and the presence of $ETCO_2$, and listen for an audible leak, evidence of airway obstruction, and bilateral breath sounds.

READ MORE
Laryngeal mask airway insertion, Chapter 161

e) **Troubleshooting**
i) Difficulty ventilating through the LMA
(1) Possible etiologies:
(a) Inadequate anesthetic depth.
(b) Epiglottis has folded over during insertion, thus obstructing the airway.
(c) Tip has migrated into the airway rather than the esophageal inlet, thus malpositioning the LMA.
(d) Tip has folded backward during placement rather than advancing into the esophageal inlet, thus causing malposition of the LMA.
ii) With correct placement, an ovoid swelling in the anterior of the neck is often visible.
iii) When in doubt, take it out, ensure adequate depth, and reinsert.



When in doubt of LMA proper positioning, take it out, ensure adequate depth, and reinsert.

f) **Ventilation**
i) Spontaneous ventilation is preferred for the Classic LMA and Unique LMA.
(1) Well tolerated when anesthetic depth is appropriate for the level of stimulation.
(2) Changes in respiratory pattern (eg, sudden increase in tidal volume or respiratory rate) suggest inadequate depth of anesthesia.
(3) Positive-pressure ventilation can be done safely, with some caveats.
(a) To avoid gastric insufflation, keep peak airway pressures <20 cm H_2O (Classic, Unique, Flexible) or <30 cm H_2O (ProSeal, Supreme); this is because the pressure at the LES is ~20 cm H_2O.
(b) Pressure-controlled ventilation may help to avoid excessive peak pressures.
(c) Pressure support ventilation may also be useful in the spontaneously breathing patient in order to maintain adequate tidal volumes and, possibly, reduce work of breathing.

g) **Emergence and removal of LMA**
i) Ensure the patient is breathing spontaneously and not requiring ventilatory support.
ii) No need to suction the airway because the cuff provides adequate protection from secretions.

(1) Suctioning may trigger airway reactivity or laryngospasm if the patient is lightly anesthetized.

iii) Leave the cuff inflated on removal.

(1) Prevents pooled secretions on the posterior surface of the cuff from falling into the airway.

iv) The LMA can be removed either when the patient is deeply anesthetized or when the patient is fully awake.

(1) Awake removal of LMA takes place during Stage 1 of anesthesia.

(a) Do not disturb the patient until swallowing is observed. This facilitates a smooth emergence.

(b) Remove the LMA only when the patient can open his or her mouth on command.

(2) "Deep" removal of LMA takes place during Stage 3.

(a) If the patient is inadequately anesthetized (Stage 2), there is a risk of laryngospasm; vigilance must be used when monitoring for this complication throughout the emergence phase.

(b) After confirming a deep plane of anesthesia (eg, assess for central gaze of eyes, dilated pupils [assuming no other factors affect pupillary size]), remove the LMA from the oral cavity.

(c) Consider 100% oxygen for a brief period before removing LMA to optimize apneic time in the event of transient upper airway obstruction after LMA removal.

(d) Consider placing the patient in a lateral decubitus position after removal of LMA to facilitate outward drainage of oral secretions.

READ MORE
Chapter 6: Emergence and postoperative issues in anesthesia, for more discussion on the stages of anesthesia.

h) **Complications associated with supraglottic airway device use**

i) Aspiration

(1) Regurgitation of gastric contents occurs rarely, and aspiration of gastric contents even more rarely, in part because of LMA design and appropriate patient selection for LMA use.

(2) If regurgitation does occur, place the patient in the head-down (Trendelenburg) position.

(a) This encourages gastric contents to flow out the mouth.

(3) Leave the LMA in place.

(4) Suction down the airway tube with a soft suction catheter.

(a) If secretions are aspirated from the airway, consider suctioning via fiberoptic scope or replacing the LMA with an ETT.



Regurgitation and aspiration are risks with LMA use. Should regurgitation occur, leave the LMA in place and first suction the airway tube.

ii) Cuff trauma

(1) The cuff of the LMA can exert traumatic pressure on vulnerable anatomic structures, including the

(a) Mucosa

(b) Hypoglossal nerve[12]

(c) Lingual and superior laryngeal nerves

(2) Avoiding cuff trauma

(a) Use the minimal amount of air to create an adequate seal.

(i) Do not exceed the recommended maximum cuff volumes.

(ii) Additional air rarely improves a poorly ventilating LMA.

1. It is better to remove and replace the LMA.

(b) Avoid any tension on the airway tube (eg, via the anesthetic circuit) because it may be translated distally to the cuff and thus to the tissues against which the cuff approximates.



Additional air in the cuff rarely improves LMA function. Traumatic pressure may lead to nerve injuries, which can have devastating consequences.

iii) Laryngeal injuries
(1) There are rare cases of arytenoid cartilage dislodgement, vocal cord injury, and/or paralysis.
(2) Proper placement of the LMA obviates direct contact with glottic structures.
(3) The period of greatest resistance to LMA advancement occurs when the tip slides behind the tongue.
a) Typically, there is a notable decrease in resistance once this is achieved.
b) Persistent resistance to advancement should be treated with caution.
(i) Removing the LMA to the level of the palate and readvancing is advised.

i) **Other uses for supraglottic airway devices**
i) Airway emergencies
(1) The LMA Classic, Unique, ProSeal, and Supreme are part of the ASA difficult airway algorithm.
(a) May be used in situations of difficult mask ventilation and/or intubation.[2]
(b) Depending on the clinical scenario, the availability and use of a rescue airway with gastric decompression capability may be favored.
(2) Fiberoptic conduit
(a) The exit point of the LMA airway tube through which the fiberoptic scope emerges from the LMA permits visualization of the glottic opening or epiglottis.
(i) This allows examination of vocal cord function during certain surgeries (eg, thyroid procedures).
(ii) Also provides a conduit for fiberoptic intubation.
(b) Modifications of the disposable Unique have been suggested to facilitate fiberoptic intubation.
(i) Removing the airway bars at the distal end of the airway tube and shortening the tube to ensure complete passage of the ETT balloon after railroading (defined as advancing the ETT over a fiberoptic scope or Aintree catheter, which has been properly positioned in the airway).[2,11-15]
(ii) Use of an Aintree intubation catheter can also facilitate fiberoptic intubation through an LMA.[16]
(iii) Certain brands of supraglottic airways may make use of an airway tube of larger diameter, thus accommodating passage of a larger ETT.

CHAPTER SUMMARY FOR SUPRAGLOTTIC AIRWAY DEVICES	
Definition	Seals off the laryngeal inlet from the gastrointestinal inlet using a cuffed mask connected to a tube
Selection	There are a variety of first- and second-generation devices available. Each has strengths and weaknesses that should be understood prior to use.
Insertion	• Place the tip of the mask firmly against the palate. • Advance the LMA along the palate and into the posterior pharynx with the initial direction of force directed toward the operator's umbilicus. • Keep advancing until resistance is encountered at the upper esophageal sphincter.
Completion	• Reinflate the mask with just enough air to create a seal. • A slight rise of the LMA out of the mouth on inflation suggests good position.
Emergence	• Avoid suctioning the airway. • Leave the cuff inflated on removal. • If removing with the patient awake, leave the patient undisturbed until swallowing is observed. Remove the LMA only when the patient opens his or her mouth on command.

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23 Fiberoptic Intubation§

Adam W. Baca

The term fiberoptic intubation refers to a technique of establishing endotracheal intubation using the assistance of a fiberoptic device capable of bending light and providing a view of the patient's glottic aperture. This includes handheld flexible bronchoscopes as well as video laryngoscope technology. The key to successful airway management using a fiberoptic technique depends upon several factors. These include extensive knowledge of airway anatomy, adequate local anesthesia in the airway (if awake), a thorough preoperative airway examination, correct patient positioning to facilitate intubation, and the ability to maneuver the fiberoptic scope through the airway. If proceeding with an awake fiberoptic intubation, the patient must also be cooperative and well informed of the anesthetic plan.

1) **Indications for fiberoptic intubation (FOI)**[1]
   a) **Patient with history of known difficult intubation or physical exam that is suggestive of such.**
   b) **Mandibular hypoplasia**
   c) **Fixed cervical spine disease (eg, ankylosing spondylitis)**
   d) **Acromegaly**
   e) **Awake intubation should be considered in the following situations if a difficult airway is anticipated:**
      i) High aspiration risk
         (1) Full stomach/NPO guidelines not met
         (2) Poorly controlled GERD
         (3) 2nd/3rd Trimester pregnancy
         (4) Poorly controlled diabetes with gastroparesis
      ii) Undesirable neck movement (unstable or uncleared cervical spine)
      iii) When self-positioning for surgery is desirable
      iv) High risk for difficult ventilation
      v) Severe obesity
      vi) Lower and/or upper airway obstruction (eg, tracheal compression, airway edema, foreign body, laryngeal or pharyngeal tumors, neck masses, hematomas, Ludwig angina, airway burns)
      vii) Radiation to the neck after cancer surgery
      viii) Facial trauma
      ix) Congenital syndromes (eg, Pierre Robin sequence, Treacher Collins syndrome, Down syndrome)

READ MORE
Obesity, Chapter 64

2) **Relative contraindications to fiberoptic intubation**
   a) **Lack of time**
      i) Fiberoptic techniques can require significant setup time, especially if planning on keeping the patient awake (gather and test equipment, patient positioning, anesthetizing the airway).
      ii) If the patient is decompensating, becoming increasingly altered or obtunded, then other options, including surgical airway, should be considered.
   b) Patients who are unwilling or unable to cooperate or refuse the procedure
      i) Inability to cooperate may be due to altered mental status, agitation, cognitive disability, or anxiety.

c) Active bleeding from upper or lower airway which is not relieved (or sometimes made worse) by suction.
d) **In patients who have limited mouth opening, nasal FOI is an option.**

3) **Preparation for fiberoptic intubation**
   a) **Checklist for fiberoptic cart**
      i) Video laryngoscope or fiberoptic bronchoscope (FOB) and light source
      ii) Endotracheal tube (ETT) and empty syringe to inflate ETT cuff
         (1) Have various sizes available; if patient has any tracheal obstruction or subglottic stenosis, a smaller ETT may be necessary.
      iii) Placing the ETT in a bottle of warm water or saline can soften the tube. This may result in less mucosal damage and bleeding into the airway.
      iv) Intubating/endoscopic oral airway[2]
         (1) Berman
            (a) Tubular along the entire length.
            (b) Available in different sizes.
            (c) Less rotational stability compared to Ovassapian airway.
            (d) Split along its side allows for lateral removal of the Berman airway to be removed once ETT is in place.
         (2) Ovassapian
            (a) Designed to provide an open air space in the oropharynx, to protect the FOB from being bitten by the patient
            (b) Available in one size
            (c) Accommodates an ETT up to 8.5 mm in inner diameter
            (d) Best suited and specifically designed for fiberoptic intubation
         (3) Williams
            (a) Designed for "blind" orotracheal intubation.
            (b) Available in two sizes, 90 and 100 mm.
            (c) Due to its design, the tip of FOB cannot be maneuvered in AP or lateral direction.
            (d) At times, this airway must be partially withdrawn to allow better exposure of the vocal cords.
            (e) ETT circuit adapter must be removed prior to withdrawing a Williams airway.
      v) Tongue depressor
      vi) Gauze (4″ × 4″)
      vii) Lubricant
      viii) Suction
      ix) Auxiliary source of oxygen
      x) Emergency airway equipment, including cricothyroidotomy kit and/or a trained physician who can perform a surgical airway expeditiously.
   b) **Preparation and inspection of the fiberoptic scope**
      i) Place the scope and all the necessary equipment on the cart next to the patient's bed, so it is easily accessible.
      ii) Test the adjustment focus.
      iii) Test all the operator's functions on the scope: anteflexing and retroflexing.
      iv) Choose and attach the appropriate size ETT to the scope.
         (1) Generally, the ETT is at least 1.5 mm larger in internal diameter than the diameter of FOB (the fiberoptic shaft diameters are ~3.5-5.5 mm).
         (2) Remove the connector from ETT prior to doing so, as it gives you more length on the scope and more maneuverability (do not lose the connector).
         (3) Lubricate the shaft of the scope prior to attaching the tube.



Placing an ETT in a warm solution prior to intubation softens the tube, and it is less likely to cause mucosal damage.

v) Turn on the light source.
vi) Connect the port to the oxygen.
(1) This improves oxygenation and displaces some secretions during FOI.
(2) It is also possible to connect the suction to the appropriate port on the scope, but it is not very powerful.

4) **Operation of a fiberoptic scope**[3]
a) **Overview of device (Fig. 23-1)**
b) **Manipulation of scope tip (for a right-handed individual)**
i) The head of the fiberoptic scope is held in the right hand, with the right thumb on the control lever.
(1) Alternatively, FOB could be held in the nondominant hand, if it is more comfortable for the operator.
ii) Anteroflexion
(1) Pull the right thumb down, to curve the distal end of the scope upward.
iii) Retroflexion
(1) Push the right thumb up, to curve the distal end of the scope downward.
c) **Directing movement of fiberoptic scope**
i) While the right hand is positioned at the control section of the fiberoptic scope, hold the bronchoscope shaft in the left hand about 15 cm from the distal tip.

FIGURE 23-1 Overview of a Fiberoptic Bronchoscope



(1) It is crucial to keep bronchoscope taut between the right and the left hands so that the orientation of the tip is the same as that of the control lever.

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Difficult airway management, Chapter 21

5) **Preparation of the patient for awake fiberoptic intubation**
   a) **Premedication**
      i) Glycopyrrolate 0.2-0.3 mg IV can be administered 15-20 minutes prior to FOI as an antisialogogue.
         (1) It is important to administer the drug early in order for it to be effective during the FOI attempt.
   b) Explain to the patient the reason for the awake intubation and how it will proceed.
   c) Judiciously sedate the patient, being careful not to oversedate him/her.
      i) There are multiple techniques for sedation; choose one that is most comfortable (**Table 23-1**).[4]
   d) **Apply anesthesia to the airway.**
      i) The airway can be anesthetized by topicalization or nerve blocks or a combination of both (**Table 23-2**).
         (1) Topicalization of oral pharynx can be done with atomizers, nebulizers, or direct application of lidocaine or with aerosol benzocaine.
         (2) For nasal route, cocaine can be used to topicalize the nasal passages, as well as cause vasoconstriction to decrease the possibility of epistaxis.
            (a) Phenylephrine (Afrin) spray can be mixed with lidocaine for vasoconstriction.
   e) **Common airway nerve blocks[5]**
      i) Glossopharyngeal nerve (cranial nerve IX) and lingual branch block
         (1) Carries the sensory innervation of the pharynx, tonsils, and posterior third of the tongue.
         (2) The block of this nerve depresses the gag reflex during the awake (FOI).
         (3) In most patients, the local anesthetic applied topically to the back of the tongue is sufficient.
            (a) Usually, 5% lidocaine jelly used as lollipop is sufficient.



The glossopharyngeal nerve block depresses the gag reflex during the awake fiberoptic intubation.

TABLE 23-1 Profiles of Available Sedation Drugs

Drug	Dose	Frequency	Side Effects	Reversal Agents
Midazolam	0.25-4 mg IV	1-5 min	Respiratory depression	Flumazenil, 0.2 mg IV q1min up to 1 mg
Fentanyl	Load up to 1 µg/kg, then 12.5-50 µg IV	5-10 min	Respiratory depression	Naloxone, 40-400 µg IV q5-10 min
Remifentanil	0.01-0.5 µg/kg/min IV	Infusion	Respiratory depression	
Dexmedetomidine	0.2-0.7 µg/kg/h IV	Infusion	Hypotension and bradycardia	
Ketamine	10-50 mg IV		Hallucinations, which can be attenuated with midazolam administration, sialogogue, arrhythmias	

TABLE 23-2 Profiles of Available Anesthetics

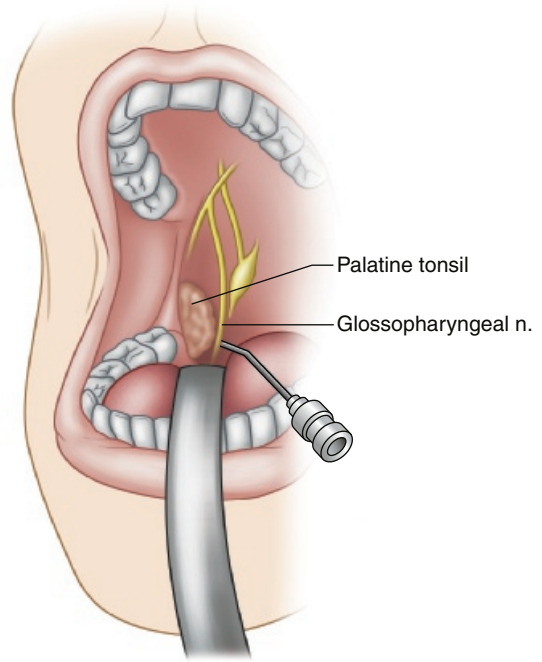
Drug	Maximum Dose	Additives	Route	Contraindications	Complications
Cocaine 4%	1.5 mg/kg		Nasal	Patients with ischemic heart disease, preeclampsia, on monoamine oxidase inhibitors	Tachycardia, hypertension
Benzocaine spray	100 mg		Oral	Local anesthetic allergy	Methemoglobinemia Caution: not to exceed recommended dose of a single 0.5-2 s spray
Lidocaine • 4% • 2% viscous • 5% jelly	5 mg/kg with epinephrine or 7 mg/kg without epinephrine	• Afrin spray (oxymetazoline) • 1% Phenylephrine	Oral	Local anesthetic allergy (although extremely rare in amide category)	CNS toxicity Cardiovascular toxicity

i) If this does not produce satisfactory anesthesia, injection of 2 cc of 1% lidocaine with 25-gauge needle at the caudal part of tonsillar pillar bilaterally will block the nerves.
   (1) Use a tongue depressor to move the tongue out of the way in anteroinferior direction (**Fig. 23-2**).

ii) Superior laryngeal nerve block
   (1) Innervates larynx (provides sensory innervation to the epiglottis, arytenoids, and vocal cords).
   (2) Methods of anesthetizing the distribution of the superior laryngeal nerve.
      (a) Atomizer
      (b) Nebulizer
      (c) Trickling local anesthetic down the posterior tongue
      (d) Depositing local anesthetic through the fiberoptic scope
      (e) Blocking the nerve directly
   (3) The superior laryngeal nerve can be blocked as it passes into the larynx through the thyrohyoid membrane (**Fig. 23-3**).
      (a) Fully extend the patient's neck. The hyoid bone is identified by palpation.
      (b) Use syringe with ~3 mL of 1% lidocaine, fitted with 25-gauge needle.
      (c) Insert the needle until it rests on the lateral portion of hyoid bone.
      (d) Withdraw it slightly and walk it off the hyoid bone in the caudad direction, and advance a couple of millimeters until it passes through the thyrohyoid membrane (slight resistance should be felt).
      (e) After negative aspiration for air and blood, inject 2-3 mL. Repeat on the other side.

iii) Transtracheal nerve block
   (1) Provides anesthesia to the area between carina and vocal cords.
   (2) The block should be performed about 1 minute prior to the start of bronchoscopy.
   (3) Use 10-cc syringe, filled with 3 cc of 4% lidocaine with 23- or 23-gauge needle.

FIGURE 23-2 The Palatoglossal Arch



Intraoral Approach to the Glossopharyngeal Nerve.

(4) Place patient in the supine position with neck extended (**Fig. 23-4**).
(5) Introduce the needle at the level of cricothyroid membrane, directed posteriorly, perpendicular to the skin, continuously aspirating.
(6) Once you have positive air aspiration, inject local anesthetic, as the patient inhales, and remove the needle quickly. The patient will cough.
(7) Differences in method
  (a) Some advocate using a catheter technique and advancing the catheter once positive aspiration is achieved and leaving it in to later inject local anesthetic through the catheter.
  (b) Others advocate avoiding transtracheal block and spraying local anesthetic through the fiberoptic scope as the scope is advanced through the airway.

f) **Patient positioning**
  i) Supine position, with the endoscopist at the head of the table
  ii) Sitting position, with the endoscopist facing the patient

g) **Oxygenation and suction**
  i) Oxygen via nasal cannula in spontaneously breathing patient.
  ii) Oxygen flow (10 L/min) through the suction port in asleep patient.
  iii) Preoxygenation is critically important when performing asleep fiberoptic intubation.



Give supplemental oxygen via nasal cannula while performing fiberoptic intubation.

FIGURE 23-3 Superior Laryngeal Nerve Block

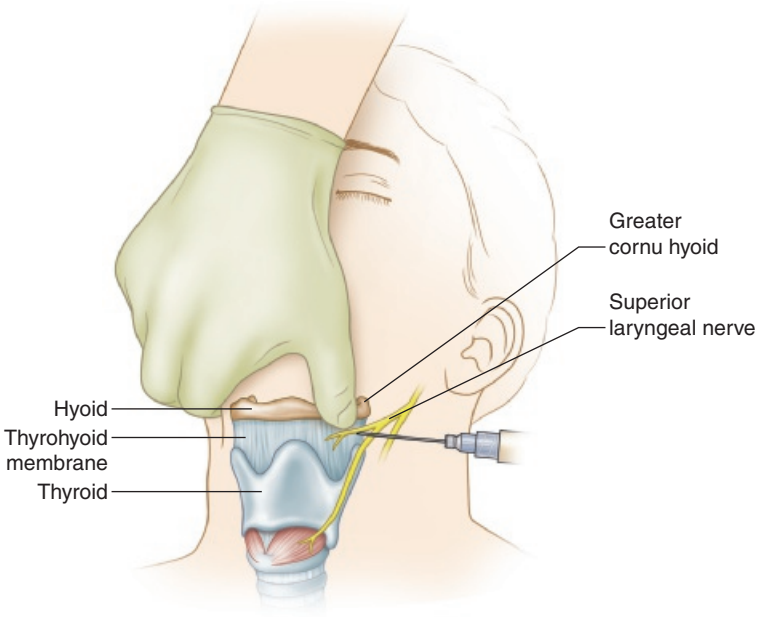
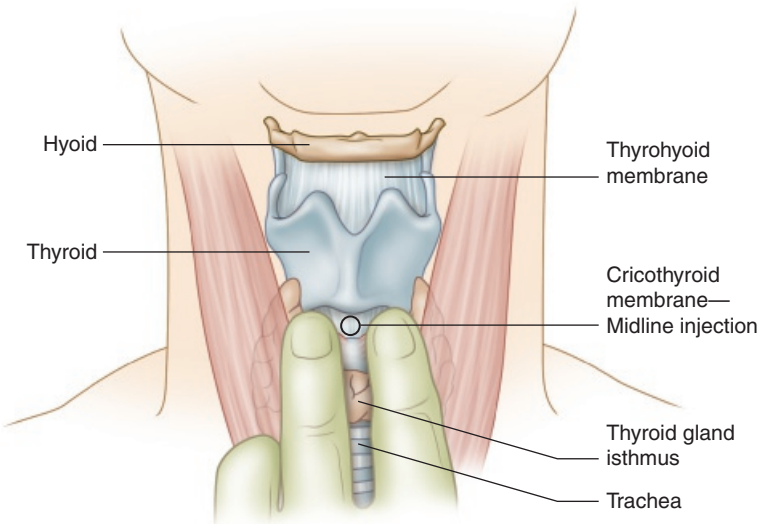


FIGURE 23-4 Transtracheal Block



6) **Performing fiberoptic intubation**[6]
   a) **Place ASA standard monitors and any additional monitors as necessary.**
   b) Position the OR table and use a step stool so that the proceduralist is able to keep the fiberoptic scope taut in order to maximize the coronal plane rotational control.
   c) Prepare equipment by inserting the fiberoptic scope through the ETT, and secure the ETT with the tape at the proximal portion of the flexible portion of the scope and set aside.
   d) An anesthesia "Time Out" should be performed prior to starting procedure.
   e) Gently suction the oropharynx.
   f) Place well-lubricated intubating airway in the oral cavity in the midline.
      i) If patient tolerates this well, then there is adequate anesthesia to the oropharynx.
      ii) Alternatively, test the adequacy of oropharynx analgesia with the tongue depressor.
         (1) Ideally, patient should not have any gag reflex prior to the start of the procedure.
   g) **Ask the assistant to pull the tongue gently and give jaw thrust.**
      i) This maneuver is not always needed but will facilitate glottic exposure by increasing pharyngeal space by elevating the tongue and epiglottis away from the posterior pharyngeal wall.
   h) **Insert the distal tip of the scope in the middle of the patient's mouth or in the nostril (if using nasal approach).**
      i) Once the FOB is inserted into the patient, look through the eyepiece or watch the video monitor.
      ii) As the FOB is advanced, the tip of the airway and the patient's soft palate and uvula will come in view.
      iii) Slowly anteflex (thumb down) the tip of the scope until you visualize the epiglottis or the glottis.
      iv) Advance the tip under the epiglottis until cords come into the view.
      v) Once the vocal cords are identified, keep them in the middle of the screen. Use fine adjustments including rotation of the body of FOB and manipulations of the tip control lever advanced through the cords. Keep in mind that the posterior aspect of the glottic aperture is the widest and easiest to pass through.
      vi) Advance the scope until you see carina.
      vii) Do not advance the scope further.
      viii) Loosen the ETT from the proximal end of the fiberoptic scope, and advance it carefully over the shaft of the FOB into the airway.
      ix) Stop advancing once the ETT is 22-24 cm at the teeth or about 3-4 cm above the carina.
      x) Withdraw the FOB.
      xi) Inflate the cuff gently.
      xii) Connect ventilator circuit.
      xiii) Reconfirm the correct placement by the presence of end-tidal CO_2.
   i) Induce general anesthesia if patient is awake.
   j) If positioning a double-lumen ETT, it is essential to identify tracheal rings.
      i) They will be complete anteriorly, to guide the left or right main bronchus.
7) **Difficulties and troubleshooting**[7]
   a) **Inability to advance FOB into the trachea**
      i) Uncooperative patient
         (1) Optimizing sedation and topical analgesia.
         (2) Consider performing the procedure with the patient anesthetized.

READ MORE
Atlas of anesthesia procedures: awake fiber optic intubation, Chapter 164



Nasal fiberoptic intubation may be easier because the pathway to the airway is more direct. Using the right nare reduces epistaxis as the tube bevel is pointed away from and less likely to shear nasal turbinates.



Jaw thrust will improve your view. Keeping the scope "taut" at all times will improve the maneuverability.

ii) Poor visualization
  (1) Scope is foggy
    (a) Prior to starting, put FOB into the warm solution (use the same warm bottle as for the ETT).
    (b) Use commercially available defogging solution.
    (c) Touch the distal end of the scope to buccal or other mucosa to clear it.
  (2) Too many secretions or blood
    (a) Administer antisialogogue early.
    (b) Suction the mouth prior to inserting the FOB.
    (c) Clean tip as necessary.
    (d) Secretions can be suctioned through the working channel of the FOB.
      (i) Alternatively, one can insufflate oxygen through the suction channel to help keep secretions away and improve exposure.
      (ii) Be aware of possible barotrauma to the lungs if FOB advanced through a narrowed airway, limiting the oxygen escape from the lungs.
iii) The presence of a large, floppy epiglottis interferes with the passage of FOB to the trachea.
  (1) Ask an assistant to provide jaw thrust or pull the tongue forward to move the epiglottis away from the posterior pharyngeal wall. An awake patient can assist by protruding his or her tongue.

b) **Inability to advance ETT into the trachea over FOB**[8]
  i) Too tight of a fit between FOB and ETT.



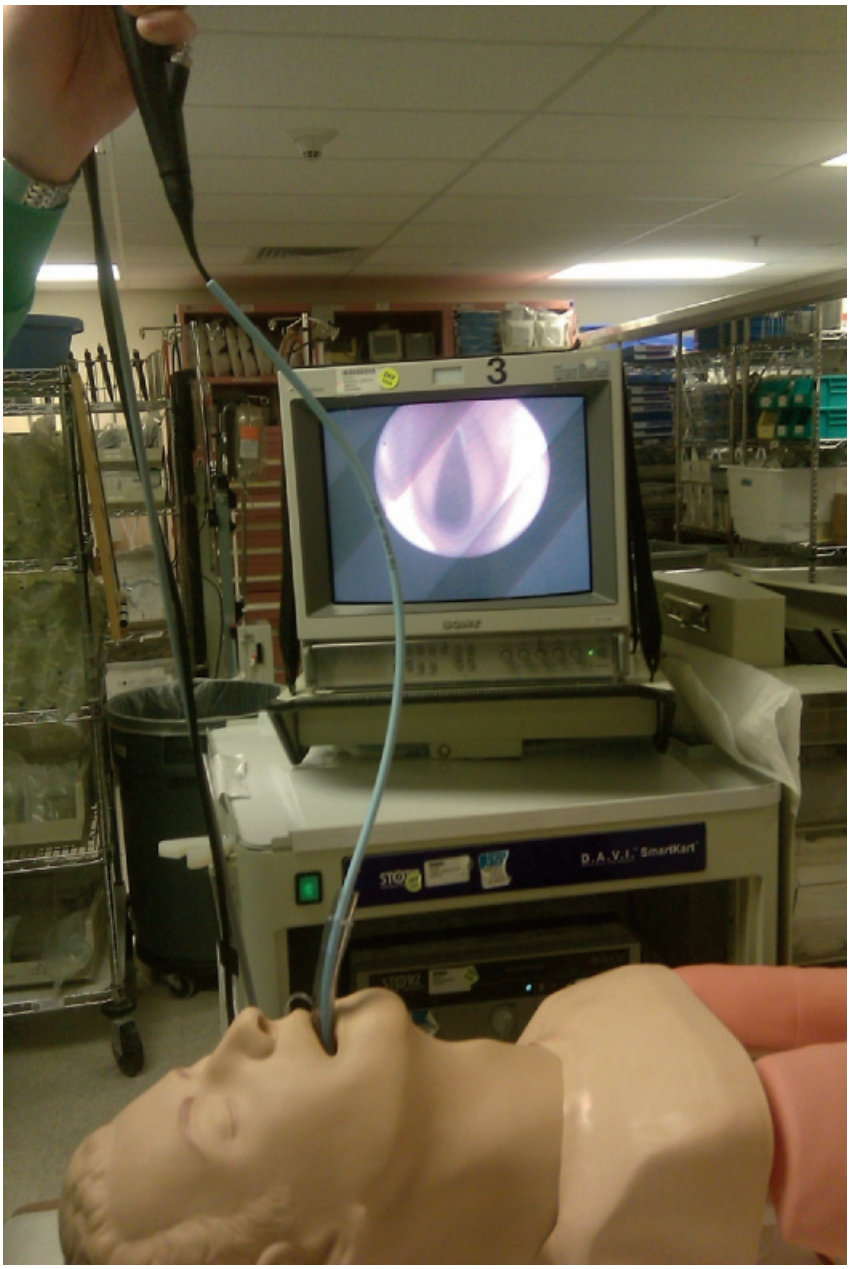
Assemble airway equipment in one place prior to starting fiberoptic intubation. Familiarize yourself with all the necessary equipment and its location during elective cases, so you are prepared in case of emergency.

FIGURE 23-5 Aintree Intubation Catheter



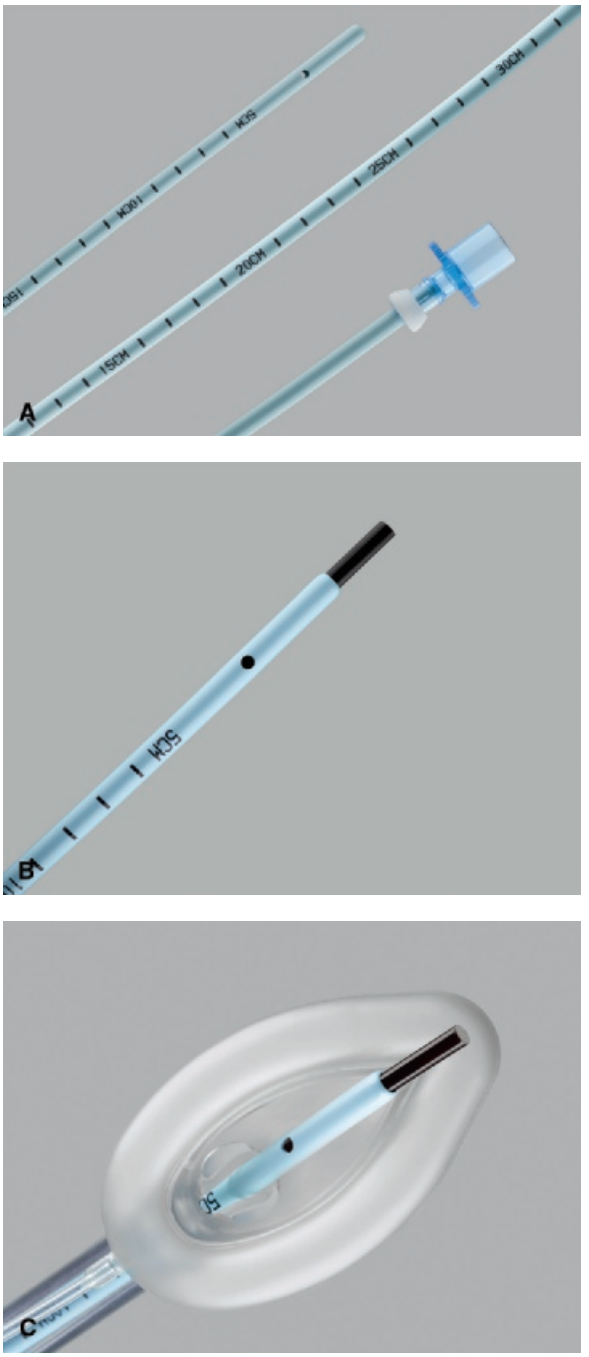
Proximal and distal ends of Aintree intubation catheter. Proximal end has a removable piece (shown attached), which allows for ventilation if necessary during exchange procedure. Catheter is usually 56 cm in length and has 4.7 mm internal diameter, which allows for a passage of small fiberoptic scope.

FIGURE 23-6 Using Aintree Intubation Catheter With Fiberoptic Scope



Aintree intubation catheter is inserted with fiberoptic bronchoscope (FOB) guidance through a laryngeal mask airway (LMA). After Aintree is placed in the trachea, FOB and LMA are removed and endotracheal tube is inserted over Aintree. Placement is confirmed again with FOB through Aintree, and then Aintree is removed and circuit is connected to the endotracheal tube.

FIGURE 23-7 Intubating Oral Airways: **(A)** Ovassapian, **(B)** Berman, and **(C)** Williams Intubating Oral Airways



(1) Consider using larger ETT or smaller FOB.
(2) Consider better lubrication.

ii) Consider using a smaller ETT, as patient's airway might be too narrow.
iii) Consider removing intubating airway.
iv) Consider rotating ETT 90 degrees counterclockwise, as the most common site of ETT being caught is on the right arytenoid cartilage.
v) FOB is not taut.
(1) Always keep FOB straight (one hand on the proximal end of the scope with the thumb on the plane control lever and the other hand on the distal end of the scope, with hands maximally apart).
vi) Consider using FOI in an Aintree ETT (**Figs. 23-5 and 23-6**).

c) **Inability to remove FOB**
i) Tip of the FOB has gone through the Murphy eye of the ETT.
(1) Withdraw FOB and ETT together (**Fig. 23-7**).
(a) Take out FOB.
(b) Repeat the procedure.
(2) Pass the scope through the ETT under direct vision prior to initiation of intubation.
ii) Not a straight insertion of the vocal cord (see Section 7.b.v.1).
iii) Too tight of a fit of an unlubricated ETT.

CHAPTER SUMMARY FOR FIBEROPTIC LARYNGOSCOPY

Indications	History of difficult intubation, "cannot intubate–cannot ventilate" scenario, facial abnormalities, compromised airway (upper or lower obstruction), undesirable neck movement, airway tumors, high risk of dental injury
Contraindications	Lack of sufficient time if doing awake fiberoptic intubation; bleeding or secretions obstruct airway visualization or an uncooperative patient.
Equipment	Fiberoptic scope, light source, endotracheal tube and airway cart
Complications	Damage to vocal cords (as ETT is inserted blindly over the FOB), failure to withdraw FOB due to tight fit, passage of FOB through Murphy eye Agitation and/or labile hemodynamic response due to inadequately prepared patient

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24 Basic Pharmacology

Konrad Meissner

A thorough understanding of the basic principles of pharmacology is paramount for the proper administration of anesthetic and analgesic drugs. As clinicians, we do not only rely on general dosage strategies but need insight into variables that cause, at times massive, variability of drug effects and side effects. Finally, there is growing evidence that the choice of drugs given for anesthesia may influence long-term outcomes.

1) **Pharmacokinetic and pharmacodynamic principles**
   a) Pharmacokinetics is the quantitative study of the absorption, distribution, metabolism, and elimination of drugs[1] (**Table 24-1**).
   b) Pharmacodynamics describes either concentration-effect relationships or the time course of pharmacological effect after dosing (**Fig. 24-1**).
2) **Drug administration (Table 24-2)**
   a) **Intravascular**—intravenous (IV) or intra-arterial
   b) **Extravascular**—intramuscular, sublingual, enteral, rectal, superficial, subcutaneous, pulmonary, pleural, or peritoneal
   c) **Regional**—spinal, epidural, perineural, or compartmental
3) **Drug absorption**
   a) Absorption is the process by which unchanged drug moves from the site of administration to the site of measurement within the body, usually the venous bloodstream.
   b) Monitoring intact drug in blood or plasma is a useful method of assessing entry of drug into the systemic circulation[1] (**Table 24-1**).
   c) **Most anesthetic drugs are administered IV or via inhalation.**
      i) With IV administration, the drug absorption process is eliminated, providing rapid therapeutic blood concentrations.
         (1) Toxicity or other adverse drug reactions will also be seen rapidly.
      ii) Drugs administered via inhalation require low molecular weight but, aside from gases, also include fluids and even solid compounds applied as aerosols (1-µm particles).
         (1) When combined with the high blood flow and large total surface area of the alveoli, drugs move very rapidly to the blood.
   d) **Intrathecal administration delivers drugs directly to the site of action (spinal cord),** allowing very low doses to be given and potential reduction of systemic side effects.
      i) Alternatively, epidural or perineural dosing requires larger doses, thereby increasing the risk of systemic side effects and possible toxicity.
      ii) Always consider density and temperature with respect to cerebrospinal fluid in order to assure (or rule out) intrathecal spread.
4) **Drug distribution**
   a) Distribution is the process of transfer of a drug from the site of application (usually the bloodstream or the bronchoalveolar system) to peripheral compartments, including the effect site.



Classical pharmacokinetics characterizes "what the body does to the drug," whereas classical pharmacodynamics assesses "what the drug does to the body."

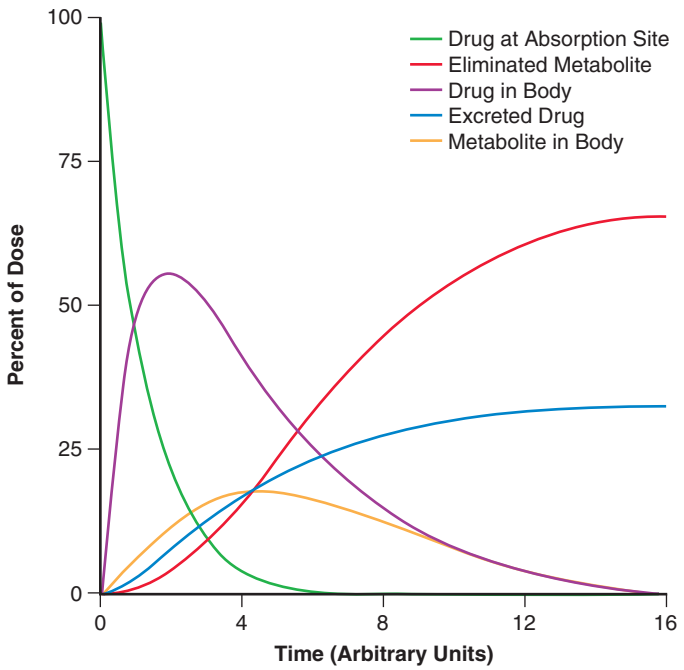


As a rule of thumb, the lower the oral bioavailability of a drug, the higher the risk of substantial interpatient and intrapatient pharmacokinetic variability.

TABLE 24-1 Comparison of Different Methods of Drug Administration

Type of Administration	Time of Onset	First-Pass Metabolism
Transdermal	Slow	No
Oral	Slow	Yes
Sublingual/buccal	Rapid	No
Subcutaneous	Slow	No
Intramuscular	Slow	No
Intravascular	Immediate	No
Inhalational	Intermediate	No
Rectal	Slow	Small amount
Intrathecal	Immediate	No
Epidural/perineural	Rapid	No

FIGURE 24-1 Time Course of Drug and Metabolite.



The amount in each compartment is expressed as a percentage of the dose administered. In this example, all the dose is absorbed. Redrawn with permission from Rowland M, Tozer T. *Clinical Pharmacokinetics: Concepts and Applications*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 1995.

TABLE 24-2 Basic Pharmacokinetic Principles

	Description	Clinical Relevance
C_{max}	Maximum concentration of a drug measured after administration	Together with tmax, C_{max} is a surrogate marker for the rate of absorption. ↑ rate of absorption may ↑ toxicity. ↓ rate of absorption may lead to delayed onset of drug effect.
Steady state (C_{ss})	Concentration of a drug or a chemical in a body fluid—usually plasma—at the time a "steady state" has been achieved and rates of drug administration and drug elimination are equal.	The effect of a drug (pharmacodynamic, toxicodynamic) can only safely be evaluated after drug concentrations reach steady state (usually after 5 terminal half-lives).
Bioavailability (*F*)	The percentage of dose entering the systemic circulation after administration of a given dosage form	• Oral bioavailability is usually <100%. Incomplete absorption from the gut or significant first-pass metabolism in the liver decreases drug levels in the blood and hence bioavailability. • In many cases, pharmacogenomics/ polymorphisms of the enzymes and transporters involved affect oral bioavailability. • Many drugs with low oral bioavailability such as immunosuppressants will require therapeutic drug monitoring. • Under normal circumstances, bioavailability after IV administration is always 100%.
AUC	Area under the plasma concentration vs time curve	AUC is a surrogate marker of exposure. For many drugs, AUC can be correlated with pharmacodynamic effects and can be used to establish bioequivalence of different drugs with the same active compound.
Volume of distribution (Vd)	The volume into which a drug appears to have been dissolved after administration.	A larger Vd will require a larger loading dose to achieve a given concentration at the presumed site of action.
Clearance (Cl)	Clearance of a drug from the body, expressed in volume/unit time	Pathological conditions such as renal disease alter the time course of clearance.
Elimination half-life ($t_{1/2}$)	The amount of time that it takes for the concentration of a drug to be reduced by half in the body.	• 5 terminal half-lives are required for a drug to reach steady state. • Terminal half-lives determine dosing intervals (usually close to one terminal half-life to avoid accumulation).
Drug-drug interaction	One drug changing the effect of another, given subsequently, earlier, or simultaneously	Mechanism includes all versions of pharmacokinetics and pharmacodynamics, including uptake, transport, metabolism, clinical, and side effects.
Drug antagonists	Antagonizing drug effects occur at different levels of drug action. Specific time courses of initial drug and antagonist must be taken into account.	Opioid or benzodiazepine antagonists may have shorter half-lives than the drug to be antagonized, potentially leading to respiratory depression or sedation after the antagonist is eliminated.

TABLE 24-2 Basic Pharmacokinetic Principles *(Continued)*

	Description	Clinical Relevance
First-pass effect	The biotransformation and/or excretion of a drug by intestinal and hepatic, including biliary, mechanisms following absorption of the drug from the gastrointestinal tract, before drug gains access to the systemic circulation.	When dosing drugs orally, it is important to know that higher doses will be required than when the same drug is administered IV.
Zero-order kinetics	Mechanisms of chemical reaction in which the reaction velocity is apparently independent of the concentration of all the reactants.	• Saturation of enzyme metabolism is possible. • At saturation, a small increase in dose will result in large increase in plasma concentration, for example, alcohol/phenytoin.
First-order kinetics	The velocity of a chemical reaction is proportional to the concentration of the reactant.	
Elimination rate constant (k_{el})	The velocity of a chemical reaction is proportional to the product of the active masses (concentrations) of the reactants.	• Elimination rate determines how fast a drug is eliminated from the body and is an important component of terminal half-life. • The elimination rate may be significantly altered by induction or inhibition of drug-metabolizing enzymes (induction: lower exposure, less drug effect; inhibition: higher exposure, higher risk of drug toxicity).

i) Distribution processes may be influenced by physiologic barriers and other processes, including
   (1) Different components of blood-organ barriers
   (2) Several kinds of active or passive transporters, including diffusion, pinocytosis, ATP-binding cassette (ABC) transport proteins (acting as efflux and uptake transporters), and their individual expression
   (3) Variations in ventilation-perfusion ratios
   (4) Diffusion barriers
   (5) Interaction with other drugs

ii) As a rule of thumb, initial plasma levels for drugs given IV tend to be highest in well-perfused areas, first and foremost the brain and heart, followed by muscle, skin, and finally fat (eg, thiopental).

iii) Equilibration of drug and their metabolites into all compartments may take hours.



Five terminal half-lives are required for a drug to reach steady state.

5) **Transport across membranes**

a) Many drugs are too large to pass through aqueous membrane channels and must cross the lipid part of the membrane in order to reach their effect site, which may be on the inside of the cell (eg, local anesthetics).

b) Almost all drugs are weak acids or weak bases and are thus ionized or nonionized at physiologic pH.

Nonionized drug = lipid soluble = crosses membranes more easily

6) **Drug metabolism and elimination**
   a) Elimination is the process of irreversible loss of drug from the site of measurement.
   b) This can occur by two processes, either excretion or metabolism.

> **EXCRETION** = the irreversible loss of an unchanged drug
> **METABOLISM** = the conversion of one chemical species to another

   c) Some drugs require metabolism, as the metabolites are the active drug form (eg, codeine is metabolized to produce morphine).[2]
   d) Whether or not metabolism results in active or inactive drug is highly variable due to genetic variation and/or drug-drug interaction (DDI).[3]
7) **Binding of drugs to plasma proteins**
   a) Drug concentrations are measured in plasma and therapeutic doses adjusted accordingly.
   b) Many drugs reversibly bind to plasma proteins.
   c) The degree of protein binding has important pharmacokinetic and pharmacodynamic implications, because it is the unbound moiety that readily diffuses across biological membranes and reaches the receptor site.
   d) The degree of plasma binding is often expressed as percentage of drug bound, which is complemented by the free fraction of drug.[4]
   e) Factors affecting protein synthesis, loss, and absorption may all affect drug effect.[5]
   f) The plasma binding of many drugs may decrease due to a variety of pathologic states, including liver disease, burns, and malabsorption syndromes. This may in turn increase free drug concentration levels.[6]
   g) Because of this, measurement of total drug concentrations alone may be misleading and cause an inappropriate increase in therapy, which might be potentially hazardous, or to administration of subtherapeutic dosages.



Drug plasma binding may decrease in many pathologic states, including liver disease, burns, and malabsorption syndromes. This may increase free drug plasma levels, causing measurement of total drug concentrations to be misleading.

8) **Drug-Drug interactions (DDIs)**
   a) DDIs may occur when two or more drugs are coadministered and the effects of the combined drugs can be increased, weakened, or harmful.[7]
   b) DDIs can become clinically relevant on pharmacokinetic (metabolism and transport), pharmacodynamic (receptor level), and pharmacogenetic levels (see below).[8]
   c) Understanding and intentionally including DDIs into the anesthetic plan comprises core anesthetic management, as clinicians often exploit additive or synergistic effects of anesthetics during balanced anesthesia.[9]
   d) On the other hand, adding anesthetic drugs in the presence of an existing medication may change toxicity profiles and put patients in danger.[10,11]
9) **Pharmacogenetics**
   a) Genetic variations (polymorphisms) in individuals lead to changes in pharmacokinetics and the pharmacodynamics of drugs. They are referred to as pharmacogenetic changes.
   b) Polymorphisms in drug-metabolizing enzymes, transporters, and/or pharmacological targets of drugs may profoundly influence the differences between the dose-response relationships of different individuals and may be relevant for anesthesia.[12,13]
   c) Since current practice of anesthesia rarely targets known interindividual variations, standard dosing is often applied. Clinicians must be aware of both unexpected lack of effect and potential side effects, particularly when combining drugs during anesthetic care.[14]

d) **With respect to the cytochrome P450 2D6 enzyme, there are slow and fast metabolizers.**
   i) Slow metabolizers accumulate substrates quickly when treated with standard doses, potentially resulting in toxicity.
   ii) Fast metabolizers eliminate the drug quickly and may have less or no drug effect.
   iii) Accordingly, slow metabolizers will require much smaller doses and fast metabolizers much higher doses than "normal" metabolizers, that is, for codeine to be transformed to morphine.[15]

e) As more information becomes available, it is likely that pretesting pharmacogenetic variables before elective cases will increase patient safety due to a decrease in adverse drug reactions.

Understanding the pharmacological properties of each drug administered allows the practitioner to predict interactions and avoid adverse outcomes. Taking into account the unknown interaction and variability potential, however, calls for caution, particularly when applying numerous drugs at once, such as in anesthesia.

10) **Pediatric pharmacology**
   a) Infants and children differ markedly from adult patients because their metabolism is developing and their principal physiologic functions are different.
   b) Volumes of distribution (blood, extravascular, etc.), flows (cardiac output, cerebrospinal fluid turnover, etc.), and barrier properties are different from those of adult patients.
   c) For example, many cytochrome P450 isoforms are not mature until the age of 1 year, and some children are deficient until the age of 4 years.[9] Sedatives may require dose adjustments depending on pediatric age, with small children requiring less and adolescents requiring even more than adults (eg, midazolam).[16,17]
   d) Clinical pharmacology research in the pediatric population is lacking, with knowledge in this area trailing that in adults or geriatric patients. This leaves us to use principles defined in adult bodies when deciding on dosing regimens, which may not apply to the very young.

11) **Geriatric pharmacology**
   a) Geriatric patients comprise an increasing part of the population, with a continuous rather than sudden change in anesthetic pharmacology.[18,19]
   b) Aside from specific medical needs and multimorbidity, geriatric patients are quite variable in condition and medical needs—ranging from healthy subjects to frailty and end-of-life situations.[20]
   c) Specific pharmacologic considerations include the choice between neuraxial and general anesthesia with respect to overall outcome and neurologic outcome in general, which remains undecided to date.[21]
   d) Inhaled anesthetics come with a lower MAC in the aged population: whereas the MAC of isoflurane for a 5-year-old is 1%, it is only 0.25% for a 95-year-old.[22] Dose of intravenous anesthetic drugs, opioids, and benzodiazepines should also be decreased by 25%-50%.

12) **Pharmacology of pregnant patients**
   a) Changes in physiology occur during pregnancy, which may unpredictably alter drug bioavailability, distribution, and clearance.[23]
      i) For example, MAC is decreased ~30% in pregnancy.
   b) PK and PD data during pregnancy for many drugs are lacking with some requiring dose adjustments that are not reflected in dosing guidelines. A thorough review on the subject exists.[23]

READ MORE
OB overview and maternal coexisting disease, Chapters 107 and 110

CHAPTER SUMMARY FOR BASIC PHARMACOLOGY

Drug Administration	Options include intravascular, extravascular, and regional.
Drug Absorption	Unchanged drug moves from the site of administration to the site of measurement within the body.
Drug Distribution	Reversible transfer of drug to and from the site of measurement
Transport Across Membranes	Nonionized form is more lipid soluble and crosses membranes more easily.
Drug Elimination	Excretion or metabolism
Binding to Plasma Proteins	Unbound moieties diffuse across biologic membranes more easily.
Pharmacokinetics	"What the body does to the drug"
Pharmacodynamics	"What the drug does to the body"
Pharmacogenomics	Genetic variations (polymorphisms) in individuals that lead to changes in PK and PD of drugs are referred to as pharmacogenetic changes.
Drug-Drug Interactions (DDIs)	When two or more drugs interact to decrease effectiveness or cause toxicity of one or more of the drugs
Pediatric Pharmacology	Anesthetic drugs require dose adjustments depending on pediatric age, with small children requiring less and adolescents requiring even more than adults.
Geriatric Pharmacology	Anesthetic requirements decrease with aging.
Pharmacology for Pregnancy	Pregnancy influences both PK and PD of drugs

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25 Local Anesthetics

Ellile Pushpanathan • Andrea J. Traynor

Local anesthetics are widely used for anesthesia and analgesia. Knowledge of onset, duration, and side effects of individual agents is essential for optimal efficacy. For more information on specific local anesthetic drugs, refer to the Appendix of Drug Information.

1) **Mechanism of action**
   a) Local anesthetics bind to voltage-gated sodium channels, preventing the influx of sodium ions necessary for nerve depolarization.
2) **Structure-activity relationships**
   a) All local anesthetics are weak bases with pKa values above physiologic pH. The pKa of substance is the pH at which it is present in 50:50 ionized-unionized forms. The higher the pKa for a particular local anesthetic, the more ionized it will be at physiological pH.

READ MORE
Appendix, Local anesthetics, Chapter 211

   b) The lipophilic (nonionized) form binds receptors and is active.
      i) In the presence of acidosis (eg, in areas of active infection), more drug is in the hydrophilic (ionized) form, often resulting in poor-quality local analgesia or anesthesia.
   c) These compounds are more stable for storage at acidic pH, especially epinephrine-containing solutions. Only the basic, nonionized, form binds receptors.
      i) The addition of sodium bicarbonate can increase speed of onset by 3-5 minutes and decrease pain with subcutaneous injection.[1,2]
   d) **Differential blockade**
      i) Clinically used local anesthetics appear to affect different types of nerve fibers in different ways.
         (1) For example, blockade of small-diameter sympathetic fibers typically occurs first, followed by loss of pinprick or light touch, followed by blockade of large-diameter motor fibers.
         (2) However, different agents (eg, ropivacaine) may affect motor function less than other agents (eg, lidocaine).
      ii) The mechanism of differential blockade is poorly understood, and its use is an active area of research.[3]



The amount of protein binding of local anesthetics affects duration of action and can be responsible for different clinical effects observed between individuals.[3]

   e) **Duration of action**
      i) Amide local anesthetics bind to alpha$_1$–glycoprotein.
      ii) The amount of protein binding affects duration of action and can be responsible for different clinical effects observed between individuals.[3]
3) **Types of local anesthetics**
   a) All have a lipophilic (unsaturated aromatic ring) moiety and a hydrophilic (hydrocarbon chain) moiety connected by an ester or amide linkage.
   b) **Esters**
      i) Procaine, chloroprocaine, cocaine, tetracaine, benzocaine

TABLE 25-1 Maximum Safe Dose of Local Anesthetic

Local Anesthetic	Dose (mg/kg) Without Adrenaline	Dose (mg/kg) With Adrenaline
Chloroprocaine	10	15
Lidocaine	3	6
Bupivacaine	2	2.5
Ropivacaine	3	4
Prilocaine	6	8

From Willliams D, Walker J. A nomogram for calculating the maximum dose of local anaesthetics. *Anaesthesia*. 2014;69(8):847-853. Reprinted by permission of John Wiley & Sons, Inc.

c) **Amides**
   i) Lidocaine, etidocaine, prilocaine, mepivacaine, bupivacaine, ropivacaine, dibucaine



Increasing the pH by adding basic solutions (eg, bicarbonate) speeds onset of action of local anesthetics.

4) **Pharmacokinetics**
   a) **Systemic absorption**
      i) Depends on the vascularity of the injection site
         (1) Amount of systemic absorption is as follows: tracheal > intercostal > caudal > epidural > brachial plexus > femoral/sciatic > subcutaneous.[2]
      ii) The addition of vasoconstrictors such as epinephrine at the injection site decreases systemic absorption of local anesthetic.
         (1) This leads to increased duration of action and decreased systemic toxicity.
         (2) Also allows a greater maximum dose per kg to be administered (**Table 25-1**).
   b) **Distribution**
      i) Determined by tissue perfusion, protein binding, and tissue mass
   c) **Metabolism**
      i) Amides
         (1) Metabolized in the liver by microsomal enzymes
      ii) Esters
         (1) Metabolized by plasma cholinesterases



Patients undergoing outpatient surgery in the lithotomy position are at increased risk of transient neurologic symptoms after lidocaine spinal anesthesia.

5) **Clinical uses**
   a) Local infiltration
   b) Topical anesthesia
   c) Peripheral nerve block
   d) Spinal, epidural, or caudal anesthesia
   e) Arrhythmia therapy
   f) Pain management
6) **Adverse effects/toxicity (Table 25-2)**
   a) **Toxic effects occur with large doses**
      i) Cardiovascular
         (1) Hypotension
         (2) Arrhythmias, including bradycardia, atrioventricular block, and ventricular fibrillation
      ii) Respiratory
         (1) Depression of hypoxic drive and inhibition of bronchial smooth muscle relaxation
      iii) Neurological
         (1) Perioral numbness, tinnitus, seizures, coma



Perioral tingling is an early sign of local anesthetic toxicity. Its presence should warrant immediate attention and prompt investigation of other indicators of local anesthetic toxicity.

TABLE 25-2 Dose-Dependent Systemic Effects of Lidocaine

Plasma Concentration (μg/mL)	Effect
1-5	Analgesia
5-10	Light-headedness Tinnitus Numbness of tongue Systemic hypotension Myocardial depression
10-15	Seizures Unconsciousness
15-25	Coma Respiratory arrest
>25	Cardiovascular depression

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b) **Transient neurologic symptoms**[3]
   i) Transient pain or sensory abnormalities in the back, buttocks, or lower extremities.
   ii) Risk factors
      (1) Lidocaine spinal anesthesia
      (2) Lithotomy position
      (3) Outpatient anesthesia
   iii) Not dose dependent.
   iv) Symptomatic treatment with nonsteroidal anti-inflammatory drugs may improve symptoms.
   v) Symptoms are usually self-limited and resolve within 3 days.[3]



Prilocaine may precipitate methemoglobinemia, especially in infants.



Large doses of hyperbaric lidocaine delivered via small-gauge spinal catheter have been associated with cauda equina syndrome.

c) **Cauda equina syndrome**
   i) Diffuse injury to cauda equina nerve roots resulting in bowel/bladder dysfunction and paraplegia.
   ii) Cause is unclear but thought to be due to direct nerve damage by high-concentration local anesthetics.
   iii) Risk factors
      (1) High-concentration (5% or greater) hyperbaric lidocaine
      (2) Small-gauge intrathecal catheters
         (a) Microcatheters were removed from the market after multiple cases of cauda equina syndrome with hyperbaric lidocaine.

d) **Allergic reactions**
   i) Very rare with amides
   ii) Allergic reactions to ester local anesthetics are usually due to cross-reactivity to *p*-aminobenzoic acid (PABA) because the ester linkage resembles PABA.

7) **Treatment of toxicity**
   a) Cardiovascular collapse from bupivacaine is exceptionally difficult to treat and may require cardiopulmonary bypass.
   b) Toxicity may develop up to 1 hour following LA administration.[4]
   c) **20% lipid emulsion has been successful in treating local anesthetic toxicity and should be used early if clinical suspicion is present.**[5]
      i) Administer 20% lipid emulsion over 2-3 minutes. Give a bolus of 100 mL for patients >70 kg and bolus of 1.5 mL/kg if <70 kg while continuing chest compressions and other supportive measures.[5]
         (1) May be repeated after 3-5 minutes to a maximum of 1-2 additional doses.[4,5]

READ MORE
Local anesthetic toxicity, Chapter 149

ii) Following bolus administration, an infusion of either 200-250 mL over 15-20 minutes (>70 kg) or 0.25 mL/kg/min if <70 kg should be initiated until the patient is hemodynamically stable.[6,7] Do not exceed a maximum cumulative dose of 12 mL/kg.[4,5]

CHAPTER SUMMARY FOR LOCAL ANESTHETICS

Mechanism of Action	Reversibly bind Na^+ channels on nerves → prevents influx of Na^+ ions and inhibits depolarization
Common Uses	Local and regional anesthesia, labor analgesia, treatment of arrhythmias
Adverse Effects (Cause)	Excessive plasma concentration Possible direct neurotoxicity in susceptible patients
Adverse Effects (Clinical Findings)	Seizures, arrhythmias, respiratory depression
Adverse Effects (Prevention)	Avoid inadvertent intravascular injection of large doses Consider risk factors when choosing a local anesthetic for spinal injection
Treatment of Toxicity	Symptomatic treatment with oxygenation, ventilation, and cardiovascular support as needed Rapid treatment with intralipid

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26 Neuromuscular Blockade and Anticholinesterases

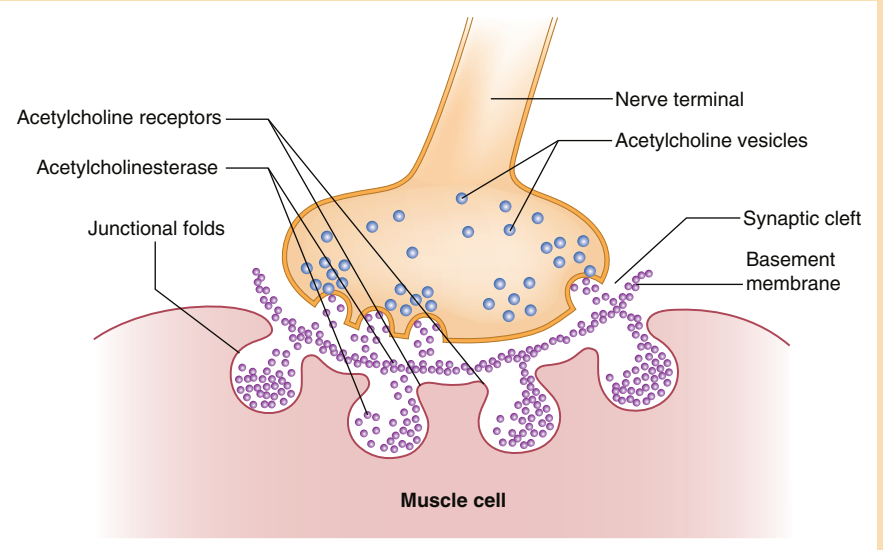
Debra J. Faulk

Neuromuscular blocking (NMB) drugs are widely used in anesthesia and critical care. They are useful for facilitating surgery, airway management, and ventilator management in critically ill patients. NMB drugs can be extremely dangerous because respiratory mechanics may be impaired. Anticholinesterase drugs reverse the effects of nondepolarizing NMB agents and are also used outside of anesthetic practice to treat other neurologic and muscle disorders. These agents increase cholinergic transmission at the neuromuscular junction.

Neuromuscular Blockade

1) **Mechanism of action**
   a) Physiology of neuromuscular transmission (**Fig. 26-1**).
   b) Acetylcholine (ACh) is released from the motor neuron and binds to the nicotinic ACh receptor at the motor endplate on the skeletal muscle cell.
   c) A conformational change occurs in the receptor, allowing sodium ions to enter the membrane and depolarize the cell.[1]
   d) NMB agents act by blocking the binding of ACh at the nicotinic ACh receptor. Depending on the choice of muscle relaxant, this will prevent neurotransmission by either causing prolonged activation of the receptor or blocking its activation. Based on their mechanisms of action, NMB agents are broadly classified into two categories: depolarizing and nondepolarizing.
      i) Depolarizing muscle relaxants
         (1) Succinylcholine is the only available depolarizing muscle relaxant in clinical use.
            (a) It is a noncompetitive agonist with a molecular structure similar to that of ACh.
            (b) Receptor binding induces a conformational change similar to that produced by ACh, resulting in depolarization.
            (c) Unlike acetylcholine, succinylcholine is not hydrolyzed by acetylcholinesterase. It remains at the neuromuscular junction longer than does ACh and may repeatedly bind to the ACh receptor resulting in prolonged depolarization, inability for repolarization, and resultant phase I block.
            (d) Muscle depolarization occurs in an uncoordinated manner, which is observed as fasciculations.
      ii) Nondepolarizing muscle relaxants
         (1) Competitive antagonists
            (a) Compete with ACh at the receptor-binding site.
            (b) Once bound, a conformational change cannot occur, and muscle depolarization is prevented.
2) **Monitoring neuromuscular blockade**[1,2]
   a) The depth of neuromuscular blockade is typically measured using a peripheral nerve stimulator.
      i) The ulnar nerve (stimulation of adductor pollicis) and the facial nerve (stimulation of orbicularis oculi) are the most common locations for monitor placement.

FIGURE 26-1 Physiology of Neuromuscular Transmission



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b) **Train-of-four (TOF) stimulation** occurs when the stimulator delivers four successive 200-μs stimuli in 2 seconds (2 Hz).
   i) The ratio of the height of the first twitch to the fourth is the TOF ratio (TOFR).
   ii) The twitches elicited from TOF stimulation fade as depth of neuromuscular blockade increases.
   iii) When three of four twitches are observed, 70%-75% of the receptors are blocked, and when one twitch is visible, 85%-90% of receptors are blocked.[1]



Absence of fade with train-of-four stimulation does not completely exclude residual neuromuscular blockade. Clinical weakness may still occur and must always be assessed.

c) **Tetany**
   i) A continuous 5-second stimulus of 50-100 Hz is delivered to elicit tetany.
   ii) Qualitatively, fade is more obvious during sustained tetanic stimulation than TOF count.
   iii) Quantitatively, tetanic fade at 50 Hz is first noticed at TOFR < 0.7. Adequate recovery is considered to be TOFR > 0.9. TOFR may therefore be a more reliable means of assessing adequate return of neuromuscular function than tetanic fade.[3]
   iv) Posttetanic potentiation
      (1) Occurs when the TOF is measured after tetany. The observed evoked response after tetanic stimulation is called posttetanic potentiation.
      (2) This is due to increased ACh present at the motor endplate.
      (3) It is most useful during profound neuromuscular blockade that is too deep to elicit a single twitch or TOF response.

(a) After administration of large doses of nondepolarizing NMB drug.
(b) When sudden movements are unwanted or may be catastrophic.
(4) Provides an estimated time to return of response.

3) **Metabolism**
   a) **Depolarizing muscle relaxants**
      i) Diffuse away from the ACh receptor and are metabolized by plasma cholinesterase (pseudocholinesterase).
      ii) Abnormal pseudocholinesterase
         (1) Approximately 1:2000 patients has a genetic mutation in the pseudocholinesterase enzyme, rendering it unable to metabolize succinylcholine.[1]
         (2) In these patients, the duration of action can be 3-6 hours.[1,2]
         (3) Dibucaine is a local anesthetic that inhibits normal pseudocholinesterase and has limited effect on the abnormal variant.
            (a) The ratio of inhibition (dibucaine number) indicates the amount of normal pseudocholinesterase and has historically been helpful in the diagnosis of abnormal pseudocholinesterase.
            (b) Mutations in the pseudocholinesterase gene have been characterized, and newer molecular diagnostic techniques may be more informative than the dibucaine number in identifying mutations in structure, function, or synthesis of pseudocholinesterase enzyme.[4]
      iii) Nondepolarizing muscle relaxants
         (1) Diffuse away from the ACh receptor and are gradually metabolized and excreted.
            (a) For more information on the metabolism and excretion of specific agents, refer to the appendix of drug information.

4) **Reversal of blockade**
   a) **Depolarizing muscle relaxants**
      i) Do not have specific reversal agents.
      ii) Restoration of neuromuscular function depends on metabolism by pseudocholinesterase.
   b) **Nondepolarizing muscle relaxants**
      i) Restoration of neuromuscular function depends on redistribution, metabolism, and excretion in addition to administration of an anticholinesterase drug or selective relaxant binding agent.



When using NMB drugs, awareness is always possible. Take care to ensure that the depth of hypnosis is appropriate to prevent awareness.

5) **Clinical uses**
   a) Intubation
   b) Muscle relaxation to facilitate surgery (ie, abdominal surgery).
   c) Prevent movement in cases where movement could be catastrophic (ie, intraocular or intracranial surgery).
   d) Facilitate mechanical ventilation, prevent ventilator desynchrony, or decrease work of breathing in critically ill patients.
   e) Management of patients with severe acute respiratory distress syndrome (ARDS).



Administration of a sedative or hypnotic agent with a comparable duration of action to the muscle relaxant is imperative when neuromuscular blockade is administered.

6) **Adverse effects/toxicity**
   a) **Awareness**
      i) Awareness is the retention of memory, pain, and consciousness while skeletal muscles are paralyzed.
      ii) NMB drugs do not have sedative, analgesic, or hypnotic properties, making awareness possible, if not combined with an anesthetic, leading to posttraumatic stress disorder.

b) **Depolarizing muscle relaxants**
   i) Hyperkalemia
      (1) Succinylcholine increases the potassium level by 0.5-1 mEq/L and is relatively contraindicated if the patient's serum potassium level is >5.5 mEq/L.
      (2) Hyperkalemia following succinylcholine administration can cause cardiac arrest, particularly in susceptible patients.
         (a) Risk factors
            (i) Burns, immobility, chronic infection, massive trauma, spinal cord injury, muscular dystrophy, and other neuromuscular diseases.
   ii) Increased intraocular pressure (IOP)
      (1) This concern is controversial since succinylcholine has been used clinically in patients with open globe injuries, and very little clinical evidence to support its avoidance exists.[5]
   iii) Increased intracranial pressure
      (1) May be related to increased $PaCO_2$ due to increased carbon dioxide production from fasciculations.
      (2) May be attenuated with a small defasciculating dose of nondepolarizing NMB agent.
   iv) Increased intragastric pressure
      (1) Related to fasciculations and can be prevented with a defasciculating dose of a nondepolarizing neuromuscular blocking agent.[2]
      (2) Aspiration risk may be offset by concurrent increase in lower esophageal sphincter tone assuming normal gastric anatomy and position within the abdominal cavity.
   v) Myalgias
      (1) Independent of fasciculations, myalgias can be decreased with the administration of a small dose of nondepolarizing muscle relaxant prior to administration of succinylcholine.
   vi) Bradycardia
      (1) Most common when a second dose of succinylcholine is given about 5 minutes after the first
      (2) May be prevented by atropine and nondepolarizing muscle relaxants[2]
   vii) Triggers malignant hyperthermia in susceptible patients.



When considering the increased intraocular and intracranial pressure effects of succinylcholine, it is important to remember that laryngoscopy with *inadequate* anesthesia and/or muscle relaxation may be far more deleterious than the small (and often controversial) side effect of the drug.[1,2]



The importance of securing an airway while minimizing the risk of gastric aspiration must be considered. Use of succinylcholine may be clinically indicated.[1]

READ MORE
Malignant hyperthermia, Chapter 67

c) **Persistent neuromuscular blockade**
   i) Can cause postextubation respiratory failure.
   ii) Quantitative monitoring is the only reliable measure to confirm adequate return of neuromuscular function (TOFR > 0.9).
   iii) When assessing by qualitative means, return of neuromuscular function must be assessed via peripheral nerve stimulation (TOF monitoring or tetany) in conjunction with clinical criteria such as sustained head lift for >5 seconds prior to extubation. Patients should continue to be monitored clinically for signs of recurarization in the postoperative recovery period.

7) **Drug interactions, medical conditions, and function of NMB agents**
   a) A number of drugs have been seen to interact with NMB agents.
      i) This can result in enhancement or inhibition of blockade.
      ii) A summary of interactions for agents that may be most relevant to the anesthesiologist is described in **Table 26-1**.

TABLE 26-1 Drug Interactions With Neuromuscular Blocking Agents

Drug	Effect on Action of Depolarizing Neuromuscular Blocking Agent (Succinylcholine)	Effect on Action of Nondepolarizing Neuromuscular Blocking Agent	Comments
Volatile anesthetics	↑	↑	Dose dependent
Antibiotics (except penicillins and cephalosporins)		↑	Aminoglycosides most likely to have effect
Furosemide		↑	Probably due to drug's inhibition of cAMP production
Magnesium	↑	↑	More profound with nondepolarizing agents; use extreme caution in peripartum patients treated with magnesium
Lidocaine		↑	
Quinidine	↑	↑	
Lithium	↑		
Anticonvulsants (phenytoin, carbamazepine)		↓ (chronic) ↑ (acute)	Chronic treatment has pharmacodynamic effect (higher plasma concentrations are required for desired effect)
Cyclosporine	↑		
Local anesthetics	↑ duration		Ester local anesthetics can prolong succinylcholine by competing for plasma cholinesterase
Corticosteroids		↑	Prolonged treatment in mechanically ventilated critically ill patients
Echothiophate	↑ duration		Inhibits both pseudocholinesterase and acetylcholinesterase; used for treatment of glaucoma

↑ refers to enhancement of blockade.
↓ refers to inhibition or decreased duration of blockade.
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b) Special consideration for several populations, comorbid disease states, and physiologic parameters should be remembered when using NMB agents and their reversal drugs.
   i) Variable effect of nondepolarizing NMB agents may be seen with either increased or decreased responsiveness.

ii) Patients with disease states producing neurologic impairment, immobility, or destruction of NMJ may be at risk for hyperkalemic response to depolarizing NMB drugs.

iii) A summary of conditions and their potential response to NMB agents is described in Table 26-2.

Anticholinesterases and Reversal of Neuromuscular Blockade

1) **Mechanism of action**

a) Anticholinesterase drugs inhibit the acetylcholinesterase enzyme, which increases the amount of acetylcholine (ACh) available to compete for the ACh receptor.

TABLE 26-2 Medical Conditions That Affect Muscle Relaxant Function

Medical Condition	Response to Depolarizing Agent	Response to Nondepolarizing Agent	Comments
Hypothermia		Prolonged duration	
Elderly		Prolonged duration, decreased dosage required	Due to altered pharmacodynamics and decreased elimination
Multiple sclerosis	↑ risk of hyperkalemia	Resistance	Up-regulation of ACh receptors
Amyotrophic lateral sclerosis and other motor neuron diseases	↑ risk of hyperkalemia	Resistance	Up-regulation of ACh receptors
Guillain-Barre syndrome	↑ risk of hyperkalemia	Sensitive	Loss of motor units at neuromuscular junction
Myasthenia gravis	Resistant	Extreme sensitivity	Anticholinesterases used to treat disease can make reversal difficult and prolong action of depolarizing blockade
Myotonic dystrophy	↑ risk of hyperkalemia	Resistant	Neostigmine can cause prolonged muscle weakness
Eaton-Lambert syndrome	Sensitive	Sensitive	Neostigmine is ineffective
Muscular dystrophy	↑ risk of hyperkalemia, rhabdomyolysis, and malignant hyperthermia	Usually normal	Possible sensitivity to nondepolarizing agents in the presence of muscle weakness
Burn injury	↑ risk of hyperkalemia after the first 24 h	Resistance	Up-regulation of ACh receptors
Spinal cord injury	↑ risk of hyperkalemia after first 24 h		
Immobility and critical illness	↑ risk of hyperkalemia	Resistance	Can be associated with persistent weakness when used in critically ill patients

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b) Sugammadex is a modified gamma-cyclodextrin that selectively encapsulates aminosteroid NMB agents rendering them unavailable for binding to the ACh receptor.
   i) Reverses rocuronium, vecuronium, and pancuronium. Affinity is greatest for rocuronium.

2) **Clinical uses and common agents**
   a) **Reversal of neuromuscular blockade**
      i) Neostigmine and edrophonium
      ii) Sugammadex
   b) **Treatment of myasthenia gravis**
      i) Pyridostigmine (most common) and neostigmine
   c) **Treatment of Alzheimer dementia**
      i) Donepezil, rivastigmine, and galantamine[6]
   d) **Treatment of central cholinergic syndrome**
      i) Physostigmine
   e) **Treatment of glaucoma**
      i) Echothiophate
         (1) Irreversible inhibitor of cholinesterase.
         (2) Also inhibits plasma cholinesterase and can increase the duration of succinylcholine[7]

READ MORE
Myasthenia gravis, Chapter 71

3) **Pharmacology**[1]
   a) Anticholinesterases
      i) Commonly used agents, neostigmine and edrophonium, follow a two-compartment model after IV injection.
         (1) A rapid distribution phase with peak plasma concentrations is seen within 5-10 minutes following bolus administration of neostigmine or edrophonium. This is followed by a more gradual decline in plasma concentrations associated with drug elimination.
         (2) If maximum doses of neostigmine (60-80 μg/kg) or edrophonium (1.0-1.5 mg/kg) fail to produce adequate neuromuscular recovery, further doses will not improve recovery, as all acetylcholinesterase has already been antagonized. At this point, recovery of neuromuscular function is dependent on metabolism and elimination of remaining NMB drug.[7]
      ii) All are water-soluble, ionized compounds with excretion by the kidney.
         (1) Impaired renal function will decrease clearance of anticholinesterase drugs, as much or more, than NMB drugs. Therefore, no dose adjustments are needed for renal impairment.
   b) Sugammadex
      i) Not metabolized
      ii) Excreted unchanged by the kidneys
      iii) Changes pharmacokinetics of rocuronium from hepatobiliary elimination of unbound drug to predominantly renal excretion of the rocuronium-sugammadex complex[8]

4) **Reversal of neuromuscular blockade**
   a) Anticholinesterases are ineffective if profound neuromuscular blockade is present such that all potential ACh receptors are occupied.
      i) It is advisable to wait until as much spontaneous recovery as possible has occurred, prior to administering the anticholinesterase.[9]
      ii) If three or four twitches are observed with train-of-four (TOF) stimulation, recovery with neostigmine will be much more effective and rapid than if one of four twitches is present.
   b) Sugammadex is seen to be effective in reversing even profound levels of rocuronium blockade.

i) Dosing is dependent on the depth of blockade at the time of reversal.

c) Evidence of effect

i) Objective evidence of neuromuscular recovery with quantitative monitoring provides the only reliable measure of residual NMB.

(1) Train-of-four ratio (TOFR) 0.7 has traditionally defined the threshold indicating adequate recovery from neuromuscular blockade.

(2) Recent evidence supports TOFR 0.9 to more reliably indicate adequate recovery of pharyngeal and upper airway function.[9]

ii) Clinical indicators of neuromuscular recovery have traditionally included sustained head lift >5 seconds, grip strength, and tidal volume. These may be unreliable as sole predictors of adequate recovery.

(1) Head lift >5 seconds is not specific for recovery of respiratory function and sustained head lift can be seen with TOFR < 0.5.



Excessive doses of neostigmine can cause paradoxical neuromuscular relaxation.

d) Excessive doses of neostigmine or administration in the absence of residual NMB drugs can cause paradoxical neuromuscular relaxation.[5,7]

i) Acetylcholinesterases increase presynaptic nerve terminal ACh release increasing ACh levels at the neuromuscular junction even further.

ii) Inhibition of acetylcholinesterase by neostigmine leads to an increased amount and prolonged exposure of the ACh receptor to acetylcholine at the neuromuscular junction.

(1) Can produce phase 2 block.

(2) Prolonged channel opening makes the receptor more susceptible to open channel blockade.

iii) Neostigmine acts as a competitive antagonist at the ACh receptor.

5) **Adverse effects/toxicity**

a) ACh is the neurotransmitter for the entire parasympathetic nervous system.

b) Increased ACh due to cholinesterase inhibition allows ACh to bind parasympathetic muscarinic receptors with side effects in multiple systems.

i) Cardiovascular—bradycardia, QT prolongation, bradyarrhythmias, asystole

ii) Pulmonary—bronchospasm, increased secretions

iii) Gastrointestinal—increased peristaltic activity, increased secretions

(1) Neostigmine may cause increased nausea and vomiting.

(2) However, this is controversial, and a meta-analysis did not show a difference with neostigmine use.



Physostigmine crosses the blood-brain barrier and can cause diffuse CNS excitation.

iv) Genitourinary—increased bladder tone

v) Ophthalmic—constricted pupils, decreased intraocular pressure

vi) Neurologic—diffuse CNS excitation

(1) Seen only with physostigmine, an anticholinesterase that crosses that blood-brain barrier.

c) Most adverse effects caused by ACh binding to muscarinic receptors as described above can be prevented with the concurrent administration of an antimuscarinic medication such as glycopyrrolate or atropine.

i) Sugammadex reverses neuromuscular blockade by chelating NMB agents and does not produce the adverse muscarinic effects seen with anticholinesterases.

d) **Cholinergic crisis**

i) Patients present with all of the above muscarinic effects.

ii) Seen most commonly in patients with myasthenia gravis undergoing treatment with anticholinesterases.

READ MORE
Myasthenia gravis, Chapter 71

e) **Organophosphate poisoning**[10]
   i) Anticholinesterases are used as nerve gas agents and pesticides.
   ii) Excessive muscarinic activity as described above (cholinergic crisis) is observed.
   iii) Central nicotinic effects are also seen.
      (1) Can result in respiratory depression
   iv) Treatment
      (1) Medical management of organ system dysfunction.
      (2) Atropine may be needed in large doses or as an infusion to counteract adverse muscarinic effects.
      (3) Pralidoxime reactivates acetylcholinesterase, but must be given early in the course of treatment before permanent inactivation of the enzyme occurs.
   v) Nicotinic effects require supportive care.
      (1) Central nicotinic overstimulation can lead to headaches, ataxia, cardiovascular or respiratory impairment, tremor, weakness, convulsions, and coma.

6) **Anticholinergic medications used with anticholinesterases**[1]
   a) Edrophonium has an onset of 1-2 minutes and is best used in conjunction with atropine (0.007 mg/kg).
   b) Neostigmine's peak onset is 7-11 minutes and is best used with glycopyrrolate 0.007 mg/kg or atropine 0.015 mg/kg.

CHAPTER SUMMARY

Mechanism of Action	NMB: Depolarizing: Binds receptor and facilitates prolonged muscle depolarization Nondepolarizing: Competitively binds receptor, resulting in inhibition of ACh binding Anticholinesterases inhibit cholinesterase → ↑ ACh available at the ACh receptor
Common Uses	NMB: Facilitation of intubation and surgical conditions Patient immobility, ventilatory support for critical care Anticholinesterases: Reversal of neuromuscular block, treatment of myasthenia gravis, glaucoma, central anticholinergic syndrome, Alzheimer dementia
Adverse Effects	NMB: Depolarizing: Hyperkalemia, trigger malignant hyperthermia, increases in IOP/ICP/IGP, myalgias, bradycardia especially with repeat dosing Nondepolarizing: Prolonged respiratory muscle weakness Anticholinesterase: Increased ACh produced by acetylcholinesterase binds to muscarinic receptors

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27 Benzodiazepines

Stacy L. Fairbanks

Benzodiazepines are sedative and hypnotic drugs that can be used for anxiolysis, anterograde amnesia, skeletal muscle relaxation, control of seizures, sedation, and induction of anesthesia. Benzodiazepines are widely used in anesthesia and can be used safely in a wide variety of patients. This chapter discusses the indications, contraindications, pharmacokinetics, and pharmacodynamics of benzodiazepines in the setting of perioperative medicine.

1) **Overview**[1]
   a) Benzodiazepines are $GABA_A$ agonists that are widely used perioperatively for amnesia, anxiolysis, sedation, and hypnosis.
   b) An overview of this class of drugs will be provided here. For more information on specific drugs, including dosing guidelines, refer to the Appendix of Anesthesia Drug Information, Chapter 214.

READ MORE
Appendix of anesthesia drug information, Chapters 211-225

2) **Mechanism of action**
   a) Benzodiazepines bind to specific receptor sites on the alpha subunits of the gamma-aminobutyric acid (GABA) receptor.
      i) Binding of benzodiazepines enhances the affinity of the receptor for GABA. The maximum effect of benzodiazepines is dependent on the prevailing levels of GABA.
      ii) This causes increased chloride conductance and hyperpolarizes postsynaptic neurons, resulting in inhibition of synaptic transmission.
   b) **Structure/activity relationships**
      i) See **Figure 27-1**.
      ii) Representative medications include midazolam, lorazepam, and diazepam.
   c) **Pharmacokinetics**[2]
      i) Absorption
         (1) Rapidly absorbed from the gastrointestinal tract with oral dose
         (2) Rapidly enter the CNS and other highly perfused organs with IV injection (small size and high lipid solubility at physiologic pH)
      ii) Distribution
         (1) Highly lipid soluble
            (a) Midazolam has an imidazole ring in structure, which allows the preparation of acidic aqueous solutions. Once exposed to body pH 7.4, the diazepine ring closes and midazolam becomes lipid soluble, and it is rapidly distributed. It is minimally painful on injection.
         (2) Highly protein bound (to albumin)
            (a) Disease states that cause hypoalbuminemia (cirrhosis, malnutrition, chronic renal failure) result in increased proportion of unbound drug and lower dosage required to achieve the desired effect.

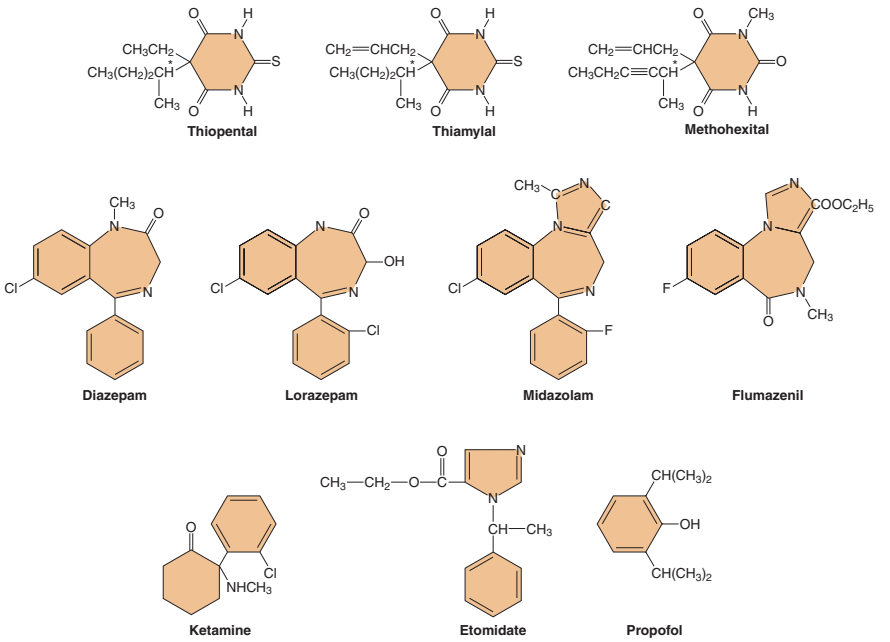


Diazepam and lorazepam are not soluble in water and, thus, are traditionally formulated in propylene glycol and ethanol. This can lead to pain on injection and long-term infusions lead to risk of glycol toxicity.



Diazepam and midazolam are metabolized by hepatic microsomal enzymes and have active metabolites.

FIGURE 27-1 Chemical structures of currently available nonopioid intravenous anesthetics.



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d) **Metabolism**
   i) Diazepam and midazolam are metabolized by cytochrome P450s in the liver and have active metabolites.
   ii) Lorazepam is metabolized by glucuronidation only and does not have active metabolites.

3) **Special considerations**
   a) Benzodiazepines decrease cerebral metabolic oxygen requirements ($CMRO_2$) and cerebral blood flow (CBF).
   b) Unlike barbiturates and propofol, midazolam treatment does not result in an isoelectric EEG.
   c) May cause anterograde amnesia (decreased ability to acquire new information)

4) **Clinical uses**
   a) **Preoperative anxiolysis (especially for pediatric patients)**
   b) **Anticonvulsant**
   c) **Sedation for procedures or in critical care units**
   d) **Alcohol detoxification/withdrawal**
   e) **Skeletal muscle relaxation (spinal cord mediated)**
      i) Not effective for surgical relaxation



Oral midazolam in children has an onset of action within 10 minutes, peaking at 20-30 minutes.



Benzodiazepines potentiate the sedative effects of other medications (especially opioids).

5) **Adverse effects/toxicity**
   a) **Dependence**
   b) **Fatigue**
   c) **Drowsiness**
   d) **Depression of airway muscle reflexes and tone**
      i) Can increase airway obstruction in patients with obstructive sleep apnea or in elderly patients
   e) **Decreased ventilation** (dose dependent)
      i) Most pronounced in patients with chronic pulmonary disease, in the elderly, and in conjunction with other respiratory depressants
   f) **Potentiates the sedative effects of other medications** (especially opiates)
   g) **Decreased heart rate, systemic vascular resistance, and blood pressure with large (ie, anesthetic induction) doses**
   h) **Postoperative use could potentiate postoperative delirium in patients at risk.**[3]



Flumazenil is a specific benzodiazepine antagonist used to treat benzodiazepine overdose.



Due to the rapid clearance, there is high potential for flumazenil to be cleared more quickly than the benzodiazepine, causing recurrent sedation.

6) **Treatment of toxicity**[4]
   a) **Flumazenil is a specific benzodiazepine antagonist**. It reverses benzodiazepine effects, including ventilatory depression.
      i) Titrate the drug to the individual, with an initial dose of 4-20 µg/kg IV (0.2 mg) and additional doses of 0.1 mg given at 1 minute intervals up to 1 mg. Typically effective doses are 0.3-0.6 mg.
      ii) Flumazenil has very rapid clearance and a short elimination half-life. Duration of action is 30-60 minutes. May need to be repeated depending on the duration of action of the benzodiazepine administered. Alternatively, an infusion of 0.1-0.4 mg/h can be used.
      iii) May precipitate withdrawal seizures in patients on antiepileptic medications
      iv) May increase intracranial pressure in head-injured patients

CHAPTER SUMMARY FOR BENZODIAZEPINES

Definition	Benzodiazepines are sedative and hypnotic drugs widely used in the perioperative time period
Mechanism of Action	Bind to sites on the alpha subunit of the GABA receptor, increasing affinity of receptor for GABA, resulting in inhibition of synaptic transmission
Uses	Used for sedation, anxiolysis, and induction of anesthesia
Treatment of Toxicity	Benzodiazepine overdose can be treated with flumazenil, a specific benzodiazepine antagonist agent

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28 Opioid Agonists and Antagonists

Daniel S. Orlovich

Opioids are used for pain management, anesthesia, and critical care. Opioids are the primary agents used to treat moderate to severe pain. This chapter provides a brief overview of the mechanism of action and clinical pharmacology of commonly used opioids in perioperative practice.

1) **Mechanism of action**[1]
   a) **Opioids bind to opioid receptors, which are coupled to G proteins and inhibit adenyl cyclase.**
      i) Inhibition of adenyl cyclase leads to inhibition of voltage-gated calcium channels and opening of potassium channels.
      ii) This results in increased intracellular potassium, resulting in hyperpolarization and decreased neurotransmission.
   b) **Receptor subtypes.** The physiologic function of opioid receptors is to bind endogenous opioids and provide endogenous analgesia in response to pain.
      i) Mu receptors are in the brain, spinal cord, and periphery and are the primary receptors responsible for the analgesic and adverse effects of opioids, especially morphine.
         (1) Mu_1 subtype—analgesia.
         (2) Mu_2 subtype—hypoventilation, bradycardia, and physical dependence. No specific endogenous ligands have been identified for Mu_2 receptors.
      ii) Kappa receptors inhibit neurotransmission via type N calcium channels.
         (1) Responsible for dysphoria and diuresis
            (a) Diuresis occurs because binding to the kappa receptor inhibits the release of vasopressin.
      iii) Delta receptors modulate mu receptor activity.
         (1) Endogenous opioids include enkephalins, endorphins, and dynorphins.
2) **Structure/activity relationships**
   a) The term *opiate* is used to describe compounds derived from the poppy (*Papaver somniferum*).
      i) The prototype opiate is morphine.
   b) The term *opioid* refers to completely synthetic or semisynthetic modifications of morphine.
   c) Representative opioid medications include hydromorphone, meperidine, fentanyl, alfentanil, sufentanil, and remifentanil.
3) **Administration of opioids**
   a) There are multiple routes of administration, including oral, parenteral, intramuscular, neuraxial, transdermal, and transmucosal.
   b) **Parenteral administration**
      i) Distribution
         (1) Onset and duration of action depend on the lipid solubility and ionization of the drug.

READ MORE
Opioids, Appendix of Drug Information, Chapter 215



The term "narcotic" is derived from the Greek word for stupor. It refers to any medication that cause dependence and therefore does not refer only to opioids.

(2) Drugs diffuse across the blood-brain barrier and redistribute to inactive tissue sites such as skeletal muscle and fat.
(3) High doses of lipid soluble drugs (eg, fentanyl) lead to saturation of active sites and buildup of the drug in the plasma, resulting in a longer duration of action.

READ MORE
Obstetrical anesthesia overview, Chapter 107

ii) Metabolism[1-3]
(1) Most opioids are metabolized in the liver and excreted by the kidney.
(2) An exception is remifentanil, which is rapidly metabolized by plasma esterases.
(3) Many opioids have long-acting, pharmacologically active metabolites.
(a) Normeperidine is the active metabolite of meperidine and has renal elimination.
(i) Accumulation of normeperidine can cause seizures and should be used with caution in patients with renal failure.
(b) Morphine-6-glucuronide is an active metabolite of morphine and can accumulate in neonates and those with renal failure.



Patients who have received neuraxial morphine should have respiratory monitoring due to the risk of delayed respiratory depression.



Hypothermia and pruritus can occur after neuraxial opioids and may be treated with naloxone.

c) **Neuraxial Administration**
i) Neuraxial opioids act on mu receptors in the substantia gelatinosa of the spinal cord.
ii) Epidural administration requires diffusion across the dura to bind receptors in the spinal cord.
iii) Spinal administration requires lower doses because diffusion across various tissues and fascia is not necessary to reach the site of action.
(1) The typical spinal dose to produce a desired effect is 5-10 times less than the epidural dose.
iv) Pharmacokinetics for neuraxial opioids.
(1) Lipid-soluble opioids (fentanyl) are more likely to have systemic or fat absorption and thus have a shorter duration of action.
(2) Water-soluble opioids (morphine) have a longer onset but longer duration, due to increased time needed to diffuse across the dura.
(3) The duration of action of neuraxially administered opioids depends on diffusion of the drug out of the cerebrospinal fluid (CSF) and into the blood and not on metabolism.



Particular attention should be paid toward certain patients who are at increased risk of respiratory depression including those with obesity, obstructive sleep apnea, and who are receiving parental opioids, sedatives, hypnotics, or magnesium.

v) Effects of neuraxial opioids
(1) Analgesia—due to effect on Mu_1, $Delta_1$ and $Delta_2$ receptors
(2) Pruritus—due to effect on Mu_2 receptors
(3) Urinary retention—due to effect on Mu_2 receptors
(4) Hypoventilation—due to effect on Mu_2 receptors
(a) May be delayed up to 12 hours, especially after neuraxial morphine administration
(5) Hypothermia—due to effect on Mu_2 receptors
(6) Nausea and vomiting—due to effect on Mu_2 receptors



Opioids delivered via the spinal route have several advantages over parenteral opioids including increased potency, decreased daily dose requirement, decreased central nervous system depression, decreased incidence of ileus, and decreased potential for abuse.

d) Transdermal/transmucosal administration.
i) Both routes avoid first-pass metabolism.
ii) Fentanyl can be administered via either route due to its high potency and solubility in both water and lipids.

iii) The transmucosal route offers the advantage of rapid onset.
iv) The transdermal route is frequently used for treatment of chronic pain.

e) **Equivalent Dosing (Table 28-1).**

f) **Single bolus dose pharmacokinetics** (Table 28-1, Fig. 28-1).
   i) Opioids have various time to peak effect after an IV bolus.
      (1) Alfentanil and remifentanil have the fastest peak to peak effect followed by fentanyl and then hydromorphone.
      (2) Morphine has the slowest peak effect.
   ii) After 20 minutes, remifentanil has the greatest decrease in percent of peak effect followed by alfentanil and fentanyl.

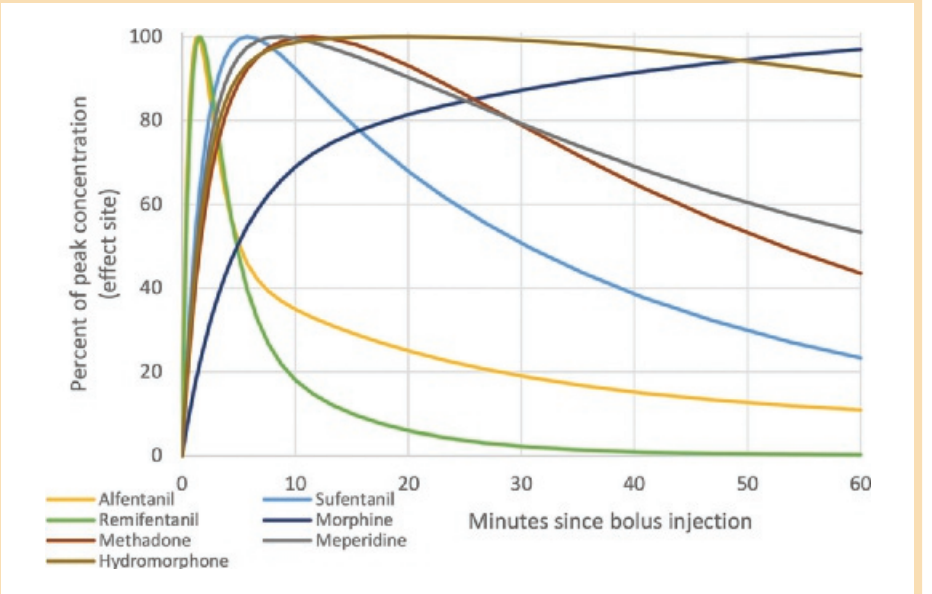
g) **Intravenous infusion dose pharmacokinetics** (Fig. 28-2).
   i) Opioids administered as an infusion, not a single bolus, have various times required to have a 50% drop in concentration at peak site.

TABLE 28-1 Intravenous Dosing of Opioid Medications

Drug	Approximate Analgesic Equivalent	Peak Onset	Duration of Single Bolus	Notes
Alfentanil	500 µg	1-2 min	5-10 min	Fastest onset of all opioids, thus used for brief periods of intense stimulation
Fentanyl	50 µg	3-5 min	30-60 min	
Hydromorphone	0.75 mg	5-15 min	2-4 h	Metabolite provides no analgesia but may cause neuroexcitation
Meperidine	37.5 mg	5-15 min	2-4 h	Caution with renal failure since active metabolite lowers seizure threshold Avoid using with monoamine oxidase (MAO) inhibitors since it may cause central nervous system excitation or depression May cause tachycardia given atropine-like effect It is the only opioid with negative inotropic effects
Morphine	5 mg	10-20 min	4-5 h	Caution with renal failure since active metabolite is renally excreted and causes analgesia May release histamine and cause vasodilation and hypotension
Remifentanil	50 µg	3-5 min	5-10 min	Unique since it is only opioid broken down by nonspecific esterases in blood and not affected by renal dysfunction nor significantly affected by hepatic dysfunction
Sufentanil	5 µg	3-5 min	20-45 min	

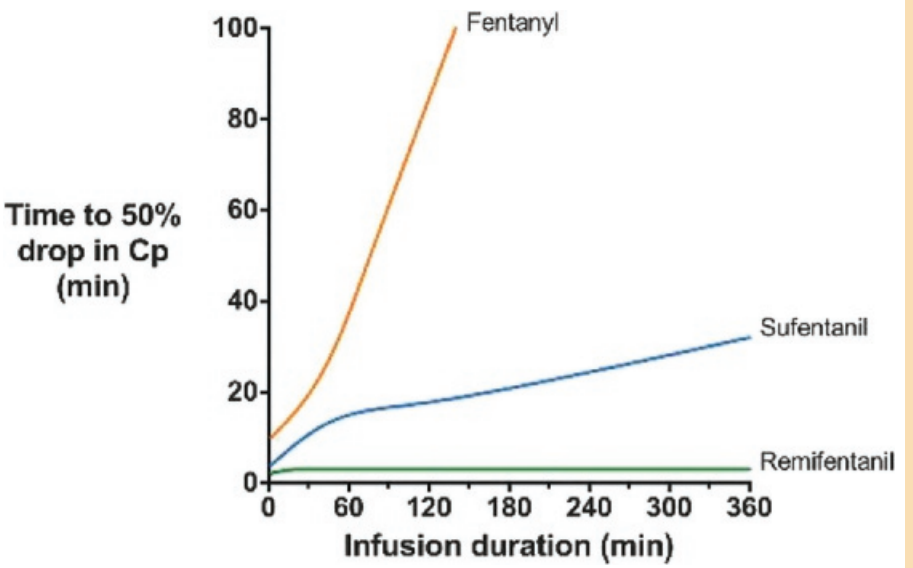
Modified with permission of McGraw-Hill, from Yaksh H, Wallace M. Opioids, analgesia and pain management. In: *Goodman and Gilman's the Pharmacological Basis of Therapeutics*. 13th ed. New York, NY: McGraw-Hill; 2018; permission conveyed through Copyright Clearance Center, Inc. Ref.[4]

FIGURE 28-1 Single Bolus Pharmacokinetics



Special thanks to Steven L. Shafer MD for the above image. Reprinted with permission from Shafer SL, Varvel JR. Pharmacokinetics, pharmacodynamics, and rational opioid selection. *Anesthesiology*. 1991;74(1):53-63. Ref.[5]

FIGURE 28-2 Relationship Between Infusion Duration and Time to 50% of Plasma Concentration



Reproduced with permission from Hughes MA, Glass PSA, Jacobs JR. Context-sensitive half-time in multicompartment pharmacokinetic models for intravenous anesthetic drugs. *Anesthesiology*. 1992;76:334.

ii) Remifentanil is unique opioid, in that it has a relatively constant time required to have a 50% drop in concentration. Thus, it is considered "fast off" medication.
iii) A fentanyl infusions exhibit a unique profile. For example, if it is infused for about 2 hours, it takes roughly 80 minutes to decrease in the plasma concentration by 50%, whereas remifentanil and sufentanil take roughly 5-10 minutes and 20 minutes, respectively. Thus, the duration of the fentanyl infusion can be considered "less forgiving," and a long infusion may lead to long duration of action.

4) **Special considerations and drug interactions**
   a) The combination of monoamine oxidase inhibitors and meperidine can result in a severe reaction including fever, convulsions, hemodynamic instability, and coma.
5) **Clinical uses**[1]
   a) **Pain management**
   b) **Sedation for procedures or critical care**
   c) **Anesthesia for cardiac surgery**
   d) **Cough suppression**
   e) **Adjuncts to anesthesia**
      i) The use of opioids leads to decreased minimum alveolar concentration (MAC) requirements for volatile and other intravenous anesthetics.
6) **Adverse effects/toxicity**[1,2]
   a) **Physical dependence**
   b) **Drowsiness**
   c) **Decreased ventilation**
      i) Opioids produce a dose-dependent change in ventilatory response to increased $PaCO_2$.
   d) **Potentiation of sedative effects of other medications**
   e) **Analgesic tolerance**
      i) May limit clinical utility
   f) **Bradycardia** (dose dependent)
      i) Meperidine is an exception. It has an atropine-like structure and tends to cause tachycardia.
   g) **Skeletal muscle rigidity**
      i) Especially seen with rapid IV administration.
      ii) Ventilation may be difficult and require administration of a muscle relaxant.
   h) **Histamine release**
      i) Most commonly associated with morphine
      ii) Causes vasodilation and hypotension
   i) **Miosis**
   j) **Decreased gastrointestinal (GI) motility**
      i) Constipation and miosis continue even with development of opioid tolerance to analgesic effects.
   k) **Decreased fetal heart rate variability** when administered to pregnant patients.
7) **Agonist/antagonists**
   a) Representative drugs in this class: nalbuphine, buprephenone, buprenorphine, and butorphanol.
      i) These medications may be used for opioid use disorder.
      ii) There is no agreed upon consensus about proper management of patients who present for surgery on buprenorphine. Broadly speaking, multimodal agents such as clonidine, ketamine, ketorolac, celebrex, and gabapentin as well as regional anesthesia



Meperidine should be avoided in patients with renal failure and history of seizures and those taking monoamine oxidase inhibitors.

READ MORE
Acute pain management, Chapter 132



The opioid agonist/antagonist nalbuphine is very effective for the treatment of pruritus due to neuraxial opioids.

and local anesthesia by the surgeon are recommended. One strategy is to consider if the patient takes <12 mg of buprenorphine then to continue it perioperatively. If the patient is on >12 mg then taper it for 2-3 days to 12 mg and then augment with 2-4 days of high-dose opioids.[6]



Patients who have received a long-acting opioid who require opioid antagonism may require repeat doses or an infusion of naloxone.

b) Agonist/antagonist medications have complex interactions with receptors.

c) These drugs have analgesic effects when used alone but with a ceiling effect, beyond which increased doses do not produce desired results or adverse effects.
   i) Can result in analgesia with minimal respiratory depression

d) When combined with a full agonist acts as an antagonist.
   i) Nalbuphine 2.5-10 mg IV is very effective in relieving pruritus induced by neuraxial opioids.

8) **Treatment of toxicity**

a) **Naloxone is a specific opiate receptor antagonist**, binding the receptor without causing activation.

b) The effective dose is 1-4 μg/kg IV, and the duration of action is 30-45 minutes.
   i) Dose may need to be repeated or an infusion started (5 μg/kg/h) after administration of a long-acting opiate.



Abrupt reversal of analgesia may cause a catecholamine surge as reflected by tachycardia, cardiac dysrhythmias, and hypertension.[7]

c) **Side effects of naloxone**
   i) Reversal of analgesia, nausea, vomiting, and increased sympathetic nervous system activity, including tachycardia, hypertension, pulmonary edema, and cardiac dysrhythmias.

CHAPTER SUMMARY FOR OPIOID ANALGESICS AND ANTAGONISTS

Mechanism of Action	Bind opioid receptors, and inhibit adenyl cyclase via G protein pathway, causing decreased calcium influx and activation of potassium channels, resulting in decreased neuronal activation, decreased neurotransmitter release, and analgesia.
Common Uses	Pain management, sedation, cough suppressant
Adverse Effects	Respiratory depression, pruritus, sedation, physical dependence, decreased gastrointestinal motility, miosis, potentiation of other sedative medications, skeletal muscle rigidity
Considerations With Neuraxial Opioids	Delayed respiratory depression is possible (morphine), pruritus may be severe and treated with nalbuphine, and spinal dose is usually 5-10 times less than epidural.
Treatment of Toxicity	Effects may be reversed with opioid receptor antagonist, naloxone. Naloxone has a shorter half-life than most opioids and may need redosing.

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29 Nonsteroidal Anti-inflammatory Drugs and Acetaminophen

Marisa A. Wiktor

Nonsteroidal anti-inflammatory drugs (NSAIDs) are commonly used analgesic medications that are composed of two categories: nonselective and selective cyclooxygenase (COX)-2 inhibitors. Frequently, NSAIDs are used as either first-line therapy in pain management or are administered as adjuvant drugs in conjunction with other analgesics; these medications primarily exhibit their effects by blocking thromboxane and prostaglandins. They have various adverse effects, the most concerning being gastrointestinal and cardiovascular. Acetaminophen is accepted as a first-line medication for the treatment of mild pain and pyrexia. Efficacy of acetaminophen can be increased when combined with NSAIDs, COX-2 inhibitors, or opioids.

1) **Overview**
   a) NSAIDs are used for pain management and treatment of inflammatory conditions.
   b) Acetaminophen is an effective and safe agent for mild pain.
   c) An overview of this class of drugs is provided here. For more information on specific drugs, refer to the Appendix of Anesthesia Drug Information, Chapter 217.
2) **Mechanism of action**[1-3]
   a) NSAIDs inhibit cyclooxygenase (COX), the enzyme responsible for converting arachidonic acid to thromboxane A2 and prostaglandins.
   b) COX has two separate gene products:
      i) (COX-1) is always functional, or constitutive.
      ii) (COX-2) is inducible.
   c) Thromboxane A2 activates platelets and increases platelet aggregation.
   d) Prostaglandins are compounds that mediate a wide variety of physiologic processes, including platelet aggregation and inflammation.
   e) Acetaminophen's exact mechanism of action is unknown but is believed to inhibit prostaglandin production and affects nitric oxide (NO), serotonergic, opioid and cannabinoid pathways, without evidence of reducing inflammation.
3) **Structure-activity relationships**
   a) **Two categories of NSAIDs**
      i) Nonspecific COX inhibitors (ie, aspirin, ketorolac, ibuprofen, acetaminophen)
      ii) Specific COX-2 inhibitors (rofecoxib, valdecoxib, celecoxib)
4) **Pharmacokinetics**
   a) Typically, well absorbed and have low first-pass metabolism.
   b) Highly protein bound agents with a small volume of distribution
   c) NSAIDs are metabolized in the liver and excreted by the kidney.
5) **Special considerations**
   a) **COX-2 inhibitors**[1]



NSAIDs nonspecifically inhibit COX-1 and COX-2, the enzyme responsible for converting arachidonic acid to thromboxane A2 and prostaglandins, respectively.

Selective COX-2 inhibitors are specific to COX-2 inhibition and prevent the formation of prostaglandins.

i) COX-2 is inducible mainly at inflammatory sites, and COX-2 inhibitors were developed to treat inflammation and pain. COX-2 inhibitors are as effective as conventional (nonselective) NSAIDs for the treatment of pain.
ii) Rofecoxib was withdrawn from the market after reports of increased incidence of myocardial infarction and cerebral vascular accidents.
iii) Valdecoxib was withdrawn from the market due to an association with life-threatening skin reactions (Stevens-Johnson syndrome) and increased cardiovascular events.
iv) Celecoxib is available and does not appear to have a higher incidence of cardiovascular events compared with traditional NSAIDs[4].



Large doses of acetaminophen may cause hepatic necrosis and failure.

b) **Acetaminophen**
i) Acetaminophen is an effective analgesic and antipyretic that lacks anti-inflammatory properties.
ii) Does not cause gastric irritation or inhibition of platelet aggregation
iii) May contribute to analgesic-induced nephropathy
iv) High dose acetaminophen (single dose >1500 mg/dose or >4000 mg/day) can cause hepatic failure.
(1) Hepatotoxicity can occur when taken with alcohol.
(2) *N*-acetyl cystine antagonizes the hepatotoxic effects and is an effective antidote when given within 8 hours of overdose.



Single-dose/short-term use of ketorolac can increase postoperative site bleeding in cases that leave abraded edges, for example, tonsillectomy, total joints, and plastic surgery.[5]

6) **Clinical uses**[1-3]
a) Postoperative pain management/multimodal analgesia
b) Closure of patent ductus arteriosus in neonates (indomethacin)
c) Long-term treatment of various inflammatory disorders, that is, arthritis, osteoarthritis, rheumatoid arthritis, and ankylosing spondylitis
d) Treatment of pyrexia
e) Treatment of familial adenomatous polyposis (celecoxib)



ASRA consensus guidelines:

NSAIDs alone should not interfere with the placement of neuraxial blocks or the timing of neuraxial catheter removal.[6]

7) **Adverse effects/toxicity (Table 29-1)**
a) Renal impairment
b) Bleeding

TABLE 29-1 Adverse Effects of NSAIDs

System	Adverse Effect
Central nervous system	Headache, aseptic meningitis, hearing disturbances
Cardiovascular	Hypertension, thrombosis (myocardial infarction), heart failure
Respiratory	Rhinitis, dyspnea, bronchospasm, asthma exacerbation, angioedema
Gastrointestinal	Gastric/duodenal ulcer, esophagitis, NSAID enteropathy
Hematological	Platelet inhibition/dysfunction with potential for bleeding/coagulopathy, anemia
Dermatological	Stevens-Johnson syndrome, erythema multiforme, morbilliform rash
Renal	Renal insufficiency, papillary necrosis, interstitial nephritis, edema
Hepatic	Hepatitis
Skeletal	May inhibit bone growth/healing

i) Due to platelet inhibition
ii) Aspirin causes irreversible platelet inhibition.
iii) Patients may need platelet transfusion to reverse the effect of aspirin on platelets in specialized cases (eg, patients with intracranial bleed).

c) Gastrointestinal irritation
   i) Possible gastric bleeding
d) Asthma (susceptible individuals)—by blocking synthesis of prostaglandin E_2
e) Hypertension

CHAPTER SUMMARY FOR NSAIDS

Definition	NSAIDs are commonly used medications for the treatment of pain and inflammatory disease
Mechanism of Action	NSAIDs inhibit COX enzyme, the enzyme responsible for converting arachidonic acid to prostaglandins
Uses	Used for treatment of pain and inflammatory diseases. Useful adjuvant medication for multimodal treatment of pain
Adverse Effects/Toxicity	Renal impairment and bleeding can occur with NSAID use. May aggravate asthma in susceptible individuals

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30 Intravenous Induction Agents

Kristin L. Richards • Jerry Ingrande

Intravenous (IV) induction agents are medications that cause a rapid loss of consciousness. The choice of agent is determined by the procedure and the patient's coexisting diseases. These medications can be used to induce general anesthesia, as a sole medication for a short procedure, or to provide sedation. Some of these medications can be given as infusions for maintenance of general anesthesia either in place of or in combination with inhalational agents.

1) **Overview**
   a) Propofol, thiopental, etomidate, and ketamine are intravenous induction agents.
   b) Dexmedetomidine, although not an induction agent, is included in the discussion. The mechanism of action, clinical use, physiologic effects, and pharmacokinetics of each medication are included.
   c) Barbiturates, benzodiazepines, propofol, and etomidate act via GABA.
      i) GABA decreases the excitability of neurons by acting at the GABA-A receptor complex (**Fig. 30-1**).
      ii) When GABA binds to its receptor, it allows chloride to enter the cell, which hyperpolarizes the cell, and as a result the neuron resists stimulation by excitatory neurotransmitters.
   d) Ketamine is a NMDA receptor antagonist and blocks the action of glutamate, which is an excitatory neurotransmitter.
   e) Dexmedetomidine is a centrally acting alpha-2 agonist and decreases sympathetic outflow and causes sedation and some analgesia.
2) **Propofol**
   a) **Propofol (2,6-diisopropylphenol) is a water-insoluble IV anesthetic clinically available as a 1% emulsion containing 10% soybean oil, 2.25% glycerol, and 1.2% egg phosphatide**. It is widely used, due to its fast onset and rapid recovery pharmacokinetic profile.
   b) **Mechanism of action**
      i) Selective GABA receptor agonist
   c) **Clinical use**
      i) Induction
         (1) Propofol is given as an IV bolus (1.5-2.5 mg/kg) for induction of general anesthesia. Its dose should be reduced in the elderly population and administered slowly.
         (2) Although not reported to induce histamine release, it should be used cautiously in persons with egg allergy.
            (a) Pain after injection is often reported.
            (b) The mechanism of pain on injection is unclear; however, its incidence is greater when propofol is given through small veins on the dorsum of the hand as opposed to large veins in the antecubital fossa.
      ii) Maintenance
         (1) Propofol is commonly used as a continuous infusion for maintenance of both general anesthesia (100-200 µg/kg/min) and conscious sedation (25-75 µg/kg/min).
         (2) Supplementation with nitrous oxide can reduce maintenance requirements.

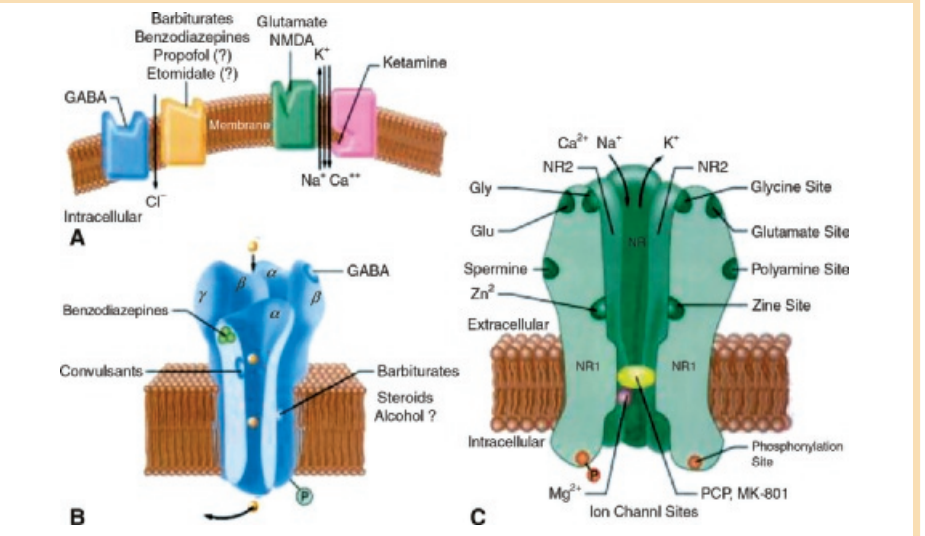


The addition of local anesthetics (ie, 1%-2% lidocaine) to propofol may decrease pain upon injection.



Propofol is often used as an infusion, alone, combined with other intravenous medications, or in combination with inhalational agents.

FIGURE 30-1 Gamma Aminobutyric Acid A (GABA-A) Receptor Complex.



A. Reprinted from Van Hemelrijck J, Gonzales JM, White PF. Use of intravenous sedative agents. In: Rogers MC, Tinker JH, Covino BG, et al., eds. *Principles and Practice of Anesthesiology*. St. Louis: Mosby; 1992:1131, with permission from Elsevier. **B.** Altered with permission from Leeson TD, Iversen LL. The glycine site on the NMDA receptor: structure-activity relationships and therapeutic potential. *J Med Chem*. 1994;37:4053. Copyright © 1994 American Chemical Society.

d) **Effects on major organ systems**
   i) Central nervous system
      (1) Decreases cerebral blood flow (CBF), cerebral metabolic activity, and intracranial pressure (ICP).[1]
      (2) Large doses may decrease cerebral perfusion pressure secondary to decreases in CBF.
   ii) Cardiovascular system
      (1) Causes direct myocardial depression and decreased systemic vascular resistance (SVR) that are both dose and concentration dependent
         (a) Doses should be reduced for the elderly or those who are volume depleted.
      (2) Attenuates the baroreflex mechanism[2]
   iii) Respiratory System
      (1) Produces respiratory depression, causing apnea in ~25% of subjects after an induction dose.
      (2) There is a decreased ventilatory response to hypercarbia and hypoxia, which is exaggerated in the presence of opioids.
   iv) Other effects
      (1) Propofol has antiemetic properties that have popularized its use for outpatient procedures or for maintenance of anesthesia in subjects who are prone to postoperative nausea and vomiting.
      (2) It does not trigger malignant hyperthermia (MH) and is considered the induction and maintenance agent of choice in MH susceptible patients.[3]



Propofol causes direct myocardial depression and decreases SVR, and doses should be significantly reduced in the elderly or volume-depleted patients.



Propofol has antiemetic properties.

e) **Pharmacokinetics**
   i) Propofol's rapid recovery profile after a bolus dose is attributed to its rapid distribution from the vessel-rich group (eg, brain), to inactive tissues (muscle and fat).

(1) Initial distribution half-life is 1-8 minutes with an elimination half-life of 2-8 hours.[3]
(2) Metabolites are cleared via the kidneys.
(3) Propofol infusion syndrome is a rare clinical syndrome. Risk factors include infusions of >4 mg/kg/h for more than 48 hours.[4] Symptoms include bradycardia, metabolic acidosis, rhabdomyolysis, hyperlipidemia, and hepatomegaly. Prolonged infusions can turn urine green.

2) **Thiopental**

a) **Thiopental is a highly alkaline derivative of barbituric acid with a pKa of 7.6 and pH of 9.**[3]

i) Reconstitution of thiopental with acidic solutions (ie, lactated Ringer's; nondepolarizing neuromuscular blocking drugs) may result in precipitation of the compound.



Reconstitution of thiopental with acidic solutions can cause precipitation

b) **Mechanism of action**

i) Like propofol, the sedative-hypnotic actions of thiopental are due to its agonism of the GABA receptor.

c) **Clinical use**

i) Induction

(1) The induction dose of thiopental is 3-5 mg/kg in adults and 5-6 mg/kg in children.[3]
(2) Not commonly given in multiple doses or via infusion because of its long terminal half-life, the potential for saturation of inactive tissues (muscle, fat), resulting in delayed awakening.
(3) Pain upon injection is rare.



Thiopental is highly basic; it can cause tissue necrosis when administered through an arterial catheter or subcutaneously (eg, an infiltrated IV catheter).

d) **Effects on major organ systems**

i) Central nervous system

(1) Thiopental reduces cerebral metabolic oxygen requirements, CBF, electroencephalogram (EEG) activity, and ICP.[1]
(2) The reduction of cerebral metabolic oxygen requirement exceeds CBF.
(a) Thiopental can be used for neuroprotection during periods of incomplete ischemia.
(b) There is no benefit in the setting of global cerebral ischemia.[5]

ii) Cardiovascular system

(1) Decreases cardiac output and SVR and causes direct myocardial depression[3]
(a) The decreased cardiac output is a function of decreased venous return secondary to peripheral vasodilatation.
(b) Dose should be reduced in patients with hemodynamic compromise.

iii) Respiratory system

(1) Causes dose-dependent respiratory depression, with depression of central ventilatory centers[1]
(2) Decreases the ventilatory response to hypercarbia

e) **Pharmacokinetics**

i) The rapid induction of anesthesia after a bolus dose of thiopental is attributed to its rapid distribution into the effect site.
ii) Redistribution of drug from the central compartment into a large peripheral compartment reservoir accounts for the rapid awakening after a single induction dose.
iii) Thiopental has a low hepatic extraction ratio, and hepatic clearance is dependent on the intrinsic metabolic activity of the liver.[3]

3) **Etomidate**

a) **Etomidate is an imidazole-containing IV induction agent that undergoes intramolecular rearrangement at physiologic pH.**[3]

i) Because of its instability at physiologic pH, etomidate is formulated in a 0.2% aqueous solution containing 35% propylene glycol.

b) **Mechanism of action**
   i) Etomidate acts via GABA-A receptors[6]
c) **Clinical use**
   i) Induction
      (1) Etomidate is given as an IV bolus (0.2-0.4 mg/kg) for induction of general anesthesia.
      (2) Not given in multiple doses or as a maintenance infusion as it leads to adrenal suppression.
d) **Effects on major organ systems**
   i) Central nervous system
      (1) Reduces cerebral metabolic oxygen requirement, CBF, and ICP.
      (2) Cerebral perfusion pressure is maintained because of the hemodynamic stability after etomidate administration.
   ii) Cardiovascular system
      (1) Minimal changes in BP, SVR, HR, and cardiac output after administration.
      (2) For these reasons, etomidate is often administered to patients with cardiopulmonary compromise.
   iii) Endocrine system
      (1) Etomidate causes inhibition of 11-beta-hydroxylase, resulting in adrenocortical suppression after its administration.[3]
      (2) This must be taken into consideration in patients dependent on an intact stress response (ie, septic shock or hemorrhage).
   iv) Other effects
      (1) Associated with a high incidence of postoperative nausea and vomiting.
      (2) The propylene glycol formulation results in significant pain upon injection.
      (3) Associated with myoclonus after bolus doses, which can be attenuated with prior administration of opioids.
e) **Pharmacokinetics**
   i) Moderately lipid soluble and almost entirely unionized at physiologic pH, allowing for rapid effect site distribution and subsequent redistribution to peripheral tissues.
      (1) This accounts for the rapid induction and rapid awakening after a single IV bolus.[1]
   ii) Metabolism occurs via hydrolysis of the ester side chain in the liver.



Etomidate causes minimal change in hemodynamics and is often used in patients with cardiopulmonary compromise.



Etomidate causes adrenal suppression. Caution is advised in patients who are dependent on stress response.



Etomidate is painful upon injection, can cause myoclonus after a bolus dose, and is associated with postoperative nausea and vomiting.

4) **Ketamine**
   a) **Ketamine is a water-soluble compound with a pKa of 7.5. It is structurally related to phencyclidine.**
   b) **Mechanism of action**
      i) Interacts with the NMDA receptor, in addition to opioid, noradrenergic, nicotinic, and muscarinic acetylcholine receptors.
      ii) Ketamine's analgesic, amnestic, and psychomimetic effects are due to antagonism of the NMDA receptor on inhibitory interneurons in the cortex, amygdala, and hippocampus. Unconsciousness results from an uncoordinated increase in neural activity.
      iii) In the spinal cord, ketamine decreases arousal by blocking NMDA glutamate-mediated nociceptive signals from peripheral afferent neurons in the dorsal-root ganglion to projecting neurons.
   c) **Clinical use**
      i) Induction
         (1) Ketamine is given as an IV bolus dose of 1-2 mg/kg for induction of general anesthesia.
         (2) For patients without IV access, 4-8 mg/kg may be given intramuscularly.



Ketamine is an NMDA antagonist

ii) Other uses
(1) Ketamine is also used in low doses as an adjunct to narcotics for pain relief, especially for chronic pain. Doses of 0.1-0.5 mg/kg are often given for this purpose.

d) **Effects on major organ systems**



Ketamine can be used for pain relief either as a bolus or infusion. It has a growing role in chronic pain management.

i) Central nervous system
(1) Produces a dissociative anesthetic state characterized by analgesia and amnesia while consciousness is maintained.
(2) High incidence of psychomimetic reactions after awakening from ketamine (emergence delirium, hallucinations, and alterations in mood and affect).
(3) The effects of ketamine related to pain can be best described as antihyperalgesic, antiallodynic, or tolerance-protective.
(4) Unlike the other induction agents, ketamine is reported to be a potent cerebral vasoconstrictor, increasing CBF by 60%. Thus, ketamine is traditionally considered to *increase* cerebral metabolic oxygen requirement, CBF, and potentially ICP; however, this may not be a valid generalization.
(a) Caution is advised in patients with intracranial pathology.



Ketamine increases CBP and ICP. Caution with intracranial pathology.

ii) Cardiovascular system
(1) Produces a sympathomimetic effect with an associated increase in HR, BP, and cardiac index[3]
(2) Can cause direct myocardial depression and in the absence of autonomic control, these effects may be unmasked

iii) Respiratory system
(1) Ketamine has direct bronchodilator properties.
(a) The induction agent of choice in the setting of acute bronchospasm/asthma[3]
(2) In contrast to the other IV induction agents, ketamine is most likely to preserve protective airway reflexes.
(3) An increase in oral secretions may require concomitant use of an antisialogogue.



Ketamine is a bronchodilator, is likely to preserve airway reflexes, and increases oral secretions.

e) **Pharmacokinetics**
i) Highly lipid-soluble, which facilitates its rapid transfer into the brain.[1] This, combined with the drug-induced increase in CBF, is responsible for its rapid onset of action.
ii) Rapidly redistributed to peripheral tissues.
iii) Duration of ketamine-induced anesthesia is ~10-20 minutes after a single bolus dose.[3]
iv) Metabolized by the cytochrome P-450 system to form norketamine, an active metabolite.
v) It has a high hepatic extraction ratio, and alterations in hepatic blood flow may affect the metabolism of ketamine.

5) **Dexmedetomidine**

a) **Dexmedetomidine is a selective alpha-2 adrenoreceptor agonist with an alpha-2 to alpha-1 ratio of 1600:1.**

b) **The anxiolytic and hypnotic responses are associated with its effects in the locus ceruleus and are mediated by the alpha-2_A receptor.**[7]

c) **Clinical use**
i) Sedation
(1) Dexmedetomidine is approved for use as a continuous infusion (0.2-1 μg/kg/h) for sedation of mechanically ventilated patients in the ICU.
(2) Dexmedetomidine is not used for induction of general anesthesia.



Dexmedetomidine is not an induction agent. It is a sedative that acts as an alpha-2 agonist.

(3) It is used "off-label" for sedation during diagnostic and therapeutic procedures such as fiberoptic intubations and as an IV adjuvant during regional anesthesia.

d) **Effects on major organ systems**

i) Central nervous system

(1) Dexmedetomidine has sedative, anxiolytic, and analgesic effects.

(a) The level of sedation is comparable to that of midazolam.[8]

(2) The analgesic effects of dexmedetomidine reduce postsurgical opioid requirements.

ii) Cardiovascular system

(1) Increases the potential for hypotension and bradycardia because of its high alpha-2 adrenoreceptor selectivity.[3]

(2) Dexmedetomidine blunts the hemodynamic effects of direct laryngoscopy and improves hemodynamic stability when given as an anesthetic *adjunct* during neurosurgical procedures.



Dexmedetomidine can cause hypotension and bradycardia.

iii) Respiratory system

(1) Produces less ventilatory depression compared to other sedative-hypnotic and opioid anesthetics.[9]

(2) Bolus administration may reduce minute ventilation.

e) **Pharmacokinetics**

i) Short distribution half-life (6 minutes)

ii) Short elimination half-life (2 hours)[7]

iii) Metabolized in the liver and eliminated by the kidneys

(1) Clearance is decreased with hepatic failure.[7]

Induction Doses and Physiologic Effects of IV Induction Agents

Drug	Induction Dose (mg/kg)	Physiologic Effects	Other Effects
Propofol	1.5-2.5	↓ CBF ↓ $CMRO_2$ ↓ ICP ↓ SVR	• Dose-dependent respiratory depression • Antiemetic properties • May cause pain upon injection
Thiopental	3-5	↓ CBF ↓ $CMRO_2$ ↓ ICP ↓ SVR	• Dose-dependent respiratory depression • May be used for EEG burst suppression • Concomitant administration with acidic medications may cause precipitation
Etomidate	0.2-0.3	↓ CBF ↓ $CMRO_2$ ↓ ICP ↔ SVR ↔ HR	• Myoclonus seen on injection • Adrenocortical suppression • High incidence of PONV
Ketamine	1-2	↑ CBF ↑ $CMRO_2$ ↑ ICP ↑ HR	• Preserves airway reflexes • Dissociative anesthetic • Direct bronchodilator • May increase oral secretions
Dexmedetomidine	Not used for induction Sedation dose: 1 µg/kg given over 10 min; followed by an infusion of 0.2-1 µg/kg/h	↓ HR ↓ BP ↔ MV ↔ RR	• Minimal ventilatory depression • Anxiolytic, sedative, and analgesic effects • Decreases opioid requirements

↑, increase; ↓, decrease; ↔, no change/minimal change; CBF, cerebral blood flow; $CMRO_2$, cerebral metabolic rate for oxygen; ICP, intracranial pressure; SVR, systemic vascular resistance; HR, heart rate; MV, minute ventilation; RR, respiratory rate.

CHAPTER SUMMARY FOR IV INDUCTION AGENTS

Common Uses	Induction of anesthesia and sedation
Mechanism of Action	Primarily through GABA (propofol, thiopental, etomidate), NMDA (ketamine), and alpha-2 (dexmedetomidine) receptors
Considerations	Propofol has fast-onset, rapid recovery pharmacokinetics favorable for ambulatory surgery. Etomidate has minimal cardiovascular effects and may be good for patients with cardiopulmonary compromise. Ketamine has bronchodilator properties that may be good in the setting of acute bronchospasm. Dexmedetomidine is used "off-label" for sedation during diagnostic and therapeutic procedures (eg, fiberoptic intubation) and as an adjuvant during regional anesthesia.
Adverse Effects	Thiopental is highly basic and can cause tissue necrosis if administered intra-arterial or subcutaneously. Etomidate can cause adrenocortical suppression and PONV. Ketamine can have psychomimetic effects and increases $CMRO_2$, CBF, and ICP. Dexmedetomidine can cause hypotension and bradycardia.

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31 Inhaled Anesthetic Agents

Olivia Romano • Shamsuddin Akhtar

Inhaled anesthetics are the most common agents used to provide general anesthesia and include nitrous oxide (N_2O) and the halogenated volatile anesthetics (isoflurane, desflurane, and sevoflurane). Inhalational anesthetics produce unconsciousness, amnesia, and skeletal muscle relaxation via their actions in the brain and spinal cord.

1) **Mechanism of action**
   a) Inhaled anesthetics reduce neuronal transmission via inhibition of excitatory neuronal activity and augmentation of inhibitory neuronal activity.
   b) Inhalational anesthetics disrupt the connectivity of different parts of the brain, leading to an anesthetic state.
   c) Anesthetic state is achieved when inhaled anesthetics achieve a certain partial pressure in the CNS.
   d) The partial pressure at which anesthetic state is achieved varies with each inhaled anesthetic. This is similar to the effect-site concentration achieved in CNS by intravenous anesthetics.[1]
   e) Volatile anesthetics enhance the action of $GABA_A$ and glycine receptors, block the action of glutamate at NMDA receptors, and inhibit sodium and calcium channels. Nitrous oxide blocks NMDA receptors and enhance the background activity of potassium channels[1]:
      i) Effects in the brain cause hypnosis and amnesia.
      ii) Effects in the spinal cord cause skeletal muscle relaxation and inhibition of movement.[2]



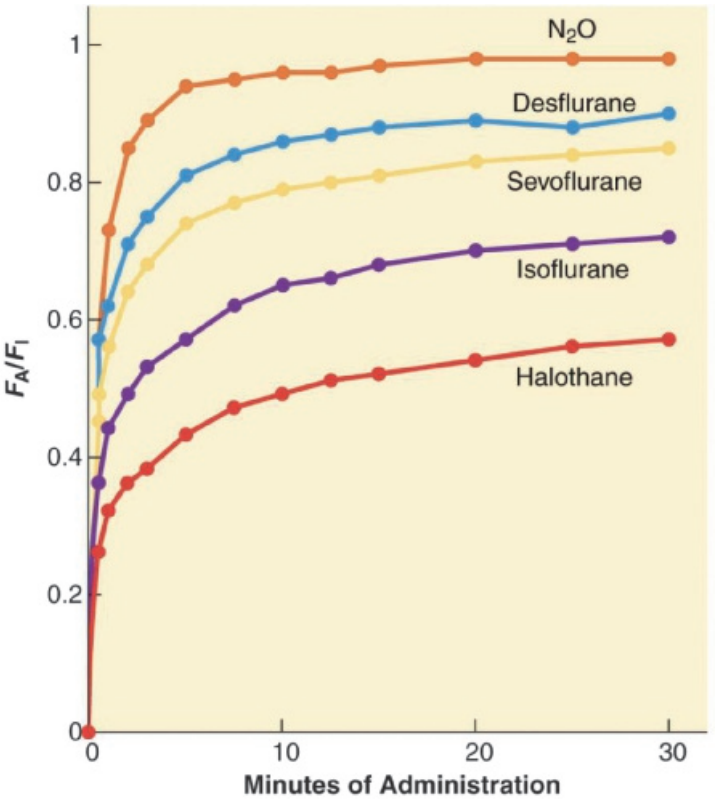
Anesthetic state is achieved when inhaled anesthetics achieve a certain partial pressure in the CNS.

2) **Pharmacokinetics**
   a) The goal of inhalation anesthesia is to achieve optimal and constant partial pressure of anesthetic in the brain (P_{br}).
      i) In order to achieve this goal, the anesthetic must go from the anesthesia machine to the tissue (brain).
      ii) This creates a gradient that eventually leads to equilibrium between the alveolar partial pressure of the anesthetic (P_A) and the partial pressure of the anesthetic in the arterial blood (P_a).
      iii) Alveolar concentration (F_A)
         (1) Measure of anesthetic concentration in the alveoli (similar to end-tidal concentration of anesthetic, F_{ET}).
         (2) F_I is the concentration of inspired anesthetic (from the anesthesia circuit).
         (3) The rate of rise of F_A/F_I determines the speed of inhalation induction (**Fig. 31-1**).
            (a) As the rate of rise in F_A/F_I increases, the speed of inhalation induction increases.
      iv) Factors affecting delivery/input
         The same factors that determine the delivery of oxygen to the tissues determine the delivery of anesthetic to the tissues (**Fig. 31-2**).



The rate of rise of FA/FI determines the speed of inhalation induction.

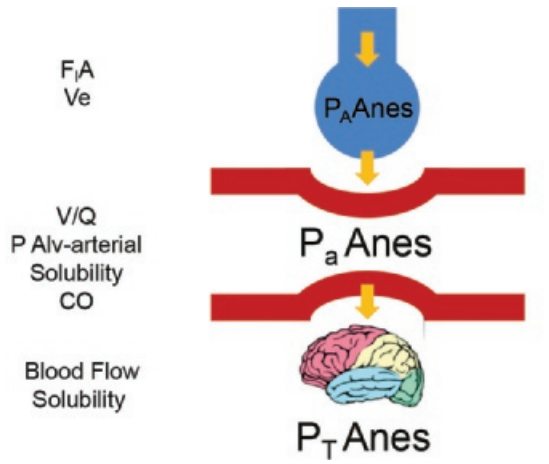
FIGURE 31-1 The rise in alveolar concentration (F_A) of anesthetic toward the inspired concentration (F_I) is most rapid with the least soluble anesthetics, N_2O (nitrous oxide), desflurane, and sevoflurane.



It rises most slowly with the more soluble anesthetics, such as halothane. All data are from human studies. Reproduced with permission from Ebert TJ, Naze SA. Inhaled anesthetics. In: *Clinical Anesthesia*. 8th ed. Philadelphia, PA: Lippincott, Williams and Wilkins; 2017:459-485.

(1) Concentration of inspired gas (F_I)
   (a) Concentration of gas that is delivered to the lungs
      (i) Controlled with the anesthetic vaporizer
(2) Fresh gas flow (FGF) rates
   (a) Increasing the FGF rates increases the total amount of anesthetic agent delivered to the patient.
(3) Breathing circuit
   (a) The volume of the circuit can affect the amount of gas delivered to the patient.[2]
   (b) Material of the circuit can cause absorption, decreasing the alveolar concentration (F_A) of the anesthetic.
(4) Concentration effect
   (a) Increasing the inspired gas concentration (F_I) will cause a more rapid rate of rise of F_A/F_I.

FIGURE 31-2 Simple model of anesthetic pharmacokinetics based on oxygen delivery model.



FIA, inspired anesthetic concentration; Ve, alveolar minute ventilation; V/Q, ventilation/perfusion matching; P alv-blood, difference in partial pressure of inhaled anesthetic between alveolus and blood; CO, cardiac output: P_AAnesth, partial pressure of anesthetic gas in alveolus; P_aAnesth, partial pressure of anesthetic gas in alveolar blood; P_TAnesth, partial pressure of anesthetic gas in brain tissue.

(b) Setting the vaporizer to a higher concentration than the desired F_A is also known as *overpressurization*.

(5) Second gas effect

(a) A high concentration of one gas (usually N_2O) will accelerate the increase in the F_A of a coadministered gas (volatile anesthetic or O_2).

(b) This effect is more profound with the more soluble volatile anesthetics.

b) **Factors affecting uptake**

i) Solubility

(1) The solubility of an anesthetic agent can be depicted through the blood:gas coefficient.

(a) The blood:gas coefficient is the ratio of concentration of a dissolved agent in the blood vs the alveoli (at equilibrium) (**Table 31-1**). It is a reflection of drug solubility.

(b) A high blood:gas coefficient (eg, halothane) indicates that a large amount of anesthetic agent will dissolve in the blood. For example, blood:gas coefficient for desflurane is 0.42 indicating that at equilibrium (37°C), a certain volume of blood will hold 42% as much desflurane as the same volume of air.

(i) The higher the blood:gas coefficient, the greater the solubility, and the slower the rise of F_A/F_I. The end result is a slower inhalation induction.

(c) A low blood:gas coefficient (eg, N_2O) indicates low solubility in the blood. This results in a faster rise in F_A/F_I and, thus, a faster inhalation induction.



The greater the solubility of the volatile anesthetic administered, the slower the induction of anesthesia, as it takes longer to develop the effective partial pressure in the blood and CNS.

TABLE 31-1 Physiochemical Properties of Volatile Anesthetics

	Halothane	Isoflurane	Sevoflurane	Desflurane	Nitrous Oxide
MAC[a] (%)	0.75	1.17	1.8	6.6	104
Vapor pressure at 20°C (mm Hg)	243	238	157	669	38 770
Blood:gas partition coefficient	2.50	1.46	0.65	0.42	0.46
Brain:blood solubility	1.9	1.6	1.7	1.3	1.1
Fat:blood solubility	51.1	44.9	47.5	27.2	2.3
Muscle:blood solubility	3.4	2.9	3.1	2.0	1.2

[a]MAC value is measured in O_2, 30-60 years, at 37°C P_B 760.
Adapted with permission from Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2017;459-485.

ii) Alveolar ventilation
  (1) Hyperventilation—increases rate of rise of F_A/F_I
  (2) Hypoventilation—decreases rate of rise of F_A/F_I

iii) Cardiac output (CO)
  (1) CO determines the pulmonary blood flow available for gas exchange in the alveoli.
  (2) If CO is high, there is rapid uptake of anesthetic agent from the alveoli, slowing the rise of F_A/F_I and slowing induction.
  (3) If CO is low, uptake from the alveoli is low, the rise of F_A/F_I is more rapid, and inhalation induction is faster.

iv) Alveolar-venous partial pressure difference (A-VD)
  (1) A-VD gradient is determined by tissue uptake. Like the blood:gas coefficient, the tissue:blood coefficient determines solubility of anesthetic agent in the tissue (eg, muscle).
  (2) Highly perfused tissues (brain, heart, liver, and kidneys) account for 75% of the CO and account for significant uptake of anesthetic from the blood in the earliest moments of induction. Once these tissues become saturated with anesthetic, venous anesthetic partial pressure rises, creating less of a gradient for uptake from the alveoli to the blood.[3]

v) Intracardiac shunt
  (1) R→L shunt—slows induction (part of the CO bypasses the lung).
    (a) Clinical significance seen with less soluble agents (eg, desflurane, N_2O).
  (2) L→R shunt—speeds induction. However, decreased anesthetic delivery to peripheral tissues negates increased uptake.[3]



The primary mode of elimination of inhaled anesthetics is exhalation. Minimal metabolism and excretion occurs with modern anesthetics.

c) **Elimination of inhaled anesthetics**
  i) Primary mode of elimination is through exhalation.
    (1) Minimal metabolism and excretion occur with modern anesthetics.
  ii) Factors that affect elimination and recovery[3]
    (1) Factors discussed above that affect uptake and delivery (AV, CO, solubility) also play a similar role in the elimination of an anesthetic.
    (2) Tissue concentration and solubility:
      (a) Tissues act as "reservoirs" for the anesthetic agent that maintains F_a as F_I is decreased to zero.

(b) Highly soluble agents (eg, halothane) will have a longer recovery time for a longer anesthetic.
(c) Poorly soluble agents (eg, desflurane) may have a shorter recovery time despite a prolonged anesthetic.
(3) Lack of a concentration effect
(a) F_I cannot be less than zero; thus, one cannot "speed" elimination such as with induction via the concentration effect.[3]

3) **Minimum alveolar concentration (MAC) (Table 31-1)**
a) **MAC is defined as the inhalation anesthetic concentration at which 50% of the population will not move after a painful or noxious stimulus (eg, surgical incision)**.
i) Approximately 1.3 MAC will prevent movement in 95% of surgical patients.
ii) End-tidal concentrations of inhaled anesthetics are a surrogate estimate of effect-site concentration and are only reasonable at steady-state conditions.[4]
b) **MAC values are an index of anesthetic potency**.
i) For example, N_2O has a low potency at MAC of 104%. Halothane has a MAC value of 0.75% and is a highly potent anesthetic agent.
c) **MAC values are additive**.
i) For example, 0.7 MAC of N_2O used with 0.5 MAC of halothane will give an effect of 1.2 MAC anesthetic.
d) Other MAC values for clinical use.[5]
i) MAC-awake (0.3-0.5 MAC)—the value necessary to prevent voluntary response to verbal or tactile stimulation
ii) MAC-TI (1.3-1.5 MAC)—prevents response to tracheal intubation
iii) MAC-BAR (1.3-2.0 MAC)—blocks autonomic response (BAR) to surgical stimulation
e) **Factors affecting MAC (Table 31-2)**
i) MAC is age dependent.
(1) MAC is greatest at infancy (month 6-12) and decreases by about 6% with each decade after 40 years of age.

TABLE 31-2 Factors Affecting MAC

Factors That Increase MAC • Increased central neurotransmitter levels (MAOIs, acute methamphetamine use, acute cocaine use, ephedrine, levodopa) • Hyperthermia • Chronic alcohol use • Hypernatremia
Factors That Decrease MAC • Anemia • Increasing age • Metabolic acidosis • Severe hypoxia • Induced hypotension (MAP < 50 mm Hg) • Decreased central neurotransmitter levels • Alpha-2 agonists • Hypothermia • Hyponatremia • Lithium • Pregnancy • Acute alcohol ingestion • IV anesthetic agents (opioids, ketamine, barbiturates, benzodiazepines) • Lidocaine • Verapamil

Adapted from Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2017:459-485.

TABLE 31-3 Cardiovascular Response to Inhalational Anesthetics

	MAP	Contractility	SVR	CO	HR
Nitrous oxide	–/↑	↓	–/↑	–	↑/–
Halothane	↓	↓	–	↓	–/↓
Isoflurane	↓	↓	↓	–	↑
Sevoflurane	↓	↓	↓	–	–
Desflurane	↓	↓	↓	–	↑

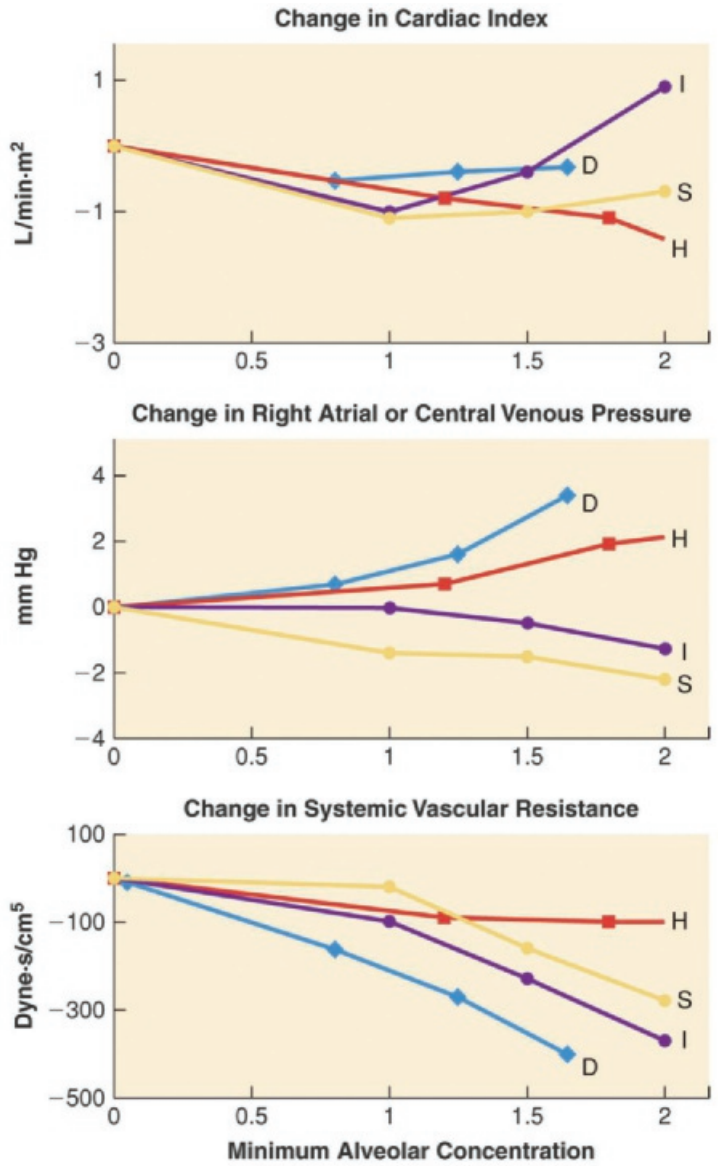
4) **Pharmacodynamics**
   a) **Neurologic effects**
      i) Cerebral blood flow (CBF)
         (1) All inhaled anesthetics increase CBF by direct vasodilation of the cerebral blood vessels.
         (2) Increases in CBF can increase intracranial pressure; however, this can be attenuated by hyperventilation.
      ii) Cerebral metabolic rate of oxygen ($CMRO_2$)
         (1) The volatile anesthetic agents decrease $CMRO_2$.
         (2) Nitrous oxide increases $CMRO_2$.


All inhaled anesthetic agents increase CBF by direct vasodilation.

   b) **Cardiovascular effects (Table 31-3; Figs. 31-3 and 31-4)**
      i) Mean arterial pressure (MAP)
         (1) All inhaled anesthetics cause a decrease in MAP.
         (2) Halothane decreases myocardial contractility, while isoflurane, desflurane, and sevoflurane decrease systemic vascular resistance (SVR).
      ii) Myocardial contractility
         (1) Volatile anesthetics universally produce concentration-dependent myocardial depression especially halothane.
      iii) Systemic vascular resistance
         (1) Isoflurane, desflurane, and sevoflurane have a dose-dependent decrease in SVR. Halothane has minimal effects on SVR.
      iv) Cardiac output
         (1) Halothane decreases CO; isoflurane, desflurane, and sevoflurane have minimal effect on CO as they decrease afterload.
      v) Heart rate (HR)
         (1) Rapid increases in desflurane (and to a lesser extent isoflurane) can cause an increase in HR (increased sympathetic stimulation).
         (2) N_2O is a direct myocardial depressant; however, it also causes sympathetic stimulation, which offsets decreases in MAP.
         (3) Sevoflurane has minimal effect on heart rate.
   c) **Ventilatory effects of inhaled anesthetics (Fig. 31-5)**
      i) All inhaled anesthetics cause a dose-dependent decrease in tidal volume (TV).
      ii) All inhaled anesthetics cause a dose-dependent increase in respiratory rate (RR).
      iii) All inhaled anesthetics cause a dose-dependent decrease in response to hypoxia and hypercarbia.
   d) **Pulmonary vascular resistance (PVR)**
      i) N_2O increases PVR.
      ii) All volatile anesthetics inhibit hypoxic pulmonary vasoconstriction.

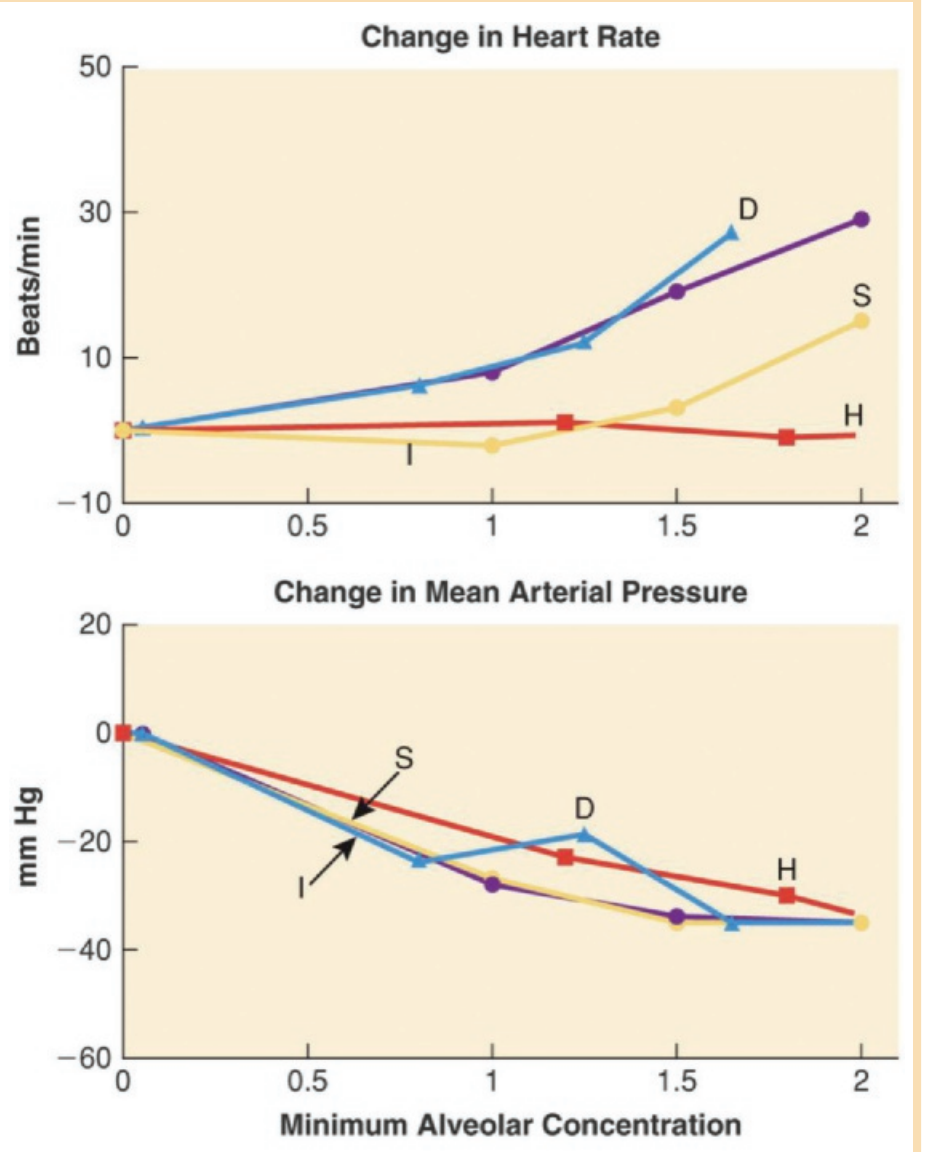

N_2O increases PVR and it should be avoided in patients with pulmonary hypertension.

FIGURE 31-3 Heart rate and blood pressure changes (from awake baseline) in volunteers receiving general anesthesia with isoflurane (I), desflurane (D), or sevoflurane (S).



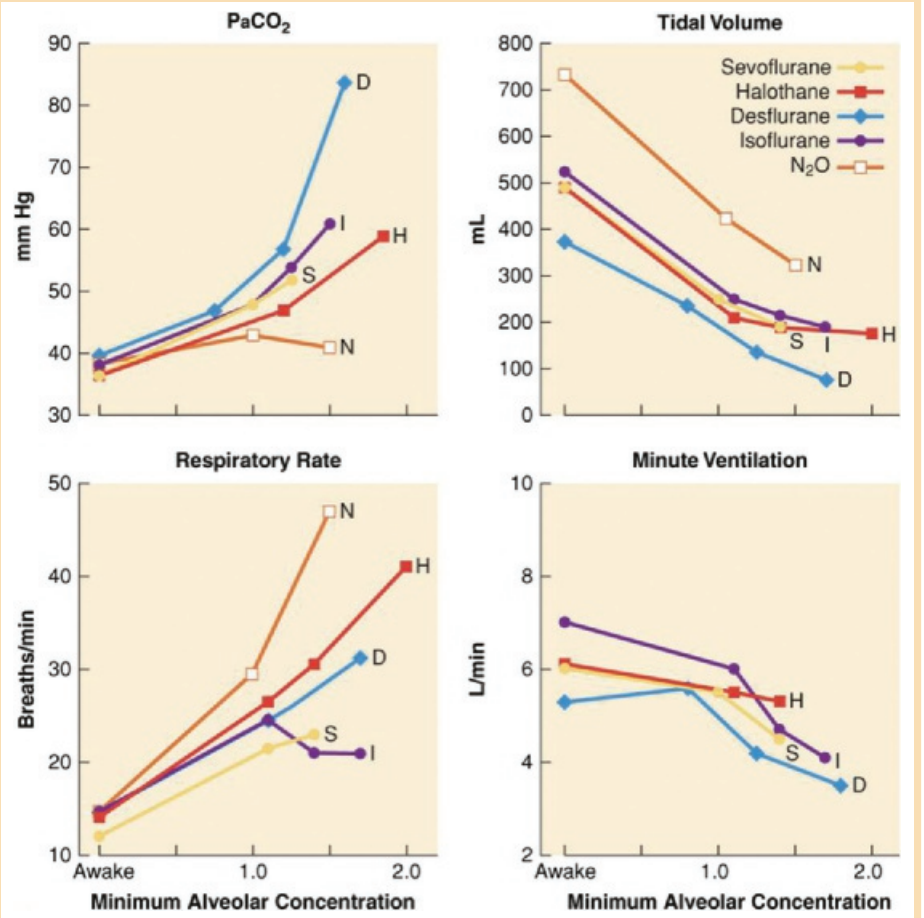
Sevoflurane produced little or no change in heart rate below 1.5 minimum alveolar concentration. All anesthetics caused similar decreases in blood pressure. Halothane (H) is presented for historical perspective. Reprinted with permission from Ebert TJ, Naze SA. Inhaled anesthetics. In: *Clinical Anesthesia*. 8th ed. Philadelphia, PA: Lippincott, Williams and Wilkins; 2017:459-485.

FIGURE 31-4 Cardiac index, central venous pressure (or right atrial pressure), and systemic vascular resistance changes (from awake baseline) in volunteers receiving general anesthesia with isoflurane (I), desflurane (D), or sevoflurane (S).



Increases in central venous pressure from desflurane might be due to venoconstriction. Halothane (H) is presented for historical perspective. Reprinted with permission from Ebert TJ, Naze SA. Inhaled anesthetics. In: *Clinical Anesthesia*. 8th ed. Philadelphia, PA: Lippincott, Williams and Wilkins; 2017:459-485.

FIGURE 31-5 Comparison of mean changes in resting $PaCO_2$, tidal volume, respiratory rate, and minute ventilation in patients anesthetized with isoflurane, sevoflurane, desflurane, or N_2O.



Anesthetic-induced tachypnea compensates in part for the ventilatory depression caused by all volatile anesthetics (decrease in minute ventilation and tidal volume and concomitant increase in $PaCO_2$). Desflurane results in the greatest increase in $PaCO_2$ with corresponding reductions in tidal volume and minute ventilation. Isoflurane, like all other inhaled agents, increases respiratory rate, but does not result in dose-dependent tachypnea. Reprinted with permission from Ebert TJ, Naze SA. Inhaled anesthetics. In: *Clinical Anesthesia*. 8th ed. Philadelphia, PA: Lippincott, Williams and Wilkins; 2017:459-485.

e) **Hepatic effects**
   i) All volatile anesthetics decrease hepatic blood flow.
      (1) Halothane decreases total hepatic blood flow more than other volatile anesthetics.
   ii) Halothane hepatitis has been described (see below).

f) **Renal effects**
   i) All volatile anesthetics and N_2O decrease renal blood flow, glomerular filtration rate, and urinary output.
   ii) Elevated fluoride levels (because of metabolism) can be seen with volatile anesthetics, particularly with sevoflurane.

iii) Fluoride-induced nephrotoxicity has been described in a previously available agent, methoxyflurane, but has not been shown with sevoflurane.

READ MORE
Malignant hyperthermia, Chapter 67 and Chapter 150

g) **Neuromuscular effects**
   i) All volatile anesthetics potentiate neuromuscular blockade by direct inhibition of nicotinic acetylcholine receptors and enhancing the function of glycine receptors in the spinal cord.
   ii) The volatile anesthetics will cause skeletal muscle relaxation.
      (1) By contrast, N_2O does not provide significant muscle relaxation.
   iii) The halogenated anesthetics are triggers for malignant hyperthermia (MH).
      (1) N_2O is **not** a trigger and can be used safely in an MH-nontriggering anesthetic.
   iv) Uterine smooth muscle is relaxed by the halogenated agents but not N_2O.



Volatile anesthetics are triggers for MH, but nitrous oxide (N_2O) is safe in MH.

5) **Inhaled agents in clinical use**
   a) **Halothane**
      i) It is a halogenated alkane.
      ii) It is the most potent and soluble of the volatile anesthetics.
      iii) It is nonpungent, making it useful for inhalational induction of anesthesia.
      iv) It is not widely used in the United States due to adverse effects.
         (1) Sensitizes the myocardium to epinephrine and can cause cardiac dysrhythmias.
         (2) Halothane hepatitis is classified as type I and type II. Type I occurs commonly and presents as asymptomatic elevation of serum transaminases. Type II is immune mediated, is less common (1:35 000 patients), and leads to fulminant hepatic failure with high mortality.[1]
   b) **Isoflurane**
      i) Intermediate blood solubility.
      ii) It is a strong coronary vasodilator and has been theorized to cause coronary steal. However, it has been safely used in patients with coronary artery disease, if the adequate blood pressure is maintained.
   c) **Sevoflurane**
      i) Pleasant odor and low solubility
      ii) Used commonly for inhalational induction in the pediatric population
      iii) Potent bronchodilator
      iv) Degraded by particular carbon dioxide absorbents (eg, Baralyme) into a vinyl ether called compound A
         (1) Compound A has shown to be nephrotoxic in rats; studies in humans have had conflicting results.
         (2) The FDA recommends administering sevoflurane with FGF rates of 1 L/min if exposure is 1 hour and 2 L/min if exposure is >1 hour.[6]
   d) **Desflurane**
      i) Ether derivative with low solubility
      ii) Rapid onset/offset of action
      iii) Pungent odor, thus can cause breath holding, bronchospasm, and laryngospasm
      iv) Causes carbon monoxide formation in desiccated carbon dioxide absorbents
   e) **Nitrous oxide**
      i) Inorganic gas with low solubility, low potency, and analgesic properties.
      ii) With a MAC of 104%, it cannot be used as the sole anesthetic for general anesthesia.

(1) Used as an adjuvant with IV agents or other volatile anesthetics
(2) May be used for analgesia for dental procedures or labor

iii) Because N_2O is 30 times more soluble than nitrogen, it rapidly diffuses into closed air-filled cavities and causes them to expand.
(1) The use of N_2O is contraindicated in patients with pneumothorax, air embolism, middle ear surgery, and intestinal obstruction.



N_2O causes expansion of closed air-filled spaces and is contraindicated in patients with pneumothorax, air embolism, middle ear surgery, and intestinal obstruction.

iv) Inhibits DNA synthesis by inactivating vitamin B_{12}–dependent enzymes
v) Increases pulmonary vascular resistance
vi) Diffusion hypoxia
(1) Due to the low solubility of N_2O, at the elimination of an anesthetic, N_2O can diffuse out of the blood into the alveoli, displacing O_2.
(2) During the termination of an anesthetic with N_2O, 100% O_2 should be used to prevent diffusion hypoxia.

CHAPTER SUMMARY FOR INHALED ANESTHETICS

Definition	Inhaled anesthetics are gases that provide amnesia, analgesia, and skeletal muscle relaxation. N_2O and halogenated anesthetic compounds are most commonly used in anesthesia.
Mechanism of Action	Via inhibition of excitatory neurotransmitters and enhancement of inhibitory neurotransmitters in the brain and spinal cord.
Pharmacokinetics and Pharmacodynamics	Higher solubility, cardiac output, and alveolar ventilation affect the speed of onset of action. All inhaled anesthetics increase CBF by direct vasodilation and decrease MAP.
Side Effects	Halogenated anesthetics can trigger MH. All inhaled anesthetics cause a dose-dependent decrease in TV and increase in RR. Volatile anesthetics potentiate neuromuscular blockade.

CO, cardiac output; CBF, cerebral blood flow; MAP, mean arterial pressure; MH, malignant hyperthermia; TV, tidal volume; RR, respiratory rate.

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32 Adrenergic Agents§

Samuel D. Gilliland

The sympathetic nervous system (SNS) is a part of the autonomic nervous system (ANS) and is responsible for the "fight or flight" mechanism. Perioperatively, pharmacologic agonism of the SNS counteracts pathophysiologic processes, which depress myocardial contractility and peripheral vascular resistance, whereas antagonism can attenuate the response to surgical stress. Adrenergic agents may also be used to treat shock and anaphylaxis, prolong local anesthetic duration, and alleviate bronchoconstriction and treat a variety of other clinical conditions. Familiarity with this class of medications and their mechanisms is vital to providing safe perioperative care.

1) **Sympathetic nervous system**
   a) **Neurotransmission**
      i) Preganglionic fibers travel from the thoracolumbar spine and synapse in sympathetic chain, peripheral ganglia, or adrenal medulla.
         (1) Acetylcholine (ACh) is the neurotransmitter.[1]
      ii) Postganglionic fibers synapse directly at target organs.
         (1) Norepinephrine (NE) is the neurotransmitter in most cases.
         (2) An exception is transmission by ACh at sweat glands.
         (3) In the adrenal glands, preganglionic fibers interact directly with chromaffin cells, which release NE and epinephrine (EPI).[1]
   b) **Adrenergic receptors**
      i) G-protein–coupled receptors selective for NE and EPI.
      ii) Subtypes have different functions and locations[1,2] (**Table 32-1**).



Acetylcholine (ACh) is the neurotransmitter for preganglionic fibers, while in most cases norepinephrine (NE) is the neurotransmitter for postganglionic fibers.

2) **Mechanism of action**
   a) **Adrenergic agonists/sympathomimetics**—use one or both mechanisms:
      i) Direct-acting—agents that directly bind a receptor, activating it
      ii) Indirect-acting—agents that cause the release of endogenous NE
         (1) Magnitude of response depends on patient's NE stores and adrenergic receptor density.
         (2) Tachyphylaxis to indirect-acting sympathomimetics is common.[3]
   b) **Adrenergic antagonists/sympatholytics**
      i) Competitively bind receptors, preventing activation by endogenous NE or EPI[4] (**Tables 32-2** and **32-3**)



Indirect sympathomimetics (eg, ephedrine) may have declining effectiveness with repetitive dosing (tachyphylaxis)

3) **Structure/activity relationships**
   a) Direct agonists are structural analogs of NE and/or EPI.
   b) Indirect agonists do not require structural similarity to NE or EPI, as they do not bind adrenergic receptors.[3]
   c) Antagonists are structurally similar to NE and EPI to allow receptor binding but are sufficiently different to prevent receptor activation.
   d) Structural variation (agonist or antagonist) imparts receptor selectivity.

TABLE 32-1 Adrenergic Receptor Types

Receptors	Sensitivity	Locations	Functions
α_1	NE > EPI	• Vascular smooth muscle • Heart • GI smooth muscle • GU smooth muscle • Liver	• Vasoconstriction • + Inotropy • Relaxation • Contraction • Glycogenolysis, gluconeogenesis
α_2	NE > EPI	• Central nervous system • Adrenergic nerve endings • Vascular smooth muscle • Platelets • Endocrine pancreas	• Sedation, decreased activity • Inhibits NE release • Vasoconstriction • Aggregation • Inhibits insulin release
β_1	EPI > NE	• Heart • Kidney	• + Chronotropy, + inotropy, increased AV conduction velocity • Increased renin secretion
β_2	EPI > NE	• Smooth muscle • Liver • Skeletal muscle	• Relaxation • Glycogenolysis, gluconeogenesis • Glycogenolysis, lactate release, K^+ uptake

From: Grecu L. Autonomic nervous system anatomy and physiology. In: Barash PG, Cullen BF, Stoetling RK, Cahalan MK, Stock MC, eds. *Clinical Anesthesia*. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2017:333-360; Gera N, Armstrong EJ, Golan DE. Adrenergic pharmacology. In: Golan DE, Tashjian AH, Armstrong EJ, et al., eds. *Principles of Pharmacology: The Pathophysiologic Basis of Drug Therapy*. Baltimore, MD: Lippincott Williams & Wilkins; 2017:132-146.

4) **Metabolism**
   a) **Adrenergic agonists/sympathomimetics**
      i) Reuptake of NE and EPI at nerve endings extraneuronal tissue and diffusion terminates their effect (**Fig. 32-1**).
      ii) When levels exceed reuptake capacity or reuptake inhibitors are present, metabolism becomes clinically significant.
      iii) NE, EPI, and structural analogs are inactivated by monoamine oxidase (MAO) and/or catechol-*O*-methyltransferase, conjugated with glucuronic acid, and excreted in urine.
      iv) Ephedrine is mainly excreted unchanged in the urine.
   b) **Adrenergic antagonists**
      i) Generally undergo liver metabolism and renal excretion. Oral administration exhibits significant first-pass metabolism.
      ii) A notable exception, esmolol, is metabolized by plasma esterases, which results in a consistent, short duration of action regardless of dose or duration of administration.[4]



MAO inhibitors may cause an exaggerated response to sympathomimetic agents.



Esmolol is a β-blocker with a short duration of action (about 9 minutes) due to its metabolism by plasma esterases.

5) **Adverse effects**—generally dose-dependent and tend to reflect response exceeding clinically desired magnitude.
   a) **Agonists**
      i) Cardiovascular—tachycardia, hypertension, myocardial ischemia, dysrhythmias, pulmonary hypertension, increased intrapulmonary shunting, cardiac failure
      ii) Pulmonary—bronchodilation
      iii) Neurological—confusion, tremor, anxiety, headache, intracranial hemorrhage
      iv) Renal—hypokalemia (β-agonism forces K+ ions intracellularly)
      v) Other—tissue necrosis (with high doses or extravasation)
   b) **Antagonists**
      i) Cardiovascular—bradycardia, hypotension, cardiac failure
      ii) Pulmonary—increased airway resistance, pulmonary edema
      iii) Neurological—sedation, stroke (due to hypoperfusion)

TABLE 32-2 Adrenergic Agonists

Drug	Clinical Receptor Selectivity				CV			Airway	Notes
	α_1	α_2	β_1	β_2	HR	MAP	CO		
Albuterol				++++	+	0/–	0/+	–	Cardiovascular effects minimized by inhalational delivery.
Clonidine		++			–	–	–	0	May be used to prolong regional anesthesia. Useful adjunct in opioid withdrawal. Exhibits rebound hypertension with discontinuation.
Dexmedetomidine		++			±	±	0/–	0	MAP increases and HR decreases with loading dose. MAP decreases and HR decreases after longer infusion. Useful for sedation
Ephedrine	+	+/0	++	+	+	+	+	–	Indirect activity exceeds direct agonism. Tachyphylaxis is common.
Isoproterenol			+++	+++	+	0/–	+	–	Useful in third-degree heart block as bridge to pacemaker implantation.
Dobutamine			+++	+	+/0	0/+	+	0	Pulmonary vasodilation improves right heart function in *cor pulmonale*.
Dopamine	++	+/0	+++	+	+	+	+	0	Receptor activity varies from patient to patient. Nausea/vomiting and tachyarrhythmias are common.
Norepinephrine	+++	+++	++		–	+	–	0	Extravasation can cause tissue necrosis.
Phenylephrine	+++				–	+	–/0	0	Can be used to control SVT via reflexive bradycardia. Decongestant and vasoconstrictive effects facilitate nasal intubation.
Terbutaline				++	+	0/–	0/+	–	Administered orally, subcutaneously, or by inhalation for asthma and preterm labor.

From: Dyck JB, Maze M, Haack C, Vuorilehto L, Shafer SL. The pharmacokinetics and hemodynamic effects of intravenous and intramuscular dexmedetomidine hydrochloride in adult human volunteers. *Anesthesiology*. 1993;78:813-820; Westfall TC, Westfall DP. Adrenergic agonists and antagonists. In: Brunton LL, Lazo JS, Parker KL, eds. *Goodman & Gilman's The Pharmacological Basis of Therapeutics*. 12th ed. New York, NY: McGraw-Hill Medical Publishing Division; 2006:277-334. Ref.5,6

6) **Clinical uses**
   a) **Agonists**
      i) Increase cardiac inotropy and chronotropy
      ii) Vasopressor
      iii) Treatment of bronchospasm
      iv) Treatment of severe allergic reactions
      v) Prolong local anesthetic duration of action (peripheral and spinal)
      vi) Sedation and analgesia

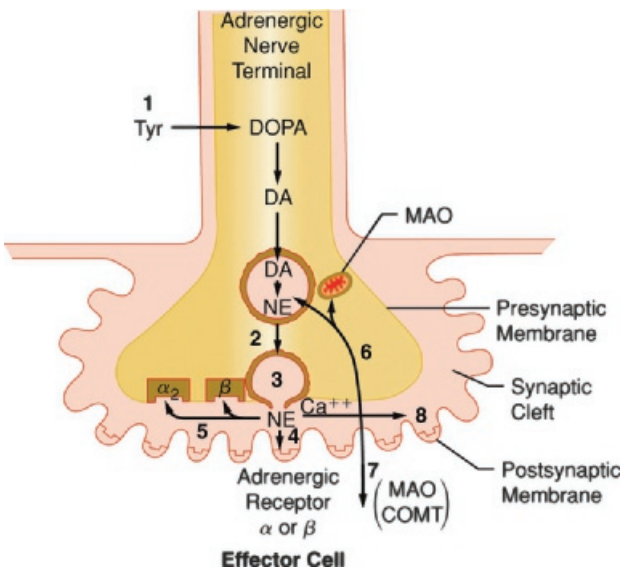
READ MORE
Appendix of drug information, adrenergic agonists, and antagonists, Chapters 220 and 221

TABLE 32-3 Adrenergic Antagonists

Drug	α_1	α_2	β_1	β_2	Elimination Half-Life (h)	Notes
Esmolol			–	–	0.15	Metabolized by plasma esterases.
Labetalol	–		–	–	5-8	α:β potency is 1:7 with IV administration.
Metoprolol			–		3-4	β_1-selectivity is abolished by large doses.
Phenoxybenzamine	–	–			24	Used in management of pheochromocytoma. Orthostatic hypotension is common.
Phentolamine	–	–			0.3	Used in management of pheochromocytoma. May cause cramping or diarrhea.
Propranolol			–	–	2-3	Decreases hepatic clearance of amide local anesthetics.

–, blocks receptor.
From: Grecu L. Autonomic nervous system anatomy and physiology. In: Barash PG, Cullen BF, Stoetling RK, Cahalan MK, Stock MC, eds. *Clinical Anesthesia*. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2017:333-360; Miller S. Sympatholytics. In: *Pharmacology & Physiology in Anesthetic Practice*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2015:474-500.

FIGURE 32-1 Schematic of the Synthesis and Disposition of NE in Adrenergic Neurotransmission (*1*) Synthesis and storage in neuronal vesicles. (*2*) Action potential permits calcium entry with (*3*) exocytosis of NE into synaptic gap. (*4*) Released NE reacts with receptors on effector cell. NE (*5*) may react with presynaptic α2-receptor to inhibit further NE release or with presynaptic β-receptor to enhance reuptake of NE (*6*) (uptake 1). Extraneuronal uptake (uptake 2) absorbs NE into effector cell (*7*) with overflow occurring systemically (*8*). MAO, monoamine oxidase; COMT, catechol-*O*-methyltransferase; Tyr, tyrosine; DOPA, dihydroxyphenylalanine; NE, norepinephrine. Reprinted with permission from Grecu L. Autonomic nervous system anatomy and physiology. In: Barash PG, Cullen BF, Stoetling RK, Cahalan MK, Stock MC, eds. *Clinical Anesthesia*. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2017:333-360.



b) **Antagonists**
   i) Antihypertensive agents
   ii) Treatment of myocardial ischemia
   iii) Management of chronic congestive heart failure
   iv) Prevention/treatment of excessive sympathetic activity
   v) Treatment of glaucoma
   vi) Treatment of benign prostatic hypertrophy
   vii) Treatment of migraine



Routine use of perioperative β-blockade is recommended in patients already taking β-blockers.

7) **Perioperative β-blockade**
   a) Early studies showed a decrease in myocardial ischemia when patients were given β-blockers in the perioperative period, which led to guidelines encouraging this practice.
   b) Subsequent randomized controlled trials, including the POISE trial, have confirmed a reduction in myocardial ischemia but at the expense of increased risk of stroke and a trend toward worsened all-cause mortality.
      i) Thus, current guidelines do not support prophylactic use of β-blockade on the day of surgery for patients not already taking β-blockers.
   c) **ACC/AHA Guidelines for Perioperative Evaluation were revised in 2014.**[7–10]
      i) Current recommendations are to continue β-blockade in perioperative period for patients already on therapy.[10]
         (1) β-Blockers should not be discontinued in the perioperative period due to increased risk of mortality.
      ii) Routine, prophylactic use of high-dose β-blockers in the absence of titration is not recommended and may be harmful to patients not already taking β-blockers.[10]



While reducing the rate of nonfatal MI, routine use of perioperative β-blockade in patients not already taking β-blockers may increase the risk of stroke and mortality and is NOT recommended.

CHAPTER SUMMARY FOR ADRENERGIC AGENTS

Neurotransmission	Preganglionic fibers use Ach. Postganglionic fibers use NE.
Mechanism of Action (Agonists)	Direct agents bind α- or β-receptors → activation. Indirect agents induce endogenous NE release.
Mechanism of Action (Antagonists)	Competitively bind α- or β-receptors preventing activation.
Common Uses	Cardiovascular modulation, bronchospasm management, and anaphylaxis treatment.
Adverse Effects	Dose-dependent response exceeding clinically desired effect.
Perioperative β-Blockade	Continue in patients already on therapy. Otherwise, do not routinely initiate treatment.

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33 Perioperative Corticosteroids

Diane Gordon • Debnath Chatterjee

Chronic corticosteroid therapy is used for a variety of medical conditions including immunosuppression for organ transplant recipients, inflammatory bowel disease, reactive airway disease, and rheumatologic disease. Exogenous corticosteroid administration can result in inhibition of endogenous glucocorticoid production by the adrenal cortex, resulting in acute adrenal insufficiency during physiological stresses such as sepsis, trauma, and surgery. This chapter provides an overview of hypothalamic-pituitary-adrenal axis (HPAA) suppression by chronic steroid therapy and the recommendations for perioperative stress-dose steroid administration in patients taking exogenous corticosteroids. Other indications for perioperative corticosteroid administration such as prevention of nausea/vomiting, reduction of inflammation, and as part of multimodal analgesia are also discussed.

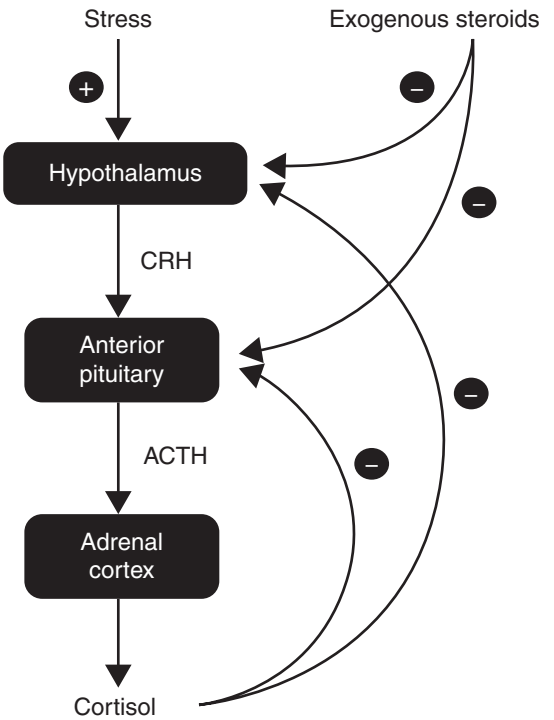
1) **Endogenous glucocorticoids**
   a) Glucocorticoids are cholesterol-derived compounds that are produced in the zona fasciculata of the adrenal cortex. They bind to the intracellular glucocorticoid receptor, which is expressed in almost every cell in the body. Cortisol, the main endogenous glucocorticoid, plays an integral role in maintaining normal cellular homeostasis; carbohydrate, lipid, and protein metabolism; cardiovascular integrity; catecholamine production; normal immune activity; and wound healing.[1,2]
   b) It is estimated that the adrenal cortex secretes 5-10 mg/m^2 of cortisol per day with levels peaking between 4 and 8 AM, depending on age. This is the equivalent of 20-30 mg/d of hydrocortisone or 5-7 mg/d of oral prednisone. During periods of severe stress, cortisol production can increase 5-10-fold, to a maximal level of ~100 mg/m^2/d.[1,2] There is considerable individual variation in the increase in cortisol secretion.
   c) The release of endogenous glucocorticoids is controlled by a negative feedback mechanism known as the hypothalamic-pituitary-adrenal axis (HPAA) (**Fig. 33-1**). The hypothalamus releases corticotropin-releasing hormone (CRH), which stimulates the pituitary gland to initiate the production of adrenocorticotropic hormone (ACTH). ACTH then stimulates the adrenal cortex to produce cortisol, which is self-regulated via a negative feedback loop that decreases CRH and ACTH secretion.[1,2]
2) **Adrenal insufficiency**
   a) Decreased glucocorticoid production results in adrenal insufficiency (AI), which can be classified into primary, secondary, or tertiary types with acute and chronic forms (**Table 33-1**). Tertiary or iatrogenic AI is the most common type, where exogenous corticosteroid administration causes suppression of the HPAA over time, resulting in low CRH and ACTH levels and subsequent adrenal cortex atrophy.[1]
   b) The exogenous corticosteroid used most frequently is prednisone, which has a relatively short half-life in plasma. The dose equivalents of other systemic corticosteroids and their relative glucocorticoid and mineralocorticoid activities are listed in **Table 33-2**.



Rule of 5s: Prednisone is five times more potent than cortisone; dexamethasone is five times more potent than prednisone.[3]

Tertiary or iatrogenic adrenal insufficiency is the most common type and occurs as a result of exogenous corticosteroid administration, which causes suppression of the hypothalamic-pituitary-adrenal axis.

FIGURE 33-1 Functional Anatomy of HPAA Functional anatomy of hypothalamic-pituitary-adrenal axis.



ACTH, adrenocorticotropic hormone; CRH, corticotropin-releasing hormone; –, negative feedback; +, positive feedback. Reprinted with permission from Liu MM, Reidy AB, Saatee S, Collard CD. Perioperative steroid management: approaches based on current evidence. *Anesthesiology*. 2017;127(1):166-172.

TABLE 33-1 Types of Adrenal Insufficiency

Type	Features	Incidence	Etiologies
Primary	Destruction or replacement of adrenal glands with inflammation, tumor, infection, or hemorrhage Decreased glucocorticoid and mineralocorticoid production Increased ACTH production	Rare	Autoimmune (most common in the United States) Infection HIV (in the United States) Tuberculosis (globally) Inflammation Cancer (breast, lung, melanoma) Acute addisonian crisis Infection, shock, hemorrhage
Secondary	Pituitary dysfunction Decreased ACTH production Intact mineralocorticoid function	Rare	Pituitary dysfunction/damage Tumor Postpartum
Tertiary	Hypothalamic or HPAA dysfunction or failure	Most common	Iatrogenic corticosteroid therapy and suppression of HPAA Hypothalamic failure or dysfunction

ACTH, adrenocorticotrophic hormone; HIV, human immunodeficiency virus; HPAA, hypothalamic-pituitary-adrenal axis.
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TABLE 33-2 Comparison of Systemic Corticosteroid Preparations

	Relative Glucocorticoid Activity	Relative Mineralocorticoid Activity	Equivalent Dose (Oral or IV, mg)	Duration of Action (h)
Cortisol[a] (hydrocortisone)	1	1	20	8-12
Cortisone	0.8	0.8	25	8-12
Prednisone	4	0.8	5	12-36
Methylprednisolone	5	0.8	4	12-36
Dexamethasone	30	0	0.75	36-54
Fludrocortisone	0	30	N/A	12-36

[a]The glucocorticoid and mineralocorticoid properties of cortisol are set as 1.
IV, intravenous.
Adapted from Wall RT. Endocrine disease. In: Hines RL, Marschall KE, eds. *Stoelting's Anesthesia and Co-Existing Disease*. 6th ed. Philadelphia, PA: Elsevier Saunders; 2012, with permission from Elsevier.

c) Patients with primary AI require glucocorticoid and mineralocorticoid replacement, whereas patients with secondary and tertiary AI have intact aldosterone synthesis and thus only require glucocorticoid replacement.

d) The clinical presentation of AI (fatigue, altered mental status, abdominal pain, nausea/vomiting, weakness, and hypotension) is masked in a patient under general anesthesia, and so a high index of suspicion is necessary to diagnose perioperative AI. Severe, persistent hypotension that is poorly responsive to fluids and vasopressor therapy is typically used to make the diagnosis.[2]

e) Etomidate, a commonly used intravenous induction agent in critically ill patients, is a potent inhibitor of cortisol synthesis (11 β-hydroxylase), which can cause immediate adrenal dysfunction that persists for 12-48 hours, even after a single dose.[4] Etomidate use in critically ill patients is associated with increased morbidity, prolonged ventilation, and intensive care unit (ICU) and hospital stay.[4]



Acute adrenal insufficiency should be suspected in a patient on chronic steroid therapy with severe, refractory hypotension.

Etomidate should be used judiciously in critically ill patients secondary to the risk of transient adrenal dysfunction.

3) **Perioperative steroid replacement**

a) **Patients with primary and secondary AI require perioperative stress-dose steroids dosed based on surgical stress** (Table 33-3).

b) The conventional wisdom of supplementing all patients receiving exogenous corticosteroids with supraphysiologic "stress-dose" steroids throughout the perioperative period has come under scrutiny secondary to the paucity of class A or B levels of evidence.[1,2,5] Additionally, multiple large doses of steroids can be associated with several adverse effects including hyperglycemia, immunosuppression, altered wound healing, hypertension, volume overload, myopathy, and acute corticosteroid-induced psychosis.[1,2,5]

c) Patients on chronic steroid therapy should be maintained on their usual preoperative steroid dose on the day of surgery.[1,2,5] The risk of HPAA suppression can be evaluated preoperatively (Table 33-4)[2,5] although opinions differ on risk factors for significant HPAA suppression including daily dose of steroid, duration of therapy, and time since cessation of chronic exogenous steroids.[2,6] Patients at low risk of HPAA suppression do not need preoperative testing or stress-dose steroids. Patients at high risk for HPAA suppression also do not need preoperative testing but should receive their usual daily

TABLE 33-3 Recommended Steroid Dosing for Patients Undergoing Surgery

Surgery	Examples	Recommended Steroid Dosing
Superficial	Dental, biopsy	Usual daily dose (UDD)
Minor	Inguinal hernia repair, endoscopy, uterine curettage	UDD + hydrocortisone 50 mg IV before incision followed by 25 mg IV q8h × 24 h and then UDD
Moderate	Lower extremity revascularization, total joint replacement, cholecystectomy, abdominal hysterectomy	
Major	Esophagectomy, total proctocolectomy, major cardiac/vascular, hepaticojejunostomy, delivery, trauma	UDD + hydrocortisone 100 mg IV before incision followed by continuous IV infusion of 200-mg hydrocortisone >24 h Or Hydrocortisone 50 mg IV q8h × 24 h, tapering dose by half per day until back to UDD + continuous IV fluids with 5% dextrose and 0.2%-0.45% NaCl (based on degree of hypoglycemia)

IV, intravenous; UDD, usual daily dose.
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dose of steroid and stress-dose steroids based on the anticipated surgical stress (**Table 33-3**).[2,5] Hydrocortisone is the drug of choice for stress-dose steroid coverage.

d) When the degree of HPAA suppression is intermediate or unknown, additional biochemical evaluation of HPAA suppression can be considered with the caveat that objective determination of patient risk by means of serum ACTH levels and/or cosyntropin testing has not been found to be sensitive or specific.[2] In cases of urgent or emergent surgery, empiric perioperative stress-dose steroid coverage is recommended.

e) Biochemical evaluation of HPAA suppression is outlined in **Table 33-5**. If the 8 AM cortisol is <5 μg/dL, the patient is likely to have HPAA suppression and needs stress-dose steroids. If the 8 AM cortisol is >10 μg/dL, the patient likely does not have significant impairment of HPAA and therefore may be continued on his or her usual daily steroid dose. If the 8 AM cortisol level is 5-10 μg/dL, further evaluation

TABLE 33-4 Risk of Hypothalamic-Pituitary-Adrenal Axis Suppression

HPAA Suppression	Examples	Recommendation
High risk	Patients treated with a glucocorticoid in doses equivalent to at least 20 mg/d of prednisone for >3 weeks or clinical features of Cushing syndrome	Administer perioperative stress-dose steroids with dosing based on surgical stress
Low risk	Patients treated with any dose of glucocorticoids for <3 weeks, morning doses of prednisone ≤5 mg/d every day or ≤10 mg/d every other day	Perioperative stress-dose steroids are not required
Intermediate risk	Any patient on chronic steroid therapy that does not fall into one of the above categories	Consider short-acting ACTH test if timing permits. Otherwise, use clinical judgment to decide if stress-dose steroids are indicated

HPAA, hypothalamic-pituitary-adrenal axis; ACTH, adrenocorticotrophic hormone.
Reprinted with permission from Liu MM, Reidy AB, Saatee S, Collard CD. Perioperative steroid management: approaches based on current evidence. *Anesthesiology*. 2017;127(1):166-172.

TABLE 33-5 Biochemical Assessment of HPAA Suppression[5]

8 AM Cortisol Level	Suggested Course of Action
<5 µg/dL	HPAA is suppressed; patient should receive stress-dose steroids
>10 µg/dL	HPAA is not suppressed; patient does not need stress-dose steroids
5-10 µg/dL	Evaluate further with ACTH stimulation test if possible; otherwise, use clinical judgment to decide if stress-dose steroids are indicated (ACTH stimulation test: serum cortisol is measured 30 minutes after 250 µg ACTH administration)

ACTH, adrenocorticotrophic hormone. Note that all suggested courses of action listed are Grade 2C evidence.
Data from Hamrahian AH, Roman S, Milan S. The management of the surgical patient taking glucocorticoids. In: Nieman LK, Carty SE, eds. *UpToDate*. Waltham, MA: UpToDate; 2018. Available at: http://www.uptodate.com/contents/the-surgical-patient-taking-glucocorticoids. Accessed December 19, 2018.

with the short ACTH stimulation test is recommended. Following administration of 250 µg of ACTH, serum cortisol is measured after 30 minutes. A cortisol level >18 µg/dL predicts an adequate adrenal reserve with no need for stress-dose steroids.[5]

f) A recent review in the rheumatology literature suggests taking a much less aggressive approach, with no supplemental steroids suggested for most groups, and hydrocortisone 50 mg intraoperatively and every 8 hours postoperatively only for patients with primary or secondary AI, or patients on a high-dose steroid regimen having major surgical procedures, or steroid-exposed patients presenting with volume-refractory hypotension.[7]

Patients on chronic steroid therapy should be maintained on their usual preoperative steroid dose on the day of surgery.

Hydrocortisone is the drug of choice for stress-dose steroid coverage.

4) **Other indications for perioperative corticosteroids**

a) **Antiemetic properties**

i) Dexamethasone is the most frequently used corticosteroid for prevention of postoperative nausea and vomiting (PONV).

ii) The mechanism of action in preventing PONV is unclear, possibly involving antagonism of prostaglandin secretion, promotion of endorphin release, or decrease of serotonin in neural and/or gut tissue.[8]

iii) Dexamethasone 4-5 mg IV should be given following induction of anesthesia for best efficacy.[9]

For prevention of PONV, dexamethasone 4-5 mg IV should be given following induction of anesthesia for best efficacy.

b) **Anti-inflammatory properties**

i) Corticosteroids decrease inflammation by incompletely understood mechanisms involving repression of inflammatory gene expression. Evidence for the effective dose for anti-inflammatory effect is lacking.

ii) In neurosurgical procedures, steroids are commonly administered to reduce tumor-associated vasogenic edema.[10]

iii) Steroids are also commonly administered to decrease airway edema after difficult intubation or when a leak is not present around the endotracheal tube. A Cochrane review of prophylactic dexamethasone administration in the ICU prior to extubation to reduce postextubation stridor showed equivocal results.[11] Multiple doses of dexamethasone begun 12-24 hours prior to extubation have been shown to be effective in decreasing postextubation stridor in high-risk adult ICU patients.[12]

c) **Reduction of postoperative pain and opioid consumption**

i) Two different meta-analyses have shown that perioperative administration of a single dose of dexamethasone at doses >0.1 mg/kg[13] decreases postoperative pain and opioid consumption after surgery.[13,14]

d) **Prolongation of the effect of regional anesthesia**

i) Perineural dexamethasone administration when used as an adjunct to brachial plexus blocks has been shown to increase analgesic duration and decrease opioid consumption, without increasing the risk of neural toxicity.[15]

e) Elderly non–steroid-exposed patients having high-risk surgery and exhibiting postoperative hypotension requiring vasopressors were found to have a high incidence of AI. Those treated with stress-dose hydrocortisone had a considerably lower mortality rate.[16]



Consider using stress-dose steroids to treat occult adrenal insufficiency causing volume-refractory hypotension in an elderly patient having high-risk surgery.

5) **Contraindications to perioperative corticosteroids**

a) Evidence regarding the efficacy of high-dose methylprednisolone therapy for acute traumatic spinal cord injury is limited, and its routine use is not recommended secondary to associated complications including infection, respiratory compromise, gastrointestinal hemorrhage, and death.[17] Some authors suggest a 24-hour infusion of high-dose methylprednisolone as a treatment option in adult patients who present within 8 hours of acute spinal cord injury.[18]

b) Routine administration of steroids to treat adults and children with severe traumatic brain injury (TBI) to lower intracranial pressure or improve functional outcomes or mortality is not recommended.[19,20] The use of high-dose methylprednisolone therapy in patients with severe TBI is associated with increased mortality and is contraindicated.[20]

c) In patients with newly diagnosed and yet-unspecified type of leukemia (and some lymphomas), even a single dose of steroids could suppress the oncologic process to the point that the diagnosis and specific type of blood cancer cannot be made resulting in a delayed or wrong diagnosis. In addition, steroid pretreatment puts the patient in a higher-risk category, and they get more intensive chemotherapy. Lastly, a dose of steroids could start or increase tumor lysis. All of these effects can cause significant morbidity and potential mortality.[21]



Steroids are contraindicated in some newly diagnosed leukemias and lymphomas. Consult oncology prior to administering any steroids.

CHAPTER SUMMARY FOR PERIOPERATIVE CORTICOSTEROIDS

Endogenous Glucocorticoids	Produced in the adrenal cortex Release is controlled by a negative feedback mechanism known as the hypothalamic-pituitary-adrenal axis (HPAA) Increased production during periods of stress (sepsis, trauma, surgery)
Adrenal Insufficiency (AI)	Tertiary or iatrogenic AI is the most common type, where exogenous corticosteroid administration causes HPAA suppression Acute AI should be suspected in a patient on chronic steroid therapy with severe refractory hypotension
Perioperative Steroid Replacement	Patients on chronic steroid therapy must be maintained on their preoperative steroid dose on the day of surgery Not all patients on chronic steroids need perioperative stress-dose steroids Risk of HPAA suppression must be evaluated preoperatively, and high-risk patients should receive stress-dose steroids based on anticipated surgical stress
Other Indications for Perioperative Corticosteroids	Dexamethasone is commonly used for prevention of PONV Dexamethasone is also used to reduce brain tumor–associated vasogenic edema and airway edema Steroids are also used as part of multimodal analgesia to reduce postoperative pain and opioid consumption
Contraindications	Limited evidence regarding the efficacy of high dose of methylprednisolone therapy for acute traumatic spinal cord injury Use in severe traumatic brain injury is not recommended In patients with newly diagnosed leukemia, steroid administration may delay diagnosis or cause tumor lysis

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34 Antimicrobials

Justin G. Meyerowitz • Andrea J. Traynor

Antimicrobials are pharmacologic agents that inhibit the growth of or kill infectious microorganisms including bacteria, fungi, or viruses. Antimicrobials are commonly administered in the perioperative period to prevent surgical site infection (SSI). The choice of agent depends on the type of surgical wound, typical microorganisms expected at the site, antimicrobial resistance patterns of the institution, and consequences of SSI.[1] Antibacterial agents are the subset of antimicrobials most commonly administered in the perioperative period, and rarely antifungal agents may also be administered (eg, in the setting of gastric or esophageal injury). It is important to consult with the surgeon about the antimicrobial requirements for a given procedure. If no antimicrobial is necessary, the reason for omission should be documented. This chapter will discuss antimicrobial agents used in the perioperative period.

1) **Choice of antimicrobial agent**
   a) **By typical microorganisms**: When used for empiric or prophylactic therapy, the antimicrobial agent should be chosen based on typical microorganisms.
   b) **Narrowest spectrum**: The narrowest spectrum agent should be chosen in order to minimize resistance and overtreatment.
   c) **By identification**: In patients with ongoing infection, perioperative antibiotics should be chosen based on identified microbe and their specific susceptibility.



Prevalent organisms and resistance patterns vary by region and institution. This information can often be obtained from your institution, county, state, or other health care authority.

2) **Type of surgical procedure**
   a) **Clean surgical wounds**
      i) Definition: Uninfected operative wound that is primarily closed, in which no inflammation is encountered and the respiratory, alimentary, genital, or uninfected urinary tracts are not entered.[2]
      ii) Choice of antimicrobial: Typically infected with skin flora (eg, *Staphylococcus aureus*), thus a cephalosporin such as cefazolin is preferred.[1]
   b) **Clean-contaminated surgical wounds**
      i) Definition: Operative wounds in which the respiratory, alimentary, genital, or urinary tracts *are entered* under controlled conditions[2]
      ii) Choice of antimicrobial: Typically requires gram-negative and/or anaerobic coverage such as a cephalosporin plus metronidazole[1]
   c) **Contaminated surgical wounds**
      i) Definition: Open, fresh, accidental wounds including those involving a major break in sterility or gross spillage from the gastrointestinal tract.[2]
      ii) Choice of antimicrobial: Typically requires coverage for skin flora, gram negatives, and anaerobes. Consult with surgeon.
   d) **Dirty or infected surgical wounds**
      i) Definition: Preexisting wounds or trauma that includes devitalized tissue, existing clinical infection, or perforated viscera.[2]

ii) Choice of antimicrobial: If culture data are available, direct antimicrobial to relevant organism. If unavailable, use empiric coverage based on infectious source.

e) **Infective endocarditis (IE) prophylaxis**[3]
   i) To reduce the risk of IE in patients undergoing dental, genitourinary, or gastrointestinal procedures
   ii) Only indicated in the highest-risk patients (see **Table 34-1**)
   iii) A single dose of amoxicillin or a cephalosporin should be given 30-60 minutes prior to the procedure. Azithromycin or clindamycin may be substituted in allergic individuals.

f) **Cesarean section**
   i) To reduce the risk of postpartum SSI, urinary tract infection, and endometritis, a single administration of preincision cefazolin is recommended.[4]
   ii) For women with penicillin allergy, combination therapy with clindamycin 900 mg plus gentamicin 5 mg/kg is recommended.[5]
   iii) For intrapartum cesarean sections and those performed at least 4 hours after rupture of membranes, a single dose of azithromycin 500 mg in addition to the above further reduces the risk of infectious complication.[6]

3) **Timing of administration**
   a) Most evidence suggests that SSI risk is optimized if antimicrobial administration occurs within 1 hour prior to surgical incision.[7,8]
   b) For cephalosporins and other antimicrobials with short infusion times, SSI risk reduction is optimized with administration within 60 minutes prior to incision.[7]
   c) For vancomycin, SSI risk reduction is optimized with administration 15-60 minutes prior to incision.[8]
   d) **Redosing** should occur if the duration of the procedure exceeds two half-lives of the antimicrobial or if there is excessive blood loss (>1500 mL).[9] See **Table 34-2** for dosing guidelines.

4) **Interactions with anesthetic agents**
   a) **Neuromuscular blocking agents and aminoglycosides**
      i) There is weak evidence that aminoglycosides such as gentamicin, particularly at supratherapeutic levels, potentiate and prolong neuromuscular blockade.



For antibiotics that require prolonged infusion time such as vancomycin or fluoroquinolones, consider arranging for infusion to begin prior to transfer to the operating room.

TABLE 34-1 Cardiac Conditions Associated With the Highest Risk of Adverse Outcome From Endocarditis for Which Prophylaxis With Dental Procedures Is Reasonable

- Prosthetic cardiac valve or prosthetic material used for cardiac valve repair
- Previous infective endocarditis
- Congenital heart disease (CHD)[a]
- Unrepaired cyanotic CHD, including palliative shunts and conduits
  - Completely repaired congenital heart defect with prosthetic material or device, whether placed by surgery or by catheter intervention, during the first 6 months after the procedure[b]
  - Repaired CHD with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device (which inhibit endothelialization)
- Cardiac transplantation recipients who develop cardiac valvulopathy

[a]Except for the conditions listed above, antibiotic prophylaxis is no longer recommended for any other form of CHD.
[b]Prophylaxis is reasonable because endothelialization of prosthetic material occurs within 6 months after the procedure.
Source: Reused with permission from Wilson W, Taubert KA, Gewitz M, et al. Prevention of infective endocarditis: guidelines from the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group. *Circulation*. 2007;116:1736-1754.

TABLE 34-2 Common Intraoperative Antimicrobials

Antimicrobial	Adults	Pediatrics	Redosing Interval	Class	Pearls
Ampicillin-sulbactam	2 g/1 g	50 mg/kg (amp)	2 h	Penicillin and β-lactamase inhibitor	
Ampicillin	2 g	50 mg/kg	2 h	Penicillin	
Aztreonam	2 g	30 mg/kg	4 h	Monobactam	
Cefazolin	2 g (3 g for >120 kg)	30 mg/kg	4 h	IGC	
Cefuroxime	1.5 g	50 mg/kg	4 h	2GC	
Cefotaxime	1 g	50 mg/kg	3 h	3GC	
Cefoxitin	2 g	40 mg/kg	2 h	2GC	
Cefotetan	2 g	40 mg/kg	6 h	2GC	
Ceftriaxone	2 g	50-75 mg/kg	24 h	3GC	
Ciprofloxacin	400 mg	10 mg/kg	12 h	Fluoroquinolone	Prolonged infusion, prolongs QTc
Clindamycin	900 mg	10 mg/kg	6 h	Lincosamide	
Ertapenem	1 g	15 mg/kg	24 h	Penem	
Fluconazole	400 mg	6 mg/kg	24 h	Azole antifungal	
Gentamicin	5 mg/kg	2.5 mg/kg	24 h	Aminoglycoside	Can prolong neuromuscular blockade
Levofloxacin	500 mg	10 mg/kg	24 h	Fluoroquinolone	Prolonged infusion, prolongs QTc
Metronidazole	500 mg	15 mg/kg	6-8 h	Nitroimidazole	
Moxifloxacin	400 mg	10 mg/kg	24 h	Fluoroquinolone	Prolonged infusion, prolongs QTc
Piperacillin-tazobactam	3.375 g	80-100 mg/kg	2 h	Penicillin and β-lactamase inhibitor	
Vancomycin	15 mg/kg	15 mg/kg	12 h	Glycopeptide	Prolonged infusion
	Penicillins	Fluoroquinolones	Cephalosporins (1/2/3-Generalion)		

Dosing parameters based on Bratzler DW, Dellinger EP, Olsen KM, et al. Clinical practice guidelines for antimicrobial prophylaxis in surgery. *Surg Infect (Larchmt)*. 2013;14(1):73-156.

ii) This effect is resistant to reversal by neostigmine but potentially reversed by calcium.[10]

5) **Adverse reactions to antimicrobials**

a) **Anaphylaxis and allergic reactions**

i) Incidence of antimicrobial allergy is 3% for sulfa drugs, 1%-1.5% for penicillins, and 0.5%-1% for other antimicrobial classes.[11]

ii) A thorough history of allergic reaction is important: anaphylaxis, hives, wheezing, or tongue swelling is concerning for true allergy.

iii) Allergy to penicillin (PCN) is a common reported drug allergy.

(1) PCN skin testing is indicated in patients with reported PCN allergy, since <10% of patients with reported PCN allergy are indeed allergic.[12,13]

(2) A chart history of PCN "allergy" leads to longer hospital stay, increased broad-spectrum antibiotic use, and increased incidence of infection with *Clostridium difficile*, methicillin-resistant *S. aureus* (MRSA), or vancomycin-resistant *Enterococcus* (VRE).[14]

iv) Cross-immunity to PCN and cephalosporins

(1) Reported cross-reactivity is highly variable (0.17%-8.4%), possibly owing to differences among cephalosporins and symptoms qualified as "allergy".[15]

(2) Choosing a less-optimal antimicrobial over a cephalosporin due to PCN "allergy" increases risk of SSI. Generally, cephalosporins can be given to patients who do not have a history of anaphylaxis to PCN or positive PCN skin test.[12–15]

b) **Red man syndrome**[16]

i) Most commonly associated with rapid IV infusion of vancomycin but also seen with ciprofloxacin, amphotericin B, rifampin, and teicoplanin.

ii) To avoid this reaction, vancomycin 1 g should be infused over at least 60 minutes.

iii) Manifested by an erythematous rash on the face, neck, and torso, pruritus, hypotension, and angioedema.

iv) Histamine-mediated degranulation of mast cells and basophils independent of IgE and complement.

v) Treat with antihistamines (diphenhydramine, 50 mg) and discontinue the infusion. Vancomycin can be re-administered at a slower rate.



Vancomycin IV requires prolonged infusion time (>60 minutes per 1 g) in order to avoid red man syndrome.

c) **Stevens-Johnson syndrome**[17]

i) Serious drug reaction manifested by fever, malaise, and characteristic mucocutaneous lesions that can progress to multiorgan failure.

ii) Antimicrobial drugs are among many classes of medications associated with this reaction.

iii) Treat by discontinuing the suspected cause and promptly consult with dermatologic and/or burn specialists

d) ***C. difficile* colitis**[18]

i) Surgical antimicrobial prophylaxis predisposes patients to *C. difficile* colitis, with up to 20% of patients acquiring colonization after single dose of SSI prophylaxis.[19]

ii) Risk factors include longer duration of prophylaxis or therapy and use of multiple antimicrobial agents.

iii) Limiting duration and breadth of coverage of antimicrobial prophylaxis can reduce the risk of *C. difficile* colitis.

e) **Other adverse reactions to antimicrobials**: Reactions to antimicrobials are highly variable and range from teratogenicity to QTc prolongation (**Table 34-3**).

TABLE 34-3 Direct Drug Toxicity Associated With Administration of Antimicrobials

Toxicity	Antimicrobial
Allergic reactions	All antimicrobials but most often with beta-lactam derivatives
Nephrotoxicity	Aminoglycosides Polymyxins Amphotericin B
Neutropenia	Penicillins Cephalosporins Vancomycin
Inhibition of platelet aggregation	Penicillins (high doses)
Prolonged prothrombin time	Cephalosporins
Bone marrow suppression (aplastic anemia, pancytopenia)	Chloramphenicol Flucytosine
Hemolytic anemia	Chloramphenicol Sulfonamides Nitrofurantoin Primaquine
Agranulocytosis	Macrolides Trimethoprim-sulfamethoxazole
Leukopenia and thrombocytopenia (folate deficiency)	Trimethoprim
Normocytic normochromic anemia	Amphotericin B
Ototoxicity	Aminoglycosides Vancomycin (auditory neurotoxicity) Minocycline (vestibular toxicity)
Seizures	Penicillins and other beta-lactams (high doses, azotemic patients, history of epilepsy) Metronidazole
Enhanced neuromuscular blockade	Aminoglycosides
Peripheral neuropathy	Nitrofurantoin (renal failure) Isoniazid (prevent with pyridoxine) Metronidazole
Benign intracranial hypertension	Tetracyclines
Optic neuritis	Ethambutol
Hepatotoxicity	Isoniazid Rifampin Tetracyclines (high doses) Beta-lactam antimicrobials (high doses) Nitrofurantoin Erythromycin Sulfonamides
Increased plasma bilirubin concentrations	Quinupristin-dalfopristin Erythromycin
Gastrointestinal irritation	Tetracyclines
Prolongation of QTc interval	Erythromycin Fluoroquinolones
Hyperkalemia	Trimethoprim-sulfamethoxazole

(*Continued*)

TABLE 34-3 Direct Drug Toxicity Associated With Administration of Antimicrobials (*Continued*)

Toxicity	Antimicrobial
Tendonitis	Fluoroquinolones
Arthralgias and myalgias	Quinupristin-dalfopristin
Photosensitivity	Sulfonamides Tetracyclines Fluoroquinolones
Exaggerated sympathomimetic effects in patients receiving monoamine oxidase inhibitors	Linezolid
Teratogenicity	Tetracyclines Metronidazole Rifampin Trimethoprim Fluoroquinolones

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CHAPTER SUMMARY FOR ANTIMICROBIAL MEDICATIONS

Definition	Antimicrobial agents are used in medicine to treat or prevent infection by bacteria, fungi, or viruses.
Selection	Narrow-spectrum agents should be chosen, when possible, to target organisms infecting tissue and to prevent development of antibiotic resistance.
Surgical Site Infection **Bacterial Endocarditis**	Consult with surgeon regarding antimicrobial requirements for a given surgical procedure. Only the highest-risk patients require endocarditis prophylaxis.
Timing	Consider dosing antibiotics within 1 hour prior to surgical incision.
Side Effects/Adverse Reactions	Anaphylaxis Minimal cephalosporin/penicillin cross-reactivity Red man syndrome (vancomycin) Limiting antimicrobial exposure minimizes *C. difficile* colitis

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35 Hematologic Agents

Kristin L. Richards

Hematologic agents are medications that modulate blood coagulation or formation of blood cells. Five to ten percent of the patients who undergo surgery are on anticoagulant medications, while procoagulant medications are frequently used in the management of bleeding, coagulopathies, and resuscitation. Anesthesiologists should be familiar with the pharmacology of these medications. However, **prior to initiating regional anesthesia on any patient on anticoagulants, the American Society of Regional Anesthesia guidelines should first be consulted.**

1) **Overview**
   a) Hematologic agents can be classified into anticoagulant medications and procoagulant medications.
   b) Anticoagulant medications are heparin, low molecular weight heparin (LMWH), warfarin, direct oral anticoagulants (DOACs), thrombolytics (such as streptokinase), glycoprotein IIb/IIIa inhibitors (such as abciximab), and adenosine diphosphate (ADP) inhibitors (such as aspirin).
   c) Procoagulants include factor VIIa, factor VIII concentrate, factor IX concentrate, prothrombin complex concentrate (PCC), protamine, antifibrinolytics (such as tranexamic acid), and desmopressin (DDAVP).
2) **Anticoagulant medications (Table 35-1)**[1]
   a) **Heparin**
      i) A negatively charged pentasaccharide that binds to naturally occurring antithrombin III, potentiating its inhibitory effect by 100-10 000 times. Heparin also has effects on other coagulation factors, including thrombin, and activated factors IX, X, XI, and XII.
      ii) Unfractionated heparin has anti-Xa to anti-IIa activity of about 1:1.
      iii) Derived from bovine lung or porcine intestine.
      iv) Side effects
         (1) Hemorrhage, allergic reactions, histamine release, heparin-induced thrombocytopenia
      v) Clinical uses
         (1) Anticoagulation in patients undergoing coronary bypass surgery, vascular surgery, and percutaneous coronary intervention
         (2) Treatment of patients with venous thrombosis and acute myocardial infarction
         (3) Prevention of deep vein thrombosis in high-risk patients undergoing noncardiac surgery[2]
         (4) Prevention and treatment of venous thrombosis and pulmonary embolism
      vi) Pharmacokinetics[1,3]
         (1) Heparin has low bioavailability due to complex protein binding.



Heparin is negatively charged and acts by binding antithrombin III and potentiating its effect on anticoagulation factors. It is reversed by protamine, a positively charged protein, that binds it making heparin inactive.

TABLE 35-1 Review of Anticoagulant and Antiplatelet Medications

	Representative Medications	Mechanism of Action/Pharmacokinetics	Monitoring	Other
Heparin	Heparin	Binds antithrombin III	aPTT, ACT	Can be reversed with protamine. Does not cross placenta
Low molecular weight heparin (LMWH)	Enoxaparin, dalteparin, fondaparinux	Derived from unfractionated heparin	Anti-Xa	
Warfarin	Warfarin	Inhibits production of vitamin K–dependent coagulation factors	PT, INR Can be reversed by prothrombin complex concentrate, vitamin K, or FFP	Routine administration is oral Crosses placenta
Direct oral anticoagulants (DOACs)	Dabigatran, rivaroxaban, apixaban, and edoxaban	Direct thrombin inhibitor: dabigatran Direct factor Xa inhibitors: rivaroxaban, apixaban, and edoxaban	Reversal agent, idarucizumab, is available for dabigatran Coagulation factor Xa (recombinant), inactivated-zhzo (Andexxa) is available to reverse rivaroxaban and apixaban	Specific coagulation assays to measure anticoagulation-dilute thrombin time for dabigatran PTT for dabigatran may provide a qualitative assessment if there is a residual anticoagulant effect Anti-Xa levels for oral Xa inhibitors PT for rivaroxaban
Thrombolytic medications	Streptokinase, alteplase, anistreplase	Produce thrombolysis by facilitating the conversion of plasminogen to plasmin		
Glycoprotein IIb/IIIa inhibitors	Abciximab, eptifibatide, tirofiban	Block fibrinogen binding at the platelet glycoprotein IIb/IIIa receptors and inhibit the final common pathway for platelet aggregation		
Adenosine diphosphate (ADP) inhibitors	Aspirin, clopidogrel, ticlopidine, dipyridamole, dextran	Prevents platelet activation, aggregation, and degranulation	Most are irreversible inhibitors; effects last for life of the platelet: 7-10 d	Aspirin is not a contraindication to regional anesthesia

(2) Administration is intravenous (IV) or subcutaneous (SC).
(3) Duration
(a) Intravenous action is immediate, while SC administration causes onset of action in 1-2 hours.
(b) Duration of action is for ~4-6 hours (IV) and 12 hours (SC), but this is highly variable and must be monitored. The precise pathway of heparin elimination is uncertain.
(4) Heparin does not cross the placenta, making its use safe in pregnancy.


The onset, duration, and clinical effect of heparin are highly variable between individuals and should be monitored with laboratory values.

vii) The amount of heparin required for a given effect is extremely variable between individuals.
(1) Activated partial thromboplastin time (aPTT) and the activated clotting time (ACT) are most commonly used to monitor heparin-induced anticoagulation.
(2) Some institutions monitor anti-Xa assays instead of aPTT due to variability of responses: low-dose regimens target 0.3-0.5 units/mL and high-dose regimens 0.5-0.8 units/mL.
viii) Considerations for surgery/anesthesia
(1) Elective surgery and regional anesthesia should be postponed until IV heparin has been discontinued for 4-6 hours or until the aPTT is normalized.
(2) If emergency surgery is required, heparin can be reversed with protamine (see below).
(3) Patients receiving low-dose SC heparin (5000 units bid) need not have surgery and/or regional anesthesia postponed. For higher SC doses, aPTT should be checked.


Subcutaneous heparin (5000 units bid) is not a contraindication to regional anesthesia.

b) **Low molecular weight heparin (LMWH)**
i) Derived from unfractionated heparin.
ii) Has a uniform molecular size, resulting in more predictable anticoagulant effects and pharmacokinetics.
iii) Representative medications include enoxaparin, dalteparin, and fondaparinux.
iv) LMWH has anti-Xa to anti-IIa activity that varies to 4:1 to 2:1.
v) The effect of LMWH is greatly prolonged with renal failure, and unfractionated heparin would be a better choice in this population.
vi) Clinical uses
(1) Treatment of acute coronary syndrome and prevention of thrombosis, especially deep venous thrombosis and pulmonary thromboembolism
(2) Prevention of DVT in high-risk patients and in the postoperative period
vii) Considerations for surgery/anesthesia
(1) Timing of surgery and regional anesthesia depends on the drug and dosage but should be delayed at least 12 hours from the last dose.
viii) Laboratory monitoring is usually not necessary, except in high-risk patient populations such as newborns, the morbidly obese, and/or those with renal failure.
(1) Monitoring is done using anti-Xa levels.
(2) Therapeutic range is 0.5-1.1 IU/mL and should be measured 4 hours after a dose.[4]


LMWH is monitored using anti-Xa levels.

c) **Warfarin**
i) Inhibits the conversion of vitamin K–dependent coagulation factors (factors II, VII, IX, and X) to their active form.
ii) Clinical uses
(1) Prevention of venous thromboembolism and prevention of systemic embolization in patients with prosthetic heart valves or atrial fibrillation and patients with thrombophilia

iii) Pharmacokinetics[3]
(1) Route of administration is oral.
(2) Despite rapid absorption after oral administration, the drug does not take effect for 8-12 hours, due to the elimination of clotting factors that are not affected by the drug. Peak effects occur in 36-72 hours.
(3) Warfarin crosses the placenta and is contraindicated in pregnancy.


Warfarin inhibits the production of vitamin K–dependent coagulation factors. It can be reversed with prothrombin complex concentrate (PCC), vitamin K, or FFP.

iv) Monitoring
(1) Anticoagulant effects vary dramatically between individuals, necessitating close monitoring via the prothrombin time (PT) and INR.

v) Considerations for surgery/anesthesia
(1) For elective procedures, anticoagulation should be discontinued for 1-5 days, which allows the INR to return within 20% of the normal range.
(2) The exact level of anticoagulation tolerable for an elective procedure depends on the type of procedure and the patient's risk of complications from discontinuation of anticoagulation.
(3) Patients with high risk of thromboembolism may require bridging therapy with unfractionated heparin, which is stopped 4-6 hours before planned surgery.
(4) PT and INR should be checked prior to regional anesthesia.
(5) For emergency or high-risk procedures, the anticoagulant effects can be reversed by administration of 2-5 mg, oral or IV, vitamin K (depending on the INR), PCC, or 1-2 units of fresh frozen plasma.


Strongly consider obtaining the PT/INR prior to elective surgery in a patient on chronic warfarin therapy.

d) **Direct oral anticoagulants (DOACs)**[5,6]
i) Direct thrombin inhibitor: dabigatran.
ii) Direct factor Xa inhibitors: rivaroxaban, apixaban, and edoxaban.
iii) Clinical uses: prevention and treatment of venous thromboembolism, stroke prevention in patients with atrial fibrillation, and postoperative prevention of DVT.
iv) A specific reversal agent, idarucizumab, is available for dabigatran.
v) Factor Xa inhibitor antidote (Andexxa) (coagulation factor Xa [recombinant], inactivated-zhzo) is indicated for patients treated with rivaroxaban (Xarelto) and apixaban (Eliquis), when reversal of anticoagulation is needed due to life-threatening or uncontrolled bleeding.
vi) Activated charcoal may mitigate the anticoagulant effect caused by DOACs if administered <2 hours after the drug was ingested.
vii) Four-factor PCCs may be useful to reduce life-threatening bleeding associated with factor Xa inhibitors.
viii) Perioperative implications
(1) Use of DOACs significantly increases the risk of bleeding in the perioperative period. They should be stopped prior to elective surgery. The data are still evolving and this complicates their management. For patients who are undergoing surgery with *high risk* of bleeding, dabigatran should be stopped 5 days before surgery, while factor Xa inhibitors should be stopped 2-3 days before surgery (based on renal function). Even for patients with low risk of surgery, factor Xa inhibitors should be discontinued at least 24 hours prior to surgery.
(2) DOACs should be stopped as per ASRA and European Society of Anaesthesiology guidelines, prior to performing neuraxial puncture or catheter removal (**Table 35-2**).[7]

TABLE 35-2 European Society of Anaesthesiology's Recommended Time Intervals Before and After Neuraxial Puncture or Catheter Removal

	Time Before Puncture/ Catheter Manipulation or Removal	Time After Puncture/ Catheter Manipulation or Removal	Laboratory Tests
UFHs (for prophylaxis, <15 000 IU/d)	4-6	1 h	Platelets during treatment for >5 d
UFHs (for treatment)	IV 4-6 h SC 8-12 h	1 h 1 h	aPTT, ACT, platelet
LMWHs (for prophylaxis)	12 h	4 h	Platelets during treatment for >5 d
LMWHs (for treatment)	24 h	4 h	Platelets during treatment for >5 d
Fondaparinux (for prophylaxis 2.5 mg/d)	36-42 h	6-12 h	(Anti-factor Xa standardized for specific agent)
Rivaroxaban (for prophylaxis, 10 mg daily)	22-26 h	4-6 h	(Anti-factor Xa standardized for specific agent)
Apixaban (for prophylaxis, 2.5 mg bid)	26-30 h	4-6 h	(Anti-factor Xa standardized for specific agent)
Dabigatran (for prophylaxis, 150-220 mg)	Contraindicated according to the manufacturer	6 h	TT
Coumarins	INR S1.4	After catheter removal	INR
Hirudirs (desinidin)	8-10 h	2-4 h	aPTT, EXT
Argatroban	4 h	2 h	aPTT, ECT, ACT
Acetylsalicylic acid	None	None	
Clopidogrel	7 d	After catheter removal	
Ticlopidine	10 d	After catheter removal	
Prasugrel	7-10 d	6 h after catheter removal	
Ticagrelor	5 d	6 h after catheter removal	
Cilostazol	42 h	5 h after catheter removal	
NSAIDs	None	None	

[a]All time intervals refer to patients with normal renal functions. Prolonged time interval in patients with hepatic insufficiency. Reprinted with permission from Gogarten W, Vandermeulen E, Van A, et al. Regional anaesthesia and antithrombotic agents: recommendations of the European Society of Anaesthesiology. *Eur J Anaesthesiol*. 2010;27(12):999-1015.

e) **Thrombolytic medications**
   i) Drugs that produce thrombolysis by facilitating the conversion of plasminogen to plasmin.
   ii) Representative medications include streptokinase, alteplase (recombinant tissue plasminogen activator), and anistreplase (anisolylated plasminogen-streptokinase activator complex).
   iii) Clinical uses
      (1) Treatment of acute coronary syndromes, pulmonary embolism, and acute ischemic stroke

f) **Glycoprotein IIb/IIIa inhibitors**[1]
   i) Block fibrinogen binding to the platelet glycoprotein IIb/IIIa receptors and inhibit the final common pathway for platelet aggregation.
   ii) Representative medications include abciximab, eptifibatide, and tirofiban.
   iii) These agents prevent thrombus formation initiated by platelets in the pathogenesis of acute coronary syndrome, angioplasty failure, and stent thrombosis.
   iv) Clinical uses
      (1) Treatment of patients with acute coronary syndrome and those undergoing interventional cardiology procedures such as percutaneous coronary intervention.
   v) Considerations for surgery/anesthesia
      (1) Glycoprotein IIb/IIIa inhibitors should be discontinued for 24-72 hours prior to elective surgery.
      (2) There are no specific reversal agents.



There are no specific reversal agents for glycoprotein IIb/IIIa inhibitors.

g) **ADP inhibitors**
   i) The inhibition of ADP prevents platelet activation, aggregation, and degranulation.
   ii) Representative medications include aspirin, clopidogrel, ticlopidine, prasugrel, ticagrelor, and cangrelor.
   iii) Clinical uses
      (1) Prevention of thrombosis in patients with coronary artery disease and those who have undergone coronary artery stenting, peripheral vascular disease, and acute coronary syndromes. It is typically used in combination with aspirin.
   iv) Considerations for surgery/anesthesia
      (1) Because most of these drugs are irreversible inhibitors, their effects last for the life of the platelet, which is 7-10 days.
      (2) The decision to discontinue antiplatelet medications perioperatively depends on the indication for use and bleeding risk of the surgery.
      (3) In patients with recent percutaneous coronary intervention and drug-eluting stent placement, discontinuation of aspirin and clopidogrel may significantly increase the risk of stent thrombosis and acute myocardial infarction.
         (a) These patients may benefit from continuation of antiplatelet therapy.
         (b) Dual antiplatelet therapy should continue for 6 weeks after bare-metal coronary stent placement and at least 6 months after drug-eluting coronary stent placement.
         (c) If the surgical procedure carries a high risk of bleeding, especially into a closed space such as the cranium, spine, and/or posterior chamber of the eye, discontinuation may be warranted.
         (d) Consultation with the patient's cardiologist is suggested.
      (4) Patients who are taking these medications for other indications should be advised to discontinue use for 7 days prior to surgery.[8]
      (5) Regional anesthesia can be performed after discontinuation of clopidogrel for 7 days.
      (6) For aspirin therapy, regional anesthesia is not contraindicated, but special considerations should be made when the aspirin is combined with other antiplatelet medications.
      (7) If emergency surgery is required and bleeding is encountered, platelet transfusion should be considered.



Patients with recent placement of a drug-eluting coronary stent are at risk of stent thrombosis and myocardial infarction if anti-platelet therapy is discontinued.



Ideally, antiplatelet therapy should be discontinued 5-7 days prior to elective surgery or regional anesthesia.



Platelet transfusion should be considered to treat bleeding in patients on antiplatelet therapy.

2) **Procoagulant medications (Table 35-3)**[8,9]
   a) **Recombinant factor VIIa**
      i) A synthetic form of activated factor VIIa.
      ii) It produces prohemostatic effect by multiple mechanisms that include complexing with tissue factor (TF), activating factor Xa, and thrombin generation.
      iii) Clinical uses
         (1) FDA-approved for treatment of bleeding in patients with hemophilia A or B and inhibitors to factors VII or XI.
         (2) Used as an off-label medication for life-threatening hemorrhage.
      iv) Usual dose is 20-70 µg/kg IV. Hemophiliac patients require a higher dose (200-400 µg/kg).
      v) Side effects
         (1) Thrombosis, including thrombotic stroke, acute myocardial infarction, pulmonary embolism, and clotting of indwelling devices have been reported.
   b) **Factor VIII concentrate**
      i) Indicated at times for patients with hemophilia A. Each FVIII unit per kg of body weight will raise the plasma factor VIII level by ~2%.
         (1) Be aware that patients can develop factor inhibitors, which are IgG antibodies directed against the deficient factor. It is more common with hemophilia A than hemophilia B.

TABLE 35-3 Review of Procoagulant Medications

Medication	Mechanism of Action/ Pharmacokinetics	Administration
Recombinant factor VIIa	Synthetic form of activated factor VIIa	Usual dose is 50-100 µg/kg IV
Factor VIII concentrate	Factor VIII, indicated at times for hemophilia A	
Factor IX concentrate	Factor IX, indicated at times for hemophilia B	
Prothrombin complex concentrate (PCC)	Contain three or four coagulation factors (factors II, IX, and X, with or without factor VII) and, depending on formulation, low doses of protein C, protein S	Can be used to reverse warfarin or in patients with coagulopathies
Protamine	Positively charged protein that combines with negatively charged heparin, rendering it in active derived from salmon sperm	Dose is 1 mg of protamine for every 100 units of heparin in circulation Rapid injection is associated with hypotension. Can rarely also cause pulmonary hypertension. Allergic reaction is most common in patients on NPH insulin and those with fish allergies
Antifibrinolytics: aminocaproic acid, tranexamic acid, aprotinin	Lysine analogs inhibit plasminogen activator and plasmin release Aprotinin: no longer used	TXA: has been associated with seizures at high doses
Desmopressin (DDAVP)	Synthetic analog of arginine vasopressin. Increases the amount of circulating factor VIII available to bind to von Willebrand factor	Treatment of bleeding or for preop administration with hemophilia A or certain types of von Willebrand disease. Preoperative management of diabetes insipidus

ii) Cryoprecipitate contains factor VIII, vWF, fibrinogen, factor XIII, and fibronectin. Cryoprecipitate provides ~80 units of FVIII in 30-40 mL.
iii) Fresh frozen plasma: 1 mL of FFP contains 1 unit of factor VIII activity. FFP is not often used in the perioperative management of hemophilias.

c) **Factor IX**
i) Indicated at times for patients with hemophilia B (Christmas disease). Each factor IX unit per kilogram of body weight infused will raise the plasma factor IX level by 1%, with a half-life of 18-24 hours.
ii) Number of units of factor IX required = (weight of patient) × (% factor level desired).

d) **Prothrombin Complex Concentrate (PCC)**[9]
i) PCCs contain either three or four coagulation factors (factors II, IX, and X, with or without factor VII) and, depending on formulation, low doses of coagulation inhibitors such as protein C, protein S, and heparin.
ii) PCCs increase thrombin generation by interacting with negatively charged phospholipids on activated platelets.
iii) Advantages of PCC are rapid availability, small volume of infusion, avoidance of ABO incompatibility and TRALI, associated with plasma. Typical dose is 30-50 units/kg.
iv) Can reverse warfarin rapidly, but adjunctive vitamin K is recommended, as PCCs have a short half-life.
v) Used in cases of significant bleeding in patients with a coagulopathy. It can rapidly increase the concentration of clotting factors by 40%-80%, without dilution of RBCs or platelets.

e) **Protamine**
i) A specific antagonist of unfractionated heparin.
ii) It is a positively charged protein that combines with negatively charged heparin, rendering it inactive.
iii) Derived from salmon sperm
iv) The dose is 1 mg of protamine for every 100 units of heparin in circulation (as calculated by the ACT).
v) Side effects
(1) Hypotension
(a) Rapid injection is associated with histamine release and hypotension. This may be attenuated by slow injection in a peripheral rather than central injection site.
(2) Pulmonary hypertension and right heart failure
(a) Rare effect due to thromboxane and serotonin release
(b) Manifested by arterial hypoxemia and pulmonary edema
(3) Allergic reactions
(a) Most common in patients receiving protamine-containing insulin (NPH) and those with fish allergies


Rapid injection of protamine is associated with hypotension and should be avoided.

f) **Antifibrinolytics**[10]
i) Aminocaproic acid and tranexamic acid are lysine analogs.
Both act by reversibly blocking the lysine binding sites of plasminogen to fibrin (plasminogen-fibrin binding site) and stopping the lysis of fibrin.
(1) Seizures have been noted after tranexamic acid, particularly when high doses are administered.
(2) They were conventionally used in patients with hemophilia A and von Willebrand disease.
(3) They reduce perioperative blood loss and decrease usage of packed red blood cells in cardiac, orthopaedic, and hepatic surgeries.
(4) They are also used as part of massive transfusion protocol in select trauma patients.

ii) Aprotinin
   (1) A nonspecific protease inhibitor that inhibits the intrinsic coagulation cascade, complement activation, fibrinolysis, and bradykinin formation
   (2) Extracted from bovine lung
   (3) No longer used due to increased risk of mortality as shown in Blood Conservation Using Antifibrinolytics in a Randomized Trial (BART).

g) **Desmopressin (DDAVP)**[11]
   i) A V2 analog of arginine vasopressin.
   ii) It stimulates the release of ultra large von Willebrand factor multimers from endothelial cells. Increased vWF might improve platelet adhesion to sites of vascular injury.
   iii) Clinical uses
      (1) Treatment of bleeding or for preoperative administration in patients with hemophilia A or certain forms of von Willebrand disease
      (2) Administered in patients with platelet dysfunction related to antiplatelet drugs or uremia
      (3) Perioperative management of diabetes insipidus[8]
      (4) 0.3-0.5 μg/kg IV is given 30 minutes prior to a surgical procedure (for hemophilia A and most types of von Willebrand disease).



DDAVP is indicated for treatment of bleeding, in certain instances, as well as in the treatment of diabetes insipidus.

CHAPTER SUMMARY FOR HEMATOLOGIC AGENTS

Definition	Hematologic agents are used to control the coagulation of blood and/or degradation of blood clots by modulating various aspects of the coagulation cascade.
Mechanism of Action	**Anticoagulants** interfere with proteins responsible for blood clotting or platelet aggregation such as antithrombin III (heparin) or production of vitamin K–dependent clotting factors (warfarin), glycoprotein IIb/IIIa inhibitors (abciximab), and ADP inhibitors (aspirin). **Thrombolytics** are used to dissolve formed blood clots by facilitating conversion of plasminogen to plasmin (streptokinase). **Procoagulants** such as activated factor VIIa, protamine, antifibrinolytics (aminocaproic acid), and desmopressin improve coagulation functions.
Special Considerations	Consult the ASRA guidelines prior to placement of neuraxial anesthetic blocks in patients taking anticoagulant medications.

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36 Perioperative Fluid Management

Tzevan Poon

Competent perioperative fluid management is of the utmost importance because intravascular fluids affect all physiological and biochemical processes essential for life. The goals of fluid management are to provide adequate delivery of oxygen to tissues and to maintain the environment necessary for proper function of cells and organ systems. Crystalloids and colloids should be thought of not only as volume replacements but also as pharmacologic agents, with different compositions and physiological effects.

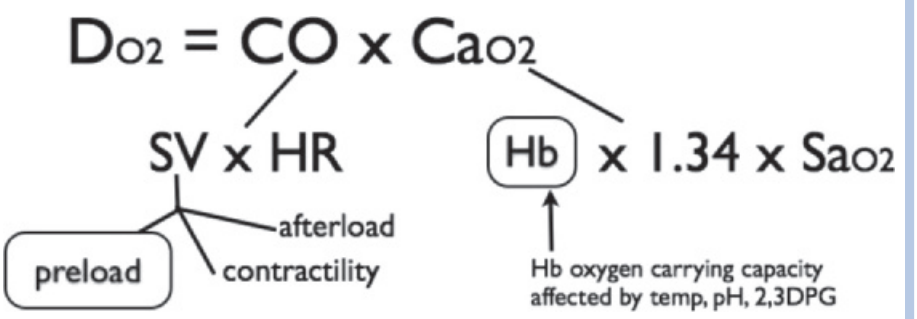
1) **Determinants of O_2 delivery to tissue**
   a) Generation of flow (cardiac output).
   b) O_2 content of blood.
   c) Preload and hemoglobin (Hb) are most often manipulated by intravascular fluid therapy (**Fig. 36-1**).
      i) A key assumption is that the vascular system (ie, vascular autoregulation and normal endothelial permeability) is intact.
2) **Fluid compartments**
   a) Circulating blood volume makes up a small percentage of body weight and total body water (**Fig. 36-2**).
      i) Yet, one should appreciate the profound influence this small fraction has on homeostasis.
   b) Estimated circulating blood volume should be compared with blood loss during surgery or trauma.
   c) The type and amount of fluid therapy used to treat blood/fluid loss have a greater physiologic effect as the proportion of fluid therapy to circulating blood volume increases (**Table 36-1**).



Inadequate or inappropriate fluid therapy can lead to poor perfusion, tissue hypoxia, and worsening acidosis.

3) **Classical calculation of fluid requirements**
   a) One common method of perioperative fluid management calculates the fluid requirement needed to meet estimated demands and losses (**Tables 36-2a and 36-2b**).
      i) This method is often referred to as a "liberal" fluid management strategy.
         (1) Calculated results often lead to greater volumes being infused than are actually lost during surgery.
         (2) This approach can help one gain a sense of the magnitude of fluids needed during a case.
            (a) However, evidence from many different surgical populations suggests that following a "liberal" fluid infusion strategy may lead to higher cardiac, pulmonary, renal, gut, and wound complications than more "restrictive" fluid strategies.[1,2]
            (b) Thus, the use of classical fluid management should be done with caution.
4) **Modern perioperative fluid management**
   a) The current intraoperative environment requires a sophisticated, individualized approach to fluid management.

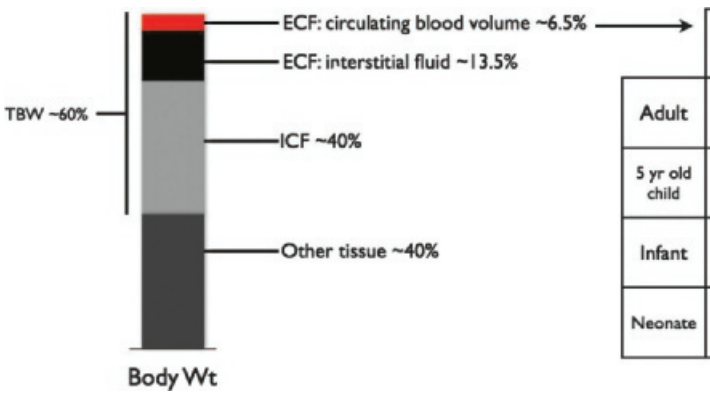
FIGURE 36-1 Determinants of O_2 Delivery to Tissue.



CaO_2, O_2 content of arterial blood; CO, cardiac output; DO_2, delivery of O_2; Hb, hemoglobin; HR, heart rate; SaO_2, arterial oxygen saturation; SV, stroke volume.

b) Clear goals should be used to determine the type of fluid, the amount, and the method in which it should be given (**Fig. 36-3 and Table 36-3**).
c) Fixed and dynamic variables influence fluid requirements and include the following:
   i) The patient's physiology and comorbidities
   ii) Anesthetic technique and intraoperative course
   iii) Type of surgery
d) Different data streams should be used as necessary to gather information.
   i) Functional reserve
   ii) Tissue oxygenation

FIGURE 36-2 Distribution of Body Fluid Compartments Relative to Body Weight TBW, total body water; ECF, extracellular fluid; ICF, intracellular fluid.



	blood volume (ml/kg)	example
Adult	~65	70 kg 4550 mL
5 yr old child	~75	20 kg 1500 mL
Infant	~80	13 kg 1400 mL
Neonate	~85	3 kg 255 mL

TABLE 36-1 Physiologic Variables and Blood Components Affected by Fluid Administration

Factors Affected by Fluid Therapy	Comments
Temperature	Patients are often hypothermic on arrival to the operating room. Fluid administration can cause or exacerbate hypothermia and acidosis. Hypothermia and acidosis significantly decrease platelet function and coagulation factor activity. Moderate to severe hypothermia in severely injured trauma patients is associated with higher morbidity and mortality, independent of other risk factors.[1]
pH	NS has an average pH of 5.6 but can have a pH as low as 4.5. Large infusions of NS lead to hyperchloremic metabolic acidosis. LR has less effect on pH, but large volumes can also lead to acidosis.
Electrolytes	Electrolyte derangements occur with changes in pH, temperature, and large-volume administration of crystalloid, colloid, and blood products.
RBC	Hemodilution, temperature, and pH affect RBC concentration and function. Stored PRBCs have lower pH and 2,3-DPG levels (decreased O_2 delivery) and have higher ammonia, PCO_2, and microaggregate levels. Massive transfusion of PRBCs can lead to hypocalcemia (due to citrate additive, though this is relatively transient) and hyperkalemia (due to RBC cell lysis).
Platelets and Coagulation Factors	Hypothermia and acidosis often seen with massive fluid infusions can cause or further exacerbate platelet dysfunction and coagulopathy. Certain disease states may cause baseline platelet dysfunction (eg, uremia, liver failure, preeclampsia/HELLP syndrome, massive trauma), and patients with these diseases may be more affected by fluid therapies.
Albumin	Albumin has important drug interactions and oncotic pressure effects. Preexisting hypoalbuminemia and concomitant third spacing are worsened by the hemodilution effects of large infusions.
WBC	Artificial colloids (eg, hetastarch) and LR significantly activate neutrophil activity and may play a pivotal role in resuscitation injury.
Inflammatory Cytokines	Blood products and artificial colloids increase inflammatory cytokines. The effect is unclear but is an area of active research.

HELLP, hemolysis, elevated liver enzymes, low platelets; LR, lactate Ringer's; NS, normal saline; (P)RBC, (packed) red blood cell; WBC, white blood cells.

   iii) Fluid responsiveness
   iv) Other hemodynamic variables

e) One must learn the strengths and weaknesses associated with each monitor. Avoid overreliance on any one.

f) Taken together, "goal-directed," patient- and procedure-oriented fluid management approaches have been shown to decrease morbidity and mortality in a wide range of populations.[1–3]

g) Enhanced recovery after surgery (ERAS) pathways:
   i) Practice bundles for major surgeries instituted by multidisciplinary healthcare teams (surgeons, anesthesiologists, physician assistants and nurse practitioners, and nurses).
   ii) ERAS pathways modify PO intake from strict overnight fasting to allowing carbohydrate drinks up to 2 hours prior to surgery (reducing preoperative hypovolemia); champion repleting fluids to maintain euvolemia over giving large volumes of intravenous fluids.
   iii) Seek to decrease large incisions by using laparoscopic or minimally invasive approaches.

TABLE 36-2A Intraoperative Fluid Requirements Based on Classical Indexes

Factors	Calculation	Example: Richard is a 65-y old, 70-kg Man Undergoing Small Bowel Resection, NPO for 10 h with 24-h Bowel Prep. (All Values for Crystalloid Administration)		
Step 1 Calculate preoperative fluid deficit	Use the "4-2-1 rule" = 4 mL/kg for the first10 kg 2 mL/kg for the second 10 kg 1 mL/kg for the remaining kg × no. of h NPO (shortcut: wt + 40 = mL/h)	4(10) + 2(10) + 1(50) = 110 mL/h or via shortcut 70 + 40 = 110 mL/h Thus, 110 × 10 h NPO = *1100 mL* GI losses from bowel prep ~**900 mL** **Total deficit = 2000**		
	Give ½ of volume in the 1st hour, and ½ over the next 2 hours.	1st hour	2nd hour	3rd hour
		1000 mL	500 mL	500 mL
Step 2 Estimate compensatory volume expansion from anesthetic technique	6-8 mL/kg	–500	–500	–500
Step 3 Calculate maintenance	Use the "4-2-1 rule"	110	110	110
Step 4 Calculate insensible, third space losses	Minimal tissue trauma = 0-2 mL/kg/h Moderate tissue trauma = 2-4 mL/kg/h Severe tissue trauma or large exposed surface area = 4-8 mL/kg/h	4 × 70 = 280	6 × 70 = 420	6 × 70 = 420
Step 5 Estimate blood loss	1:3 ratio replacement for blood: crystalloid 1:1 ratio replacement for blood: colloid	0	~100 EBL = 300	~200 EBL= 600
Step 6 UOP		100	110	75
	Totals (mL/h)	1990	1940	2205

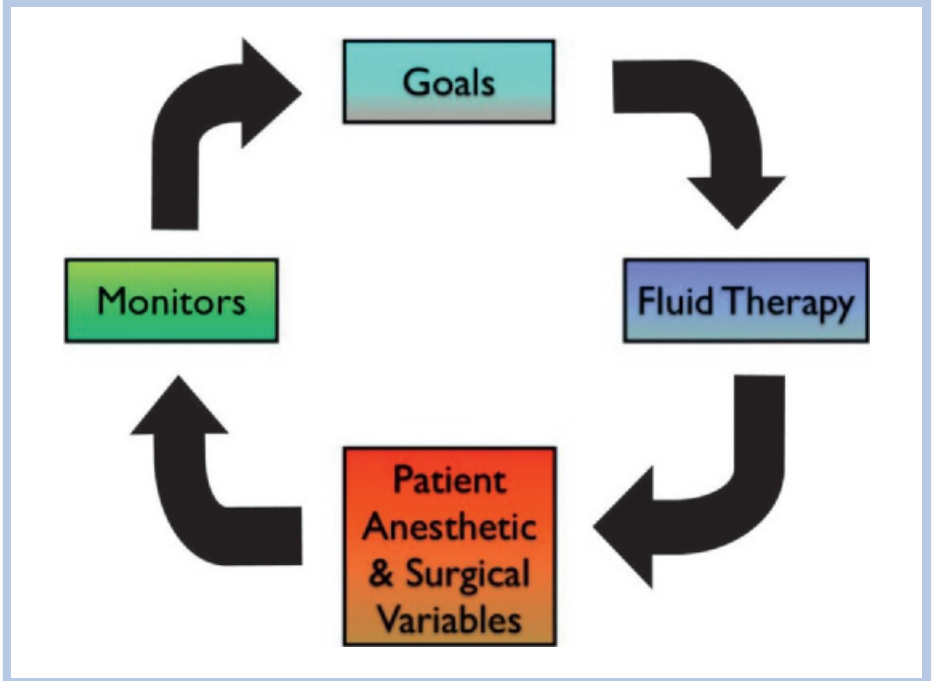
EBL, estimated blood loss; GI, gastrointestinal; MAP, mean arterial pressure; NPO, nothing per os; NS, normal saline; UOP, urine output.

TABLE 36-2B Intraoperative Fluid Requirements Based on Classical Indexes

Guidelines to Consider if Giving Fluid Therapy Based on Calculated Fluid Requirements

- Use sound medical judgment regarding the patient's current condition, medical history, and type of surgery and anesthetic when considering the type and amount of fluid to give.
- In general, try to maintain MAP within ±20% of baseline; UOP 0.5-1 mL/kg/h; core temperature 36°C-37°C.
- Be aware that even if vital signs continue to be around normal limits, this may not fully account for blood or insensible losses, especially in healthy patients with robust compensatory mechanisms.
- Use fluids that can be used for volume expansion for intraoperative maintenance. Do not use 1/2 NS or other fluids with less osmolarity as it will further increase interstitial fluid gain with little or no addition to effective intravascular circulating volume.

FIGURE 36-3 The Feedback Loop of Continuous Goal-Directed Fluid Management.



iv) Combined, large randomized trials using ERAS pathways have shown a 50% relative risk reduction in complications and reduced length stay in colorectal surgery, with benefits also found in liver, pancreatic, gastric, esophageal, thoracic, major urologic, gynecologic, orthopedic, and even emergency surgery.[2]

5) **Types of IV fluids**
   a) **Crystalloids: commonly used for resuscitation (Table 36-4)**
      i) Normal saline (NS)
         (1) Lower pH, higher osmolarity, and higher chloride content than plasma
            (a) Large-volume administration (>2-3 L) leads to hyperchloremic metabolic acidosis:
               (i) This can take hours to days to resolve in patients with acute and/or chronic renal failure.
               (ii) Lactated Ringer's (LR) and Normosol cause less perturbation to chloride and acid-base homeostasis.
      ii) Lactated Ringer's (LR)
         (1) The lactate in LR is rapidly converted into pyruvate and shuttled into the Krebs cycle mostly by liver cells.
            (a) During severe liver failure, overwhelming lactic acidosis, or, more infrequently, very large-volume infusions of LR, *net* conversion can be diminished.
         (2) LR contains a physiologic amount of potassium.

TABLE 36-3 Continuous Goal-Directed Fluid Management: Expanded View of Factors Involved in Decision-Making

Fixed and Dynamic Variables	Monitors and Other Data Streams	Overall Goals of Fluid Management	Fluid Therapies

• Cardiac function
• Vascular function
• Pulmonary function
• Renal function
• Preoperative intravascular volume depletion
• Anesthetic plan: technique and pharmacologic agents
• Type of IV access and other resources available (fluid warmers, rapid infusers, etc.)
• Patient positioning
• Type of surgery
• Anatomic changes during surgery (eg, thoracotomy, pneumoperitoneum, etc.)
• Duration of surgery
• Outcomes data from prior studies pertaining to patient or surgery
	• Preoperative studies
	• Preoperative functional status
	• Physical exam findings
	• NIBP
	• Dynamic A-line BP and respiratory variability
	• Pulse oximetry respiratory variability
	• UOP
	• Temperature probes
	• Intraoperative lab data
	  ○ ABG
	  ○ MVO_2 saturation
	  ○ Electrolytes
	  ○ Lactate
	  ○ Coagulation labs/TEG
	• CVP
	• PAC
	• Continuous MVO_2 sat
	• Continuous pulse Hb monitoring
	• TEE
		• Adequate delivery of oxygen (DLO_2)
		  ○ Preload
		  ○ Adequate Hb
		  ○ Cardiac indices
		  ○ SvO_2 sat >65%
		• Normothermia
		• Acceptable:
		  ○ pH
		  ○ Coagulation
		  ○ Electrolyte
		  ○ Environment
		  ○ Glucose levels
		• Unique requirements of surgery or patient
		• Prevention of fluid excess (possibly leading to pulmonary edema or CHF)
			• Crystalloids
			• Colloids
			• PRBCs
			• FFP
			• Platelets
			• Fibrinogen
			• Electrolyte replacements
			• Choice of IV access
			• Rapid infusers/ warmers

Accounting for and synthesizing the issues in this table enables a global perspective on the patient, the procedure, monitors, and the goals of fluid therapy.
ABG, arterial blood gas; A-line, arterial line; BP blood pressure; CHF, congestive heart failure; FFP, fresh frozen plasma; Hb, hemoglobin; NIBP, noninvasive blood pressure; MVO_2, mixed venous oxygen; PRBCs, packed red blood cells; TEE, transesophageal echocardiogram; TEG, thromboelastogram; UOP, urine output.

iii) Balanced crystalloids (Normosol, Plasma-Lyte)
   (1) Currently, slightly more expensive than other crystalloids but much less expensive than colloids.
   (2) A reasonable choice for resuscitation crystalloid for critically ill patients who have severely diminished compensatory mechanisms in response to anion gap and nongap metabolic acidosis.

TABLE 36-4 Characteristics of Commonly Used Crystalloids by pH, Osmolarity, and Ion Content

Crystalloid	pH	mOsm/L	Na^+	Cl^-	Ca^{2+}	$Lactate^{-a}$	K^+	Mg^{2+}	$Gluconate^{-b}$	$Acetate^-$
NS (0.9% saline)	5.6 (4.5-7.0)	308[c]	154[d]	154						
LR	6.6 (6.0-7.5)	274	130	109	3	28	4			
Balanced crystalloid (Normosol, Plasma-Lyte)	7.4 (6.5-7.6)	295	140	98			5	3	23	27

[a]Under normal circumstances, lactate is converted to pyruvate and shuttled back into the Krebs cycle by lactate dehydrogenase (LDH), an enzyme predominantly found in the liver. There are also other isoenzymes in organs such as the kidneys, striated muscle, lungs, and heart.
[b]The majority of parenteral gluconate is excreted unchanged by the kidneys (60%-80%), with much of the rest converted into CO_2 during the production of adenosine triphosphate (ATP) or into glucose through gluconeogenesis. Acetate is mostly oxidized by the liver into CO_2 by way of acetyl-CoA during ATP production (~70%), while a lesser percentage is recycled into ketone bodies or glucose through gluconeogenesis.
[c]Normal range of human plasma osmolarity is ~275-295 mOsm/L, whereas, at the extremes of overhydration and dehydration, plasma osmolarity can reach limits of 260 and 310 mOsm/L, respectively.
[d]All units of ions are mEq/L.
LR, lactated Ringer's; NS, normal saline.

(3) Contains a physiologic amount of potassium and more pH balanced than normal saline. In multiple randomized trials (specifically in patients undergoing renal transplant, a population at high risk for perioperative hyperkalemia), balanced crystalloids and LR have been shown to be safe and more effective in *preventing* hyperkalemia, with less rise in plasma potassium thought due to less cellular potassium fluid shifts.[4,5]

iv) Replacement of intravascular volume with crystalloid

(1) Classically, it has been taught that ~3-4 L of crystalloid (vs only 1 L of colloid) should be replaced for every liter of blood loss.

(2) Recent human data have shown that tissue equilibration may be slower than previously thought (20-30 minutes after infusion) and that this process is complex and dynamic.[6]

(3) Furthermore, robust clinical trial data have shown that titrating crystalloids and colloids to similar hemodynamic goals leads to a crystalloid:colloid ratio of only 1.3-1.6:1.0.

(a) This ratio is stable over hours to days.[7]

b) **Additional forms of crystalloid** are available and have a diverse array of uses.

i) Various mixtures of ½ NS

(1) D5 or 20 mEq/L of KCl may be added.

(2) Used as maintenance fluids before and after surgery.

(3) These hypotonic forms provide minimal intravascular volume effect and are usually not used intraoperatively.

ii) Isotonic sodium bicarbonate (300 mEq/L) in D5W plus *N*-acetylcysteine.

(1) Although initially thought to be superior, large randomized trials have demonstrated that sodium bicarbonate with or without *N*-acetylcysteine confers no additional benefit over saline alone in the prevention of contrast-induced nephropathy.[8]

iii) Hypertonic 3% or 7.5% saline

(1) Commonly administered to patients with brain injury to decrease intracranial pressure (ICP)

(2) Thought to osmotically promote outflow of brain interstitial fluid from the enclosed calvaria

(3) May be more effective than mannitol and has a good safety profile

(a) Imaging and autopsy studies have not shown evidence of central pontine myelinolysis from this therapy.[9]

(4) Hypertonic saline may be given through a central line as a 250-mL bolus every 6 hours as needed.

(a) Gradually achieve a plasma sodium goal of 145-155 mEq/L and an osmolarity of 310-320 mOsm/L.

(i) Increase plasma sodium no more than 10-20 mEq/L/24 h.

iv) Highly concentrated 23.4% sodium chloride

(1) May emergently reverse transtentorial herniation and temporize the situation until more invasive interventions can be performed (eg, craniectomy).

(2) Mechanism of action is similar to lower concentration hypertonic saline administration.

(3) May lead to transient hypotension after initial bolus (ie, seconds to minutes).

(a) However, heart rate, mean arterial pressure, and cerebral perfusion pressure generally remain stable.

(b) ICP may continue to decrease for up to 24 hours.[10]

(4) Given centrally in 30- or 60-mL boluses over 15-20 minutes.

c) **Colloids**: Provide oncotic pressure effects in addition to volume and are further classified as protein (human serum albumin or modified gelatin solutions) or nonprotein colloids (dextrans and hydroxyethyl starches).
   i) Albumin
      (1) The most expensive resuscitation fluid with the exception of blood products.
      (2) Well tolerated, and not associated with anaphylaxis, increased infection, or coagulopathy (aside from hemodilution effects) even at high volumes.
   ii) Crystalloids vs albumin in primary resuscitation: *The saline vs albumin fluid evaluation (SAFE) trial.*[11]
      (1) A large (6997 patients), multicenter, double-blinded randomized, controlled study.
         (a) Designed to address the safety of albumin administration.
         (b) Albumin did not confer overall mortality benefit or harm to a large representative group of critically ill patients.
            (i) Did not increase or decrease the risk of organ failure, duration of mechanical ventilation, or duration of renal replacement therapy.
         c) A statistically significant increase in mortality in traumatic brain injury patients was observed.
            (i) An *ad hoc* follow-up study showed that this increase involved only the most severely injured (GCS 3-8) patients.[12]



Consider caution in using albumin in patients with traumatic brain injury.

   iii) Gelatins: Not sold in the United States. Trials have shown similar in safety to albumin but with much shorter effects (3-6 hours).[12]
   iv) Dextrans
      (1) Have plasma-expanding volume effects. Also have robust anticoagulant properties. They have a low but relatively higher association with anaphylactoid reactions than other colloids.[12] They are not used for resuscitation.
   v) Hydroxyethyl starch
      (1) Available in the United States as high-molecular-weight hetastarch (450 kDa) and medium-molecular-weight pentastarch (200 kDa).
      (2) Hetastarch and pentastarch: Generally safe for *healthy* patients in volumes <2 L. However, avoid use in critically ill patients and in large-volume resuscitations as secondary outcomes from high-quality randomized controlled trials have shown hydroxyethyl starch use leads to significant increases in acute kidney injury, need for renal replacement therapy, red cell transfusion requirements, and 90-day mortality in septic patients.[13]



While hydroxyethyl starch is generally safe in volumes <2 L in healthy patients, they should NOT be used during larger-volume resuscitation or in critically ill patients.

   vi) Blood products
      (1) Have oncotic effects that expand intravascular volume similar to colloids

d) **Rational use of crystalloids and colloids**
   i) Crystalloids should usually be the initial fluids used for resuscitation. They are inexpensive, readily available, safe, and effective for intravascular volume replacement in a wide variety of surgical and critically ill populations.
   ii) Albumin should be considered the second-line volume agent due to cost and large randomized trial data that show no benefit to mortality or end-organ dysfunction in critically ill patients.
   iii) Despite being equivalent in terms of mortality and other major endpoints, there continues to be a large gap in our knowledge on what indication is better suited for albumin.

(1) Albumin may be considered if a large amount of crystalloid (>3-4 L) has already been given and the patient still appears intravascularly depleted.
(2) Elderly or extremely frail patients (with plasma albumin <2.5 g/dL) might not tolerate large volumes of fluid resuscitation and may benefit from early albumin administration.
(3) Giving albumin in special situations may provide benefits to certain patients such as after paracentesis with >4 L of ascites removed, during plasmapheresis, or resuscitation in the setting of nephrotic syndrome or severe diarrhea with albumin <2 g/dL.
(4) The choice to give crystalloid or colloid is only one factor among many other considerations when administering fluid therapy.



Surgeries involving large (and sometimes unexpected) amounts of blood loss will often use not just one type of fluid but a combination of crystalloids and colloids, including blood products.

6) **Pearls and pitfalls for select surgical populations**

a) **Neonates**
i) Require dextrose (D5) and should go no more than 3-4 hours without fluids
ii) Monitor blood glucose levels.
iii) Limit fluids with a mini-infusion device.
iv) Remove all air bubbles from lines to prevent any possibility of air emboli.

b) **Preeclampsia**
i) Patients usually have notable fluid deficits and decreased uteroplacental blood flow.
ii) Despite intravascular volume depletion, these patients are at risk for pulmonary edema.
iii) Randomized controlled trials to guide fluid management in patients with preeclampsia are lacking.
iv) Most practitioners limit crystalloid administration to 1-2 L.
(1) Some use colloid prior to spinal anesthesia.
(2) Arterial BP monitoring should be considered in patients with severe preeclampsia.
(3) Other invasive monitoring (eg, PA catheter, TEE) may be helpful to guide fluid management in patients with renal failure in the setting of severe preeclampsia.

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Pregnancy and hypertension, Chapter 109

c) **Cerebral edema and/or intracranial neurosurgical patients**
i) Dextrose-containing fluids are contraindicated because they can worsen neurological outcome.[14]
(1) Hyperglycemia and oxidative stress lead to increased glycolytic metabolism and toxic derivative production.
(2) The optimal level of glucose is unknown, but it is generally accepted that values >180 mg/dL should be treated with insulin.



Dextrose-containing fluids should be avoided in patients with neurologic injury or undergoing intracranial neurosurgical procedures.

d) **Congestive heart failure (CHF)**
i) Judicious fluid management should be the rule in CHF patients.
ii) Expect fluid redistribution to stress the heart in the first 24-96 hours post surgery.
iii) Consider invasive monitoring and intensive care unit (ICU) postoperative care.

e) **Acute respiratory distress syndrome (ARDS) (23)**
i) Patients with ARDS may need emergency surgical services.
ii) Fluid-restrictive therapy has been shown to reduce morbidity in these patients in the ICU setting (20). However, this study had goals of CVP < 4 mm Hg or PAOP < 8 mm Hg, which may be difficult to achieve.

iii) A fluid-restrictive approach may be beneficial, but this may be complicated by other factors and conditions such as sepsis.
iv) Invasive monitoring should be considered to guide fluid therapy.

f) **Lung resection**
i) Although multifactorial in origin, postpneumonectomy pulmonary edema (PPE) rates increase with excessive fluid therapy.
ii) Minimize fluids to ~1.5 L for an entire case.
(1) Use vasopressors intraoperatively as needed.
iii) Fluid restriction may also be beneficial during the first 72 hours postoperatively.
iv) Blood transfusion should be avoided.
(1) It is linked to worse perioperative pulmonary inflammatory changes and increased risk for PPE.

g) **Anuric renal failure**
i) These patients are unable to compensate for fluid overload and are often challenging.
ii) Consider invasive BP monitoring with an arterial line.
iii) Central venous access.
(1) If possible, avoid cannulating upper extremity central veins as these may be sites for future arteriovenous fistulae (AVF).
(2) No IV catheters or BP cuffs should be placed on same side as an arteriovenous fistula unless absolutely necessary.
(a) Every effort should be made to protect patients' fistula when anesthetized.

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Diseases of the liver and biliary tract , Chapter 56

h) **Liver failure/hepatic transplantation**
i) Patients often have multisystem organ dysfunction.
ii) A wide variety of possible complications and comorbidities may be present (24).
(1) Cerebral edema
(2) Hyperdynamic ventricular function
(3) Cardiomyopathy
(4) Pulmonary hypertension
(5) Hepatopulmonary syndrome
(6) Hepatorenal syndrome
(7) Effective circulating volume depletion
(8) Electrolyte perturbations
(9) Anemia
(10) Coagulopathy

READ MORE
Burn injuries, Chapter 68

i) **Burns**
i) These patients often have massive fluid requirements due to widespread loss of epidermal barrier and circulating oncotically active proteins
ii) The Parkland formula is frequently used as an *initial guide* for fluid requirements for burns >30% total body surface area:
(1) BSA (use % as a whole number not as fraction) × wt (kg) × 4 = total mL over 24 hours.
(a) Give ½ of volume over 8 hours and second ½ over the next 16 hours.
(2) More importantly, titrate infusions to hemodynamic parameters or to a urine output of 0.5-1 mL/kg/h or more.
iii) Albumin is often given to replace large intravascular protein losses from injury after the first 24 hours.
(1) However, timing and amount vary widely among institutions.

j) **Liposuction**
i) The "tumescent technique" consists of infiltration of the surgical region with very dilute lidocaine 0.1% (1 mg/mL) and very dilute epinephrine (1:1 000 000 = 1 mg/L) in large volumes of saline.

ii) The area becomes swollen (tumescent), which helps minimize bleeding, decreases lidocaine absorption, and achieves effective local anesthesia in the surgical region.
iii) Maximum lidocaine dose for this technique is up to 55 vs 7 mg/kg maximum for 1%-2% lidocaine with 1:100 000 epinephrine.[15]
iv) Minimize any additional fluids that may be given intravenously.
v) Cardiopulmonary complications may be increased when liposuction volumes exceed 5 L due to large fluid shifts.

k) **Laparoscopic gastric bypass surgery**
i) Transient intraoperative oliguria frequently develops due to increased SVR and compression of intra-abdominal renal cortices and the inferior vena cava secondary to pneumoperitoneum.
(1) Effects are reversed when pneumoperitoneum is abolished.
ii) A liberal management strategy is often used for these patients. However, care must be taken to avoid perioperative cardiopulmonary complications.

l) **Massive trauma**
i) Patients require close attention in order to avoid the "lethal triad" of acidosis, hypothermia, and coagulopathy.

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Trauma and massive transfusion, Chapter 125

CHAPTER SUMMARY FOR PERIOPERATIVE FLUID MANAGEMENT

Overview	Management of perioperative fluid infusions is best served by a goal-directed approach. This may be straightforward or highly complex depending on the clinical situation.
Crystalloids	Crystalloids are most commonly used for intravascular volume expansion. Classically, 3-4 L crystalloid are administered for every liter of blood loss, but modern clinical trials show this is often excessive.
Colloids	Colloids provide oncotic pressure in addition to intravascular volume. Colloids are categorized as protein (albumin or gelatin solutions) and nonprotein (dextrans or HES). Normal saline and albumin have been shown be safe in a wide variety of critically ill populations. Many complex surgical cases that involve large fluid requirements often require a combination of crystalloid and colloid (including blood products). Other types of colloids (hetastarch, dextrans) should be avoided in critically ill patients and, in general, used sparingly in healthy populations.
Key Points	Classical calculations on fluid requirements may be helpful in giving an overall sense of the volume needed, but can lead to overinfusion. Large randomized controlled trials suggest that the ratio of the volume effects for blood to albumin to crystalloid is not 1:3 as has been traditionally taught, but 1:1.4. All intravenous infusions have the potential to adversely affect the patient when given in excessive amounts (**Table 36-1**). ERAS protocols are pathways of bundled best-evidence perioperative care. In terms of fluid management, these protocols seek to maintain euvolemia and to avoid the detrimental effects of hypo- and hypervolemia.

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37 Water, Electrolyte, and Acid-Base Disturbances§

Claudia R. Fernandez Robles • Shamsuddin Akhtar

Patients undergoing surgery, especially those with critical conditions, can present with significant changes in water, electrolytes, and acid-base disturbances. Changes in either water homeostasis or electrolyte balance can lead to hemodynamic changes, arrhythmias, and neuromuscular dysfunction. Proper diagnosis and treatment of these disturbances are important in managing patients in the perioperative period.

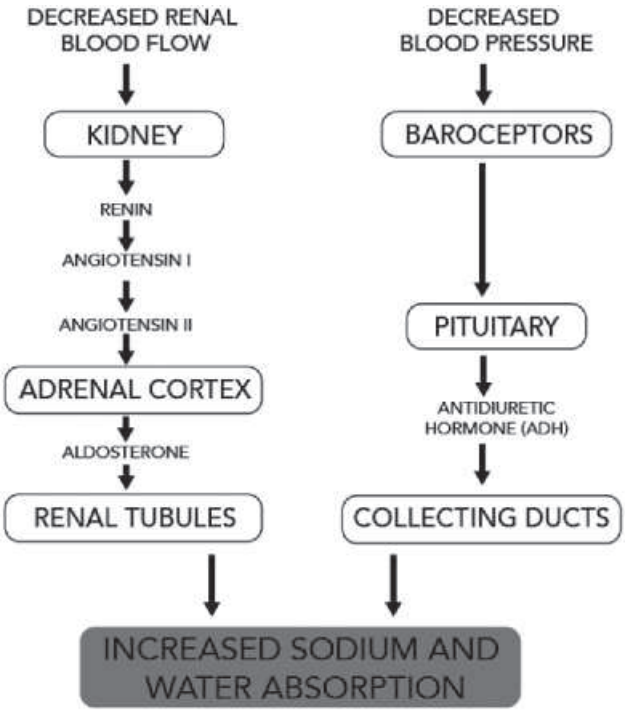
1) **Overview of water, electrolyte, and acid-base disturbances**
   a) **Incidence/prevalence**
      i) Preoperative electrolyte disturbances are not common in healthy patients; the statistics are different for the critically ill patients.
         (1) Hyponatremia is present in 30%-40% of the critically ill patient[1] and about 4%-7% on the ambulatory patients.[2]
            (a) Preoperative hyponatremia is a predictor of increase morbidity and mortality.[3–5]
         (2) Acid-base disturbances are documented with an incidence of as high as 56% in critically ill patients.
   b) **Clinical implications**
      i) Water, electrolyte, and acid-base disturbances affect the overall cellular milieu; they can have major influence on normal physiologic function when left untreated.
      ii) Arrhythmias
         (1) Primary electrolyte and pH abnormalities can lead to cardiac conduction abnormalities and arrhythmias, which can eventually cause hemodynamic collapse.
         (2) Intravascular volume abnormalities can further contribute to electrolyte and pH abnormalities.
            (a) Distention of the atrial wall is a common cause of arrhythmias.
      iii) Hemodynamic changes
         (1) Electrolyte disturbance, if severe, can lead to hemodynamic instability.
         (2) A pH < 7.2 leads to ineffective adrenergic receptor activity and results in a suboptimal environment for catecholamine action.[6]
      iv) Impact on enzymatic function
         (1) Most enzymes require a narrow pH environment for optimal functional activity. For example:
            (a) pH changes affect the oxygen-hemoglobin dissociation curve (Bohr effect).
               (i) Creates decreased peripheral dissociation and availability of oxygen in extreme alkalosis
      v) Impact on work of breathing
         (1) Severe hypocalcemia, hyper- and hypokalemia, and hypophosphatemia can lead to respiratory muscle weakness and paralysis.
         (2) Pulmonary edema from fluid overload can lead to increased *V/Q* mismatch, shunt, and hypoxemia.



Hypocalcemia, hyper- and hypokalemia, and hypophosphatemia can lead to respiratory muscle weakness and paralysis.

2) **Water disturbances**
   a) Total body water (TBW)
      i) Normal values: 80% in premature infants, 70%-75% in term infants, 65%-70% in toddlers, and around 60% in adults.
      ii) The intravascular volume and the TBW do not demonstrate a consistent relationship.
      iii) Some disease, such as cirrhosis and anasarca, may result in an increase in the TBW, with a decrease intravascular volume.
   b) **Hypervolemia**
      i) Hypervolemia with *increased* intravascular volume
         (1) Causes
            (a) Congestive heart failure
            (b) Renal failure
            (c) Primary hyperaldosteronism
      ii) "Hypervolemia" with *decreased* intravascular volume
         (1) Capillary leak
            (a) Sepsis, anaphylaxis, burn injury
         (2) Hypoalbuminemia
            (a) Decreased production
               (i) Malnutrition, cirrhosis
            (b) Increased loss
               (i) Nephrotic syndrome, enteropathy, exudative ascites
      iii) Mechanisms
         (1) Drop in blood pressure leads to suppression of carotid baroreceptors, which causes the release of antidiuretic hormone (ADH) from the pituitary. ADH acts on collecting ducts to increase tubular resorption of water.
         (2) Activation of tubuloglomerular apparatus activates the renin-angiotensin system to increase water/sodium resorption as well as increased vasomotor tone (see **Fig. 37-1**).
      iv) Signs
         (1) Peripheral edema
         (2) Pulmonary edema
            (a) Hypoxia
         (3) Congestive hepatopathy
            (a) Increased liver enzymes (LFTs), coagulopathy
         (4) Hypertension
      v) Treatment
         (1) Treat the etiology of the water retention.
         (2) Intravascular hypervolemia.
            (a) Diuretics
            (b) Preload reduction
            (c) Aldosterone antagonists (spironolactone and eplerenone)
   c) **Hypovolemia**
      i) Can be associated with both hyper- and hyponatremia.
      ii) Causes.
         (1) Gastrointestinal: diarrhea, emesis
         (2) Renal losses
         (3) Hemorrhage
         (4) Insensible water losses
      iii) If the volume loss (from water loss alone or water combined with sodium loss) is not replaced, then hypernatremia is the result.
         (1) If there is access to continued water ingestion, then stimulation of ADH release (see **Fig. 37-1**) leads to water retention and can result in hyponatremia.

FIGURE 37-1 Effect of Hypovolemia and Hypotension on Renin-Angiotensin-Aldosterone System, Resetting Increased Sodium and Water Absorption



iv) Preoperative assessment.
(1) Dry mucosa
(2) Peripheral vasoconstriction—delayed capillary refill
(3) Increased sympathetic activity to increase cardiac output—tachycardia and increased cardiac contractility and increased systemic vascular resistance (SVR)
(4) Hypotension
(a) Late sign, due to initial compensatory mechanism
(5) Electrolyte and acid-base derangements can accompany hypovolemia.
v) Treatment.
(1) Preoperative optimization with intravenous hydration to improve blood pressure and heart rate.
(2) Continue intraoperative volume repletion with isotonic fluids.

3) **Electrolyte disturbances**
a) **Sodium**
i) Hypernatremia (Na > 145 mEq/L)[7,8]
(1) Etiology
(a) Hypovolemic hypernatremia: diuretics, gastrointestinal losses, head injury
(b) Euvolemic hypernatremia: diabetes insipidus, fever, medications (eg, lithium, phenytoin, ethanol, hypertonic saline)



Consider preoperative optimization of the hypovolemic patient with hydration using isotonic fluids.

(c) Hypervolemic hypernatremia: iatrogenic, Cushing syndrome, hyperaldosteronism

(2) Signs and symptoms

(a) Usually asymptomatic, unless Na > 160 mEq/L

b) Anorexia, nausea, vomiting, altered mental status, weakness, seizures, coma

(3) Treatment

(a) Treat the cause.

(i) Hydration. The free water deficit can be calculated.

(ii) Free water deficit (in L) = 0.6 × weight (kg) × ([current Na/140] − 1)

(b) If the patient has lost both water and solutes (eg, sodium), then intravenous (iv) or po administration of an isotonic solution is the best plan.

(c) Reduce Na levels at a rate no more than 1 mmol/h or 10 mmol/d.

ii) Hyponatremia. Na < 135 mEq/L[8]

(1) Etiology

(a) Hypovolemic hyponatremia: cerebral salt wasting syndrome, diuretics, gastrointestinal loss, mineralocorticoid deficiency, osmotic diuresis, third spacing

(b) Euvolemic hypernatremia: glucocorticoid deficiency, low sodium intake, syndrome of inappropriate antidiuretic hormone hypersecretion (SIADH), water intoxication

(c) Hypervolemic hyponatremia: heart failure, hepatic failure/cirrhosis, nephrotic syndrome, renal failure

(d) Pseudohyponatremia[9]

(i) Hyponatremia with isotonic osmolality

(ii) Common causes: hyperglycemia, multiple myeloma, or macroglobulinemia



Hyponatremia should be corrected at a rate no more than 1 mmol/h to maximum of 6-8 mmol/d.

(2) Signs and symptoms

(a) Acute hyponatremia can lead to brain edema and herniation.

(3) Treatment[10]

(a) Treat the cause.

(b) Fluid restriction if hypervolemia is present.

(c) Hypertonic fluids if acute severe hyponatremia. Correction of 4-6 mEq/L is sufficient to reverse impeding herniation.

(d) Increase Na levels at a rate no more than 1 mmol/h to maximum of 6-8 mmol/24 hours.

(e) Rapid correction of hyponatremia can lead to the osmotic demyelination syndrome (ODS; formerly called central pontine myelinolysis or CPM).



Rapid correction of hyponatremia can lead to the osmotic demyelination syndrome (ODS)

b) **Potassium**

i) Hyperkalemia. K > 5.5 mEq/L.[11,12]

(1) Etiology

(a) Kidney disease

(b) Endocrine disorders: diabetes mellitus, adrenal insufficiency, hypoaldosteronism

(c) Metabolic acidosis

(d) Massive blood transfusion

(e) Tissue injury: rhabdomyolysis, burn injury, trauma

(f) Medications: inhibitors of renin-angiotensin-aldosterone system, spironolactone, NSAIDs, and succinylcholine

(i) Succinylcholine increases K levels by 0.5 mEq/L. It can result in hyperkalemia in patients with spinal cord injuries, burn, and prolonged immobilization.

(2) Signs and symptoms
  (a) Very few signs or symptoms until plasma potassium is >6.5 mEq/L,[13] unless rise has been rapid
  (b) Muscle weakness leading to flaccid paralysis
    (i) Including respiratory muscles
  (c) ECG conduction abnormalities
    (i) Peaked T waves and shortened QT interval, with lengthening of PR and QRS intervals
    (ii) Eventual loss of P waves and QRS widening, leading to "sine wave"
    (iii) Ventricular arrhythmias
(3) Treatment[13,14]
  (a) Above 6.0 mEq/L. Three main steps:
    (i) Membrane stabilization: intravenous calcium gluconate or calcium chloride
    (ii) Decrease serum potassium:
      (1) Insulin (10 units accompanied with 50 mL of Dextrose 50% intravenously. Close monitoring of glucose required)
      (2) Beta-agonist: albuterol inhalers
      (3) Sodium bicarbonate: only if acidemia is present
    (iii) Remove potassium: hemodialysis,
  (b) Chronic hyperkalemia management
    (i) Diuretics: loop diuretics or thiazide diuretics
    (ii) Cation exchange resin: Kayexalate.
    (iii) Decrease po intake of potassium



A serum potassium above 6.5 mEq/L is life threatening and requires immediate treatment.

ii) Hypokalemia. K < 3.6 mEq/L[15]
(1) Etiology
  (a) Decreased total body stores.
    (i) Gastrointestinal loss: emesis, gastric tube drainage, diarrhea, laxative, and VIPoma
    (ii) Renal losses: associated with metabolic alkalosis
    (iii) Poor intake
  (b) Hyperaldosteronism.
  (c) Hypomagnesemia is associated with increased potassium excretion.
  (d) Medications: beta-adrenergic agonists, xanthines, diuretics, mineralocorticoids, penicillins, amphotericin B, and cisplatin.
(2) Signs and symptoms
  (a) K 3.0-3.5 mEq/L: often no symptoms
  (b) K < 3.0 mEq/L: common symptoms include muscle weakness, cramps, paresthesias, and premature ventricular contractions
  (c) K < 2.5 mEq/L: rhabdomyolysis, A-V block, ventricular tachycardia, or ventricular fibrillation
(3) Treatment[15,16]
  (a) Identify the cause.
  (b) Replenish the potassium deficit.
    (i) 10 mEq replacement leads to approximately an increase in plasma potassium of 0.1 mEq/L.
    (ii) po and iv formulations
      1. 40-100 mEq/d of potassium chloride will maintain K levels in patients taking diuretics.
      2. IV potassium is irritating to veins, and boluses can lead to lethal arrhythmias; therefore the maximal iv replacement rate is 20 mEq/h through a central line in a monitored setting
  (c) Replete magnesium deficit.



Intravenous potassium administration can irritate veins and cause lethal arrhythmias. Administer through a central line no faster than 20 mEq/h.

c) **Phosphate**

i) Hyperphosphatemia[17,18]

(1) Causes

(a) Endogenous

(i) Tumor lysis syndrome

(ii) Rhabdomyolysis

(iii) Massive hemolysis

(iv) Lactic acidosis

(v) Diabetic ketoacidosis

(b) Exogenous administration

(i) Laxatives

(ii) Vitamin D toxicity

(c) Decreased excretion of phosphate from renal failure

(d) Increased renal tubular absorption of phosphate

(i) Hypoparathyroidism

(ii) Acromegaly

(iii) Bisphosphonates

(iv) Familial humoral calcinosis (rare genetic disorder)

(e) Pseudohyperphosphatemia

(i) Hyperglobulinemia

(ii) Hyperlipidemia

(iii) Hyperbilirubinemia

(2) Signs and symptoms

(a) Accelerates vascular calcification

(b) Can lead to cardiac arrhythmias secondary to hypocalcemia

(3) Treatment

(a) If mild and chronic, can be treated with phosphate binders (sevelamer, lanthanum, magnesium).

(b) If moderate, intravenous fluid administration with the goal of increasing renal excretion of phosphate.

(c) If severe, hemodialysis may be required.



Hyperphosphatemia can lead to acute hypocalcemia, leading (in severe cases) to arrhythmia, seizures, tetany, and death.

ii) Hypophosphatemia: phosphorus <2.5 mg/dL[19]

(1) Etiology

(a) Intracellular shift

(i) Increased insulin secretion

(ii) Acute respiratory alkalosis

(iii) Hungry bone syndrome

(iv) Refeeding syndrome

(v) Medications: insulin, adrenaline, salbutamol, intravenous iron repletion

(b) Decreased intestinal absorption

(i) Decreased phosphate intake

(ii) Antacids

(iii) Steatorrhea and chronic diarrhea

(iv) Malabsorption

(c) Increased urinary secretion

(i) Hypercalcemia

(ii) Fanconi syndrome

(iii) Hyperparathyroidism (1 and 2 degrees)

(2) Signs and Symptoms

Decreased oxygen release from hemoglobin because of decreased intracellular levels of 2,3-diphosphoglycerate (DPG) and a shift of the oxygen-hemoglobin dissociation curve to the left and decrease ATP.

(a) Encephalopathy

(b) Respiratory and skeletal muscle weakness
(c) Decrease cardiac contractility
(d) Arrhythmias
(e) Smooth muscle weakness manifesting in ileus
(f) Hypercalciuria
(g) Red blood cell hemolysis
(h) Abnormal platelet function
(i) Decrease leucocyte function
(j) Insulin resistance

(3) Treatment
(a) Calculate the urinary fractional excretion of phosphate.
(i) If higher than 5% in setting of hypophosphatemia, then renal wasting is the cause, and replacement is needed.
1. Intravenous replacement only for phosphorus level <1 mg/dL or severe symptoms.
a. Can precipitate with calcium and cause lethal hypocalcemia
2. Nonsymptomatic patients should be corrected with oral phosphate replacement (2.5-3.5 g or 80-110 mmol/d).
(b) If other causes are present, usually treatment of the etiology will correct the deficit.
(c) Consider replacement of vitamin D.

d) **Calcium**

i) Hypercalcemia[20,21]

(1) Etiology
(a) Primary and secondary hyperparathyroidism
(b) Malignancy-related
(c) Vitamin D intoxication
(d) Increased bone turnover
(i) Hyperthyroidism, immobilization, thiazides, vitamin A intoxication
(e) Renal failure
(f) Aluminum intoxication, milk-alkali syndrome
(g) Sarcoidosis
(h) Lithium
(i) Familial hypocalciuric hypercalcemia

(2) Signs and symptoms[21]
(a) Neuromuscular: generalized weakness, fatigue, mild depression, confusion.
(b) Renal: nephrolithiasis, polyuria, renal insufficiency
(c) Cardiovascular: shortened QT interval, prolonged PR, arrhythmias, cardiac arrest
(d) Gastrointestinal: anorexia, constipation, nausea, vomiting
(e) Other: bone demineralization, arthralgia, bone pain, pruritus

(3) Treatment
(a) Treat the cause.
(b) If emergent, intravenous hydration with normal saline.
(c) Consider iv furosemide (once hydration is adequate).
(d) Follow other electrolytes closely during this process.

(4) Other treatments
(a) Bisphosphonates
(b) Calcitonin
(c) Glucocorticoids, in dosages of 40-100 mg qd in four divided doses
(i) Increase urinary calcium excretion and decrease intestinal calcium absorption.
(ii) Effective antitumor agent in certain malignancies.

(d) Dialysis
(e) Gallium nitrate
(i) Inhibits bone resorption
(ii) Nephrotoxin
(f) Mithramycin
(g) po and iv phosphates
(h) Parathyroid surgery for primary hyperparathyroidism



Approximately 50% of total serum calcium is protein bound; the remaining is ionized, which is the biological active form.

ii) Hypocalcemia. iCa < 2.12 mmol/L
(1) Etiology
(a) Acute hypocalcemia
(i) Transfusions: sequelae of chelation by citrate in banked blood product
(b) Critical illness: sepsis, burns, acute renal failures, pancreatitis
(c) Hypoalbuminemia
(i) Decreased binding to albumin reduces total calcium concentration (normal *ionized* calcium concentration).
(d) Metabolic alkalosis
(i) Increases calcium's binding to protein decreasing ionized calcium concentration
(e) Parathyroid disorders
(i) Hypoparathyroidism
(ii) Pseudoparathyroidism
(iii) Vitamin D deficiency
(iv) Hungry bone syndrome



Chvostek sign is ipsilateral facial twitching by tapping on facial nerve anterior to the ear.

(2) Signs and symptoms
(a) Central nervous system: numbness and circumoral paresthesia, confusion, and seizures
(b) Cardiovascular: hypotension, prolonged QT
(c) Musculoskeletal: weakness and fatigue, skeletal muscle spasm, laryngospasm
(i) Chvostek sign: tapping on facial nerve anterior to the ear results in ipsilateral facial muscle twitching.
(ii) Trousseau sign: inflating blood pressure cuff above systolic pressure for several minutes results in muscular contraction of the hand.



Trousseau sign is muscular contraction of the hand after inflating blood pressure cuff above systolic pressure for several minutes.

(3) Treatment
(a) If hypoparathyroidism
(i) Vitamin D or calcitriol supplement
(ii) Calcalcium supplement
(b) Hypomagnesemia
(i) Supplement po vs iv magnesium
(c) If critically low:
(i) Confirm hypocalcemia with an ionized calcium measurement.
(ii) Administered 500-1000 mg of calcium chloride or gluconate, and repeat as needed.
(iii) Check phosphorus and magnesium levels and treat as needed.



Avoid hyperventilation in the setting of hypocalcemia as respiratory alkalosis can increase calcium binding to serum proteins decreasing ionized calcium (which is the active form).

4) **Acid-base disturbances**
   a) **Arterial blood gas can be used to assess**
      i) Acid-base disturbances and causes
      ii) Adequacy of ventilation
      iii) Adequacy of oxygenation
   b) **Assessing acid-base disturbances (see Fig. 37-2)**
      i) Step 1: Assess pH
         (1) Normal: pH 7.35-7.45
         (2) Acidemic: pH < 7.35
         (3) Alkalemic: pH > 7.45
      ii) Step 2: Assess $PaCO_2$
         (1) Normal 35-45 mm Hg
         (2) Primary respiratory acidosis >45 mm Hg
         (3) Primary respiratory alkalosis <35 mm Hg
      iii) Step 3: Assess HCO_3
         (1) Normal 22-26 mmol/L
         (2) Primary metabolic alkalosis >26 mmol/L
         (3) Primary metabolic acidosis <22 mmol/L
      iv) Step 4: Calculate serum anion gap (SAG) to help determine cause
         (1) $SAG=Na^+ - (Cl^- + HCO_3)$
         (2) High anion gap acidosis >12 mEq/L (see **Table 37-1**)
         (3) Normal anion gap acidosis (see **Table 37-1**)
      v) Step 5: Identify type of compensation if present (see **Fig. 37-1 and Table 37-2**)
      vi) Step 6: Identify a mixed acid-base condition if present (see **Table 37-3**)
   c) **Assessing adequacy of ventilation**
      i) Bohr dead space equation to assess adequacy of ventilation
         (1) $(P_ACO_2 - ETCO_2)/P_ACO_2$
            (a) Normal <0.3
            (b) Elevated >0.3



Avoid thiazide diuretics in the setting of hypercalcemia: they enhance renal tubular resorption of calcium.

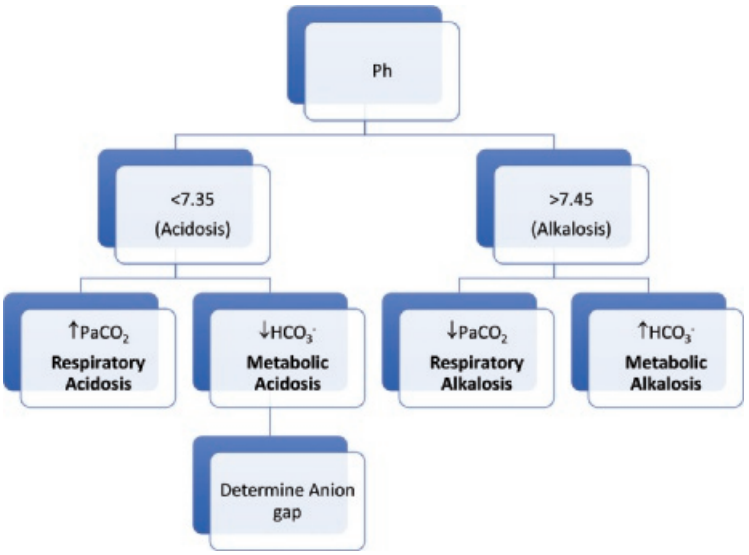


Causes of anion gap acidosis are "CUTE DIMPLES" and non–gap acidosis are "FUSED CARS."

TABLE 37-1 Metabolic Acidosis Differential Diagnosis

High Anion Gap Acidosis > 12 mEq/L (CUTE DIMPLES)	Normal Anion Gap Acidosis 8-12 mEq/L (FUSED CARS)
Cyanide	Fistula-pancreatic
Uremia	Uretogastric conduits
Toluene	Saline administration
Ethylene glycol	Endocrine disorders—*hyperparathyroidism*
Diabetic ketoacidosis	Diarrhea
Isoniazid	Carbonic anhydrase inhibitors—*acetazolamide*
Methanol	Ammonium chloride
Paraldehyde/propylene glycol	Renal tubular acidosis
Lactic acidosis	Spironolactone
Salicylates	

FIGURE 37-2 Stepwise Approach to Determine Acid-Base Disorder



(2) Differential diagnosis due to decreased perfusion
  (a) Hypotension
  (b) Low cardiac output
  (c) Pulmonary embolus
  (d) High PEEP
  (e) Pulmonary blebs or cysts

d) **Assessing adequacy of oxygenation**
  i) P/F ratio
    (1) Ratio of PaO_2 mm Hg: FiO_2
    (2) The lower the ratio, the worse the oxygenation
      (a) Acute respiratory distress syndrome (ARDS)[22]
        (i) Mild ($PaO_2/FiO_2 \leq 300$ mm Hg)
        (ii) Moderate ($PaO_2/FiO_2 \leq 200$ mm Hg)
        (iii) Severe ($PaO_2/FiO_2 \leq 100$ mm Hg)

TABLE 37-2 Calculate Expected Compensation Based on $PaCO_2$

Calculated $PaCO_2$ in Respiratory Conditions	Calculated $PaCO_2$ in Metabolic Conditions
$PaCO_2$ *increased* by 10 mm Hg • **Acute respiratory acidosis** will decrease pH by 0.08. • **Chronic respiratory acidosis** will decrease pH by 0.03.	Metabolic acidosis $PaCO_2 = 1.5 \times HCO_3 + 8 \pm 2$
$PaCO_2$ *decreased* by 10 mm Hg • **Acute respiratory alkalosis** will increase pH by 0.08. • **Chronic respiratory alkalosis** will increase pH by 0.03.	Metabolic acidosis $PaCO_2 = 0.7 \times HCO_3 + 20 \pm 1.5$

TABLE 37-3 Determine If Mixed Acid-Base Disorder Is Present

Identify if a Mixed Acid-Base Condition Is Present
Step1: Calculate delta gap: • Measured serum anion gap (SAG) – normal SAG
Step 2: Calculate delta-delta: • Add the delta gap to the measured bicarbonate from the basic metabolic panel taken at the same time the arterial blood gas was analyzed.
Step 3: Compare delta-delta to normal HCO_3 • If delta-delta <22, there is a non–anion gap metabolic acidosis. • If delta-delta >26, there is an additional metabolic alkalosis.

(3) A-a gradient
(a) Calculating P_AO_2 (alveolar)
(i) $PAO_2 = (P_B - 47)(FiO_2) - (P_aCO_2/R)$
(b) A-a gradient
(i) $P_AO_2 - P_aO_2$ (arterial oxygen measured from blood gas)
(ii) Large gradient due to shunt or dead space
(c) Response to supplemental oxygen
(i) Dead space: P_aO_2 will improve with supplemental O_2.
(ii) Shunt: P_aO_2 will NOT improve with supplemental O_2 once the shunt fraction is >50%.
1. Atelectasis
2. Pulmonary edema
3. Endobronchial intubation
4. Aspiration
5. Airway obstruction

e) **Metabolic alkalosis**[23]
i) Causes
(1) Renal: diuretics, Bartter syndrome, Gitelman syndrome
(2) Nonrenal: nausea, vomiting, suction, gastroenteritis, hyperaldosteronism
(3) Other: alkali intake, milk-alkali syndrome, severe potassium depletion

f) **Metabolic acidosis**
i) Decide if there is an anion gap present
(1) Anion gap = [sodium] – [chloride + bicarbonate].
(2) If >12, then gap acidosis is present.
(a) Adjust for hypoalbuminemia
(i) Add 2 to the calculated gap for every decrease in plasma albumin level of 1 g/dL below normal.
(ii) Albumin behaves as an unmeasured anion.

g) **Causes**
i) Anion Gap: **"CUTE DIMPLES"**
(1) Cyanide
(2) Uremia
(3) Toluene
(4) Ethylene glycol
(5) Diabetic ketoacidosis
(6) Isoniazid
(7) Methanol

(8) Propylene glycol
(9) Lactic acidosis
(10) Salicylates

ii) Nongap acidosis: "FUSED CARS"
(1) Fistula-pancreatic
(2) Uretogastric conduits
(3) Saline administration
(4) Endocrine disorders—*hyperparathyroidism*
(5) Diarrhea
(6) Carbonic anhydrase inhibitors—*acetazolamide*
(7) Ammonium chloride
(8) Renal tubular acidosis
(9) Spironolactone
(10) Other causes
(a) Hyperalimentation (eg, TPN)
(b) Hyperaldosteronism
(c) Hyperchloremic acidosis from liberal administration of intravenous 0.9% sodium chloride administration

h) **Treatment**

i) Respiratory acidosis and lactic acidosis.
(1) Treat the cause.

ii) If the pH is <7.2, then rapid correction may be necessary to avoid impending hemodynamic collapse.

iii) Traditionally, sodium bicarbonate has been utilized.
(1) 2 mmol/kg of iv sodium bicarbonate, administered over 15 minutes, can increase the pH by 0.14.
(2) However, clinical studies demonstrate no improvement in outcome from such a strategy.[24]
(3) In fact, multiple studies demonstrate *worsened* intracellular acidosis in situations of lactic acidosis from hypoperfusion, because of the lack of removal of the resulting carbon dioxide if the patient is not ventilated adequately.

iv) In many ways, a better choice for acidosis treatment is THAM.
(1) THAM
(a) Tris–hydroxymethyl aminomethane
(b) Buffer compound that chelates protons and is then cleared via renal secretion
(2) The amount of THAM (0.3 M solution) needed to correct acidosis, in mL units, can be calculated:
(a) Multiply the patient's body weight (kg) by the base deficit (mmol/L)
(3) No benefit to utilizing sodium bicarbonate in treatment of respiratory acidosis
(a) Hypoventilation
(i) The cause of respiratory acidosis
(b) The inability to clear carbon dioxide, which is the way to clear carbonic acid (the resulting compound when sodium bicarbonate combines with H^+ ions) from the circulation
(i) Leads to lactic acidosis

v) Renal etiology acidosis
(1) Consider treatment with sodium bicarbonate infusion.

CHAPTER SUMMARY FOR WATER, ELECTROLYTES, AND ACID-BASE DISORDERS

Water Disturbances	**TBW** is 55%-60% of an adult total body mass			
	Hypervolemia can occur in the setting of increased or decreased intravascular volume. Treat the cause of the water retention.			
	Hypovolemia can be associated with both hyper- and hyponatremia. Hydrate with iso-osmolar fluids.			
Electrolyte Disturbances				
Disturbance	**Diagnostic Information**	**Physiologic Effects**	**Anesthetic Implications and Concerns**	**Treatment**
↑ Na^+	>145 mEq	Mental status Δ's	Mental status Δ's	Replace free H_2O deficit
↓ Na^+	<135	Mental status Δ's		iv NS or ½ NS
↑ K^+	>5.5	Myocardial depolarization changes	Arrhythmias	Kayexalate or dialysis
↓ K^+	<3.5	Myocardial depolarization changes	QT prolongation and torsades de pointes	iv or po potassium
↑	>4.5 mg/dL	Hypocalcemia	Hemodynamic collapse	iv hydration; po binding agents; dialysis
↓	<2.5 mg/dL	Muscle weakness; encephalopathy	Respiratory failure	po phosphate
Acid-Base Disorders	Obtain arterial blood gas sample to evaluate. a) pH > 7.4, then alkalosis b) pH < 7.4, then acidosis **Respiratory acidosis:** PCO_2 of 10 mm Hg decreases pH by 0.08. **Metabolic acidosis:** If base deficit is a negative number, there is a component of metabolic acidosis. Gap acidosis: "CUTE DIMPLES" Non-gap acidosis "FUSED CARS"			

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38 Blood Component Therapy

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Administration of blood components is often necessary in the perioperative period. Blood component therapy should be used to treat specific indications, bearing in mind the potential complications associated with transfusion. Vigilance is absolutely critical as complications are most often due to human error.

1) **Pretransfusion testing**
   a) When preparing to transfuse blood, diagnostic testing is required in order to prepare crossmatch-compatible blood.
      i) A patient blood sample should be sent to the blood bank preoperatively.
   b) **Type and screen**
      i) Determines the ABO type, presence or absence of the Rhesus (Rh) D antigen on the patient's RBCs, and screens the patient's plasma common clinically significant anti–red blood cell (RBC) antibodies.
      ii) ABO type
         (1) Determined by cell surface glycoprotein antigens present on the RBC surface.
         (2) Patients may be type A, B, AB, or O.
         (3) Type O patients lack type A and B antigens.
      iii) RhD antigen
         (1) 85% of patients express the RhD antigen from the Rh group and are commonly referred to as "Rh positive."
         (2) Patients who are RhD negative are at risk of alloimmunization (synthesis of antibodies against foreign antigens) when exposed to the RhD antigen.
      iv) Antibody screen
         (1) Commercially available RBCs are mixed with the patient's plasma in order to detect anti-RBC alloantibodies with the potential to cause hemolysis.
         (2) If positive, RBCs in "panels" are mixed with the patient's plasma in order to identify antibody specificity.
            (a) If the antibody recognizes a rare antigen, identification can require testing with several RBC panels and take an extended period of time.
   c) **Type and crossmatch**
      i) Crossmatching the blood involves mimicking the transfusion by mixing the donor's RBC with the recipient's blood and assessing for potential antibody and RBC-antigen reactivity causing binding or hemolysis.
         (1) Ensures compatibility of donor and recipient blood samples
   d) **Time to obtain blood**
      i) Type and screen can usually be performed within 45 minutes and the crossmatch within an additional 15 minutes if the antibody screen is negative.
      ii) If antibodies are present, these steps can take much longer, especially if the antibodies are uncommon.
   e) **Autologous blood donation**
      i) If time permits, patients may donate their own blood preoperatively.

ii) Patients who elect autologous blood donation may become anemic and require iron and erythropoietic agents.
iii) Autologous blood should be meticulously handled as human errors still occur with its administration.


If a patient requires emergent blood transfusion, type O, RhD-negative pRBCs and AB plasma can be given.

f) **Directed donor blood donation**
i) A family member or friend may be designated by the patient to donate blood, which may be used if needed, provided compatibility can be assured.
ii) This technique does not decrease the incidence of transfusion-transmitted infection as the patient may not be aware of the donor's infection status.[1]

2) **Packed red blood cells (pRBCs)**
a) The purpose of packed RBC (pRBC) transfusion is to increase the O_2-carrying capacity of the blood.
b) The decision whether to transfuse pRBC is multifactorial. The patient's coexisting disease and risk of ongoing blood loss should be considered.
i) For example, a patient with coronary artery disease may require transfusion at a higher hemoglobin value due to the need to maintain myocardial oxygen delivery.[2]
c) **pRBC transfusion parameters**
i) There is no absolute laboratory value that necessitates blood transfusion.
ii) Transfusion is rarely indicated for hemoglobin concentrations >10 g/dL.[3]
iii) Transfusion is usually indicated for hemoglobin concentrations <6 g/dL.[3]
iv) Within the range of 6-10 g/dL, the risk of ongoing blood loss, hemodynamic status, and patient's coexisting disease must be considered.


The decision to transfuse pRBC depends on the potential for ongoing blood loss and the patient's coexisting medical conditions.

d) In the absence of continued blood loss, one unit of pRBCs should increase the hemoglobin concentration by 1 g/dL and the hematocrit by 3%.
e) **ABO compatibility**
i) ABO blood type is determined by the presence or absence of A and/or B antigens on the surface of the RBC.
(1) Patients who lack both antigens are type O.
(2) Patients have circulating antibodies to ABO antigens that they lack.
a) For example, type O patients have circulating anti-A and anti-B antigens and type A patients have anti-B antibodies.
(3) ABO-compatible transfusion is essential. This is because ABO incompatibility leads to immediate and potentially life-threatening hemolysis.
a) Type O patients are universal RBC donors, and type AB patients are universal RBC recipients.[4]
b) If type-specific blood is not available, type O, usually RhD negative, pRBC may be given.


Transfusion of the wrong blood type may lead to potentially fatal hemolytic transfusion reactions.

3) **Plasma (fresh frozen plasma [FFP])**
a) FFP contains all plasma proteins and clotting factors.
b) Stored at −18°C to −30°C and must be thawed prior to administration.
c) **Indications for FFP**
i) Correction of microvascular bleeding in patients with abnormal coagulation parameters (INR > 2.0, aPTT > 2 times normal).[1]

ii) Correction of known coagulation factor deficiencies when factor concentrates are unavailable.
iii) Antithrombin deficiency in patients receiving heparin
iv) Correction of microvascular bleeding in patients who have been transfused greater than one blood volume.
v) Rapid reversal of warfarin therapy if prothrombin complex concentrates are not available.

d) Recent studies in trauma patients who require massive transfusion have shown decreased morbidity and mortality with earlier administration of plasma (1:1:1 ratio).[5]
e) The goal of plasma administration is to achieve 30% of clotting factor concentration. The starting dose is 10-15 mL/kg.
f) **ABO compatibility**
i) Compatibility requirements are different than with pRBC because the *plasma* contains anti-A and/or anti-B antibodies in patients who lack these antigens.
ii) Plasma transfusions should be ABO compatible.
iii) For instance, a patient with type AB blood should receive only type AB plasma, but a patient with type O blood can receive types O, A, B, or AB plasma.[4]



In patients with bleeding, the surgical field should be scanned for inadequate surgical hemostasis, and the presence of microvascular bleeding indicative of coagulopathy should be discussed with the surgeon.

4) **Platelets**
a) Platelets are usually available as an individual unit collected via apheresis from a single donor. Alternatively, an equivalent number of platelets can be isolated and pooled from six separate whole blood donations.
b) Platelet units are stored at room temperature for 5 days, which increases the likelihood of bacterial contamination and decreases shelf life.
c) Transfusion of a single apheresis unit increases the platelet count by 30-60 000 cells/mm^3 in the absence of platelet destruction/consumption.[6]
d) **Indications**
i) Platelets are rarely indicated if a patient's platelet count exceeds 100 000 cells/mm^3.
ii) Platelet transfusion should be considered in the presence of excessive microvascular bleeding with platelet counts <50 000 cells/mm.[1,4]
iii) May also be necessary in the presence of platelet dysfunction (antiplatelet therapy, uremia, postcardiopulmonary bypass) and microvascular bleeding.
e) **ABO compatibility**
i) ABO-incompatible platelet units can generally be transfused safely in adults if titers of anti-A and anti-B antibodies in the accompanying plasma are confirmed to be low. However, as ABO antigens are also expressed on platelets, the platelet count increase after transfusion may be lower than after ABO-compatible transfusion.[7]



When transfusing blood products in women of child-bearing age, either give RhD-negative products or ensure RhD compatibility.[4]

5) **Cryoprecipitate**
a) Made from thawing FFP at 4°C.
b) Contains high levels of fibrinogen, von Willebrand factor, and Factor VIII
c) **Indications**
i) Treatment of microvascular bleeding in the presence of fibrinogen deficiency, which most commonly occurs due to disseminated intravascular coagulation (DIC) or massive transfusion.
ii) Treatment of congenital fibrinogen deficiencies or bleeding in patients with von Willebrand disease, where factor concentrates are unavailable.[3]
iii) Ideally, a fibrinogen level should be obtained before administering cryoprecipitate.

(1) Patients whose fibrinogen concentrations exceed 150 mg/dL usually do not require cryoprecipitate.
(2) When fibrinogen concentrations are <80-100 mg/dL, cryoprecipitate is usually indicated.[3]

d) **ABO compatibility**
i) Because cryoprecipitate has only a small amount of plasma, ABO compatibility is not necessary.[4]

6) **Complications of transfusion**

a) **Human error**
i) Due to recent advances in detecting potentially infectious agents, complications due to human error are significantly more common than transfusion-related infections.
ii) Catastrophic outcomes during blood product administration are usually associated with multiple errors during the process.[8,9]
iii) In order to prevent errors, it is critical that all persons involved in administering blood products exercise extreme caution, including verifying blood samples sent to the lab against a patient's armband and verifying blood prior to transfusion.



Vigilance is essential when transfusing blood products as clerical errors can result in potentially fatal ABO-incompatible transfusions. Patient identity and the correct blood product assignment should be confirmed as a critical safety check prior to initiating transfusion.

b) **Acute hemolytic reaction**
i) The most serious complication of blood product administration.
ii) Usually caused by ABO incompatibility, resulting in immune-mediated intravascular hemolysis due to recipient anti-RBC antibody interaction with donor erythrocytes.
iii) Signs/symptoms of acute hemolytic reaction:
(1) Anxiety, fever, chills/rigors, nausea, back and flank pain, hematuria, hypotension, DIC, hemoglobinemia, and acute renal failure.[6]
(2) Many symptoms are masked by general anesthesia.
iv) If you suspect an acute hemolytic reaction:
(1) Immediately stop the transfusion.
(2) Begin supportive care, which includes the following:
(a) BP support with pressors and inotropes as needed.
(b) Preservation of renal function with fluid administration and diuresis.
(c) Alkalinization of urine with sodium bicarbonate and administration of corticosteroids is of unclear benefit.[10]
(3) Obtain urine and plasma hemoglobin, repeat type and screen, coagulation parameters, blood counts, direct antiglobulin test LDH, and haptoglobin.[6]
(4) Send the blood component being infused to the blood bank with a sample of the patient's blood to confirm or rule out incompatibility.



Prior to transfusing blood products, always check the patient's armband. Verify that the unit is the correct blood type and intended for that patient.

READ MORE
Crisis management: Transfusion reaction, Chapter 156

c) **Delayed hemolytic reaction**
i) Caused by extravascular hemolysis of donor erythrocytes.
ii) Due to the presence of antibodies in recipient serum developed after previous transfusions or pregnancy at levels too low to be detected during the crossmatch.[6]
iii) Signs/symptoms of delayed transfusion reaction



If you suspect a transfusion reaction, immediately STOP the transfusion.

(1) Occur ~1 week after a seemingly compatible transfusion.
(2) Include anemia, mild fever, increased unconjugated bilirubin, jaundice, hemoglobinuria, decreased haptoglobin, and spherocytosis on the blood smear.[6]

iv) Because the hemolysis is extravascular, the reaction is much less severe than in an acute hemolytic reaction, and the symptoms are self-limited.[6]

d) **Transfusion-related acute lung injury (TRALI)**

i) An acute respiratory distress syndrome occurring within 2-6 hours after transfusion.[11-13]

ii) Characterized by noncardiogenic pulmonary edema manifesting as hypoxia and bilateral infiltrates on chest radiograph.[11-14]

iii) It's true incidence is unknown, because it is difficult to distinguish from other forms of acute lung injury, and it often occurs in patients with multiple coexisting illnesses.[13]

(1) TRALI is not rare; its estimated occurrence is 1 in 2000 to 1 in 5000 transfusions of blood or blood products.[13]

iv) TRALI is the leading cause of death from transfusions in the United States.[13]

v) Treatment of TRALI.

(1) Supportive care with most patients requiring mechanical ventilation. Small tidal volumes are recommended.
(2) Hypotension is generally responsive to IV fluid.
(3) Despite the presence of pulmonary edema, diuretic administration can worsen the patient's condition.[12]



When administering blood products, urine output and color and peak airway pressures should be periodically assessed for signs and symptoms of transfusion reaction.[1]

e) **Infectious risks**

i) Bacterial contamination

(1) Bacterial contamination of blood products is most common with platelets because platelet units are stored at room temperature.
a) Incidence of 1 in 12 000 platelet transfusions[15]
(2) The most frequent contaminating organism is *Yersinia enterocolitica* for pRBC and *Staphylococcus aureus* for platelets.
(3) The clinical presentation ranges from mild fever to acute sepsis leading to death.
(4) Suspect bacterial contamination and consider antibiotic therapy in patients who develop a fever within 6 hours after platelet transfusion.[15]

ii) Viral transmission

(1) The incidence of viral transmission has decreased substantially over the past 20 years.
(2) Currently, the estimated risk per unit of blood transfused of acquiring human immune deficiency virus is 1 in 1 780 000 and 1 in 1 600 000 for hepatitis C virus.[16]
(3) Other viral pathogens that can be transmitted via transfusions are cytomegalovirus, B19 parvovirus, human T-lymphotropic virus, West Nile virus, Zika virus, and hepatitis B virus. It is currently believed that SARS-CoV-2 cannot be transmitted via transfusion.

iii) Others

(1) Other potentially transmissible infectious diseases include malaria, babesia, variant Creutzfeldt-Jakob disease, and Chagas disease.[7]

CHAPTER SUMMARY FOR BLOOD COMPONENT THERAPY

Blood Component/ Contents	Indication/Guidelines	Complications/Warnings
pRBC (Red Blood Cells)	Increase O_2-carrying capacity Almost always indicated for Hgb < 6 Almost always not indicated with Hgb > 10 1 unit pRBC → ↑ HCT 3%, Hgb 1 g/dL	Warm blood ABO compatibility essential
Plasma (Coagulation Factors)	Treatment of microvascular bleeding in the setting of coagulopathy (INR >2.0; aPTT > two times normal) Reversal of specific agents such as warfarin, in absence of prothrombin complex concentrates For trauma, evidence supports earlier plasma administration	May require time to thaw ABO compatibility essential
Cryoprecipitate (Fibrinogen, von Willebrand Factor, Factor VIII)	Treatment of fibrinogen deficiency (usually <80-100 mg/dL) Commonly used in the setting of DIC	
Platelets	Treatment of severe thrombocytopenia in the setting of bleeding, surgery, or platelet destruction Usually not required with platelet counts >100 000 cells/mm^3 Usually given for platelets <50 000 cells/mm^3, especially in surgical patients	Single-donor apheresis unit most common Potential for bacterial contamination
Blood components should be given to treat specific indications and not for volume replacement.		
Extreme vigilance is required by all persons involved in blood product replacement to prevent errors.		

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39 Alternatives to Blood Product Replacement

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Transfusion of allogeneic blood products carries known risks and is associated with increased morbidity and mortality. Additionally, there are occasions where transfusion of blood products is not possible or practical due to extreme difficulty in obtaining blood or patient refusal. Alternative techniques may be employed to avoid transfusion, bearing in mind that these may only be temporizing measures. The inability to treat coagulopathy and/or increase oxygen carrying capacity when indicated may result in life-altering morbidity and/or death.

1) **Overview**
   a) **Risks of allogeneic blood product transfusion**[1]
      i) Transfusion-transmitted infections.
         (1) Although blood safety continues to improve, a transmission risk of known (HIV, HCV, HBV, bacterial) and emerging (West Nile virus, Zika virus) infectious agents remains.
      ii) Transfusion reactions
      iii) Intraoperative transfusion is associated with increased morbidity and mortality in surgical patients.[1,2]
   b) **Difficult crossmatch**
      i) Occasionally, the situation arises where obtaining crossmatched blood is difficult or nearly impossible due to the development of anti–red blood cell (RBC) antibodies (alloimmunization).
      ii) Patients at risk for alloimmunization.[3]
         (1) Alloimmunization in transfusion medicine refers to the development of antibodies to human donor cell antigens.
         (2) Patients who are at high risk are those who have had multiple blood transfusions or pregnancies.
   c) **Patient refusal of blood products**
      i) Informed consent
         (1) Patients should be informed about blood transfusion when appropriate and the consequences (including death) of blood product refusal.
         (2) A thorough discussion of alternatives to blood transfusion, including the limitations of each technique, should be undertaken.
         (3) Alternatives that are acceptable to the patient should be clearly documented.
2) **Maintenance of normovolemia**
   a) One of the primary goals of any anesthetic should be maintenance of normovolemia.
   b) IV crystalloid or colloid should be given as volume replacement, using fluid losses (blood loss, urine output, evaporative losses) and, if available, invasive monitoring (arterial waveform pulse pressure variation or transesophageal echocardiography) as a guide.



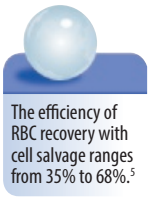
Transfusion of allogeneic blood carries risk of infectious disease transmission and is associated with increased morbidity and mortality in surgical patients.



Patients should always be informed that a possible consequence of blood product refusal is extreme anemia resulting in death.

READ MORE
Perioperative Fluid Management, Chapter 36

3) **Intraoperative cell salvage ("cell saver")**
   a) **Procedure**[4]
      i) Blood is suctioned from the operative field and collected in an anticoagulant solution.
      ii) RBCs are separated from cellular debris other contaminants via centrifugation.
      iii) Cells are then washed, suspended in saline, and reinfused to the patient.
         (1) Reinfusion is usually via bagged aliquots with a hematocrit of 50%-60%.



The efficiency of RBC recovery with cell salvage ranges from 35% to 68%.[5]

   b) **Indications for intraoperative cell salvage**[6]
      i) Procedures where blood loss is expected to be high
         (1) Those procedures in which estimated blood loss is >1000 mL, procedures for which 2 units of pRBC are routinely crossmatched, or procedures where 20% of patients are routinely transfused.
         (2) Common procedures include cardiovascular surgeries, liver transplantation, spinal instrumentation, joint arthroplasty, and aortic reconstruction.
      ii) Patients who do not accept allogeneic blood transfusion, such as Jehovah's Witnesses.
      iii) Patient with limited or no crossmatched blood availability.
   c) **Contraindications**[6]
      i) Absolute contraindications are few and related to risk of red cell lysis and end organ damage from administration of lysed RBCs.
         (1) Presence of hypotonic solutions such as sterile water or antibiotic irrigation or contamination with intravascularly toxic solutions including hydrogen peroxide, alcohol, or povidone-iodine.
         (2) Contamination with hemostatic agents (topical thrombin, fibrin glues, bovine-derived collagens) or bone cement (methyl methacrylate).
      ii) Relative contraindications include the presence of cells or other substances that have the potential to cause harm if reinfused to the patient. However, there are few data to support these restrictions, and the risks of cell salvage use must be considered against the benefits of avoiding allogeneic RBC transfusion. Some relative contraindications include the following:
         (1) Malignancy
            (a) Use of leukoreduction filtration significantly decreases tumor cell contamination, and cell salvage has successfully been used in oncologic surgery with no difference in outcomes.[7]
         (2) Infection
         (3) Urine, bone chips, fat, or bowel contents in surgical field
         (4) Hematologic disorders such as sickle cell disease and thalassemia
         (5) Presence of amniotic fluid
            (a) Cell salvage has been used for cesarean section without complications and may be considered in certain high-risk situations.[8]
      iii) Potential complications[3,8]
         (1) Coagulopathy from the anticoagulants used during blood processing
            (a) Washing steps incorporated in modern cell salvage equipment have substantially reduced this concern.
         (2) Dilution of clotting factors and platelets can occur since only RBCs are returned to the patient.
         (3) Contamination of the cell salvage contents with bacteria or debris from the surgical field can occur resulting in systemic infection.
         (4) Air embolism is possible if blood is directly returned from the machine or a rapid infusion device and not properly monitored.

iv) Advantages
(1) Compatibility testing is not required.
(2) Reduced risk for clerical errors related to transfusion, unless the machine is not properly operated.
v) Disadvantages
(1) Requires specialized equipment and trained personnel
(2) Not applicable in all surgeries

4) **Acute normovolemic hemodilution (ANH)**[3,9]
a) Involves collection of autologous whole blood immediately prior to surgery and maintenance of normovolemia with IV administration of colloid or crystalloid.
i) Blood should be collected in standard blood storage bags containing anticoagulant solution and the volume regulated using weight measurements to prevent overfilling.
ii) The volume of colloid administered should be equal to the volume of blood withdrawn.
iii) When crystalloid is administered, the volume should be three times the volume of blood removed.[6,9]
b) After ANH, when blood is lost, it has less RBC mass, thereby effectively decreasing operative blood loss. The autologous blood removed contains RBCs, coagulation factors, and platelets, which can be returned to the patient as needed.
c) **Candidates for this procedure include the following**[9]:
i) Patients who have a likelihood of transfusion >10%
ii) Those with preoperative Hgb of at least 12 g/dL
iii) Those with absence of coronary, pulmonary, renal, or liver disease
iv) Those with absence of severe hypertension
v) Those with absence of infection and low risk of bacteremia
d) **Advantages**
i) There is virtually no potential for ABO incompatibility provided that the blood does not leave the operating room.
ii) There is also no need for compatibility testing.
iii) The cost associated is lower than that of allogeneic, banked blood.
e) **Disadvantages**[9]
i) Decreased oxygen delivery may result in organ ischemia, although this is very unlikely in patients without cerebrovascular or coronary artery disease.

5) **Pharmacologic alternatives**
a) **Erythropoietic agents**[10,11]
i) Recombinant human erythropoietin (rhEPO) with iron, folate, and vitamin B12 supplementation can be used preoperatively or postoperatively to increase RBC production.
a) Intravenous iron instead of oral supplementation may be required in patients with anemia of chronic disease due to decreased GI iron absorption.
ii) Advantages
(1) May be used in patients at high risk for anemia and blood loss.
(a) Examples include chronic renal failure and anemia.
(2) Accepted by many patients who otherwise refuse blood transfusion.
iii) Disadvantages
(1) The onset of action of rhEPO is 4-6 days, therefore administration should occur several days prior to surgery.[12]
(a) Not helpful in the acute situation or when blood loss is unexpected.
(2) Use of erythropoietin is associated with increased risk of thrombotic cardiovascular complications; therefore, its use must take this risk into consideration.

b) **Recombinant activated factor VII (rFVIIa)**[13-15]
   i) rFVIIa is identical in structure and function to human factor VIIa.
   ii) Developed to prevent or control bleeding in patients with hemophilia A or B with inhibitors to factors VIII and IX, respectively.
   iii) rFVIIa acts in multiple pathways of the clotting cascade by forming complexes with tissue factor (extrinsic pathway) that then activates factor X (common pathway) as well as factor IX (intrinsic pathway).
   iv) FDA-approved indications for rFVIIa include treatment of bleeding in hemophilia patients with inhibitors or those with Glanzmann thrombasthenia. However, given its prothrombotic potency, it is now used in many different clinical situations, including treatment of life-threatening hemorrhage.
   v) There are no specific guidelines for rFVIIa therapy in the setting of acute hemorrhage, but it is often administered for continued life-threatening coagulopathy refractory to standard treatment.
      (1) The dose is 50-100 mg/kg IV, with most patients responding to the drug within 30 minutes.
         (a) It can be repeated every 2 hours until hemostasis is achieved, but the vast majority of patients require only one dose.
         (b) rFVIIa is less effective in the presence of acidosis, hypothermia, and inadequate clotting factors.[15]


rFVIIa may be beneficial in cases of intractable hemorrhage complicated by coagulopathy.

   vi) Advantages
      (1) rFVIIa is a pharmacologic agent and not a blood product.
      (2) Does not carry risk of transfusion-transmitted disease.
      (3) Accepted by many patients who decline allogeneic blood transfusion.
   vii) Disadvantages
      (1) Arterial and venous thrombotic events are the most commonly associated complications and include cerebrovascular accidents, myocardial infarction, pulmonary embolism, and clotting of indwelling devices.[16]
      (2) Each administration of rFVIIa carries significant cost, >$10 000 per vial.

c) **Fibrinogen concentrates**
   i) Fibrinogen concentrates are lyophilized powder preparations manufactured from pooled human plasma that are reconstituted with sterile water at the time of administration.
   ii) These concentrates are FDA approved for the treatment of acute bleeding episodes in patients with congenital fibrinogen deficiency, including afibrinogenemia and hypofibrinogenemia. However, they are beginning to be used off-label as an adjunct to cryoprecipitate for fibrinogen supplementation.
   iii) Hypofibrinogenemia can occur as a result of acute trauma, obstetric hemorrhage, and dilutional coagulopathy from resuscitation after large volume blood loss. Treatment with fibrinogen supplementation is important to decrease ongoing blood loss and decrease transfusion requirements.[17-19]
   iv) Advantages
      (1) Decreased risk of transfusion-transmitted disease as preparations undergo pasteurization or solvent-detergent treatment.
      (2) Uniform dose of fibrinogen can be administered in a small volume (50 mL)
      (3) Available for immediate use. Preparations are stable at room temperature and do not require determination of a patient's blood type prior to administration.
   v) Disadvantages
      (1) Fibrinogen concentrates are more costly relative to equivalent amounts of cryoprecipitate (~$740/g fibrinogen).[20]

6) **Management strategies for patients who decline blood transfusion**[1]
   a) **Minimize blood loss**
      i) Meticulous surgical technique should be discussed with the physician performing the procedure.
      ii) Minimizing phlebotomy and using point-of-care laboratory testing can significantly reduce blood loss.
   b) **Minimize O_2 consumption**
      i) Mechanical ventilation with or without muscle paralysis reduces the work of breathing.[21]
      ii) Other strategies include hypothermia, analgesics, and sedation.[10,21]
   c) **Maximize O_2 delivery**
      i) Tissue O_2 delivery can be increased by increasing inspired oxygen (FiO_2), maintaining normovolemia, and increasing cardiac output, using inotropes if necessary.[10]
         (1) A pulmonary artery catheter with the capability to measure cardiac output and mixed venous SpO_2 can be helpful.[10]
      ii) **Hyperbaric O_2** can also be used to increase blood O_2 content if available.[22]



One of the most common perioperative situations where patients refuse blood products is the care of the Jehovah's Witness.

7) **The Jehovah's Witness (JW)**
   a) **Definition**
      i) JWs are patients who object to receiving blood or blood products on religious grounds.
      ii) There are over one million JWs in the United States and over six million worldwide.[23]
   b) **Preoperative informed consent**
      i) Whenever possible, discuss patient's wishes alone (ie, without the presence of family or religious personnel).
      ii) Patients should be informed about blood transfusion when appropriate and the consequences (including death) of blood product refusal.
      iii) Some patients will accept intraoperative cell salvage, ANH, or fractionated products.
      iv) These options need to be discussed thoroughly and the patient's wishes understood prior to the operative procedure.
   c) **Alternative techniques and the JW patient (Table 39-1)**
      i) Many techniques have variable acceptance in JW patients.
      ii) Preoperative discussion and documentation of acceptance or refusal of various techniques is absolutely essential.

TABLE 39-1 Alternatives to Blood Transfusion and the JW Patient[a]

Alternative Technique	Acceptance by JW Patients	Comments
Crystalloid	Generally accepted	May be used for volume expansion
Hetastarch	Generally accepted	May be used for volume expansion
Albumin	Acceptance varies	Derived from human sources
Cell salvage Normovolemic hemodilution	Acceptance varies	Pt may desire continuous circuit with his/her body
Cryoprecipitate	Generally refused but may vary with the patient	Use has been reported, discuss with patient
Erythropoietic agents	Acceptance varies	May contain small amounts of human albumin
Recombinant factor VIIa	Generally accepted	Should be discussed in cases where risk of coagulopathy is high

[a]Patients vary widely with their acceptance of techniques. Whenever possible, ALWAYS confirm the patient's wishes prior to administration.

CHAPTER SUMMARY FOR ALTERNATIVES TO BLOOD TRANSFUSION

General Principles	Patients may decline blood product transfusion based on religious grounds. Rarely, crossmatched blood is difficult to obtain.
Intraoperative Cell Salvage	Requires specialized equipment Blood suctioned from operative field, processed, and reinfused to the patient Contraindications may include malignancy, bacterial contamination, and urine and bowel contents in the surgical field.
Acute Normovolemic Hemodilution	Blood is removed from the patient and reinfused after most blood loss has occurred. Blood removed must be replaced with colloid or crystalloid.
Pharmacologic Agents	Include rhEPO and rFVIIa.
Management Strategies to Minimize Need for Blood Transfusion	Minimize phlebotomy, notify surgeon of need for minimizing blood loss with surgical technique, controlled ventilation, increase FiO_2
Jehovah's Witness	Patients who object to blood transfusion on religious grounds Discuss patient's wishes alone if possible. Clearly explain the risks associated with blood product refusal, including death. Understand and document the patient's wishes.

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40 Massive Transfusion and Resuscitation

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Massive bleeding in the operating room can be expected, as in the case of liver transplant or abdominal aortic aneurysm repair, or unexpected, as in the case of trauma, vascular injury, or postpartum. Hemorrhage can cause decreased tissue perfusion, cellular injury, and death if not treated appropriately. Significant bleeding may result in the need for transfusion of large amounts of blood products.

1) **Definition**
   a) Massive hemorrhage has been described as the loss of one's entire blood volume in a 24-hour period or half of the entire blood volume in a 3-hour period.[1]
   b) Massive transfusion is defined as transfusion of 10 units of pRBC or blood products in 24 hours.[2]
2) **Causes and triggers (Table 40-1)**
   a) Causes of massive hemorrhage requiring massive transfusion are variable. Any time of the day or night, one can be faced with the potential for massive bleeding in most major centers and community hospitals.
   b) Early transfusion of trauma patients has been demonstrated to improve outcomes[3,4]; however, identification of those who require massive transfusion can be difficult. Several scoring systems are available to assist in identification of trauma patients who may require massive transfusion.
      i) Assessment of blood consumption (ABC)[5]
         (1) Four-component scoring system (initial ED value), each component assigned 1 point: SBP $\leq$ 90, HR $\geq$120, penetrating trauma mechanism, and positive FAST (focused assessment with sonography for trauma)
         (2) ABC score $\geq$2 is considered a positive predictor of needing massive transfusion (75% sensitive and 86% specific).[5]
      ii) Massive transfusion score (MTS)[6]
         (1) Six-component scoring system (initial ED value), each component assigned 1 point: SBP $\leq$ 90, HR $\geq$120, penetrating trauma mechanism, positive FAST, INR >1.5, Hgb <11 g/dL, and base deficit $\geq$6 mmol/L.
         (2) MTS $\geq$ 2 is considered positive and has a sensitivity of 85% for predicting the need for massive transfusion. MTS < 2 has a negative predictive value of 89%.[6]
3) **Management goals in massive hemorrhage**
   a) The primary goal is maintenance of adequate perfusion and cellular function. Prevention of microvascular bleeding is a secondary goal.
   b) **Lines and monitors**
      i) Standard ASA monitors plus invasive monitors are usually required.
      ii) Central venous access
         (1) Large-bore central lines are extremely beneficial for volume replacement and can be used in conjunction with a rapid infusion device.
         (2) Central venous pressure can be used as a guide for volume replacement.

READ MORE
Central venous monitoring, Chapter 13

TABLE 40-1 Causes of Massive Hemorrhage

Cause of Massive Hemorrhage	Example
Obstetric hemorrhage	Placenta accreta
Trauma	Motor vehicle accident, gunshot wound
Surgical	Liver transplant, aortic surgery
Coagulopathy	von Willebrand disease, factor deficiency

(3) Ultrasound guidance should be used when placing central venous lines whenever possible.
(4) While placement of central access can aid in management, it can also draw attention and personnel resources away from treatment of the patient. Ensure that the patient is being adequately resuscitated while the line is being placed, or defer placement to focus on volume replacement.

iii) Intra-arterial blood pressure monitoring
(1) Offers the ability for close monitoring of BP and frequent blood draws for laboratory analysis.
(2) Should be placed early in a hemorrhagic emergency because placement may be difficult once significant peripheral vasoconstriction is present.
(3) Radial artery is the most common cannulation site, but femoral is often used in emergent or difficult situations.

iv) Adequate IV access is essential in the bleeding patient. The flow of a fluid through a catheter is proportional to the fourth power of the radius, so large-bore peripheral IV access allows for rapid transfusion.
(1) Recommended IV access includes two large-bore (14-16 g) IV cannulae (**Table 40-2**).



In cases of hemorrhage, an arterial line is extremely helpful for BP monitoring and blood draws and should be placed as early as possible.

v) Urine output
(1) Monitoring urine output is an effective indicator of the patient's intravascular volume status.
(a) Urine output <15 mL/kg/h is indicative of severe volume depletion.[7]

vi) Temperature monitoring
(1) Monitoring core temperature (eg, tympanic membrane, pulmonary artery, distal esophagus, and nasopharynx) is essential in patients with hemorrhage.
(2) Hypothermia is extremely problematic in the massively bleeding patient because decreased core temperature worsens acidosis, impairs platelet aggregation, and decreases coagulation factor enzymatic activity.

TABLE 40-2 Flow Rates Through Peripheral Intravenous Catheters of Varying Sizes

Peripheral IV Gauge	Saline Flow Rate
14 g × 1¼″	325 mL/min
16 g × 1¼″	215 mL/min
18 g × 1¼″	105 mL/min
20 g × 1¼″	60 mL/min
22 g × 1″	35 mL/min

vii) Airway management and maintenance
   (1) Massively bleeding patients may arrive in the operating room or emergency department with an endotracheal tube in place.
   (2) Severe blood loss significantly alters consciousness. The airway should be protected and managed by endotracheal intubation and ventilation.
   (3) Large volume resuscitation with crystalloid, colloid, or blood products can result in edema, altering airway anatomy and possibly increasing the risk of difficult intubation.



Temperature monitoring is vital in patients with massive hemorrhage. Every effort should be made to maintain normothermia.

c) **Maintenance of adequate perfusion**
   i) In order to assure organ perfusion, intravascular volume must be maintained by large amounts of IV fluid, appropriate amounts of colloid, and potentially blood products.
   ii) Blood products should be transfused for appropriate indications (READ MORE, Chapter 38).

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Alternatives to blood product replacement, Chapter 39

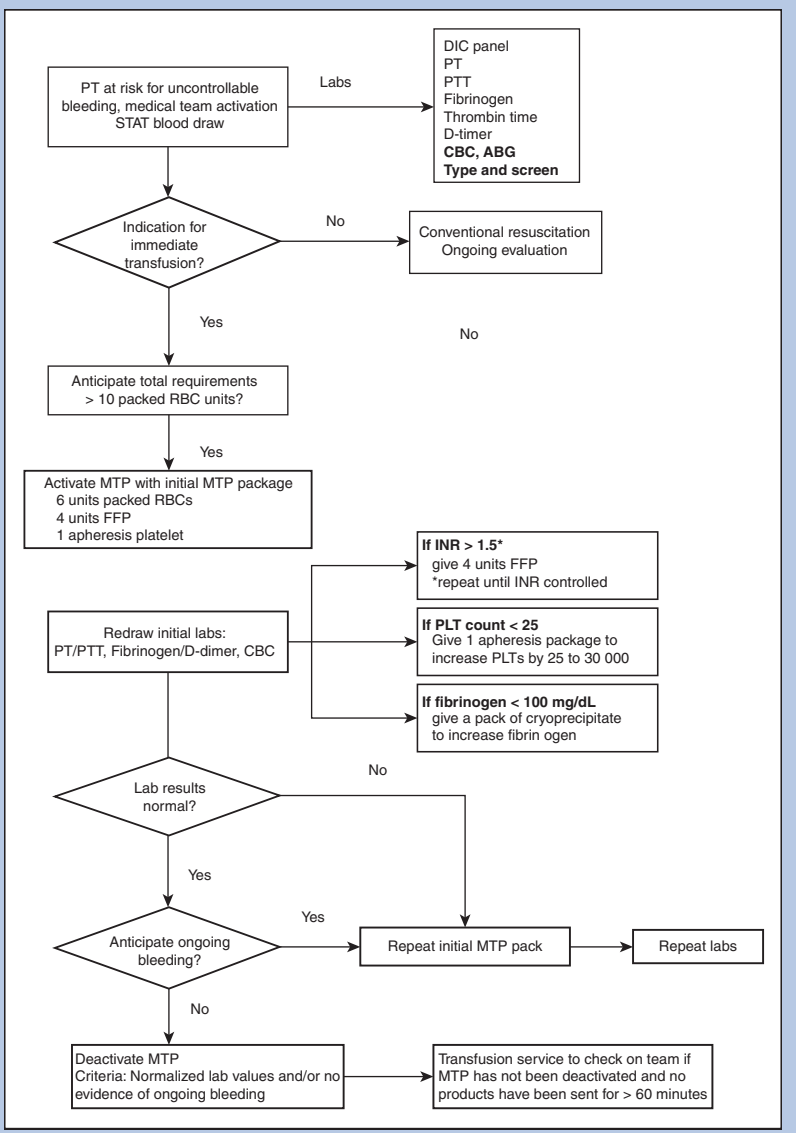
4) **Mobilization of resources**
   a) Call for help of anesthesia technicians and additional anesthesia personnel.
   b) Mobilize necessary equipment.
   c) Notify blood bank and clinical laboratory.
   d) Send blood samples for blood type and screen and activate the massive transfusion protocol (MTP), if applicable.
5) **Massive transfusion protocol/guideline**[8]
   a) Clear communication among all parties involved in caring for the massively bleeding patient is essential.
   b) An MTP that gives clear directions on laboratory data, blood product availability and transport, as well as equipment and personnel is extremely helpful.
   c) Instituting an MTP should be seriously considered in every institution where the potential to treat massive hemorrhage exists (**Fig. 40-1**).[9]
6) **Blood product replacement specific to massive transfusion**
   a) **Packed red blood cells (pRBC)**[10]
      i) Given to increase oxygen carrying capacity.
      ii) Estimated blood loss is a reasonable guide to pRBC replacement, although in a massive hemorrhage, estimating blood loss is often difficult. In this situation, RBCs are often given in response to clinical events or laboratory values such as hemoglobin with consideration of the patient's acid-base status.
      iii) Often in a hemorrhagic emergency, type-specific, crossmatched blood is not available.
         (1) If a blood type is available, type-specific, uncrossmatched blood may be given in an emergency as the risk of a hemolytic transfusion reaction from ABO-compatible, uncrossmatched blood in patients who have not been previously transfused or pregnant is extremely low.[11]
         (2) If no blood type is available and the patient requires blood emergently, type O pRBCs (universal RBC donor) and AB plasma (universal plasma donor) can be given.
         (3) Type O, RhD-negative pRBCs is preferred for all patients, but required in women of childbearing age, as exposure to Type O, RhD-positive blood risks development of anti-RhD antibodies that can cause hemolytic disease of the fetus and newborn (HDFN) in subsequent pregnancies.



The purpose of IV crystalloid and/or colloid in massive hemorrhage is to maintain adequate organ perfusion and blood flow. The purpose of pRBC transfusion is to increase oxygen delivery.

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Blood component therapy, Chapter 38

FIGURE 40-1 Algorithm for Massive Transfusion



PT, patient; DIC, disseminated intravascular coagulation; CBC, complete blood count; ABG, arterial blood gas; INR, international normalized ratio. Reproduced with permission from Goodnough LT, Spain DA, Maggio P. Logistics of transfusion support for patients with massive hemorrhage. *Curr Opin Anaesthesiol*. 2013;26(2):208-214.

(4) If a confirmed blood type is available, it is acceptable to transition to type-specific blood products after large volume transfusion of universal blood components. Type O pRBC units contain low volumes of plasma and therefore small amounts of anti-A and anti-B antibodies (isohemagglutinins), which are responsible for the majority of acute hemolytic transfusion reactions.

In the setting of massive transfusion, the small amount of transfused isohemagglutinins is diluted in the much larger recipient blood volume as well as further diluted with AB plasma, which is devoid of anti-A and anti-B antibodies. Consequently, the concentration of isohemagglutinins remains clinically insignificant, allowing transition to type-specific blood and preservation of universal blood components for other patients.[12,13]

(a) One exception to this approach applies to transfusion of neonates and small infants, where the plasma content of transfused pRBCs may comprise a clinically significant portion of recipient blood volume.

(1) Washed type O pRBC units are usually provided in these situations to decrease this risk.

(5) Type O whole blood may be transfused in certain situations such as prehospital resuscitation (military trauma, helicopter transport). These units must be confirmed to have low titers of anti-A and anti-B antibodies in order to decrease the risk of hemolytic reactions.[14] If patients are transfused low titer group O whole blood units, they should not receive type-specific blood products until low levels of anti-A and/or anti-B antibodies have been confirmed by the hospital transfusion service.[15]



In an emergency where blood is required, and a blood type is not available, type O pRBC and AB plasma may be transfused.

iv) Large volume pRBC administration is associated with coagulopathy due to dilution of clotting factors and platelets (dilutional thrombocytopenia).

b) **Plasma (eg, fresh frozen plasma [FFP])**

i) Current ASA guidelines state that plasma should be given in the presence of microvascular bleeding in patients who have been transfused one blood volume or when INR is >2.0.[10]

ii) Recent data suggest that early transfusion of FFP and platelets aids in prevention of coagulopathy associated with trauma-related massive transfusion.[3,4,16]

iii) It is unclear if this data can be extrapolated to other situations. This is an active area of research.

c) **Platelets**

i) Current guidelines recommend a platelet count prior to platelet transfusion and state that platelet transfusion is typically not indicated for platelet counts >50 000 cells/mm^3.[10]

ii) Platelets are now mostly collected from donors via apheresis. The platelet content of an apheresis platelet unit is equivalent to 6 whole blood derived platelet units (6-pack). Transfusion of a single apheresis platelet unit should increase an average-sized adult's platelet count by 30-50 000/µL.

iii) Recent information in massively bleeding trauma patients suggests that platelet and plasma transfusion early (1:1:1 ratio) in resuscitation with an attempt to approximate whole blood is associated with lower mortality.[3,4,16]

d) **Blood product ratios for massive transfusion**

i) Ratios of pRBC, plasma, and platelets for a MTP should approximate contents of whole blood.[3,4,16] Additional plasma to yield a near 1:1 pRBC/plasma ratio is obtained from the apheresis platelet unit, as the platelets are suspended in 300-350 mL of donor plasma. Examples are as follows:

(1) Patients >50 kg—6 pRBC, 4 plasma, 1 apheresis platelet

(2) Patients <50 kg—4 pRBC, 2 plasma, 1 apheresis platelet

7) **Recombinant coagulation factors and hemostatic agents**

a) Use of recombinant activated factor VII (rFVIIa, NovoSeven) may be of benefit during massive hemorrhage to treat dilutional or acute traumatic coagulopathy; however,

trials have failed to show a mortality benefit, and its use is associated with increased risk of arterial thrombosis.[17]

b) Trauma and surgical patients may experience increased clot degradation (hyperfibrinolysis) that can lead to higher rates of bleeding and increased transfusion requirements. Early administration of antifibrinolytics, such as tranexamic acid, is associated with decreased mortality in trauma victims and is recommended unless contraindicated.[18]



In massively bleeding patients, blood product replacement ratios that approximate whole blood may improve patient outcome and should be considered.

8) **Complications of massive transfusion**

a) Complications associated with blood component therapy also apply to massive transfusion.[19]

i) These include acute and delayed hemolytic reactions, transfusion-associated circulatory overload (TACO), transfusion-related acute lung injury (TRALI), and transfusion-transmitted infectious disease.

b) Other complications are more specific to large volume transfusion.

c) **Hypothermia**

i) Hypothermia with a core temperature <35°C significantly affects coagulation.

(1) The more hypothermic the patient becomes, the higher the risk of microvascular bleeding.

(2) Every effort should be made to prevent the lethal triad of hypothermia, acidosis, and coagulopathy.

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Alternatives to blood component therapy, Chapter 39

ii) In trauma patients, hypothermia is associated with an increased risk of developing disseminated intravascular coagulation.

(1) Trauma patients often arrive in the OR hypothermic. Rewarming requires substantial effort, but the difference in outcome can be significant.

iii) Every effort to maintain normothermia, including using warming devices for all IV fluids and blood products and forced-air heating devices if possible.[20]

READ MORE
Electrolyte and Acid/Base Disturbances, Chapter 37

d) **Hypocalcemia**[21]

i) Due to citrate anticoagulant in the blood products binding calcium.

ii) May manifest as decreased cardiac contractility and ECG abnormalities.

iii) Contributes to coagulopathy (calcium is Factor IV in the clotting cascade).

iv) Carefully monitor calcium levels and treat hypocalcemia with IV calcium chloride.

e) **Hyperkalemia**[19]

i) Increased potassium is due to RBC lysis during storage.

ii) Potassium should be carefully monitored and hyperkalemia treated appropriately.

f) **Volume overload**[22]

i) Care must be taken to avoid excess volume replacement as circulatory overload can lead to pulmonary edema and other potential complications.

ii) Furthermore, excessive crystalloid administration may lead to abdominal compartment syndrome and potentially other cardiopulmonary complications.

g) **Dilutional coagulopathy**[23]

i) Administration of large volumes of products that do not contain clotting factors or platelets results in decreased circulating clotting factors and platelets, which may result in coagulopathy.

ii) Clotting factors and platelets should be administered either based on an MTP that attempts to approximate whole blood, laboratory values, or microvascular bleeding in the situation of large volume resuscitation.

h) **Decreased 2,3-diphosphoglycerate**[11]
   i) 2,3-Diphosphoglycerate is a compound that binds deoxyhemoglobin and increases oxygen delivery to cells.
   ii) Transfusion of large volumes of stored RBCs can lead to decreased 2,3-diphosphoglycerate thereby decreasing oxygen delivery to tissues.

i) **Disruptions in acid-base balance**[11,19]
   i) The addition of citrate-phosphate-dextrose solution during RBC storage decreases the pH to ~7.0.
   ii) In the normally perfused liver, the citrate is metabolized to bicarbonate, which should mitigate the acidic pH of the transfused blood.
   iii) In situations where the liver is not adequately perfused, this process may be altered which has the potential for exacerbation of metabolic acidosis.
   iv) The clinical significance of the low pH associated with stored RBC is unclear, but it is possible that transfusion of large volumes may significantly contribute to metabolic acidosis.

CHAPTER SUMMARY FOR MASSIVE TRANSFUSION

Causes	May be surgical, trauma, or obstetric
Definition	Massive transfusion is the administration of 10 or more units of blood products in 24 h
Goals	Adequate tissue perfusion, oxygen delivery, and prevention of coagulopathy
Blood Product	Should be based on specific indications
Replacement	Consider ratio of pRBC/plasma/platelets of 1:1:1, which closer approximates whole blood, especially in massively bleeding trauma patients
Complications	Risks associated with blood product replacement apply Specific risks for massive transfusion include hypothermia, hyperkalemia, hypocalcemia, dilutional coagulopathy, volume overload, and decreased 2,3-diphosphoglycerate

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41 Spinal Anesthesia§

Robert L. Hsiung

Injection of local anesthetic into the subarachnoid space produces rapid onset of surgical anesthesia. When coadministered with local anesthetics, intrathecal opioids may enhance the quality of spinal anesthesia and provide postoperative analgesia.[1]

1) **Indications and contraindications**
   a) **Indications**
      i) Spinal anesthesia is generally performed for surgical procedures involving the lower abdomen, perineum, and lower extremities.
      ii) It is the preferred anesthetic technique for cesarean delivery due to decreased maternal morbidity and mortality.[2]

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Obstetrical anesthesia overview, Chapter 107

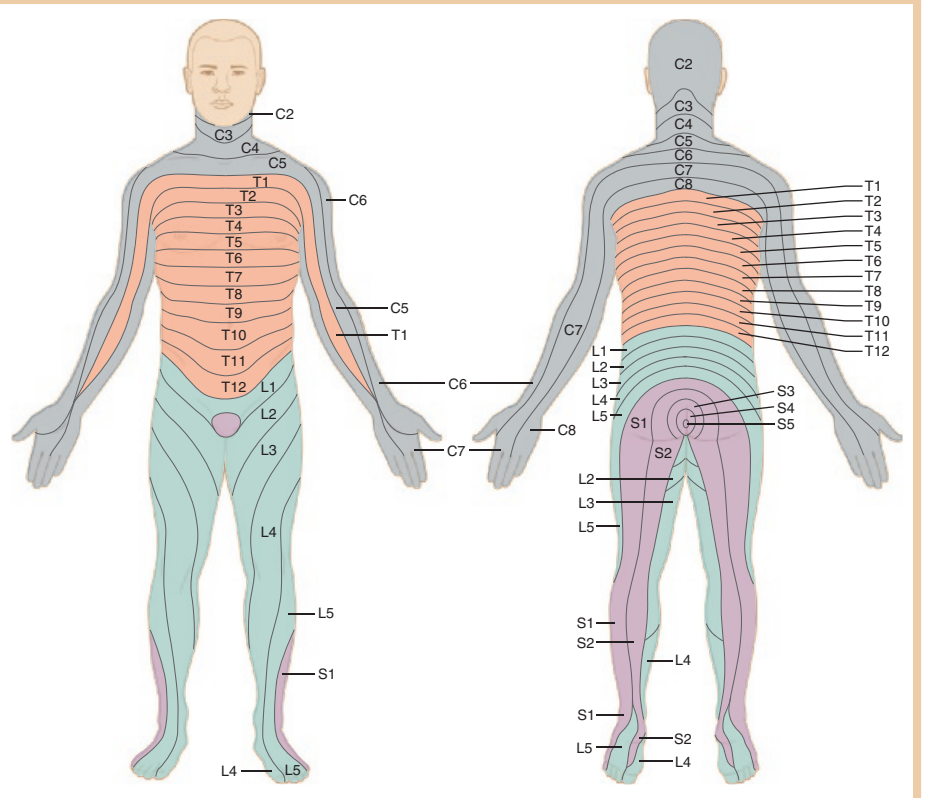
   b) **Contraindications**
      i) Absolute contraindications
         (1) Language or consent barrier
         (2) Increased intracranial pressure
         (3) Coagulopathy
      ii) Relative contraindications
         (1) Systemic infection
         (2) Inability to position
         (3) Hemodynamic profile that would not tolerate a decrease in systemic vascular resistance (eg, shock or aortic stenosis)
2) **Anatomy**
   a) **Spinal cord**
      i) Thirty-one pairs of spinal nerves consisting of paired ventral motor roots and paired dorsal sensory roots.
      ii) The area of skin innervated by the cutaneous branches from a single spinal nerve is called a *dermatome* (**Fig. 41-1**).[3]
      iii) In term infants, the caudal extent of the spinal cord, known as the *conus medullaris*, normally ends at L2.[4]
      iv) In adults, the mean level at which the conus medullaris ends is L1, with a range between T11 and L3.[5]
      v) Spinal anesthesia should not be performed above the L2-L3 interspace due to the increased potential for mechanical injury to the spinal cord.



Spinal anesthesia should not be performed above the L2-L3 interspace.

   b) **Potential anatomical paths for the spinal needle**
      i) Midline approach: skin, subcutaneous tissue, supraspinous ligament, interspinous ligament, ligamentum flavum, dura mater, arachnoid mater, and subarachnoid space
      ii) Paramedian approach: bypasses the supraspinous and the interspinous ligaments
3) **Pharmacology**
   a) **Determinates of block height**
      i) Baricity and patient position are the two major factors.
      ii) Baricity is defined as the density of the local anesthetic solution relative to the density of cerebral spinal fluid (CSF) at 37°C.

FIGURE 41-1 Sensory Dermatomes



(1) Hyperbaric solutions have a density greater than CSF.
   (a) Usually prepared by mixing local anesthetic in dextrose (eg, bupivacaine 0.75%).
   (b) In the supine position, hyperbaric solutions distribute by way of gravity toward the thoracic and sacral curvatures, producing an average block height at the midthoracic level.
   (c) Block height may be affected by solution volume and duration of time spent in block position (eg, sitting or lateral decubitus) prior to assuming the supine position.
(2) Isobaric solutions have a density equal to CSF.
   (a) Positioning affects the distribution of local anesthetic to a lesser extent, and the average block height is at the low thoracic level (eg, lidocaine 2%).
(3) Hypobaric solutions have a density lower than CSF (eg, bupivacaine 0.5%).

b) **Determinates of block duration**
   i) Local anesthetic selection (**Table 41-1**).[6]
   ii) Local anesthetic dose.
      (1) Increasing the dose results in an increased duration of spinal anesthesia.
   iii) Elimination of local anesthetic occurs through vascular absorption from the subarachnoid space and the epidural space.

TABLE 41-1 Local Anesthetics Used for Spinal Anesthesia

Local Anesthetic	Dose (mg)	Mean Peak Block	Complete Regression (min) (SD)	Dosing for Common Procedures
Short duration				
2-Chloroprocaine, 2%[a]	40	T7	114 (14)	Knee arthroscopy, hemorrhoid surgery
Intermediate duration				
Lidocaine, 5%	50	T6	130 (18)	Lower extremities, often diluted to 2.5% or less
Mepivacaine, 1.5%[a]	60	T10	212 (25)	Knee replacement
Long duration				
Bupivacaine, 0.5%[a]	10-15	T7	178 (20)	Hip replacement
Bupivacaine, 0.75%	12-15	T4	360	12 mg is used for most cesarean sections, as higher dose increases risk of high spinal and cardiorespiratory compromise

[a]Not FDA approved for spinal anesthesia. Considered "off-label" use.

iv) Baricity
(1) Isobaric bupivacaine has a longer onset and appears to provide a longer block than hyperbaric bupivacaine.[7]

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Local anesthetics, Chapter 25

c) **Differential block**
i) Sympathetic blockade often occurs a few dermatomes higher than sensory blockade, which, in turn, occurs higher than motor blockade.
ii) This effect may be secondary to local anesthetic action on the differing fiber sizes and degree of myelination.[8]



Addition of opioids to intrathecally administered local anesthetics enhances block quality and prolongs analgesia.

d) **Site of action**
i) Local anesthetics deposited in the subarachnoid space are thought to act primarily on the spinal nerve rootlets.

e) **Adjuvants**
i) Opioids (eg, fentanyl, sufentanil, morphine) may be added to local anesthetics to enhance the quality of block and prolong analgesia.[1,9,10]
ii) Clonidine may extend the duration of sensory and motor block.[11]
iii) Vasoconstrictors may prolong duration and block quality.[12]
(1) Epinephrine added to local anesthetics may produce untoward side effects such as flulike symptoms.[13]

4) **Equipment**
a) Common equipment for all regional anesthetic techniques includes a block tray, kit, or cart and an appropriate volume of local anesthetic solution.
b) Spinal anesthesia should be performed with meticulous sterile technique using appropriate hand hygiene, sterile gloves, hat, mask, and drape.
c) **Spinal needle and introducer**
i) Small gauge, pencil-point (Whitacre, Sprotte) spinal needles are recommended due to the decreased incidence of postdural puncture headache (PDPH).[14]
ii) Most practitioners use 24- to 26-G pencil-point needles.
iii) Occasionally, 22-G needles may be useful during difficult blocks (eg, elderly patients with arthritic changes in the spine, obesity). The potentially higher incidence of PDPH should be weighed against the benefit of easier placement.



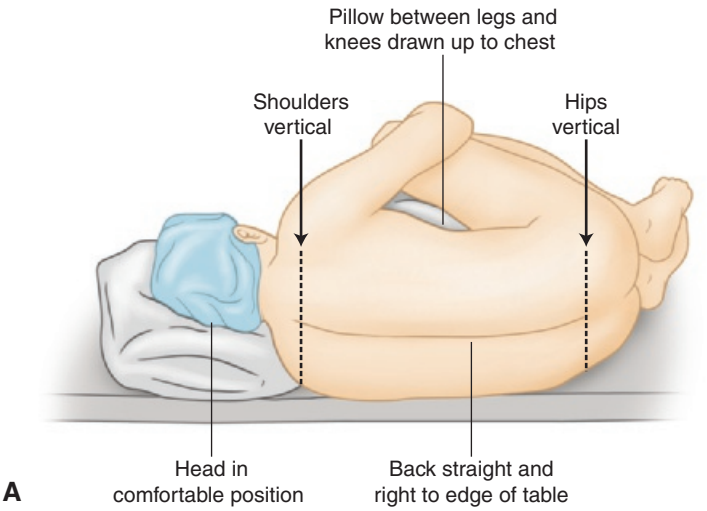
Meticulous sterile technique should always be used when performing spinal anesthesia.

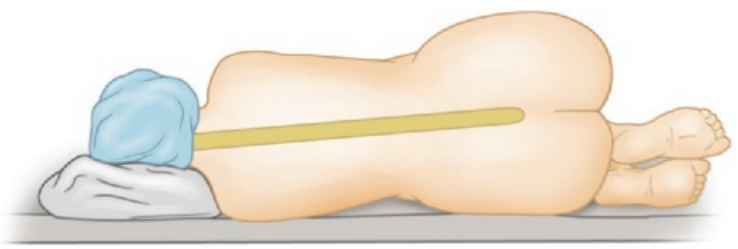
5) **Technique**
   a) **Monitoring**
      i) Spinal anesthetics should be administered in a location with monitoring capabilities.
      ii) Supplemental oxygen should be provided if sedation is to be administered.
      iii) Emergency airway equipment and resuscitation drugs should be available.
   b) **Positioning and preparation**
      i) Apply monitors and supplemental oxygen prior to administering sedation.
      ii) Patients are typically placed in the lateral decubitus or sitting positions.
         (1) Lateral decubitus (**Fig. 41-2**)[3]
            (a) The patient's back should be at the edge of the bed, while keeping the shoulders and the hips perpendicular to the bed to minimize rotation of the spine.
            (b) The knees are drawn upward toward the chest with the neck flexed and chin in a downward position, as if the patient were to be curling into a ball.
            (c) Beneficial in that it may require fewer personnel to hold the patient while the procedure is being performed.
            (d) Also maybe the most practical for those difficult to position such as patients with hip fractures or women requiring emergency cesarean delivery.
         (2) Sitting (**Fig. 41-3**)[15]
            (a) May help identify midline, particularly in obese patients.
            (b) The knees are drawn upward toward the chest or supported rather than dangling at the edge of the seat as it is lumbar flexion that increases interspace openings.
            (c) There are multiple devices that help with positioning; however, an assistant is often required to help stabilize the patient during the procedure, especially if sedation is utilized.
      iii) The anticipated injection site is prepared with appropriate antiseptic solution and draped in a sterile fashion.
         (1) It is beneficial to prepare a wide area to facilitate any subsequent attempt at a different entry point.
         (2) Allow the antiseptic solution to dry and wipe away any excess.
   c) **Identification of surface anatomy**
      i) A line drawn between the tips of the scapula estimates T7, while one drawn between the iliac crests (Tuffier line) estimates L4 (**Fig. 41-4**).
      ii) Using the index and the middle finger side by side, palpate the spinous processes by sliding side to side and up and down the midline.
      iii) The target interspace for spinal anesthesia is L4-L5 or L3-L4.
      iv) Ultrasound guidance has been used to help identify interspaces as visual estimation is often inaccurate.[16,17]

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Lumbar Neuraxial Ultrasound, Chapter 200

   d) **Midline approach**
      i) Identify the middle of the target interspace.
      ii) After informing the patient, infiltrate the skin and intended path of the spinal needle with local anesthetic. Injecting as though performing a field block can often allow one to use this as a "finder" needle in helping contact spinous process, indicating the midline.
      iii) Assuming that you are at the top edge of a spinous process, insert the introducer needle with a 10- to 15-degree angulation in the cephalad direction until it engages the interspinous ligament. Most of the spinal kits have introducer needles that are unlikely to penetrate the subarachnoid space; however in children and thin adults this is a possibility.

FIGURE 41-2 **A.** Lateral decubitus position for spinal anesthesia with differences between women **(B)** and men **(C)** on the level of subarachnoid space.





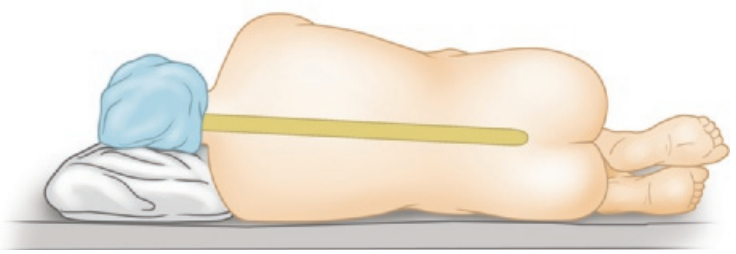
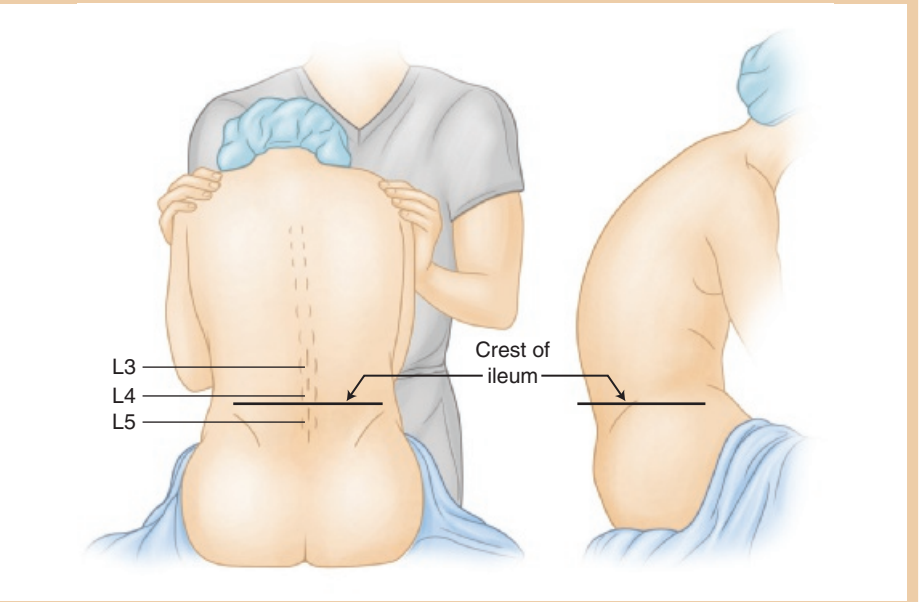


FIGURE 41-3 Correct Sitting Position Demonstrated for Spinal Anesthesia



(1) The introducer aids the thin spinal needle towards a specific direction.
(2) If redirection is necessary, the introducer needle will have to be withdrawn and also redirected.

iv) Insert the spinal needle through the introducer (**Fig. 41-5A**).
(1) Two characteristic changes in resistance may be felt: the firmness of the ligamentum flavum, followed by a decrease in resistance at the dura-arachnoid membrane interface. These pressure changes are often not noticed in most spinal placements, and thus the visual inspection of CSF is most important.
(2) The tip of the spinal needle should now be in the subarachnoid space.



Hyperbaric local anesthetic solution will make a characteristic "swirl" when mixed with CSF aspirated into the syringe.

v) Stabilize the spinal needle by resting the dorsum of the other hand against the patient's back while grasping the hub of the spinal needle between the thumb and the index finger (**Fig. 41-5B**).

vi) Remove the stylet of the spinal needle and observe for free CSF flow.
(1) Attach the syringe containing the local anesthetic solution.
(2) Gently aspirate to confirm subarachnoid placement and then slowly inject the local anesthetic solution (**Fig. 41-5C**).
(3) Some practitioners advocate to remove the stylet before entering the subarachnoid space to have the earliest identification of CSF flow.



The introducer needle may cause inadvertent dural puncture in a thin patient; therefore, utmost care must be placed when advancing any needle in this space.

vii) Remove the introducer, needle, and syringe together as a unit.

FIGURE 41-4 A Line Drawn Between the Iliac Crests Approximates L4

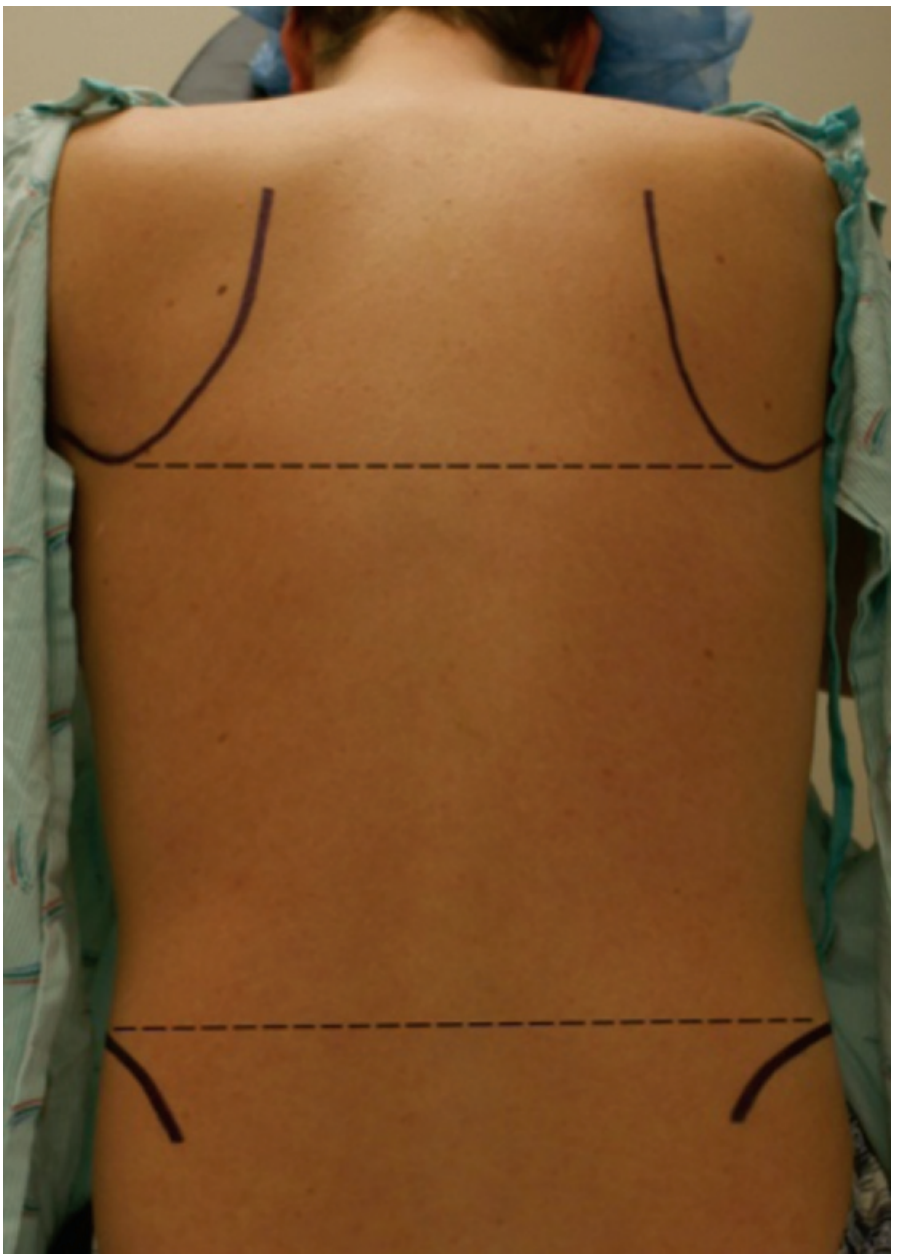


FIGURE 41-5 Spinal Needle Insertion Techniques



A



B



C

A. Using the dominant hand, insert the spinal needle through the introducer. **B.** Stabilize the spinal needle by resting the dorsum of the nondominant hand against the patient's back while grasping the hub of the spinal needle between the thumb and the index finger. **C.** Attach syringe with desired spinal medication, aspirate to confirm intrathecal location, and inject.

e) **Paramedian approach**
   i) May be helpful in patients unable to flex their spine or in elderly patients with arthritic changes.
   ii) As a starting point, insert the introducer needle ~1 cm lateral to the superior aspect of the inferior spinous process and angle 15 degrees toward midline and 45 degrees cephalad.
   iii) Insert the spinal needle through the introducer.
      (1) The first resistance encountered should be the ligamentum flavum.
      (2) If bone is contacted past the estimated depth of the spinous process, the spinal needle is most likely contacting the lamina. If it is angulated too medially, it may be contacting the spine of the spinous process.
      (3) Proceed to locate the interlaminar space by "marching off" the lamina cephalad or caudad. If it was the spinous process that was contacted, redirect the needle to a less medial direction as walking off the spinous process would cross midline and miss the desired location.
   iv) Once free-flowing CSF is achieved, proceed as above.

f) **Troubleshooting**
   i) Bony contact
      (1) It is important to have a working three-dimensional mental image of the spine.
      (2) Adjustments of the needle angle must first be preceded by withdrawing the introducer before the spinal needle is advanced again. Small changes in trajectories can translate to a large distance at full needle insertion depth. It is easy to bend the needle such that the internal needle path is different from the desired external direction.
      (3) In the midline approach, note the depth at which bone is contacted and redirect slightly cephalad.
         (a) If bone continues to be contacted at a shallower depth, the needle is likely marching up the inferior aspect of the superior spinous process, and a more caudad angle is warranted.
         (b) If bone continues to be contacted at a deeper depth, the needle is likely marching down the superior aspect of the inferior spinous process, and a more cephalad angle is warranted.
         (c) If bone is contacted repeatedly at the same depth deeper than you would expect the spinous process, the needle is likely contacting lamina off midline. Re-examining the anatomy and inserting at a slightly different location maybe appropriate.
   ii) No CSF flow
      (1) It is unlikely that the spinal needle is in the subarachnoid space, although low CSF pressure is a less likely but possible etiology.
      (2) The needle may still be in the epidural space and further advancement may be warranted, especially in larger patients.
      (3) Rotation of the needle may result in more brisk flow if there appeared to be flow earlier. Reinsertion and removal of the spinal needle stylet might remove an obstruction at the aperture.
      (4) If these measures fail, then the needle should be removed and reinserted.
   iii) Paresthesia
      (1) Immediately stop needle advancement, and remove the spinal needle stylet.
      (2) If CSF flow is obtained, the needle may have encountered a cauda equina nerve root in the subarachnoid space.[18]
      (3) Paresthesia is frequently transient, and injection of local anesthetic may proceed after needle repositioning and only if the paresthesia resolves.
      (4) If no CSF flow occurs, remove the needle and attempt reinsertion.

iv) Blood in the spinal needle
  (1) This might result from the needle entering the epidural vein or a traumatic spinal tap.
  (2) If the flow clears, it is likely CSF and continuation of drug delivery are acceptable.
  (3) If there is blood in the needle and no CSF flow is obtained, it is often best to use a new spinal needle as blood will clot the small needle and detection of free flow would be difficult.



If a paresthesia is elicited, ensure that it has resolved prior to injection of local anesthetic solution.

6) **Risks and complications**

a) **Pulmonary function changes that occur due to spinal anesthesia.**

i) Usually not clinically significant in healthy patients because the diaphragm is innervated by the phrenic nerve (C3-C5).

ii) Respiratory rate and tidal volumes remain unchanged, though a high thoracic level block can result in decreased expiratory reserve volume from deafferentation of the abdominal wall and intercostal muscles.[19]

iii) Caution is advised in performing neuraxial blocks expected to achieve a high level of anesthesia in patients with limited respiratory reserve.

iv) Respiratory arrest associated with high spinal is attributed to hypoperfusion of the medullary respiratory neurons, rather than inhibition of the phrenic nerve. Prompt hemodynamic resuscitation is associated with immediate return of spontaneous respiration.[3]

v) Complaints of dyspnea in an otherwise healthy patient who is able to vocalize normally should be addressed with acknowledgment, supplemental oxygen, and, if needed, respiratory support of a high or total spinal.



Spinal anesthesia can cause symptomatic bradycardia and even lead to cardiac arrest, especially in young patients with high parasympathetic tone. Patients with conduction abnormalities and those taking nodal blockers (eg, beta-blockers) are also at high risk.

b) **Hypotension is a frequently encountered physiologic effect of spinal anesthesia.**

i) Sympathetic efferent blockade results in arterial and venous dilation, causing a decrease in afterload and preload, respectively.

ii) Treatment involves IV fluid resuscitation and vasoactive agents (eg, ephedrine or phenylephrine).[20–22]

iii) Prevention of spinal hypotension also involves vasopressors and fluids.
  (1) Use of titrated vasoactive infusions, particularly phenylephrine, has greatly reduced the incidence of hypotension due to spinal anesthesia.[23]
  (2) Evidence suggests that crystalloid fluid coloading (administration of a bolus while the procedure is being performed) is likely superior to fluid preloading in preventing hypotension.[24]

c) **Bradycardia may result from blockade of T1-T4 sympathetic cardioaccelerator fibers and decreased venous return.**[25]

i) Treatment is recommended when the heart rate is low (<50 bpm) or dysrhythmia is observed, since sudden asystole and cardiovascular collapse may follow especially in young patients with high parasympathetic tone.[26]

ii) If this occurs, external chest compressions and fluid resuscitation are necessary to maintain coronary perfusion and circulation of medications.

iii) Pharmacologic resuscitation includes atropine, ephedrine, and epinephrine.



Patients with hand numbness and decreased grip strength following spinal anesthesia are at risk for total spinal anesthesia and should be closely monitored for respiratory depression.

d) **Postdural puncture headache (PDPH)**
   i) May occur within 12-48 hours.
   ii) Characterized by a severe fronto-occipital headache in the upright position and improves with recumbency.
   iii) PDPH is more common in women and younger patients and with use of larger gauge needles.
   iv) Severe cases may be treated with an epidural blood patch.[27]

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Postdural puncture headache and epidural blood patch, Chapter 43

e) **Total spinal**
   i) May occur when the block rises past the cervical region, potentially involving the brainstem.
   ii) Symptoms include a rapidly ascending motor-sensory block, hypotension, bradycardia, and possible respiratory arrest.
   iii) Management includes hemodynamic and airway support and administration of an amnestic/anxiolytic agent until the block resolves.
   iv) Most commonly seen as a result of inadvertent injection of an epidural dose of local anesthetic into the subarachnoid space.
   v) Is avoidable with appropriate dosing of local anesthetic.



Transient neurological symptoms have been associated with lidocaine spinal anesthesia, typically presenting the day following surgery, and resolve without sequelae.

f) **Transient neurologic symptoms (TNS)**
   i) Characterized by low back pain that radiates to the buttocks and lower extremities.
   ii) Is primarily associated with the use of lidocaine for spinal anesthesia but has been reported with other local anesthetics.[28]
   iii) Usually occurs the day following spinal anesthesia.
   iv) Risk factors include the lithotomy position, obesity, and outpatient surgery.
   v) Treatment is supportive since TNS usually resolves within 72 hours.

Any patient with persistent or recurrent neurologic deficits and/or back pain after neuraxial block resolution should be immediately examined for signs and symptoms of spinal or epidural hematoma.

g) **Spinal or epidural hematoma is extremely rare but often devastating.**
   i) Patients with pathologic defects in hemostasis or those pharmacologically anticoagulated are most at risk.
   ii) Signs and symptoms
      (1) May include persistent or residual weakness or numbness, back pain, or bladder dysfunction.
      (2) Most importantly, the reappearance of anesthesia or neurologic deficits after block regression must be investigated.
   iii) Diagnostic MRI scan and neurosurgical consultation must be obtained immediately because prognosis is poor, especially for those with symptoms >8 hours.

h) **Persistent neuropathy is extremely rare.**
   i) For spinal anesthesia, the incidence is estimated at 3.8/10 000 with permanent neurological injury estimated from 0 to 4.2/10 000.[29]
   ii) In contrast, an epidural's estimated rate of neuropathy is 2.2/10 000 with permanent injury at 0 to 7.6/10 000.[29]

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Epidural and combined spinal epidural anesthesia, Chapter 42

i) **Infection**
   i) Epidural abscess
   ii) Meningitis
      (1) Rare complication.
      (2) Risk is significantly decreased by using meticulous sterile technique, including the use of masks.[30]

CHAPTER SUMMARY FOR SPINAL ANESTHESIA

Definition	Placement of local anesthetic into the subarachnoid space
Indications	Surgical anesthesia for procedures involving the lower abdomen, perineum, and lower extremities
Advantages	Technically easy to perform, rapid onset of dense neural blockade, small volume of local anesthetic required. Inexpensive
Disadvantages	Generally fixed duration of action once injected
Potential Complications	Hemodynamic perturbations, postdural puncture headaches, total spinal, epidural hematoma, and abscess

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42 Epidural and Combined Spinal Epidural (CSE) Anesthesia§

Robert L. Hsiung

Injection of local anesthetic into the epidural space produces various degrees of anesthesia and analgesia. Advantages of epidural over spinal anesthesia include the ability to provide anesthesia or analgesia to the desired location without necessarily causing complete motor blockade and to allow a continuous effect by the placement of a catheter. Disadvantages of epidural over spinal anesthesia include a technically more difficult technique, slower onset, higher failure rate, and a potentially less dense block. A combined spinal epidural (CSE) offers the rapidity of spinal anesthesia with the added benefit of redosing and thus increasing the effect duration via the epidural catheter.

Epidural Anesthesia and Analgesia

1) **Indications and contraindications**
   a) **Indications**
      i) Epidural anesthesia or analgesia is generally performed for surgical procedures involving the chest, abdomen, pelvis, and lower extremities.
      ii) It can be used intraoperatively as the primary anesthetic, as an adjunct to general anesthesia by decreasing minimum alveolar concentration values of volatile anesthetics, or postoperatively as a continuous infusion with or without patient-controlled epidural analgesia.[1]
   b) **Contraindications**
      i) Absolute contraindications
         (1) Language or consent barrier
         (2) Increased intracranial pressure
         (3) Coagulopathy
      ii) Relative contraindications
         (1) Systemic infection
         (2) Inability to position
         (3) Hemodynamic profile that would not tolerate a decrease in systemic vascular resistance (eg, shock).
2) **Anatomy**
   a) The epidural space is located between the dura and the walls of the vertebral canal and extends from the foramen magnum down to the sacrococcygeal ligament.[2]
   b) It is bound anteriorly by the posterior longitudinal ligament, laterally by the vertebral pedicles, and posteriorly by the ligamentum flavum and vertebral lamina.
   c) Unlike the subarachnoid space, the epidural space is not a closed space.
      i) It communicates with the paravertebral spaces through the intervertebral foramina.
      ii) In addition, it becomes a series of discontinuous compartments when the dura intermittently abuts the ligamentum flavum or pedicles.
      iii) Injection of liquid or air may open up the potential space connecting these compartments.

d) The epidural space contains lymphatics, epidural fat pads, and a network of valveless veins known as Batson plexus.

3) **Pharmacology**

a) **Determinants of block spread**

i) Site of injection

(1) Thoracic injection produces symmetrical spread of anesthetic solution.

(a) The volume of local anesthetic solution should be reduced because a higher block may result in hypotension and bradycardia.

(2) Lumbar injection produces a preferential cephalad spread due to the narrowing of the epidural space at the lumbosacral junction.

(a) The larger diameter of the L5-S1 nerve roots may delay the onset or result in incomplete anesthesia.

(b) Sacral coverage does occur but it is often delayed up to an hour.

(3) Caudal injection predominantly results in sacral and low lumbar anesthesia, except in young children.[3]



The site of injection, volume, and dose are the major factors that determine spread of epidural block.



The choice of medication administered in the epidural space depends on whether the catheter is used for **anesthesia** or **analgesia** and the side effect profile tolerated by the patient.

ii) Volume and dose

(1) Volume and dose are independent variables in predicting the spread of anesthetic solution.

(2) Increasing the injected volume will increase the extent of epidural block.

b) **Determinants of block duration**

i) Local anesthetic selection[4] (Table 42-1)

(1) There are many different options for epidural medication dosing. Most include a local anesthetic and/or opioid.

(2) The selection of local anesthetic depends on the desired duration of the anesthetic and the surgical procedure.

(3) For postoperative analgesia, dilute solutions (0.05%-0.15%) of ropivacaine or bupivacaine with or without opioids are often used.

(4) Bupivacaine 0.75% solution is no longer used due to local anesthetic toxicity with inadvertent intravascular injection.

ii) Volume

(1) For lumbar injections, a recommended volume of 15 mL will, on average, produce a midthoracic block.

TABLE 42-1 Local Anesthetics Commonly Used for Epidural Anesthesia

Local Anesthetic	Complete Resolution (min)
Short duration	
2-Chloroprocaine 3%	100-160
Intermediate duration	
Lidocaine 2%	160-200
Mepivacaine 2%	160-200
Long duration	
Ropivacaine 0.5%-1.0%	240-420
Bupivacaine 0.5%-0.75%	300-460

Adapted with permission from Mulroy MF, Bernards CM, McDonald SB, et al. *A Practical Approach to Regional Anesthesia*. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009.

(2) For thoracic injections, a starting volume of 5 mL is reasonable as anything larger may cause a high block.
(3) The use of an epidural catheter allows titration of the epidural block.
(4) All dosing through an epidural catheter should be incremental and performed only after a negative aspiration for cerebrospinal fluid (CSF) and blood.

iii) Adjuvants
(1) Similar to spinal anesthesia, the addition of epinephrine to local anesthetic prolongs the duration of epidural anesthesia.
(a) The precise mechanism is unknown but may be attributed to decreased clearance of the local anesthetic due to vasoconstriction.
(b) Epinephrine is not recommended as an adjuvant for continuous infusions because of the theoretical safety concerns of spinal ischemia.
(2) Opioids improve the quality of sensory blockade, depending on dose and lipid solubility.[5,6]
(3) Addition of bicarbonate increases the speed of onset of short-acting local anesthetics and is especially beneficial in obstetrics where existing labor epidurals are often converted for use in emergent cesarean deliveries.

iv) Site of action
(1) Local anesthetic solutions injected into the epidural space are thought to act on the spinal nerves traversing the epidural space and the spinal nerve rootlets in the subarachnoid space.

4) **Equipment**
a) A regional anesthetic tray or cart that contains commonly used equipment and local anesthetics is recommended. All personnel performing the block should be familiar with the contents of the tray.
b) Epidural anesthesia should be performed with meticulous sterile technique with sterile skin cleansing solution, sterile gloves, hat, mask, and large clear sterile fenestrated drape.
c) **Epidural needle and catheter**
i) Epidural needles typically have a slightly curved tip (eg, Tuohy).
ii) Needles are usually large enough to pass a catheter, most commonly 17-18 gauge.
iii) Epidural catheters may be single or multiorifice.
d) Low-friction syringe filled with preservative-free saline, air, or a combination of both.
e) Emergency airway equipment and resuscitation drugs should be immediately available.

5) **Technique**
a) **Monitoring**
i) Epidural anesthetics should be administered in a monitored location.
ii) Supplemental oxygen should be provided if sedation is utilized.
iii) For labor analgesia
(1) Sedation is not usually administered.
(2) The ASA recommends that the parturient's vital signs and the fetal heart rate be monitored and documented, with additional monitoring based on the clinical condition of the patient or fetus.[7]

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b) **Positioning and preparation**
i) Patients are typically positioned lateral decubitus or sitting.
(1) Lateral decubitus
(a) The patient's back should be at the edge of the bed, while keeping the shoulders and hips perpendicular to the bed to minimize rotation of the spine.

(b) The knees are drawn upward toward the chest, with neck flexion, producing an arch of the back.

(2) Sitting

(a) May help identify midline, particularly in obese patients

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Spinal anesthesia, Chapter 41

ii) The anticipated injection site is prepared with appropriate antiseptic solution and draped in sterile fashion.

(1) Prepare a wide area to facilitate any subsequent attempts at another insertion point.

(2) Allow the antiseptic solution to dry and wipe away any excess.

c) **Identification of surface anatomy**

i) The most prominent spinous process at the cervical level is C7.

ii) A line drawn between the tips of the scapula estimates T7, while one drawn between the iliac crests (Tuffier line) estimates L4 (**Fig. 42-1A**).

iii) Using the index and middle finger side by side, palpate the spinous processes by sliding side to side and up and down the midline.

d) **Paramedian approach**

i) May be used for any level but commonly applied to thoracic epidurals.

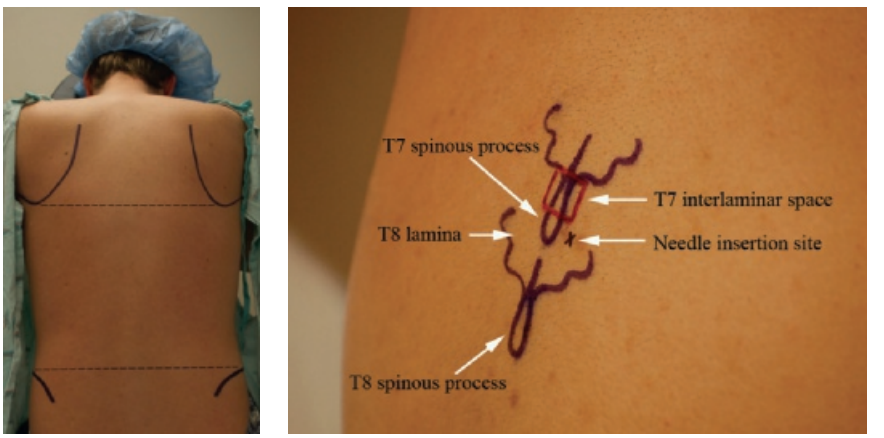
ii) Palpate the desired interspace, and after warning the patient, infiltrate the skin with local anesthetic ~1 cm lateral to the inferior aspect of the superior spinous process (**Fig. 42-1B**).

iii) To help determine the location and depth, inject along the desired pathway of the epidural needle to the lamina with the intent to contact bone (lamina and confirming spinous process as midline) while at the same time injecting local anesthetic.

iv) Insert the epidural needle perpendicular to the skin at the infiltration site until lamina is contacted.

(1) Direct the needle medially along the lamina by intermittently withdrawing the needle and redirecting it towards midline (**Fig. 42-2**).

FIGURE 42-1 Landmarks for Placement of Epidural Anesthesia



A. A line drawn between the tips of the scapula estimates T7. **B.** Thoracic epidural landmarks.

FIGURE 42-2 Epidural Needle Inserted in a Controlled Fashion



(2) If the depth decreases, this likely represents the juncture between the lamina and the lateral aspect of the spinous process.

v) Loss-of-resistance technique with saline (author's preferred technique)

(1) Remove the stylet, and attach a low-friction syringe containing preservative-free saline with or without a small visible air bubble (**Fig. 42-3**).

(2) Stabilize the epidural needle by resting the dorsum of the nondominant hand against the patient's back while grasping the needle between the thumb and the index finger.

(3) The epidural needle is then redirected in the cephalad direction and "marched" off the lamina, eventually into the ligamentum flavum. Note that the purpose of initial needle placement is not engagement of the ligamentum flavum immediately but rather to contact lamina and "walk" the needle off of the bony protection of the spinal cord. Accidental placement of the needle in the intrathecal space can occur if advanced too far, with subsequent loss of resistance becoming an inadvertent spinal tap.

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(4) Apply continuous pressure on the syringe plunger with the dominant hand while carefully advancing the epidural needle shaft with the nondominant hand. Intermittent small advancements (less than half a centimeter at a time), while assessing for resistance continuously between each advancement, is acceptable.

(5) When the epidural needle tip enters the ligamentum flavum, an increase in resistance is felt. A small air bubble when compressed would become mostly spherical and provides a visual cue in addition to tactile feel.

FIGURE 42-3 Loss-of-Resistance Technique with Saline and Air



(6) When the epidural needle tip subsequently enters the epidural space, a decrease or a loss of resistance is felt as saline is injected into the space.
(7) Stop advancement of the needle.
(8) For a continuous catheter technique, disconnect the syringe, and thread the epidural catheter 3-5 cm into the epidural space.
(9) Advance the catheter into the needle. Withdraw the epidural needle while continuing to advance the catheter in order to ensure the catheter isn't inadvertently removed. Withdraw the catheter to an appropriate final depth after the needle is removed if it is overthreaded.



Do not withdraw the epidural catheter through the needle after it has passed the end of the needle as shearing of the catheter may occur.

e) **Median or midline approach**
   i) Commonly performed for lumbar epidurals.
   ii) The target interspace is identified, and the approach is similar to the midline spinal anesthetic technique.

iii) The epidural needle is inserted in the midline of the target interspace and carefully advanced through the interspinous ligament. Unlike the paramedian technique, bone is often not contacted, and the needle is inserted at a depth less than the anticipated epidural space.

iv) Attach the low-friction syringe containing preservative-free saline, and proceed with the loss-of-resistance technique as described previously.

It is important to note and document the depth of the epidural space, which can be determined by noting the marking of the needle where loss of resistance is encountered. This will help determine how far to leave the epidural catheter within the epidural space.

f) There are other alternative techniques that may be used for identification of the epidural space such as loss of resistance to air, the "hanging drop" method, fluoroscopy-assisted (most commonly utilized for chronic pain procedures), waveform analysis,[8] and confirmation with dural puncture.[9]

g) **Test dose**

i) Aspirate the catheter prior to injecting the test dose to check for CSF or blood.

ii) Injection of up to 3 mL of 1.5% lidocaine with 1:200 000 epinephrine may help detect subarachnoid injection (rapid onset of spinal anesthesia) or intravascular placement (increased HR by ~30 bpm in <1 minute).[10]

iii) Blood pressure measurements may be necessary for patients on nodal or beta adrenergic antagonists,[11] as these patients may not mount a HR response to intravascular epinephrine.

iv) The use of an epinephrine-containing test dose to assess catheter position is controversial in obstetrical patients because the onset of a contraction may increase HR and thus cause a false-positive result. In addition, the higher concentration of lidocaine may result in urinary retention and motor block, which is undesirable in the obstetric population.[12]

Combined Spinal Epidural (CSE)

1) **Indications**

a) **Labor analgesia**

i) Provides rapid onset of analgesia from the spinal component

ii) Improved patient satisfaction in some studies[13]

iii) Lower incidence of motor block, especially if intrathecal opioids are given without local anesthetic (eg, "walking epidural")

b) **Operative procedure of uncertain duration**

i) Placed for the benefit of the fast onset of a spinal anesthetic with the option of extending the duration of anesthesia and analgesia via a catheter technique.

2) **Technique**

a) **Needle-through-needle (Fig. 42-1)**

i) Most common CSE technique

ii) The epidural space is localized with the loss of resistance to saline or air technique as described above.

iii) A spinal needle is placed through the epidural needle.

(1) These needles are longer and of smaller gauge than the usual spinal needles.

(2) It is important that the needle comes as part of a CSE kit or is tested to fit through the epidural needle with appropriate length before starting the procedure.

iv) A characteristic "pop" is often felt and free flow of CSF observed.

v) Spinal medication is administered and the spinal needle removed with care not to move the epidural needle.

Concerns regarding epidural catheter migration through the dural hole after a CSE have not been substantiated when studied.[14]

vi) The epidural catheter is then inserted as described above and secured.

b) **Separate procedures**

i) When the epidural and spinal are performed separately, they can be done at the same or different interspinous spaces.

ii) It is advisable to place the spinal portion first as it is theoretically possible to traumatize a previously placed epidural catheter when the spinal needle passes through the epidural space.



When saline is used for the epidural portion of CSE, it is important to confirm free flow of CSF to avoid the possibility of mistaking saline for CSF, resulting in failed spinal block.

3) **Troubleshooting**

a) **False loss of resistance**

i) When the characteristic loss of resistance is not felt, the epidural needle may have passed obliquely through the ligamentum flavum and into the paraspinous muscle or interspinous cysts.

ii) Using the low-friction syringe, injection of a small amount of air (0.5 mL) can help differentiate between these two scenarios as there still might be a small resistance to air.

iii) Almost no resistance will be felt in the epidural space, while resistance can be felt in the paraspinous muscles.

iv) Most importantly, a loss of resistance may represent any change in tissue interface, including fascial layers, and one can see it entering the paravertebral, subarachnoid, and intrapleural spaces.

v) Passage of a small gauge spinal needle via the epidural needle through the dura and observation of free CSF flow may be a useful confirmatory test of proper epidural needle placement.[9]



Epidural catheters should always be aspirated to rule out intrathecal or intravascular location prior to injection.



Passage of a small gauge spinal needle via the epidural needle through the dura and observation of free CSF flow may be a useful confirmatory test of proper epidural needle placement.

b) **Difficulty threading the catheter**

i) The needle may either be partially in the epidural space, occluded by epidural fat, or incorrectly positioned.

ii) First, confirm appropriate needle placement with loss of resistance to air or saline.

iii) Often, additional fluid or air creates a potential space for catheterization.

iv) Epidural needle rotation without advancement or slight advancement of the needle may be considered since the bevel may only be partially in the epidural space.

c) **Obesity**

i) The optimal position for obese patients is often in the sitting position as it allows better identification of midline.

ii) A special positioning device may be helpful, but an assistant to prevent falls and provide patient encouragement is essential.

iii) Location of interspaces may be estimated, but depth to the spinous process and lamina may be determined by a "finder" or spinal needle used for local anesthetic infiltration.

(1) Outside of the lumbar levels, it is important to direct the needles perpendicular to the skin at first to avoid accidental insertion into the intrathecal space and spinal cord.

iv) Alternatively, ultrasound can also help determine the depth and location of spinous and transverse processes allowing one to plan appropriate trajectories and expected length at correct placement.[15]

v) Ultrasound has not decreased the time to place thoracic epidurals in usual patients;[16] however, its clinical utility has become more popular.

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Lumbar neuraxial ultrasound, Chapter 200

d) **Nonspecific or nonconvincing loss of resistance**
   i) While frustrating, difficulty in determining the quality of a loss of resistance could result from excessive saline or local anesthetic injection before attempt at loss of resistance.
   ii) Using a slightly different directional approach or starting at a new interspace may be warranted.
   iii) In older patients, despite age-related changes of the ligamentum flavum and increasing incidence of spinal disease (eg, spinal stenosis), the loss of resistance is still very reliable.[17]

e) **Failure of spinal portion of CSE (Fig. 42-2)**
   i) "Tenting" of the dura resulting in a lack of definitive dural puncture.[18] This is more likely to occur when the procedure is done in the lateral position due to lower CSF pressure.
   ii) Off midline position, increasing the possibility for the spinal needle to be lateral to the dural sac.[18]
   iii) Spinal needle not long enough to puncture dura.[18,19]
   iv) Mistaken saline for CSF when LOR to saline technique is used.
      (1) Must confirm free flow of CSF in spinal needle before injection.

f) **Failure of epidural portion of CSE**
   i) Untested catheter
      (1) In the event that an epidural catheter is not placed optimally after the spinal portion of a CSE, one may encounter a situation where an epidural top up for analgesia or anesthesia is inadequate after the spinal portion of the CSE has worn off.



For patients for whom the reliability of an epidural catheter is extremely important (eg, those at risk of requiring an urgent/emergent cesarean delivery, such as severe preeclampsia, difficult airway, nonreassuring fetal heart rate tracing), it may be advisable to avoid the CSE technique in favor of epidural placement.

4) **Risks and complications**

a) The incidence and variety of complications related to epidurals and CSEs are similar to those associated with spinal anesthesia.

b) **Hypotension**
   i) Generally, slower onset and more gradual than encountered in spinal anesthesia.
   ii) Management includes administration of intravenous fluids and vasoactive agents such as phenylephrine.

c) **Unilateral epidural anesthesia**
   i) Despite attempts at directing epidural catheters during placement, they are rarely found in the midline position.[20]
   ii) Epidural injectate spread is highly variable.[21]
   iii) A unilateral block may occur if the epidural catheter tip enters an intervertebral foramen or is positioned in the anterior epidural space, especially when inserted more than 5 cm.[20] The catheter may also be in the paravertebral space.
   iv) Uniform anesthesia may sometimes be achieved by increasing the volume of local anesthetic solution, increasing the dosing frequency, or withdrawing the catheter so that only 1-3 cm remains in the epidural space.
   v) When these maneuvers fail, the epidural catheter should be removed and a replacement catheter offered.

d) **Dural puncture**
   i) Given the larger diameter of the epidural needle, inadvertent subarachnoid puncture increases the likelihood of postdural puncture headache.[22]

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Spinal anesthesia, Chapter 41.



The risk of unilateral epidural blockade is higher when the catheter is inserted >5 cm.



If an epidural catheter is not functioning properly and appropriate measures to correct problems have been taken, it is appropriate to offer the patient a replacement catheter.

ii) Most of the time, a dural puncture, also known as a "wet tap," is obvious immediately after the loss of resistance where the plunger of the syringe immediately fills up with fluid. Options then include:
(1) Remove the epidural needle and reattempt placement at a different level.
(a) Most practitioners would dose their epidural conservatively as higher pressures or volumes are thought to migrate through the hole created.[23]
(2) Placement of an intrathecal catheter, using doses appropriate for spinal anesthesia.
(a) May decrease the incidence of postdural puncture headache.[22]
(b) Only recommended at the lumbar level.

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Postdural puncture headache and epidural blood patch, Chapter 43

e) **Intravascular injection of local anesthetics**
i) Epidural catheters, especially when used for surgical anesthesia, may require large volumes of concentrated local anesthetic that may be toxic if injected intravascularly.
ii) Catheters may migrate intravascularly and should be aspirated for blood prior to injection.

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Local anesthetics, Chapter 211

f) **Respiratory depression**
i) Can be secondary to a high level of anesthesia, much like a high spinal.
ii) Intrathecal or epidural narcotics are contributory, with an estimated incidence of 0.01%-0.1% with the use of intrathecal fentanyl and/or sufentanil, and the risks appear to be dose dependent.

g) **Fetal bradycardia**
i) Association of fetal bradycardia with CSE labor analgesia is controversial and likely related to rapid onset of analgesia. Current theories suggest this results in excess maternal circulating catecholamines, thereby increasing uterine tone.[24]

h) **Epidural abscess**
i) Rare complication that may be associated with prolonged epidural catheter duration.[25]
ii) Symptoms
(1) Usually presents several days after the epidural catheter is removed
(2) Severe backache with tenderness, fever, headache, sensory and motor deficits, and bladder incontinence
(3) Erythema at catheter site and increased white blood cell count may be observed.
(4) Symptoms should prompt rapid diagnostic MRI scan.
iii) Treatment
(1) Surgical decompression via laminectomy
(2) Treatment must be initiated as soon as possible for optimal outcome

i) **Epidural hematoma**
i) This rare complication can lead to permanent paralysis and requires immediate diagnosis and intervention.
ii) Diagnosis and treatment as above for epidural abscess.
iii) Prevention: Avoid performing neuraxial anesthesia in patients receiving anticoagulants, and follow published guidelines.[26]

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Spinal anesthesia, Chapter 41

j) **Persistent neurologic symptoms (see Chapter 41, Spinal Anesthesia, for more information)**

k) Block failure[27]
i) Unlike spinal anesthetics, epidurals have a higher failure rate, possibly from needle misplacement, inadvertent catheter dislodgement, or original incorrect placement.

ii) Furthermore, they are sometimes discontinued postoperatively due to side effects from drugs.
   (1) Opioids may cause pruritus, nausea and vomiting, sedation, and respiratory depression.
   (2) Local anesthetics produce hypotension and may cause lower extremity weakness, thereby increasing the risk of falls.

CHAPTER SUMMARY

Epidural Anesthesia	
Definition	Placement of medications and/or an indwelling catheter in the epidural space, which is between the ligamentum flavum and the dura
Indications	Postoperative analgesia, labor analgesia, and surgical anesthesia
Advantages	Long duration, especially with catheter-based technique; may be used at any level in the spine for more precise location of analgesia; allows the avoidance or reduction of intravenous narcotics
Disadvantages	Technically more difficult than a spinal anesthetic and, slower onset, and a potentially less dense block; higher risk of block failure than spinal anesthesia
Potential Complications	Hemodynamic perturbations, dural puncture, epidural hematoma, or infection (rare)
Combined Spinal Epidural	
Indications	Labor analgesia, long surgical procedure where neuraxial anesthesia or postoperative epidural catheter desired
Complications	May be associated with increased fetal bradycardia[24,28] Rate of complications similar to other neuraxial techniques
Considerations	Epidural catheter is untested; potential for spinal portion to be unsuccessful due to technical issues

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43 Postdural Puncture Headache and Epidural Blood Patch§

Surbhi Malhotra

When the dura is punctured for diagnostic or anesthetic purposes, loss of cerebral spinal fluid (CSF) occurs. In some patients, this results in a postdural puncture headache (PDPH), the symptoms of which range from mild to severely debilitating. It is important for the anesthesiologist to consider the broader differential diagnosis of headache when evaluating a patient with a suspected PDPH.

1) **Pathophysiology**[1]
   a) Dural puncture results in loss of CSF
   b) The pathophysiology and mechanism of postdural puncture headache (PDPH) is poorly understood.
   c) Commonly accepted theories include:
      i) Traction on pain-sensitive structures due to pressure changes in the cranium
      ii) Cerebral venous dilation[1,2]
2) **Symptoms**
   a) **Characteristics**
      i) Onset is usually 12-48 hours after dural puncture but may be delayed and occur up to 7-14 days after the event.
      ii) A PDPH is typically located in the frontal or occipital region.
      iii) It may present with symptoms such as neck pain, visual disturbances (eg, diplopia or photophobia which may be due to cranial nerve palsies), nausea and vomiting, dizziness, and auditory changes.
   b) **Positional changes**
      i) The classic symptom of PDPH is that the headache is relieved upon assuming the supine position and worsened in the upright position.



The symptoms of a PDPH differ from those associated with other etiologies; often the headache is postural and associated with dizziness, auditory changes, visual disturbances, or neck pain.[3] However, in some cases the headache can be atypical and not postural.[4]

3) **Incidence of headache with dural puncture depends on several variables**
   a) **Needle size**
      i) In general, the larger the needle, the higher the incidence of headache.
   b) **Needle type**
      i) Pencil-point needles (Whitacre, Sprotte, and Gertie-Marx) have a lower incidence of headache than cutting-point needles (Quincke).
         (1) For example, a 26-g Quincke needle has a PDPH incidence of 5%, while a 25-g Whittacre needle has an incidence of ~1%.[5]
   c) **Bevel direction**[6]
      i) If a Quincke needle is used, care should be taken to orient the bevel of the needle with the longitudinal axis of the spine as this decreases the incidence of headache.

d) **Patient characteristics**
   i) Female patients and/or those younger than 40 years have a higher incidence of PDPH than male patients and those over 60 years old.
   ii) Morbidly obese patients have a lower incidence of PDPH.



Always consider the differential diagnosis of headache. Never assume that a headache is PDPH until other causes are ruled out by a thorough chart review, history, and physical examination.

4) **Patient evaluation**
   a) The patient must have a thorough history and physical examination.
   b) The onset, characteristics, severity, and exacerbating factors should be determined.
   c) A thorough chart review should focus on co-existing diseases and vital signs (especially temperature and blood pressure). Lab investigations should be considered when appropriate.
   d) Physical examination should be focused on ruling out focal neurologic deficits.
5) **Differential diagnosis**
   a) **Pre-eclampsia**
      i) Hypertensive disorders of pregnancy are often accompanied by headache.
   b) **Tension headache**
      i) Patients may have muscle tension headaches from prolonged positioning in bed and/or straining during labor.
   c) **Caffeine withdrawal**
      i) Patients who regularly consume caffeine may experience headache when caffeine is withheld.
   d) **Fatigue**
      i) Sleep deprivation and stress may contribute to fatigue, which may cause or exacerbate other causes of headache.
   e) **Pneumocephalus**
      i) Immediate onset of severe headache can be associated with pneumocephalus due to use of the loss of resistance to air technique for localizing the epidural space and subsequent unintended dural puncture. Because of this, many practitioners do not recommend loss of resistance to air techniques.
   f) **Subarachnoid hematoma**
      i) Sudden onset of severe headache and/or accompanying neurologic deficits may be the result of a ruptured cerebral aneurysm and/or arteriovenous malformation.
   g) **Venous sinus thrombosis**
      i) Peripartum patients are at higher risk for this condition, and many will have undergone dural puncture for labor analgesia or cesarean delivery.
      ii) This type of headache will likely not be positional or may start as a positional headache and lose this feature.
      iii) Venous sinus thrombosis should be considered in the differential diagnosis for patients with worsening headache or those whose headache is not improved by epidural blood patch.
   h) **Subdural hematoma**
      i) Decreased CSF volume from dural puncture rarely may result in bleeding from cerebral vessels.
   i) **Migraine**
      i) In patients with a history of migraine headache, it is important to differentiate between the patient's typical migraine symptoms and PDPH. The postural nature of PDPH often is a differentiating factor.
   j) **Meningitis**
      i) The presence of fever and nuchal rigidity is an ominous sign in a patient with headache.
         (1) Antibiotic therapy must be started in a timely manner.

6) **Conservative treatment**
   a) **IV and/or oral hydration**
      i) While widely prescribed for treatment of PDPH, there is no evidence supporting the benefit of hydration for PDPH.[6]
   b) **Caffeine**
      i) The effectiveness of caffeine has been debated, but studies have shown benefit to oral and IV caffeine.[7,8]
      ii) This is only the case when systemic caffeine levels are sufficient. The headache reoccurs when caffeine levels decrease.
      iii) A significant amount of caffeine needs to be consumed for caffeine to be of any benefit.
         (1) Caffeine is a central nervous stimulant with side effects of insomnia, arrhythmias, and seizures and should not be used for long-term therapy.



In postpartum patients with PDPH, patient safety and the presence of social support are important concerns as they often are sleep deprived and have children to care for at home.

   c) **Supine position**
      i) Maintaining the supine position after dural puncture is not effective in preventing or treating PDPH but is effective in symptom relief.
   d) **Sphenopalatine ganglion blocks**
      i) There is a growing interest in the use of sphenopalatine ganglion blocks for treatment of PDPH.
      ii) This technique is relatively noninvasive and used for treatment of migraines and facial pain.
      iii) The current evidence for sphenopalatine blocks for PDPH treatment is retrospective, in the form of case series and with variable results in terms of efficacy.[9,10]
      iv) Patients often subsequently require an EBP.
   e) **Other treatments**
      i) There are a number of other treatments described in the literature, many of which are controversial and not widely used due to insufficient evidence.
   f) **Epidural blood patch**
      i) The most consistently effective and well-studied treatment for PDPH is an epidural blood patch (EBP).
      ii) It is considered to be the gold standard (see Section 8).[11]
7) **Natural course of PDPH**
   a) In most patients, the headache resolves between 1 and 4 weeks.
   b) Performance of daily activities is impaired in many patients.
   c) Rarely, symptoms can last much longer and are associated with chronic pain and headache syndromes.[12]
      i) The importance of this in parturients cannot be emphasized enough, as this patient population expects to care for an infant and often other children.

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Epidural and Combined Spinal Epidural Anesthesia, Chapter 42
Lumbar epidural placement, Chapter 170

8) **Epidural blood patch**
   a) **Effectiveness**
      i) The reported success rate of EBP varies significantly. In one study, the success rate of EPB in a heterogeneous population that included parturients was 75%.[13]
         (1) If a second blood patch is needed, the success rate is ~90%-95%.
         (2) It is important to counsel patients that there is a high possibility of requiring a second and even third epidural blood patch.[14]
      ii) There is some evidence that when the EBP is delayed for 24 hours after dural puncture, it is more effective.[15] However, this delay may cause significant distress to a mother. A more effective management plan is to perform an EBP early and counsel the patient that a second patch may be required.[14]

b) **Informed consent**
   i) The patient should be counseled about the possibility of repeat dural puncture.
   ii) One of the most common complaints after EBP is back pain. The incidence and association of long-term back pain associated with EBP is unknown, but a retrospective study showed that early EBP may reduce the incidence of chronic back pain by 32%.[15]
   iii) Otherwise, the risks are equivalent to the risks of epidural placement, including inadvertent dural puncture.


Patient follow-up after PDPH diagnosis is essential.

c) **Performing EBP**
   i) Meticulous sterile technique must be maintained.
   ii) Full monitoring and IV access should be utilized.
   iii) Many patients are not comfortable in the upright position, so the lateral position may be used.
      (1) In patients with potentially difficult EBP, the upright position should be considered because landmarks are easier to identify in this position. Care should be taken to minimize the time the patient spends in this position.
   iv) The epidural space is localized.
   v) Blood is obtained under sterile conditions by an assistant and passed to the person performing the epidural.
   vi) After aspiration for blood and CSF, the blood should be injected in 5-mL increments to a total of 20 mL. There is no evidence that >20 mL is more effective.[16]
   vii) The patient should then lie flat for ~1-2 hours, after which he or she is reassessed for resolution of headache.[17]
   viii) In the days following the procedure, the patient should be advised against heavy lifting and Valsalva maneuvers, which may dislodge the clot.
   ix) Patient follow-up is extremely important in order to monitor for complications and ensure resolution of the headache.

d) **EBP through an epidural catheter**
   i) The effectiveness of prophylactic EBP performed through an epidural catheter in parturients with unintended dural puncture has been examined in two studies; the results are conflicting.[18,19] The incidence, severity, and need for an EBP were reduced significantly in the most recent study.[19]
   ii) Given this conflicting information, it is recommended that EBP through an epidural catheter be performed only in patients whose epidural catheters were extremely difficult to place and are at high risk for repeat dural puncture with EBP.

9) **Contraindications to EBP**
   a) **Relative**
      i) Immunosuppression, systemic infection, coagulation disorder
   b) **Absolute**
      i) Patient refusal


If an intrathecal catheter is placed after an accidental dural puncture, extreme caution must be taken. The catheter must be clearly labeled and its presence communicated with all members of the care team.

10) **Other treatments**
   a) **Intrathecal catheter**
      i) There are a number of observational studies and meta-analyses investigating the placement of epidural catheter in the intrathecal space as a means to reduce the incidence of PDPH and subsequent need for EBP. Most of these studies are of poor quality and the results are mixed[20–22] with regard to the overall benefit of an intrathecal catheter in this situation.

ii) Extreme caution must be taken when an intrathecal catheter is in place.
   (1) Communication to all staff (nursing, obstetrics, and anesthesia) about the presence of an intrathecal catheter is paramount.
   (2) Clearly label the catheter as intrathecal in order to prevent the administration of medications intended for intravascular or epidural administration.
   (3) Care must also be taken to prevent infection.
   (4) The catheter should be managed by an anesthesiologist.
   (5) The anesthesiologist should be aware that intrathecal catheters have a high failure rate due to catheter dislodgement and/or kinking.



Patients with a perioperative or postpartum HA require a thorough evaluation to eliminate causes other than CSF leak from dural puncture.

11) **Managing a patient with a PDPH in whom EBP has been ineffective**
   a) In the vast majority of patients with PDPH, two blood patches will result in resolution symptoms.
   b) Indications for imaging studies and/or consultation with a neurologist include:
      i) Unresolved headache after two EBPs
      ii) Worsening headache severity
      iii) Changing characteristics (including loss of positional nature)
      iv) Fever
      v) Associated sensory or motor deficits.

CHAPTER SUMMARY FOR PDPH AND EPIDURAL BLOOD PATCH

Patient Characteristics	↑ risk in women, age <40 ↓ risk with obesity
Signs and Symptoms	Positional (improved with supine position) Frontal/occipital Throbbing Associated photophobia, double vision, nausea/vomiting
Differential Diagnosis	Must always be considered and patient thoroughly evaluated Includes musculoskeletal headache, caffeine withdrawal, migraine, meningitis, subarachnoid hemorrhage, and subdural hematoma
Treatment	Conservative measures have limited effectiveness Epidural blood patch is most effective

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44 Overview of Single-Injection and Continuous Peripheral Nerve Blocks

Russell R. Kinder • Robert L. Hsiung • Vanessa J. Loland

A peripheral nerve block (PNB) is the placement of local anesthetic next to a target nerve, plexus, or within a fascial plane to inhibit neural transmission. A continuous peripheral nerve block (CPNB) can provide lasting analgesia with local anesthetic infusion or repeated boluses.

1) **Basics of peripheral nerve blocks**
   a) **Peripheral nerve anatomy**
      i) The brachial plexus (C5-T1 anterior rami) innervates the upper extremity (**Figs. 44-1 and 44-2**).
         (1) Interscalene and supraclavicular blocks target the proximal upper extremity.
         (2) Infraclavicular and axillary blocks target the distal upper extremity.
      ii) The lumbar plexus (L1-L4 anterior rami, **Figs. 44-3 and 44-5**) and sacral plexus (L4-S4 anterior rami, **Figs. 44-4 and 44-5**)) innervate the lower extremity.
         (1) The lumbar plexus gives off the femoral, obturator, lateral femoral cutaneous, genitofemoral, ilioinguinal, and iliohypogastric nerves.
            (a) The lumbar plexus and branches can be blocked together or individually.
         (2) The sacral plexus contributes the sciatic nerve and the posterior femoral cutaneous nerve to the leg/thigh.
            (a) The sciatic nerve can be blocked anywhere along its course.
      iii) Intercostal nerves innervate the trunk and abdominal wall.
         (1) Intercostal, paravertebral, and pectoral blocks target the chest wall.
         (2) Rectus sheath and transversus abdominis plane blocks target the abdominal wall.
   b) **Local anesthetics and additives** (**Table 44-1**)
      i) Local anesthetics are used in varying concentrations and volumes to achieve the desired block density, onset, and duration.

READ MORE
Local Anesthetics, Chapter 25

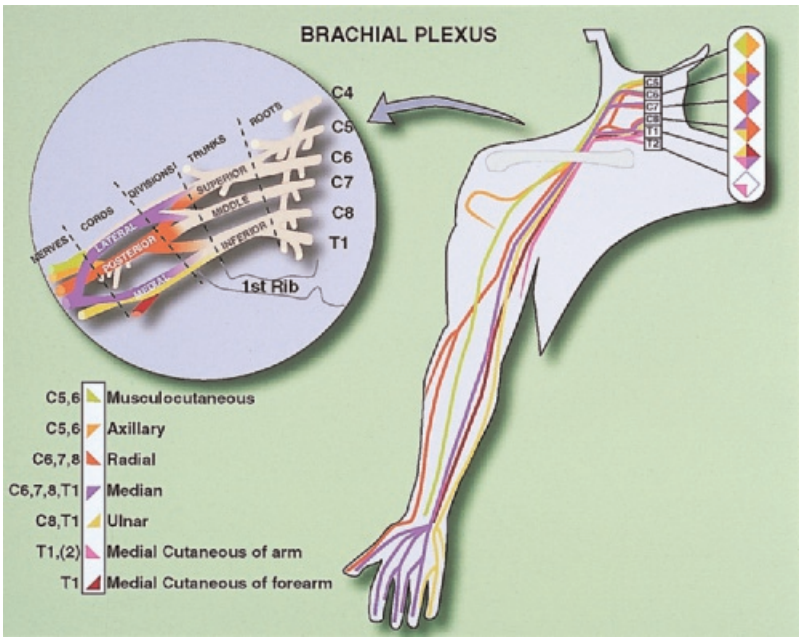
      ii) Additives[1]
         (1) The vasoconstrictor epinephrine prolongs block duration, delays systemic absorption of local anesthetics, and helps identify intravascular injection.
            (a) May increase the risk of neuronal injury
         (2) Sodium bicarbonate hastens neuronal uptake of intermediate-acting local anesthetics.
            (a) The alkalinizing effect can crystallize bupivacaine and ropivacaine.



Alkalinization of bupivacaine and ropivacaine with sodium bicarbonate can lead to precipitation and is not recommended.

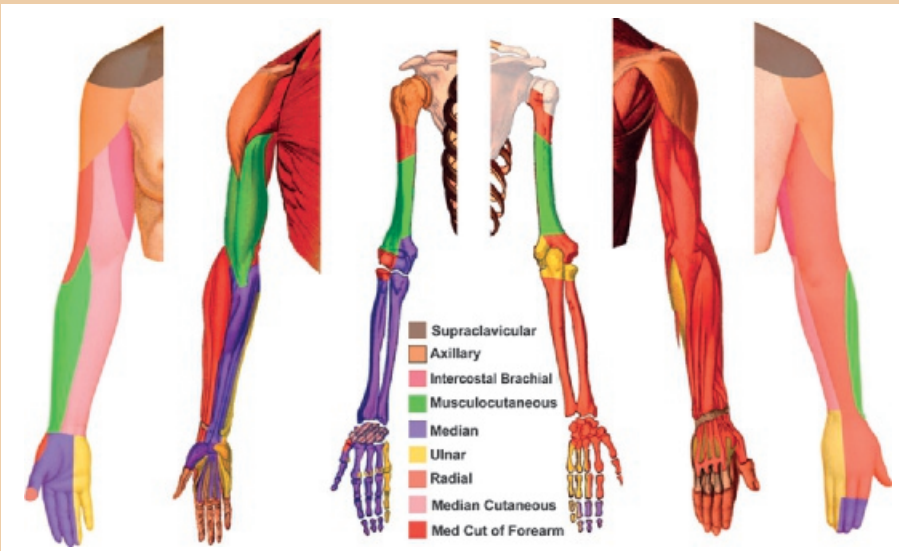
         (3) Block duration is prolonged by α-2 agonists (dexmedetomidine, clonidine),[2] opioid agonist/antagonists (buprenorphine, tramadol, naloxone),[3–5] and the steroid dexamethasone.[4,6]
         (4) Liposomal bupivacaine offers extended drug delivery, but its use in peripheral nerve blockade remains under investigation.[7,8]

FIGURE 44-1 Illustration of the Brachial Plexus From Roots to Branches.



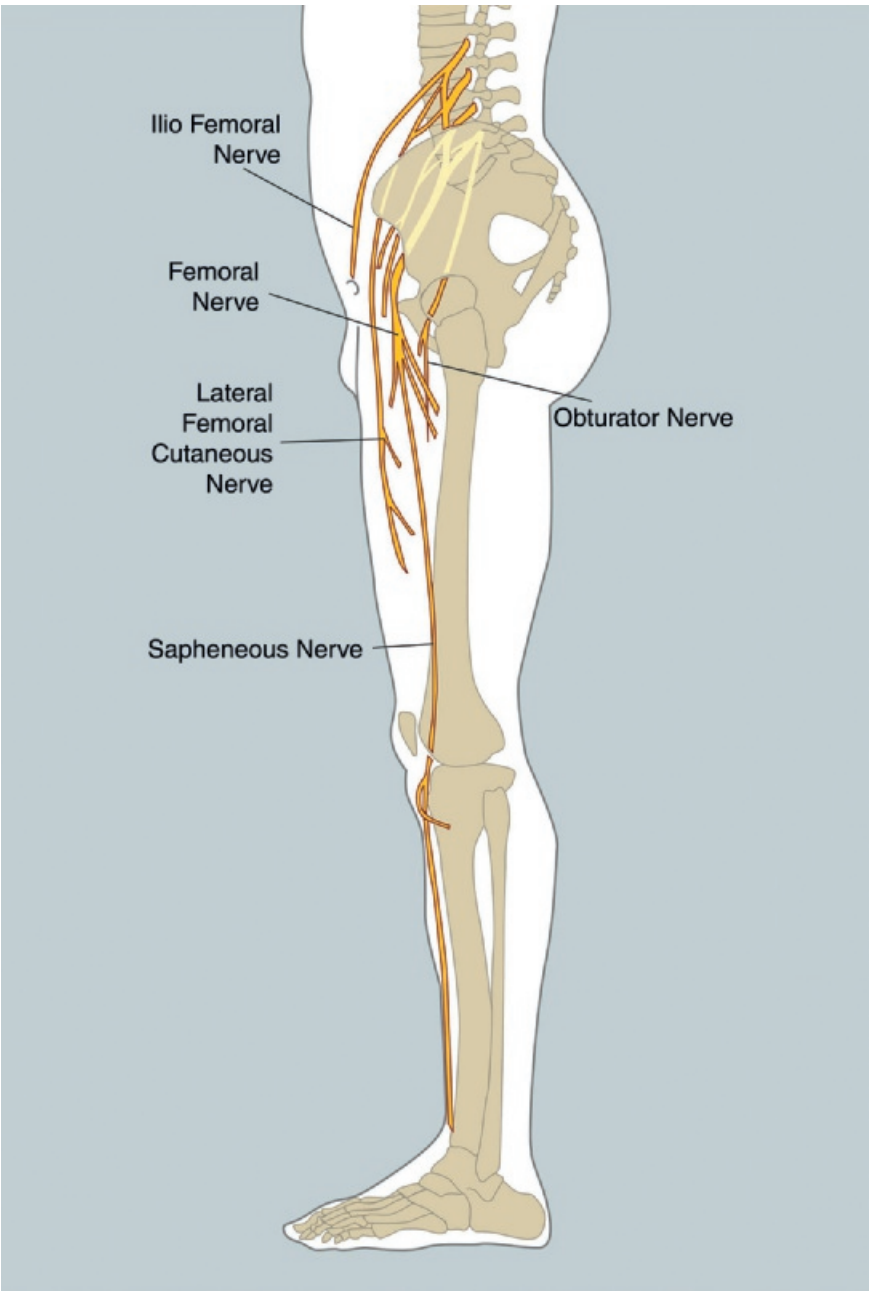
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FIGURE 44-2 Innervation of the Upper Extremity by Individual Peripheral Nerves.



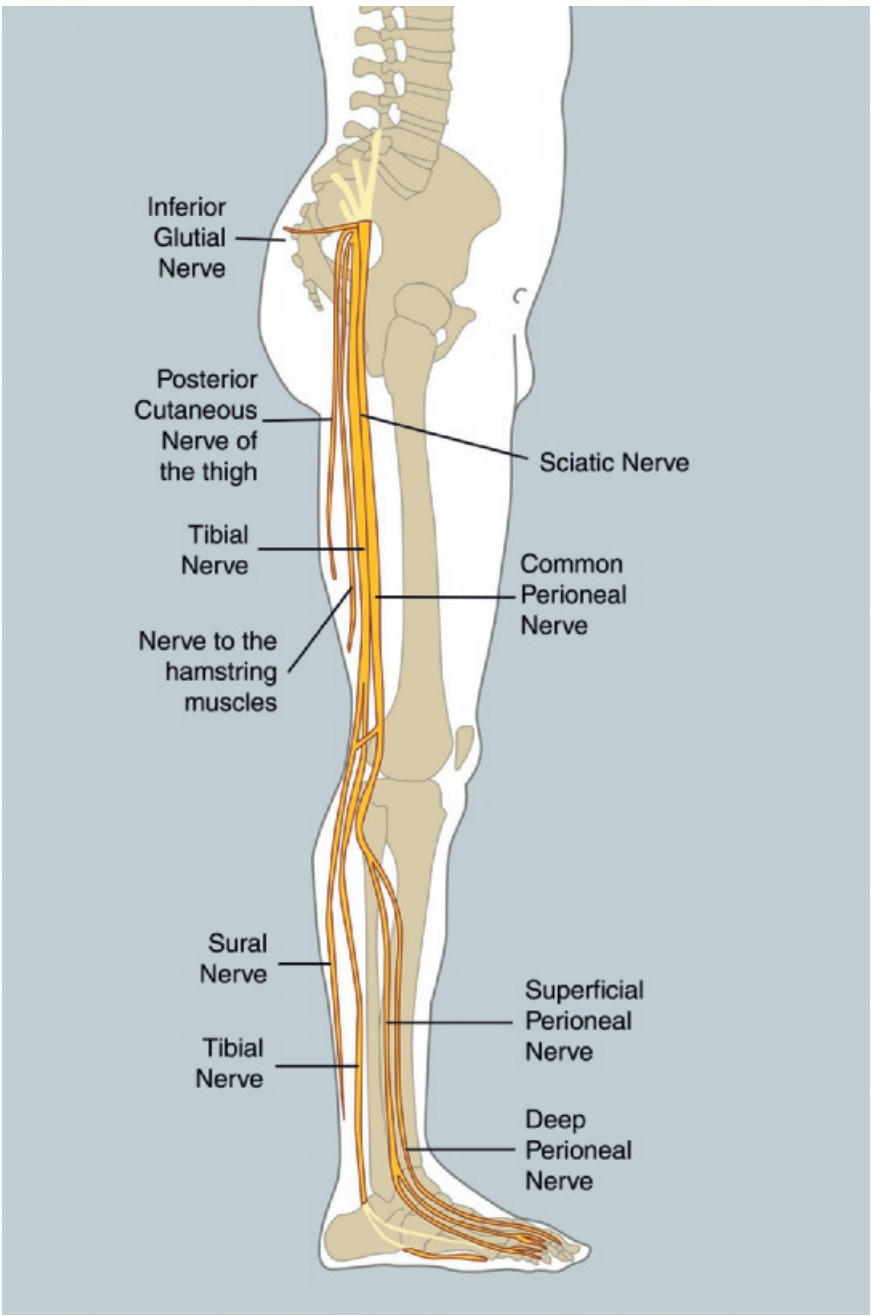
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FIGURE 44-3 Branches of the Lumbar Plexus Important in Lower Extremity Innervation.



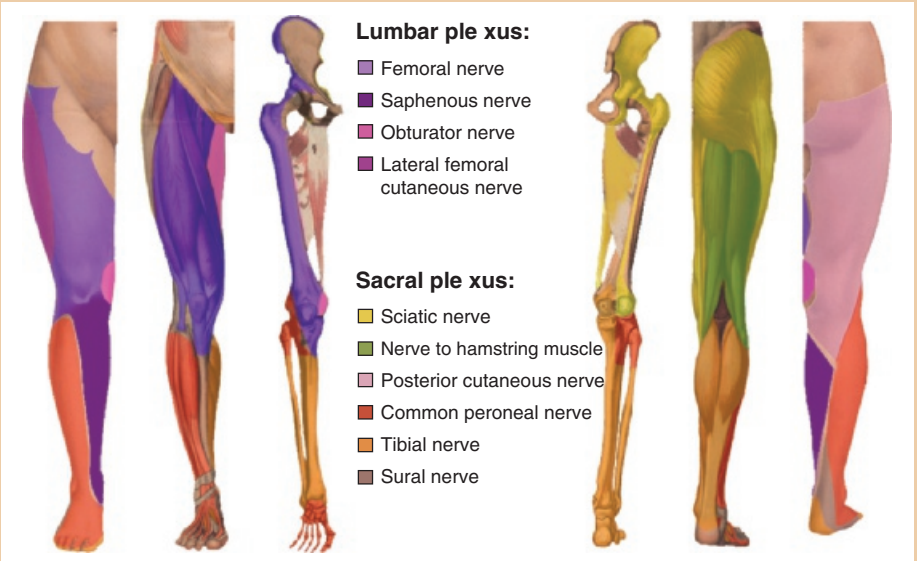
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FIGURE 44-4 Branches of the Sacral Plexus Important in Lower Extremity Innervation.



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FIGURE 44-5 Innervation of the Lower Extremity by Individual Peripheral Nerves.



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2) **Benefits and indications**
   a) **PNBs can provide surgical anesthesia and/or postoperative analgesia.**
      i) Can be used alone or in conjunction with other anesthetic techniques
      ii) As a primary anesthetic, PNBs can bypass potential complications of general anesthesia such as difficult airway, hemodynamic instability, and malignant hyperthermia.
      iii) PNBs—and especially CPNBs—provide better analgesia, greater patient satisfaction, decreased pruritus, decreased nausea/vomiting, improved sleep, and decreased length of hospitalization compared with opioids alone.[9]
      iv) There is no conclusive mortality advantage to PNB, and there is limited evidence of benefit in long-term functional outcomes and development of chronic pain.[10]
      v) CPNB may be offered to patients expected to experience at least moderate postoperative pain for at least 24 hours that will not be easily managed with oral opioids.
         (1) CPNB may help patients avoid hospitalization for pain control since they can be managed on an outpatient.[11]
         (2) CPNB may be appropriate following mildly painful procedures in patients intolerant of opioid-induced side effects.
         (3) CPNB can be used in select cases (eg, limb salvage or vascular insufficiency) to optimize perfusion to a specific area via preganglionic β-fiber sympathectomy.[12,13]
         (4) CPNB can be used to facilitate physical therapy.[14]
         (5) Recommended CPNB catheter locations for common surgical procedures are listed in **Table 44-2**.
3) **Risks and contraindications**
   a) General risks include neurologic injury, bleeding, infection, and local anesthetic toxicity. Additional site-specific risks are discussed in their respective chapters but

TABLE 44-1 Table of Common Local Anesthetics (LA) Used for Peripheral Nerve Blocks

	Common Local Anesthetics	Concentrations (%)[a]	Onset (min)[b]	Block Duration (h)[b]	Max Dose w/epi (mg/kg)[c]	Comments and Recommendations
Short-acting	Chloroprocaine/procaine	1-3	7-15	0.5-1.5	15	Good for short-term surgical anesthesia but not for post-op analgesia
Intermediat-eacting	Lidocaine	1.5-2	10-20	1-4	7	Works well for initial dose when a continuous infusion of long-acting LA is planned
	Mepivacaine	1.5-2	10-20	2-5	7	Like lidocaine, can be used as initial dose before a continuous infusion is started
Long-acting	Bupivacaine	0.25-0.5	10-30	10-14	3	Good choice for post-op analgesia as a single dose or a continuous infusion
	Ropivacaine	0.2-0.5	10-30	10-14	2.5	Less-potent alternative to bupivacaine. 0.2% is a popular choice for continuous infusions

[a]Concentration and volumes used depend on desired effect (eg, anesthesia vs analgesia), location of the block, and nerve localization technique used. The lower concentrations are typically used for continuous infusions.
[b]Onset time and block duration depend on dose of LA, block location, and the use of adjuncts.
[c]Actual plasma concentration can vary greatly, with toxicity being possible at much lower doses.

TABLE 44-2 Recommended Catheter Locations and Infusion Regimens for Common Surgical Procedures

Catheter Location	Planned Surgical Site
Interscalene	Shoulder, humerus
Supraclavicular/infraclavicular	Elbow and distal upper extremity
Lumbar plexus	Hip, thigh
Femoral	Thigh, knee, leg
Sciatic (including popliteal)	Ankle, foot
Paravertebral	Breast, thorax, abdomen

may include vascular puncture, pneumothorax, local anesthetic spread to neuraxial structures, and diaphragmatic paralysis.

i) Risks and benefits must be discussed with the patient or proxy.
ii) A thorough preoperative evaluation must be performed prior to any anesthetic technique.
iii) Motor block is an expected outcome of most PNBs. Careful patient selection and education can help limit problems such as falls.

b) **Absolute contraindications**
   i) Patient refusal or inability to consent
   ii) Allergy to local anesthetic
   iii) Active infection at site of planned needle insertion
   iv) Severe coagulopathy
      (1) Patients with acquired disorders of coagulation are usually not good candidates for PNB.

c) **Relative contraindications**
   i) Distant or systemic infection
   ii) Preexisting neuropathy

d) **Minimizing neurologic injury**
   i) The rate of nerve injury is approximately 2-5 per 10 000 blocks.[15]
   ii) Categories
      (1) Traumatic (mechanical and injection injury)
      (2) Ischemic (vascular)
      (3) Neurotoxic (chemical)
   iii) Certain operations, patient characteristics, tourniquet use, and positioning may predispose patients to nerve injuries, even without nerve blocks.
   iv) The incidence of nerve injury is relatively low, so prospective data comparing specific risk factors for injury are limited.
   v) Direct visualization using ultrasound may minimize needle-to-nerve trauma but does not reliably reduce peripheral nerve block-related neurologic complications.[16,17]
      (1) A motor response may not be elicited even with intraneural needle placement, so stimulation-only techniques can result in additional unnecessary needle passes.[18,19]
   vi) High pressure or resistance with injection may indicate intraneural placement and may contribute to injury.[20,21]
      (1) The rate of unintentional intraneural injection is around 15% and depends on the type of block.
   vii) Nerve blocks performed under general anesthesia

(1) Adverse outcomes have been reported.
(2) In some patient populations (eg, pediatrics), the benefits of PNB under general anesthesia may outweigh the risks.[22]
(3) Some trunk blocks carry a low risk of nerve injury and are commonly performed under general anesthesia.

e) **Minimizing the risk of bleeding**[23]
i) The risk of bleeding complications after PNB is not well defined.
ii) Consider patient factors and procedural factors.
(1) The American Society of Regional Anesthesia guidelines for regional anesthesia in anticoagulated patients can inform safe patient care.
(2) PNBs in noncompressible areas (eg, paravertebral or lumbar plexus) should be avoided in coagulopathic patients.
(3) Superficial PNB are considered relatively safe due to site compressibility.
iii) Ultrasound guidance can reduce the risk of vascular puncture.



Avoid PNBs in non-compressible areas (eg, paravertebral and lumbar plexus) in anticoagulated patients, as the risk for bleeding complications is increased.

f) **Avoiding infection**[24]
i) The risk of infection from PNB is low.
(1) May be increased with indwelling catheters
(2) Catheters retained longer than 4 days are at increased risk.[25]
ii) Sterile technique decreases infection rate.
iii) Risks and benefits should be carefully weighed in the setting of preexisting infection.

g) **Systemic toxicity from local anesthetics**
i) Toxicity from local anesthetics primarily affects the cardiovascular and central nervous systems (CNS).
(1) Cardiovascular: arrhythmia, hypotension, cardiovascular collapse
(2) CNS: oral numbness, dizziness, visual disturbances, seizure, coma
ii) Most cases of systemic toxicity have been attributed to inadvertent intravascular injection of local anesthetic.
iii) Dosing guidelines are discussed separately.
iv) The Local Anesthetic Systemic Toxicity (LAST) Checklist should be easily visible in any location where PNBs are performed.
(1) A crash cart with resuscitative drugs, intralipid, and airway supplies should be available.
(2) Access to cardiopulmonary bypass (CPB) can be life-saving.

READ MORE
Local anesthetics, Chapter 25; Appendix of drug information, Chapter 211

READ MORE
Emergency manual: local anesthetic toxicity

h) **Risks specific to CPNB**
i) The incidence of nerve injury following CPNB is likely no higher than with its single-injection counterpart.[26]
ii) Inadvertent catheter dislodgement: 0%-30%
(1) Catheter tunneling, use of sterile adhesive glue, and catheter anchoring decrease risk (**Fig. 44-6**)
iii) Secondary block failure (ineffective catheter infusion after successful initial placement): 0%-40%
iv) Catheter knotting, retention, or shearing[27]
(1) Avoid feeding the catheter >5 cm past the needle tip.
(2) Avoid withdrawing the catheter once it has been fed past the needle tip.
v) Infection
(1) Colonization is common but infection is rare.[28]
(2) Infusions lasting >3 days require additional vigilance.
(3) Ambulatory infusion duration is generally limited by the local anesthetic delivery system.

FIGURE 44-6 Example of a Properly Dressed Perineural Catheter Employing Liquid Adhesive, Clear Occlusive Dressings, and an Anchoring Device.



vi) Falls
(1) Highest risk with lower extremity CPNB, though all patients are at risk
(2) See "Patient Education for PNB" below.

4) **Equipment and setup**
a) **A regional anesthesia cart or tray streamlines care.**
i) Saves time
ii) Maintains cleanliness
iii) Local anesthetics, rescue medications, and supplies available
b) **Nerve blocks should be performed using sterile technique.**
i) Sterile prep with chlorhexidine, iodine, or alcohol-based cleaning solution
ii) Sterile gloves, mask, hair covering, and drapes (**Fig. 44-7**)
c) **Nerve stimulation requires an insulated needle, nerve stimulator, and grounding electrode.**
d) **Ultrasound guidance requires an ultrasound machine with appropriate transducer, sterile probe cover, and ultrasound gel.**
e) **Needle and catheter selection based on preference**



Patients with lower extremity perineural catheters and continuous local anesthetic infusions should be considered high fall risk.

READ MORE
Atlas of individual nerve block procedures, Section 15.

FIGURE 44-7 Popliteal-Sciatic Perineural Catheter Insertion Demonstrating Proper Sterile Technique. Note the use of a clear fenestrated drape that allows the practitioner to operate the nerve stimulator and observe the distal extremity for evoked motor responses.



i) Simulating needle with nonstimulating catheter
ii) Stimulating needle with stimulating catheter
iii) Nonstimulating needle with nonstimulating catheter
(1) An epidural tray could serve this purpose, but trays designed for CPNB are available.

5) **Monitoring and positioning**
   a) **Peripheral nerve blocks should be placed in a location with monitoring capabilities and emergency rescue supplies, even if sedation is not used.**
      i) ASA standard monitors applied prior to the procedure: EKG, pulse oximeter, noninvasive blood pressure, and end-tidal CO_2
      ii) Oxygen source available
   b) **Proper positioning is essential for successful block placement; please see atlas for individual blocks.**
   c) A "time out" reviewing patient identity, allergies, block location and side, planned surgery, NPO status, and special concerns should occur prior to block placement with the responsible anesthesiologist (and primary proceduralist, if different), and a second care provider present.
6) **Nerve localization techniques**
   a) **Paresthesia-seeking technique**
      i) Anatomical landmarks are used to guide the needle insertion site.
      ii) Needle-to-nerve contact elicits a paresthesia in the desired distribution.

(1) Requires patient responsiveness
(2) Proper needle placement may fail to produce a paresthesia.

iii) Needle-to-nerve contact indicated by paresthesia may reflect nerve trauma and can lead to patient discomfort and long-term sequelae.
(1) Use of this technique has declined in favor of more directed localization.

b) **Nerve stimulation technique**

i) Anatomical landmarks are used to guide the needle insertion site.

ii) Placement of an insulated needle near the target nerve elicits an evoked motor response when electric current is applied.
(1) Strength of motor response does not necessarily correlate with proximity to the nerve, although a motor response with current <0.5 mA is a commonly accepted endpoint.
(2) A positive "Raj test" occurs when the motor response ceases with injection of local anesthetic.
(3) Strong motor response with current <0.2 mA is believed to represent intraneural needle placement.[18,29]
(4) Paresthesia may still be elicited.
(5) Purely sensory nerves (eg, saphenous) will not deliver a motor response, but stimulation may induce paresthesia in the corresponding sensory distribution.
(6) Local anesthetics and electrolyte-containing solutions interfere with targeted electrical stimulation.
(a) An alternate localization technique may be required for repeat blocks.
(b) If fluid is needed for hydrodissection, D5W may be used.[30]



If fluid is needed during placement of a nerve block using nerve stimulation, a small amount of D5W may be used.

iii) CPNB using nerve stimulation[31]
(1) Stimulating catheters
(a) Prior to local anesthetic bolus, attach the perineural catheter to a stimulating lead and advance it through the placement needle.
(b) As the catheter passes through the needle, the evoked motor response should be elicited if the tip is maintained near the target nerve.
(c) Local anesthetic may be given after satisfactory catheter placement.
(2) Nonstimulating catheters
(a) Local anesthetic bolus may be given via the placement needle.
(b) Advance the perineural catheter 1-2 cm past the needle tip.

c) **Ultrasound-guided technique (Fig. 44-8)**

i) The goal is to visualize the nerve, needle, and local anesthetic spread.[32]

ii) Currently the most popular method of nerve localization has contributed to the resurgence and popularity of regional anesthesia.[33]

iii) Target nerves can be imaged in short-axis (cross-sectional) or long-axis (longitudinal) views.[34,35]

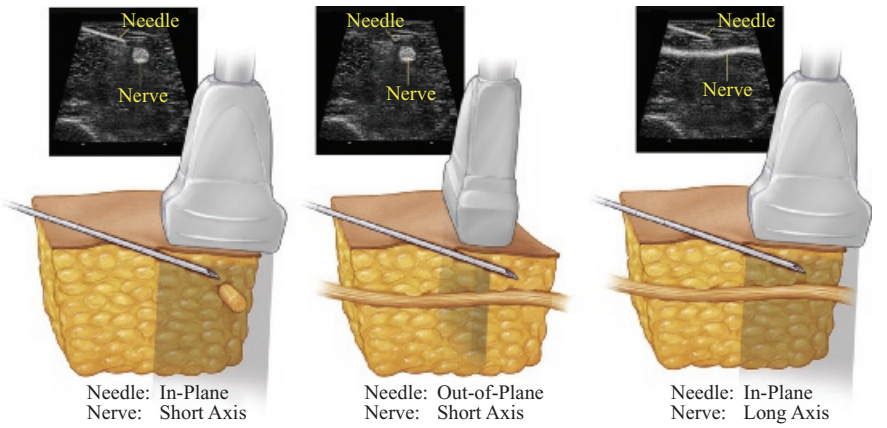
iv) Needle guidance can be performed using in-plane or out-of-plane approaches.
(1) In-plane allows for visualization of the entire needle shaft during placement.
(2) Out-of-plane delivers a cross-sectional image of the needle.

v) Fewer needle passes are needed with ultrasound guidance compared with other techniques.

vi) Real-time ultrasound guidance reduces block performance time and improves success.[36]

vii) CPNB using ultrasound guidance
(1) Proper needle placement is evidenced by local anesthetic spread around the target nerve.

FIGURE 44-8 Illustration Depicting the Various Methods Available to Perform Ultrasound-Guided Peripheral Nerve Blocks. **A.** Nerve in short axis with the needle guided in-plane. **B.** Nerve in short axis with the needle guided out-of-plane. **C.** Nerve in long axis with the needle guided in-plane.



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(2) Catheter may be placed with direct visualization or without.
(a) Final catheter position can be inferred by visualizing fluid spread injected through the catheter or by injecting a small volume (<1 mL) of air through the catheter.

viii) Due to the relatively high initial cost (>$10 000 US), ultrasound is not universally available, but improving technology may mitigate cost in the future.

ix) Learning ultrasound-guided nerve block techniques
(1) Novice learners are more comfortable using ultrasound than other localization techniques.[37]
(a) The most common error is failure to accurately identify the needle during advancement, resulting in inadvertently deep placement.
(b) Inability to visualize the needle tip may result in complications such as intravascular or intraneural needle placement and/or injection.
(c) Inexperienced practitioners are able to rapidly learn and improve using simulation.
(2) Training curricula for learning ultrasound-guided regional anesthesia are available.[38,39]



Final catheter position can be inferred by visualizing fluid spread injected through the catheter or by injecting a small volume (<1 mL) of air through the catheter.

7) **CPNB local anesthetic infusion**
a) **Bupivacaine (0.1%-0.25%) or ropivacaine (0.15%-0.4%) commonly used**
b) **Adjuncts do not provide additional benefit.**[40]
c) **Best regimen is a basal infusion plus patient-controlled bolus (Table 44-3).**
i) Basal infusion improves analgesia and sleep quality.[41]
ii) Bolus capability improves analgesia and lowers local anesthetic requirements.[41,42]

8) **Patient education for PNB**
a) Patient education should be done as part of the consent process, at the preoperative clinic, and at the surgeon's office before surgery.

TABLE 44-3 Recommended Perineural Infusion Regimens

Catheter Location	Recommended Basal Infusion (mL/h)	Recommended Bolus
Interscalene	6*8	4 mL every 30 min
Supraclavicular/infraclavicular	6-8	4 mL every 30 min
Lumbar plexus	4-6	2-4 mL every 60 min
Femoral	4-6	2-4 mL every 60 min
Sciatic (proximal approaches)	4-6	2-4 mL every 60 min
Popliteal-sciatic	6-8	4 mL every 30 min
Paravertebral	4-6	4 mL every 30 min

b) **Important for successful management of CPNB, especially in the outpatient setting**

c) **Patients and nurses should be provided with written instructions.**
   i) Provide a 24-hour contact number for questions and assistance.
   ii) Since outpatients will likely remove their own CPNB catheters (possibly with caretaker assistance), adequate instructions for removal should be provided to patient and family prior to discharge.

d) **Minimizing fall risk**
   i) Use the minimum dose/mass of local anesthetic required for analgesia.
   ii) Limit the volume or time interval of patient-controlled bolus doses or consider a decreased continuous infusion rate.
   iii) Recommend non–weight-bearing status for patients with lower extremity CPNB, or consider using a knee immobilizer or walker/crutches if weight bearing is permitted.
   iv) Educate physical therapists, nurses, and surgeons about possible CPNB-induced muscle weakness and encourage fall precautions at home and on the hospital ward (ie, signs, bed alarms, use of bedside commode).



Use the smallest effective dose of local anesthetic required for analgesia to minimize the risk of falls.

e) **Follow-up**
   i) A phone call or patient visit after surgery facilitates communication and can help head off potential problems.
   ii) Work with patients, surgeons, and care teams on continual improvement and feedback to sustain a successful regional block and catheter service
   iii) Create a database or join a registry to track successes and complications.

CHAPTER SUMMARY FOR PERIPHERAL NERVE BLOCK OVERVIEW

Definition	A peripheral nerve block (PNB) is the placement of local anesthetic next to a target nerve, plexus, or fascial plane to inhibit neural transmission. PNBs can be used for surgical anesthesia or postoperative analgesia
Benefits of PNB	• Avoidance of general anesthesia and its concomitant risks (airway complications, aspiration, medication side effects, hemodynamic instability, malignant hyperthermia) • Superior analgesia, greater patient satisfaction, decreased pruritus, decreased nausea/vomiting, improved sleep, and decreased length of hospitalization compared with opioids alone • Improved limb perfusion

Risks of PNB	Vary with the type of block and include: • Neurologic injury • Bleeding • Infection • Local anesthetic toxicity • Catheter-related complications • Site-specific risks
Techniques	• Paresthesia • Nerve stimulation • Ultrasound • Hybrid Once a nerve is located, a single injection of local anesthetic medication can be administered or a catheter can be left *in situ* for continuous infusion of local anesthetic medication
Post-placement management	• Infusion dosing • Patient education • Follow-up

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45 Part A: Vascular Diseases
Primary Hypertension and Anesthetic Considerations

Selina Read

Primary hypertension (HTN) is a significantly prevalent problem in the United States. With the newest definition of HTN, at least 75 million Americans carry a diagnosis. HTN accounts for the leading cause of modifiable cardiovascular deaths.[1] A "normal" BP is defined as <120/80 mm Hg and elevated BP is 120-129/<80 mm Hg. HTN is defined as a SBP ≥ 130 mm Hg or a DBP ≥ 80 mm Hg. Adequate understanding of the pathophysiology, treatment, and complications associated with HTN is essential to anesthetic practice.

1) **General principles**
   a) **Definition: SBP > 130 mm Hg or a DBP > 80 mm Hg**
   b) **Classification (Table 45-1)**[2,3]
      i) Primary HTN
         (1) Must rule out secondary causes of HTN including pheochromocytoma/paraganglioma, Cushing syndrome, thyroid disorders, aortic coarctation, primary hyperthyroidism, adrenal disorders, and acromegaly
         (2) Must rule out medications contributing to elevated blood pressure such as amphetamines, antidepressants, atypical antipsychotics, decongestants, oral contraceptives, immunosuppressants, and corticosteroids
         (3) Primary HTN constitutes 95% cases of HTN[3].
   c) **Risk factors**[2] **(Fig. 45-1; Table 45-2)**
2) **Complications**
   a) **Hypertensive urgency and emergency**[4]
      i) Defined as systolic BP > 180 mm Hg and/or diastolic BP > 110 mm Hg without end-organ damage (urgency) or with the presence of end-organ damage (emergency)
   b) **Perioperative considerations for uncontrolled HTN**
      i) In patients with hypertensive urgency, consider delaying elective and time-sensitive surgery after confirming proper BP measurement technique and patient compliance with antihypertensives prior to surgery.
      ii) Delay elective and time-sensitive surgery in patients with hypertensive emergency to allow further evaluation and management (see Section 4.a).
      iii) In conjunction with the patient's primary care provider, consider increasing or modifying antihypertensive therapy. Beta-adrenergic antagonists should be added in certain patient populations.
      iv) For patients with uncontrolled HTN having emergency surgery, consider invasive monitoring of perioperative hemodynamics (arterial line, central line, and possibly echocardiogram).



Persistent HTN causes remodeling of small arteries, increased vascular wall tension, and an enlargement of the medial layer of vascular wall thickness. These changes result in a chronic vascular resistance elevation, shifting autoregulatory curves and causing an exaggerated response to vasopressors[4]. Chronic HTN contributes to hypertensive heart disease, left ventricular HTN, and diastolic dysfunction.

TABLE 45-1 Classification of HTN

BP Classification	Systolic BP (mm Hg)	Diastolic BP (mm Hg)
Normal	<120	<80
Elevated—check	120-129	<80
Stage 1 HTN	130-139	80-89
Stage 2 HTN	≥140	≥90

c) **End-organ damage**
   i) Cardiovascular
      (1) Coronary artery disease
      (2) Cerebral vascular disease: Transient ischemic attacks, ischemic stroke
      (3) Peripheral vascular disease (PVD)
      (4) Left ventricular hypertrophy (LVH), diastolic dysfunction, congestive heart failure (CHF)
      (5) Retinopathy
   ii) Kidney failure
   iii) Neurologic
      (1) Hemorrhagic stroke, hypertensive encephalopathy



Blood pressures in preoperative clinic or day of surgery should not be used to diagnose HTN—refer the patient to primary care. Diastolic BP elevation may be more ominous than systolic elevations.[5]

d) **Perioperative considerations for patients with end-organ damage**
   i) Detailed preoperative assessment with a focus on end-organ damage should be performed in any patient with primary HTN. Ensuring proper blood pressure cuff size and optimization of patient conditions prior to measurement can help to improve accuracy.[2]
   ii) Appropriate referrals should be made and the patient's medical condition optimized prior to elective surgery.

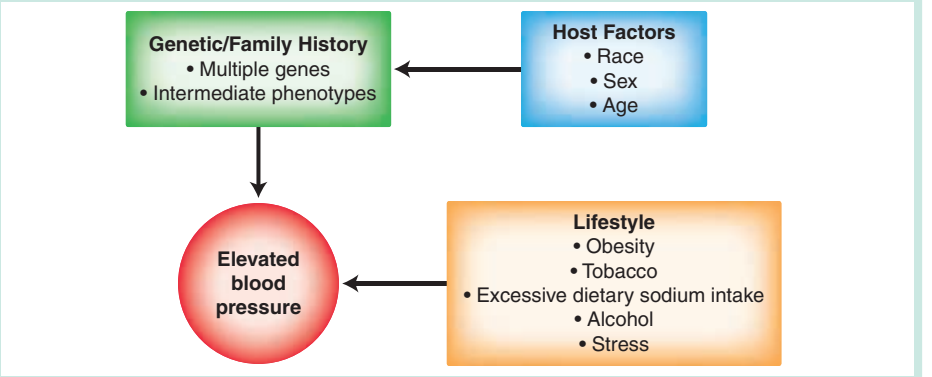
3) **Treatment and management**
   a) **Lifestyle changes**[1]
      i) Weight loss with diet modification and exercise, moderate alcohol consumption, smoking cessation
   b) **Pharmacologic**[1,5]
      i) β-Adrenergic antagonists, thiazide diuretics, calcium channel blockers, renin-angiotensin receptor agents (ARB), angiotensin-converting enzyme inhibitors (ACEIs), alpha-1 receptor antagonists

4) **Anesthetic management of the patient with HTN**
   a) **Preoperative evaluation**[1]
      i) Assess the patient's baseline BP and self-management at home.
      ii) Patients with untreated severe HTN have a higher incidence of stroke, myocardial ischemia, and kidney failure in the perioperative period because of chronic changes in the autoregulation of end-organ perfusion.[3]

TABLE 45-2 Risk Factors for Primary HTN

Increased age	Diabetes mellitus
Obstructive sleep apnea	Chronic kidney disease
Genetic/family history	Tobacco use
Overweight/obesity	Psychosocial stress
Current cigarette smoking and secondhand smoke	Physical inactivity/low fitness
Dyslipidemia/hypercholesterolemia	Low socioeconomic/educational status

FIGURE 45-1 Schematic Presentation of the Multiple Risk Factors that are Associated With Elevated BP.



iii) No clear consensus exists regarding the timing of elective surgery in patients with uncontrolled HTN. However, recommendations according to HTN stage are as follows.[1–3,6]
   (1) Elevated HTN, Stage 1 and 2 HTN: proceed with surgery.
   (2) Hypertensive urgency: consider deferring anesthesia after ensuring proper technique of measuring BP (appropriate cuff size, patient positioning) and patient characteristics (followed preoperative medication instructions; not taking illicit substances/caffeine).
   (3) Hypertensive emergency: postpone all but emergency surgery.

iv) Evaluate for end-organ damage.
   (1) Including myocardial ischemia, LVH, CHF, cerebrovascular disease, stroke, PVD, renal insufficiency
   (2) Laboratory studies[3]
      (a) Complete blood count
      (b) Coagulation studies
      (c) Basic metabolic panel to determine the extent of renal disease, electrolyte status, and blood glucose levels

v) Presume ischemic heart disease in patients with primary HTN until proven otherwise.[1]
   (1) Assess patient's exercise tolerance with the focus on dyspnea or angina.
   (2) Check ECG for abnormal conduction or signs of ischemia. Consider stress testing if it will change management.
   (3) If cardiac exam suggests valvular disease or myocardial dysfunction, consider echocardiography.

vi) Continue current antihypertensive regimen prior to surgery. Consider omitting diuretic, ACE inhibitors, and angiotensin receptor blocker (ARB) dose the morning of surgery.[7]

vii) Review antihypertensive medication for pharmacology, anesthetic interactions, and potential side effects.
   (1) ACE inhibitors and ARBs can be associated with severe hypotension with general anesthesia, especially during induction.



Patients who have taken ACE inhibitors or angiotensin II receptor antagonists the day of surgery are at high risk for intraoperative hypotension and perioperative morbidity (stroke, death, and myocardial ischemia). The decision to continue or discontinue ACEI/ARBs should be made on an individual basis taking into account patients' BP and planned surgery. Restarting the medication postoperatively is vital.[7]

(2) Complications due to cessation of antihypertensive medications[2]
(a) β-Adrenergic antagonists → myocardial ischemia
(b) Clonidine → hypertensive crisis

b) **Intraoperative management**

i) Induction[3]

(1) Direct laryngoscopy and tracheal intubation can cause significant HTN, tachycardia, and myocardial ischemia.
(2) The use of IV drugs cannot always suppress the cardiovascular responses to tracheal intubation.
(3) Management
(a) In patients at high risk for myocardial ischemia, consider deep inhalation anesthesia or injection of opioid, lidocaine, β-adrenergic antagonists, or vasodilator to suppress tracheal reflexes and reduce autonomic responses to laryngoscopy.

ii) Intraoperative hemodynamic management[8]

(1) Patients with essential HTN have an increased incidence of perioperative HTN.
(a) Volatile anesthetics can attenuate pressor responses of sympathetic nervous system resulting in dose-dependent decrease in BP, systemic vascular resistance, and myocardial depression.
(2) Intraoperative hypotension may occur, when ACE inhibitors and ARBs were not discontinued 24 hours before surgery.[7]
(a) Vasoconstrictor treatment such as low-dose vasopressin may be required to treat hypotension.[7]
(3) No volatile agents or muscle relaxants appear to be more preferable in this patient population.
(a) One should consider avoiding pancuronium because of its dose-related sympathomimetic and vagolytic effects.
(4) Cerebral autoregulation[9]
(a) Typically maintained between mean BP between 50 and 150 mm Hg, although lower limit is likely closer to 70 mm Hg.
(b) In chronic HTN, the lower BP limit of autoregulation of cerebral blood flow is shifted toward a higher BP.[10]
(c) The clinical consequence of this shift is an impaired tolerance to lower BP.
(d) Long-term antihypertensive treatment can help readapt to normal autoregulation of cerebral blood flow illustrating the importance of the preoperative baseline evaluation.[4]



Patients with preoperative uncontrolled HTN are at high risk of hemodynamic lability intraoperatively.



Due to changes in cerebral autoregulation with chronic HTN, rapid BP drops to normotensive levels can result in decreased cerebral blood flow and possibly cause ischemic brain damage.

iii) Monitoring

(1) ECG, preferably 5-lead, to recognize myocardial ischemia during periods of intense painful stimulation such as laryngoscopy and tracheal intubation
(2) Arterial line is useful for extensive surgery or in case of left ventricular dysfunction or other significant end-organ damage.
(3) Transesophageal echocardiography to monitor left ventricular function and intravascular volume replacement in major surgical cases or critical condition of the patient

5) **Postoperative management**[1]

a) **Postoperative HTN is common in patients with primary HTN.**

i) Close monitoring of BP with prompt assessment and treatment is required to decrease risk of myocardial ischemia, arrhythmias, CHF, cerebrovascular ischemia, stroke, and maintenance of surgical wound hemostasis.

b) **Adequate management of postoperative pain**
   i) Pain activates the sympathetic nervous system and is an important contributor to postoperative HTN.
   ii) Useful approaches to pain management may include multimodal intravenous analgesia and regional and/or neuraxial anesthesia.

c) **Temporary IV administration of antihypertensive agents may be necessary in the postoperative period.**
   i) Medications should be chosen based on the patient's vital signs and coexisting diseases. There is no single agent that has been proven to be superior to another.
   ii) Two common medications used to control BP in the perioperative period are labetalol or nicardipine. Labetalol is given as a bolus of 10-20 mg IV followed by 20-80 mg every 10 minutes, maximum dose of 300 mg. Nicardipine is administered as an infusion, 5-15 mg/h.
   iii) In the presence of a low heart rate and/or contraindications to β-adrenergic antagonists, hydralazine is an alternative agent.

CHAPTER SUMMARY FOR PRIMARY HTN AND ANESTHETIC CONSIDERATIONS

Definition	Primary HTN—defined as HTN, when no secondary cause for HTN can be identified such as pheochromocytoma, hyperaldosteronism, Cushing syndrome, renovascular HTN, and aortic coarctation. Primary HTN constitutes 95% of all cases of HTN.		
Classification		Systolic BP (mm Hg)	Diastolic BP (mm Hg)
	Normal	<120	<80
	Elevated	120-129	<80
	Stage 1 HTN	130-139	80-89
	Stage 2 HTN	>140	>90
	Hypertensive crisis	>180	>120
Preoperative Considerations	Review medications and assess baseline BP and end-organ damage. In patients with hypertensive urgency and emergency, consider deferring surgery for optimization.		
Intraoperative Considerations	Increased risk of perioperative HTN and BP lability. Cerebral autoregulation may be shifted requiring higher BP for cerebral perfusion.		
Postoperative Considerations	Close monitoring and management of BP to avoid myocardial ischemia and maintain surgical wound hemostasis. Adequate postoperative pain management is essential.		

BP, blood pressure; HTN, hypertension.

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46 Peripheral Vascular Disease

Ruma Bose • Shamsuddin Akhtar

Peripheral vascular disease (PVD) is defined as partial or complete obstruction of one or more peripheral arteries. Global incidence of PVD is 3%-12%. PVD is typically a disease of the elderly and frequently associated with coronary artery disease (CAD) and cerebrovascular disease. Innovative surgical techniques and treatment options are now available to patients with PVD who in the past were considered very high risk due to their comorbidities. Preoperative risk assessment and optimization, formulating an anesthetic plan for each patient, based on patient and surgical factors is important to decrease perioperative complications in this high-risk patient population. This chapter focuses on the pathophysiology of atherosclerosis, clinical diagnosis, medical and surgical management, preoperative assessment, and anesthetic management of patients with PVD.

1) Epidemiology

The worldwide prevalence of lower extremity peripheral artery disease (PAD) is 3%-12%. Based on CDC data ~8.5 million people in the United States have PAD including 12%-20% of individuals older than 60 years of age. The risk factors promoting PAD are similar to those that promote coronary artery disease. Sixty percent of patients with severe PAD who require surgery also have significant CAD in at least one coronary vessel. The medical management and risk reduction strategies are similar for both disease processes.



Symptomatic and asymptomatic PVD are predictors of cardiovascular disease and cerebrovascular disease

2) Pathophysiology

a) Atherosclerosis is caused by inflammatory process of the vascular endothelium. Hypertension causes shear stress and injury to the vessel wall. Hyperlipidemia leads to formation of lipid-rich plaques in these sites, which rupture and cause platelet activation and inflammatory changes in the subendothelial layers and intima (vasculitis). These changes cause weakening of the intimal layer, narrowing of the vessel lumen, and vasospasm.

b) Clinically, these processes translate into symptoms of limb ischemia in the extremities (claudication), gangrene in the extremities, transient ischemic attacks and stroke.

c) Ankle-brachial index (ABI) is the ratio of SBP at the ankle to that in the arm, <0.9 is indicative of PAD in the lower extremity. It is commonly used in clinical practice and epidemiological research to diagnose PAD.[1]



Ankle-brachial index is commonly used in clinical practice and epidemiological research to diagnose PAD.

3) Risk factors for atherosclerosis[2]

a) Hypertension
b) Diabetes
c) Smoking (strongest modifiable risk factor)
d) Hyperlipidemia
e) Age (prevalence of >10% in patients who are more than 60 years old)
f) Family history of early-onset PAD and CAD
g) Abdominal obesity
h) Proinflammatory and prothrombotic states.
i) Chronic kidney disease especially end-stage renal disease on hemodialysis

4) **Signs and symptoms**
   a) Primary carotid disease
      i) Neurologic presentation of carotid disease are recent history of stroke, transient ischemic attacks, amaurosis fugax, reversible ischemic neurologic deficits, weakness or numbness of limbs and face, dysarthria, aphasia, and visual field defects.
      ii) Surgery is indicated in symptomatic patients with >70% decrease in luminal diameter.
      iii) Carotid endarterectomy (CEA) in asymptomatic patients with more than 60% stenosis reduces the risk of fatal and nonfatal stroke.
   b) Peripheral vascular disease of lower extremities
      i) Acute ischemia is caused by acute vasospasm, thrombosis from plaque rupture, or embolism originated from cardiac or other sources.
      ii) The symptoms are commonly known as the 5 Ps of acute ischemia:
      Pain
      Paresthesia
      Pallor
      Pulselessness
      Paralysis
      iii) Chronic ischemia is slower in onset with claudication being the early symptoms. Progression of disease leads to pain at rest, nonhealing ulcerations, cellulitis, osteomyelitis, and gangrene.



Risk of adverse events after CEA are significantly higher in patients with ipsilateral symptoms, age more than 75 years, diastolic blood pressure more than 110 mm Hg, evidence of internal carotid artery thrombus, history of heart disease, stroke, and diabetes

5) **Diagnosis**
   a) ABI as the first screening test, followed by ABI with exercise.
   b) Duplex ultrasonography, CTA (can affect renal function) MRI, MRA, and angiography



Ankle-brachial index of <0.5 is indicative of critical limb ischemia.

6) **Medical management of PVD**

The goals of care are minimizing disability, preserving quality of life, and slowing disease progression.

   a) **Smoking cessation is the single most modifiable risk factor (Class I)**
      i) Smoking is not only a risk factor for developing PVD; it also increases the rate of disease progression to critical limb ischemia (CLI).
      ii) Smoking cessation decreases the severity of claudication and the risk of stroke or MI in this population.
   b) **Exercise**
      i) Walking until the point of maximal pain three times a week for 6 months improves disability associated with claudication and is more effective than antiplatelet therapy or angioplasty.
   c) **Statins**
      i) Statin therapy increases maximal walking time in patients with intermittent claudication and decreases the incidence of new claudication in patients otherwise at risk of developing vascular disease (Class I).
   d) **Blood pressure control**
      i) Antihypertensive therapy should be administered in patients with HTN and PAD to reduce risk of stroke and MI (Class I).
         (1) Angiotensin-converting enzyme (ACE) inhibitors or ARB with thiazide diuretic are recommended with a blood pressure goal of 130/80 (secondary prevention of TIA) and for reduction of ischemic events in PAD.

(2) Aortic disease: beta-blockers are recommended as the preferred antihypertensive drug class in patients with hypertension and thoracic aortic disease.

e) **Glycemic control**
   i) Patients with diabetes have a higher risk of disease progression.
   ii) As the level of glycosylated hemoglobin rises, so does the risk of PVD.
   iii) Glycemic control is beneficial for patients with CLI to reduce limb-related complications.

f) **Antiplatelet therapy**
   i) Current guidelines recommend either ASA in daily doses of 75/325 mg or clopidogrel 75 mg/d to reduce the risk of MACE (Class I).

g) **Phosphodiesterase inhibitors**
   i) Mechanism of action is suppression of platelet aggregation and direct arterial vasodilation.
   ii) Cilostazol is the only agent in this class approved for treatment of claudication. It improves symptoms and increases walking distance (Class I).



Endovascular treatments have better durability in aortoiliac disease as compared to femoral and popliteal disease

7) **Surgical management of chronic limb ischemia**

a) **Endovascular revascularization for chronic limb ischemia (EVT).**
   i) Revascularization in patients with claudication is indicated when the disease progresses despite medical therapy and risk reduction strategies impact quality of life.
   ii) Endovascular treatment has worse outcome compared to open repair when length of the constriction is long and there are multiple diffused lesions, poor-quality runoff, diabetes, renal failure, and history of smoking.
   iii) Revascularization techniques consist of balloon dilation, stents, and atherectomy

b) **Open surgical revascularization**
   i) Indicated for patients who have failed endovascular therapy, have vascular anatomy that is favorable for anastomosis, and have acceptable risk profile.
   ii) The superficial femoral and proximal popliteal arteries are the most common sites of stenosis.
   iii) Femoral-popliteal bypass is the most frequently performed peripheral vascular anastomosis.
   iv) Autologous vein is preferred over prosthetic graft for bypass conduit.

c) **Management of acute limb ischemia**
   i) Acute limb ischemia is described as an ischemic leg that has been cold and painful for a period of <2 weeks.
   ii) Etiology is thrombosis or embolism from another source.
   iii) Urgent diagnosis with Doppler ultrasound and management is critical to prevent irreversible ischemia.
   iv) Systemic anticoagulation with heparin is the first step in management.
   v) Catheter-directed thrombolysis or embolectomy may be indicated.
   vi) Thrombectomy should not be delayed to establish the source of embolism.



READ MORE

2016 ACC/AHA Guideline on management of patients with lower extremity PAD

d) **Salvage operations**
   i) Despite aggressive medical and surgical interventions, some patients continue to have progressive disease and may require limb debridement or amputation procedures.

8) **Anesthetic management of peripheral vascular disease**

a) The patients presenting for vascular surgery tend to be elderly and frail. They present with a host of comorbid conditions, which makes anesthetic management challenging. The procedures often are urgent, which do not provide enough time for preoperative optimization of comorbidities.

i) Preoperative considerations

(1) Major adverse cardiac events (MACE) and stroke are serious complications of patients undergoing vascular surgery and is associated with significant morbidity and mortality (30-day mortality after postoperative myocardial ischemia is 1.9%).



Major adverse cardiac events (MACE) and stroke are serious complications of patients undergoing vascular surgery

(2) Postoperative stroke is associated with eightfold increase in morbidity and mortality.

(3) *Risk factors*

(a) Recent myocardial infarction (MI < 6 weeks without coronary intervention).

(b) Age more than 62 years is a risk for stroke.

(c) History of CVA increases risk of MACE.

(d) Heart failure (increases the risk of readmission and 30-day mortality).

(e) Low ejection fraction (<30% independent predictor of mortality).

(f) Atrial fibrillation.

(g) Preoperative natriuretic peptide will independently predict cardiovascular events in the first 30 days of surgery.

(h) Valvular disease: severe aortic stenosis and mitral stenosis

(i) Aortic stenosis increases the perioperative risk (30-day mortality of 2.1% and MI). Preoperative TAVR or balloon dilation can be considered as an option (stroke risk of 1%-2%, mortality of 2%-3%).

(j) Preoperative assessment with echocardiography to assess the severity helps in planning appropriate management.

(4) Preoperative risk assessment[3]

Multivariate risk assessment models are available that take into account the patient-specific factors and surgery-specific factors to provide a realistic assessment of preoperative risk. The commonly used models are

(a) Revised Cardiac Risk Index (RCRI)

(b) National Surgical Quality Improvement Program (NSQIP)

(c) Gupta Perioperative Risk for Myocardial Infarction and Cardiac Arrest (MICA-ACS)

(5) Cardiac evaluation and optimization

(a) History and physical exam, functional capacity, activities of daily living measured in METS, and 6-minute walk test. Duke Activity Status Index (DASI) questionnaire is a better marker of exercise capacity than simple history.[4]

(b) Risk of postoperative MACE is increased in patients unable to perform activities equivalent to four METS in the absence of physical constraints. In many cases, the patients are so handicapped by their disease that an accurate indication of exercise tolerance cannot be obtained from history alone.

(c) Pharmacological stress test is indicated only if the results of the test will significantly alter the management.

b) **Cardiac intervention prior to surgery**[3]

i) Preoperative CABG/PCI do not change postoperative outcomes except in the presence of left main disease, acute angina, malignant arrhythmias, aortic stenosis, and low ejection fraction (recommended independent of their preoperative status).

ii) In the absence of active angina or other indications for immediate intervention, these patients should be medically managed through the perioperative period.



In the absence of active angina or other indications for immediate intervention, these patients should be medically managed through the perioperative period.

c) Percutaneous coronary intervention
   i) The risk of surgical bleeding and cardiac stent rethrombosis need to be considered, and a shared decision between surgeon, cardiologist, anesthesiologist, and the patient should be made.
   ii) As per guidelines by ACC/AHA, elective noncardiac surgery should be postponed if possible for 14 days after balloon angioplasty, 30 days after BMS (bare-metal stent), and 6 months after DES (drug-eluting stent)
   iii) Invasive risk reduction strategies are limited in patients for revascularization due to urgency.

READ MORE
2014 ACC/AHA Guideline on Perioperative Cardiovascular Evaluation and Management of Patient Undergoing Non-Cardiac Surgery.

9) **Management of dual antiplatelet therapy (DAPT) for noncardiac surgery**[3]
   a) Bleeding risk DAPT is 21% vs 4% on one antiplatelet agent.
   b) Low and intermediate thrombosis risk: continue aspirin, stop P2Y12 inhibitors but reinstitute at 24-72 hours after surgery.
   c) High thrombosis risk: postpone surgery/continue ASA/stop P2Y12 receptor inhibitor and consider bridging with short-acting GP2B/3A receptor antagonist.
   d) CABG and PCI are not recommended as interventions for risk reduction prior to surgery.
   e) If coronary revascularization and the surgery is urgent, then a combined procedure can be considered.



PVD patients are on antithrombotic agents, which should not be discontinued in the preoperative period

10) **Intraoperative management**
   a) Anesthetic plan is determined by procedural factors (minor-, moderate-, high-risk procedures) and patient factors (ASA status and patient comorbidities).
   b) *Embolectomy* (low-risk procedure):
      i) Local anesthesia with monitored anesthetic care.
      ii) Invasive monitors not required.
      iii) Peripheral nerve block with or without general anesthesia for vessel dissection (cut down)
   c) *Emergent limb vascularization:* general anesthesia with *invasive* monitoring
      i) Neuraxial anesthesia is contraindicated in the setting of anticoagulation for profoundly ischemic extremities.[5]
      ii) Invasive monitoring usually required. Arterial line is frequently required for close hemodynamic monitoring. Central line, PA catheter, and transesophageal echocardiography may be indicated in specific clinical scenarios.
      iii) These patients may present with severe metabolic derangements (rhabdomyolysis, acidosis, hyperkalemia, and azotemia), which can result in hemodynamic instability and significant resuscitation with vasoactive agents
   d) *Nonemergent lower extremity revascularization:*
      i) Can be performed with neuraxial blockade (subarachnoid block or continuous epidural anesthesia or general anesthesia.
      ii) Neuraxial anesthesia has been shown to demonstrate possible benefits in terms of hemodynamic stability, reduced blood loss, improved distal arterial blood flow due to sympathectomy, and decreased narcotic requirement. These patients are less hypercoagulable and have fewer thrombotic complications and have a lower rate of regrafting and embolectomy.
      iii) Anticoagulation management in the setting of neuraxial blockade is guided by the recommendations published by the American Society of Regional Anesthesia (ASRA Guidelines).[5]
      iv) There is no statistically significant difference in the rate of postoperative myocardial infarction, morbidity, and mortality in either group.



No significant difference in MACE between general anesthesia and neuraxial anesthesia in patients undergoing peripheral vascular surgery.

11) **Anesthetic considerations for carotid endarterectomy (CEA)**

a) **Specific goals for CEA**

i) Prevention of cerebral ischemia, early detection, and management.

ii) Rapid recovery from anesthesia

iii) Maintain hemodynamic stability

CEA is marked by fluctuations in blood pressure and heart rate. Carotid baroreceptors are mechanoreceptors that respond to alterations in blood pressure. The carotid sinus baroreceptors are located in the internal carotid artery and are innervated by the sinus nerve of Hering, which is a branch of glossopharyngeal nerve. In response to external pressure or stimulation, the nerve fibers stimulate the parasympathetic nervous system (via vagus nerve) and inhibit the sympathetic nervous system via a centrally acting mechanism, resulting acute bradycardia and hypotension. This can result in myocardial ischemia in patients at risk for cardiovascular events (infiltration of local anesthetic around the carotid body or around the carotid sinus by the surgeon can reduce bradycardia response this to some extent).

b) **Intraoperative monitoring**

i) Invasive blood pressure monitoring:

(1) Reflex-mediated changes can cause a rapid change in heart rate and blood pressure. The cerebral perfusion is directly affected by blood pressure fluctuation. Thus, invasive blood pressure monitoring is strongly recommended to manage acute hemodynamic changes and prevent cerebral ischemia.

ii) Monitoring for cerebral perfusion

(1) Neurological exam in an awake patient during surgery

(2) Patient under GA cerebral perfusion can be measured by a variety of monitors

(3) EEG monitoring

(4) SSEP (poor predictor of postoperative deficits)

(5) Cerebral oximetry (20% decrease below baseline during cross clamp predicts preoperative stroke (high sensitivity, low specificity)

(6) TCD (transcranial Doppler)

(7) Jugular venous bulb saturation and jugular venous lactate (measure of global function (low specificity)

(8) Stump pressure measurement



No single technique is sensitive and specific for measuring adequacy of cerebral perfusion during cross clamp.

c) **Choice of anesthetic for CEA (regional or general anesthesia)**

i) Regional anesthesia (superficial and/or deep cervical plexus block)

(1) Pros: Neurological exam is possible to determine adequacy of cerebral perfusion; shunt is not required in every patient.

(2) Con: Patient discomfort and anxiety in technically challenging procedures.

(3) No real differences in outcome when compared to general anesthesia.

(4) Deep cervical plexus block is not routinely used as the risks of subarachnoid injection (Horner syndrome, unwanted blockage of the phrenic, recurrent laryngeal and vagus nerves) outweigh the risk. Urgent conversion to general anesthesia is seen more often with deep cervical plexus blocks

ii) General anesthesia

(1) Typically performed with invasive blood pressure monitoring and some means of cerebral perfusion monitoring or routine placement of shunt.

(2) Rapid reversal of anesthetic effects to conduct neurological exam

(3) Maintain adequate cerebral perfusion and minimize cerebral metabolic rate $CMRO_2$ (avoid hyperthermia, hyperglycemia).

(4) Anticipate and treat bradycardia and blood pressure fluctuations.

TABLE 46-1 Comparison Between Regional and General Anesthesia for Carotid Endarterectomy

	Regional Anesthesia	General Anesthesia
Hemodynamics	Less hypotension	Labile blood pressure
Monitor for cerebral perfusion	More sensitive measure of cerebral perfusion	Requires various modalities, each with its own limitation
Pitfalls	Unprotected airway	Controlled ventilation
Patient discomfort	Requires patient's cooperation	
Postoperative neck hematoma	Low risk	Higher risk
Pain control	Block may not last suffice for a long incision	
Requirements for shunt	Low	Higher
Intraoperative hypertension	More frequent due to stress	Less common
PACU length of stay	Shorter	Longer

(5) Minimize coughing and straining at extubation to avoid hematoma (Table 46-1).

d) **Anesthesia for carotid stenting**
   i) Local anesthesia with monitored anesthesia care.
   ii) Balloon occlusion of the carotid causes sudden hypertension and bradycardia that can be treated by anticholinergic agents like atropine.

e) **Postoperative complications**
   i) The complications associated with CEA include myocardial infarction; stroke; cerebral hyperperfusion syndrome; nerve injury; parotitis and bleeding, which can lead to neck hematoma requiring reoperation; and late carotid restenosis.
   ii) Cerebral hyperperfusion syndrome: cerebral autoregulation is altered in cerebral blood vessels. Increase in blood flow after CEA causes increase in cerebral blood flow and cerebral edema, hemorrhage, petechiae, and seizures.
   iii) Other complications: bleeding, neck hematoma, recurrent laryngeal nerve injury

CHAPTER SUMMARY FOR PVD

Epidemiology	PV affects 30% of adult population of the United States. Ankle-brachial index (ABI) is a commonly used measure of severity of vascular disease.
Risk factors	The main risk factors associated with PVD similar to those of stroke and CAD include advanced age, hypertension, smoking, diabetes, hyperlipidemia, and renal insufficiency.
Anesthetic Concerns	Cardiac events and stroke are common in this population, making perioperative cardiac risk stratification and management the primary goal of the preoperative evaluation.
Anesthetic Management	A discussion of risk-benefit analysis of the procedure with the surgeon, patient, and family is essential. Minimally invasive techniques are available for risk reduction as well as surgical treatment that present viable options for very frail patients. Regional, neuraxial, and general anesthesia are options for anesthetic management. The goal of anesthetic management is risk assessment and optimization and to minimize intraoperative and postoperative complications.

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47 Deep Vein Thrombosis and Pulmonary Embolism

Ruma R Bose • Shamsuddin Akhtar

Deep vein thrombosis (DVT) and pulmonary embolism (PE) are major causes of significant morbidity and mortality, which are frequently, but not exclusively associated with surgery, trauma, and oncological conditions. The iliofemoral veins are the most commonly recognized source of PE. Survivors of PE or DVT may suffer from chronic thromboembolic pulmonary hypertension or postthrombotic syndrome. Anticoagulants are used to either prevent or treat DVT and PE.

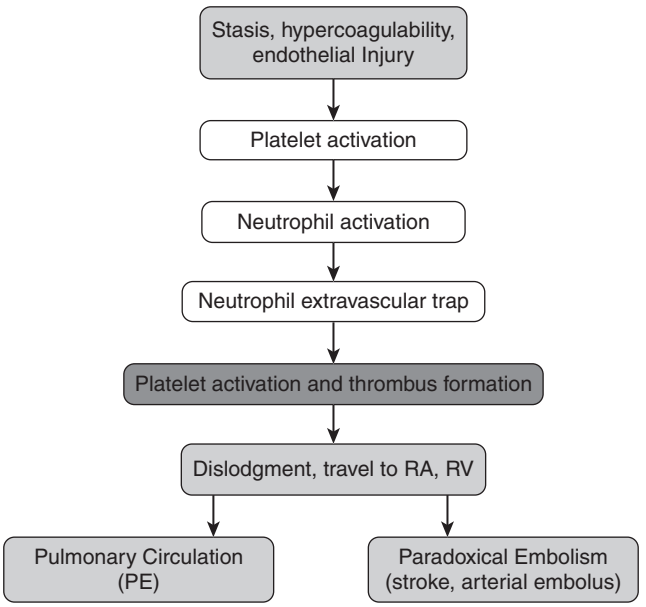
1) **Pulmonary embolism**[1]
   a) **Epidemiology**
      i) The estimated incidence of PE is around 100-300 cases per 100 000 person years, rising sharply after the age of 60 years.
      ii) It is estimated that 100 000-180 000 persons die from PE annually in the United States.



Up to 180 000 deaths in the United States are either caused or related to PE each year.

   b) **Risk factors**
      i) Patients with PE usually have identifiable risk factors for the development of venous thrombosis.
      ii) *Major risk factors that can increase the risk by 5-20 times are* surgery, major orthopedic trauma, immobility, hospital admission, late pregnancy, history of previous DVT or PE, varicose veins.
      iii) *Major risk factors that can increase the risk by 2-4 times are* cardiovascular disease, obesity, travel (>5-6 hours), thrombophilia, oral contraceptives, hormone replacement therapy.
   c) **Pathophysiology**
      i) The majority of pulmonary emboli arise from propagation of lower limb thrombi. Rare cases include septic emboli (from tricuspid or pulmonary valves), fat emboli (following long bone fracture), air, amniotic fluid, or tumor.
      ii) Pathophysiology of thrombi formation is illustrated in **Figure 47-1**.
      iii) Platelet activation at the site of injury, due to hypercoagulability or stasis, leads to neutrophil activation and recruitment, which leads to formation of extracellular traps and prothrombotic networks. Further platelet aggregation, activation, and thrombin generation lead to thrombus formation. Inflammation, antiphospholipid antibody syndrome, sedentary life style, and protein C, protein S, and antithrombin deficiencies can promote prothrombotic state and cause thrombus formation.
      iv) Venous thrombi form and flourish in an environment of stasis, low oxygen tension, and up-regulation of proinflammatory genes.
2) **Physiological impact of PE**
   a) An embolic occlusion in the pulmonary circulation increases the total dead space.
   b) A theoretical increase of $PaCO_2$ is expected but often not clinically observed, due to compensation from increased minute ventilation, triggered by medullary chemoreceptors sensing of the rise of $PaCO_2$.

FIGURE 47-1 Pathophysiology of Pulmonary Embolism.



c) When hypercapnia becomes evident, massive PE should be suspected.
d) Hypoxemia is more often seen during a PE.
   i) *V/Q* mismatch in which there is "dead space" ventilation in some parts of the lung and overperfusion in others.
   ii) Atelectasis caused by loss of surfactant within hours after PE
   iii) Right-to-left intrapulmonary shunting where there is overperfusion and decreased vascular resistance occurs in nonembolic parts of the lung.
   iv) Intracardiac shunting may occur due to increased pulmonary artery (PA) pressure and enlarged opening of preexisting patent foramen ovale.
e) A pulmonary embolus reduces the cross-sectional area of the pulmonary vasculature, resulting in increased pulmonary vascular resistance.
   i) In addition, intrapulmonary reflexes stimulate the release of humoral substances that lead to vasoconstriction throughout the lungs and thus further increases pulmonary vascular resistance.
      (1) As a result, high PA pressure could acutely develop, especially in patients with preexisting cardiopulmonary diseases, and potentially precipitate or lead to acute right ventricular failure (RVF).

3) **Classification of PE**
a) PE is typically classified into three categories (massive PE, submassive PE, and low-risk PE).
b) *Massive PE*: characterized by
   i) Extensive thrombosis affecting at least half of the pulmonary vasculature
   ii) Dyspnea, syncope, hypotension, and cyanosis are common.
   iii) Comprise 10%-15% of the cases

c) *Submassive PE*: characterized by
   i) Right ventricular dysfunction despite normal systemic arterial pressure
   ii) Comprise 20%-25% of the cases
d) *Low-risk PE*: comprise 65%-75% of the cases and have excellent prognosis

4) **Diagnosis**
   a) **Clinical presentation**
      i) Clinical features can be mild and nonspecific.
      ii) Though 90% of the embolic arise from the lower extremities, majority of the patients have no leg symptoms at the time of the diagnosis.
      iii) *Preoperative and postoperative presentation of pulmonary embolism*
      iv) Most consistent
         (1) Acute dyspnea (75%)
         (2) Tachypnea (70%)
      v) Observable with PE near pleural surface
         (1) Pleuritic or chest pain (60%)
         (2) Rales (50%)
         (3) Cough (40%)
      vi) Others
         (1) Possible wheezing on auscultation
         (2) Wide S2 splitting
         (3) Tachycardia[3]
   b) **Intraoperative presentation of pulmonary embolism**
      i) Often transient and nonspecific
      Includes the sudden onset of the following symptoms:
         (1) Hypoxemia
         (2) Low $EtCO_2$
         (3) Bronchospasm
         (4) High airway pressure
   c) **Diagnosis is difficult to establish on clinical presentations alone.**
      i) Diagnosis is based on clinical presentation, risk factors for PE (eg, Wells score, Geneva score) (**Table 47-1**), and results of investigations.



Intraoperative signs of PE include sudden onset of hypoxemia, low $EtCO_2$, bronchospasm, or high airway pressures.

5) **Investigations**
   a) **ECGs** could be abnormal, but the findings listed are not specific or sensitive.
      i) ST and T-wave change
      ii) Peaked P wave
      iii) RV strain
      iv) Right bundle-branch block (RBBB)
      v) An S wave in lead I and a Q-wave and T-wave inversion in lead III (S1Q3T3), in up to 70% of patients with PE and no preexisting cardiovascular disease
   b) **Chest x-ray** is often normal.
   c) An **arterial blood gas** (ABG) may show respiratory alkalosis, hypoxemia, and increased A-a gradient.
   d) **D-Dimer assay:**
   e) Normal D-dimer test has a high negative predictive value of up to 95% in the setting of low clinical probability.
      i) Positive D-dimer assay is highly nonspecific and is influenced by age, malignancy, postoperative state, myocardial infarction, pregnancy, infection, and peripheral vascular disease.
   f) **CT pulmonary angiogram:**
      i) Currently the standard diagnostic imaging test to evaluate a suspected PE



Normal D-dimer test has a high negative predictive value of up to 95% in the setting of low clinical probability.

TABLE 47-1 Prediction Rules of Pulmonary Embolism

Items	Points
Wells score	
Previous PE or DVT	1
Heart rate equal or >100 bpm	1
Surgery or immobilization within the past 4 wk	1
Hemoptysis	1
Clinical signs of DVT	1
Alternative diagnosis less likely than DVT	1
Clinical probability	
Two-level score	
PE unlikely	0-1
PE likely	≥2
Revised Geneva score	
Previous PE or DVT	1
Heart rate	
75-94 bpm	1
≥95	2
Surgery or fracture within the past 1 mo	1
Hemoptysis	1
Active cancer	1
Unilateral lower limb pain	1
Age > 65 y	1
Pain on lower limb deep venous palpation and unilateral edema	1
Clinical probability	
Two-level score	
PE unlikely	0-2
PE likely	≥3

ii) Current scanners have increased the detection rate of subsegmental PE, virtually eliminating nondiagnostic studies.
iii) They may also help establishing the diagnosis that is not apparent on chest x-ray, for example, pneumonia, emphysema, pulmonary fibrosis, or aortic pathologies.

g) **V/Q scan**
i) Used to be the first-line study but has now become a second-line test especially for patients who cannot tolerate intravenous contrast (pregnancy, renal failure, anaphylaxis to intravenous contrast)
ii) It is associated with high false-positive results up to 50%.
iii) A negative scan is of high value and can essentially rule out PE. Currently, the test is reserved for patients with major renal impairment or pregnancy.

h) **Magnetic resonance (MR) contrast-enhanced imaging**
i) MR pulmonary angiogram may detect large proximal PE but is not reliable for smaller segmental and subsegmental PE.

i) **Transesophageal echocardiography**
i) Echocardiography is *not* a reliable diagnostic test for acute PE; however, it is very useful for detecting conditions that may mimic PE.
ii) The best-known indirect sign of PE is the McConnell sign (dilation of right ventricle [RV], depressed right ventricular function with apical sparing or hyper kinetic

apex). Severe tricuspid regurgitation. Thrombus might be visualized in the cardiac chambers or in the PA. Absence of thrombus does not exclude the diagnosis of PE.

j) Pulmonary angiography
   i) Gold standard test to diagnose PE; however, it is rarely performed and superseded by high-resolution CT angiogram.
   ii) Used when other imaging modalities are nondiagnostic or contradicted, and a high clinical suspicion for PE persists with hemodynamic instability. It may also be considered if catheter thrombolysis is planned.

6) **Decision tree for DVT/PE**[2]
   a) Step 1—Based on signs and symptoms of DVT/PE, the likelihood of thrombosis is determined by clinical scoring system, Wells score/Geneva score (**Table 47-1**).
   b) Step 2—If probability of VTE is low, D-dimer testing is recommended (if D-dimer is negative, no further workup is required). If D-dimer is positive, testing CTAP (PE) and compression ultrasonography (DVT) is recommended.
   c) Step 3—If VTE is likely based on clinical scoring system, further testing CTAP (PE) and compression ultrasonography is recommended for DVT. Other diagnostic modalities are V/Q scan, MRI.

7) **Treatment for PE**[3]
   d) Patients with stable vital signs, normal BP, and no evidence of RV dysfunction generally responded well with anticoagulation and other supportive measures, such as supplemental oxygen.
      i) Anticoagulation: three main strategies
         (1) Treat with unfractionated heparin, low molecular weight heparin (LMWH), or fondaparinux (at least 5 days) and bridged to warfarin
         (2) Parenteral heparin therapy switched to direct thrombin inhibitor (dabigatran) or an antithrombin agent (edoxaban)
         (3) Oral anticoagulation monotherapy with rivaroxaban (3-week loading) or apixaban (1-week loading) followed by maintenance therapy



Most serious side effect of anticoagulation is bleeding.

      ii) Fibrinolysis with tissue plasminogen activator (tPA) (100 mg as continuous infusion over 2 hours)
         (1) Reserved for massive PE with acute RVF and hemodynamic instability
         (2) Intracranial hemorrhage risk may be as high as 3.0%.
         (3) In one study, fibrinolytics did not reduce mortality or recurrent PE at 90 days
         (4) Not advised in hemodynamically stable submassive PE due to increased risk of hemorrhage
         (5) When full dose of thrombolysis may not be advisable, catheter-directed thrombolysis can be considered.
      iii) Pulmonary embolectomy
         (1) Reserved for massive PE in whom fibrinolysis is contraindicated or has failed or for patients with hemodynamic or respiratory compromises requiring cardiopulmonary resuscitation.
      iv) Inferior vena cava (IVC) filter
         (1) Filters are reserved for two primary indications:
         (2) Active bleeding that precludes anticoagulation
         (3) Recurrent venous thrombosis despite intensive anticoagulation
         (4) Patients who are at high risk, with right heart failure, who are not candidate for fibrinolysis and prophylaxis may be considered for IVC filter.



Intracranial hemorrhage risk may be as high as 3% with tPA.

Contraindications to fibrinolysis include recent surgery, trauma, and intracranial pathology.

(5) IVC filters can themselves be nidus for thrombus formation and contribute to recurrent DVTs or caval thrombosis. Retrievable filters are now placed whenever possible.



IVC filters can themselves be nidus for thrombus formation and contribute to recurrent DVTs. Retrievable filters are now placed whenever possible.

8) **Deep vein thrombosis**[4,5]

a) **Epidemiology**

i) The annual incidence of VTE is ~1:1000 in Western populations.

ii) Thirty-day mortality is about 10%.

iii) Lower extremity DVT is 10 times more common than upper extremity DVT.

b) **Pathophysiology**

i) Risk factors for thrombus formation (stasis, endothelial injury, hypercoagulability, and some hematological disorders predispose to venous thrombosis)



Chronic thromboembolic pulmonary hypertension develops in 2%-4% of patients with acute PE.

c) **Diagnosis**

i) Clinical presentation

(1) Often nonspecific

(2) Low predictive value (~15%)

ii) Symptoms

(1) Local swelling

(2) Discoloration of the legs

(3) Increased warmth over the skin

(4) More visible surface veins

(5) Calf or leg pain or tenderness

(6) It is no longer advisable to elicit a Homan sign.



Contrast venography is the gold standard test to diagnose deep venous thrombosis.

Doppler ultrasonography is the diagnostic test of choice.

d) **Lab studies**

i) A positive D-dimer test. It should be utilized as an exclusion test.

(1) Doppler ultrasonography

(a) It is a diagnostic test of choice.

(b) In the setting of high Wells score (>2), sensitivity and specificity is more than 95%.

ii) Contrast venography

(1) Considered as the gold standard test to diagnose DVT

iii) Other

(1) Spiral computed tomography (CT)

(2) Impedance plethysmography

(3) Magnetic resonance contrast imaging (MRI)

e) **Management of DVT**

i) The primary objectives of treatment of VTE are to prevent and/or treat the following complications:

(1) Prevent further clot extension

(2) Prevention of acute PE

(3) Reducing the risk of recurrent thrombosis

(4) Treatment of massive iliofemoral thrombosis with acute lower limb ischemia and/or venous gangrene

(5) Limiting the development of late complications, such as the postthrombotic syndrome, chronic venous insufficiency, and chronic thromboembolic pulmonary hypertension

ii) Anticoagulation

(1) Low-dose heparin followed by warfarin at least 612 weeks

(a) Duration of prophylaxis depends on the identification of risk factors.

(b) For thrombosis that is "provoked" by identifiable risk factors, anticoagulation is continued for 3 months as recurrence is unlikely cases.

(c) For thrombosis that is "unprovoked" where the likelihood of recurrence is high, the anticoagulation should be continued for prolonged period of time.

(2) Direct oral anticoagulants, comprising the thrombin inhibitor dabigatran etexilate and the factor Xa inhibitors rivaroxaban, apixaban, and edoxaban, have been introduced for long-term prophylaxis. These agents overcome many disadvantages of vitamin K antagonists.

f) Routine perioperative prophylaxis

i) The best treatment for VTE is prevention.

ii) Proper prophylaxis may decrease perioperative VTE risk more than 10-fold.

iii) The methods of thromboprophylaxis proven to be clinically effective and relevant are low-dose anticoagulants and sequential compression devices.

iv) All patients should be well hydrated, and early mobilization after surgery is mandatory.

(1) Nonpharmacological strategies

(a) Early ambulation should be encouraged.

(b) Mechanical compression devices should be initiated prior to induction of the anesthesia and continued intraoperatively and then into the postoperative period and have to be worn at least 18-20 hours a day to be effective.

(2) Pharmacological strategies (**Table 47-2**)[3]

(a) Based on the ACCP guidelines, prophylaxis is recommended by both level of procedural thromboembolic risk and individual patient risk factors.

(b) In general, prophylaxis should be initiated ~2 hours preoperatively and continued for 5-10 days postoperatively. Prophylaxis should be extended for 30 days after a high-risk surgical procedure.

(i) LMWH is as effective, if not slightly more effective, when compared with low-dose unfractionated heparin with fewer side effects and usually require only daily dosing.

(ii) Low-dose unfractionated heparin administered at a dose of 5000 units subcutaneously 2 hours preoperatively and every 8 or 12 hours postoperatively reduces the risk of developing a DVT by ~60% with a similar reduction in PE-associated mortality.

(iii) Duration up to 5-10 days of VTE prophylaxis is recommended in selected high-risk general or gynecological surgery patients, and extended prophylaxis 30 days is indicated after major orthopedic procedures.

(iv) LMWH is preferable to warfarin, and aspirin alone is not recommended in any patient population.

TABLE 47-2 Recommendations for Thromboprophylaxis

Low risk	No thromboprophylaxis is recommended.
Moderate risk	• An effective form of thromboprophylaxis is recommended. • This should be either in the form of an effective low-dose anticoagulant or an effective mechanical method. • The thromboprophylaxis should ideally be started before the risk commences and should continue as long as the risk persists.
High risk	An effective form of thromboprophylaxis is mandatory, and consideration should be given to using a combination of low-dose anticoagulants and mechanical devices. Continuation of thromboprophylaxis beyond the period of hospitalization should be considered.

CHAPTER SUMMARY FOR DEEP VENOUS THROMBOSIS	
Prevalence	It is estimated that 100 000-180 000 persons die from PE annually in the United States
Risk Factors	Surgical patients, especially elderly, and patients undergoing orthopedic procedures are at high risk of VTE
Causes	Causes of venous thrombosis can be classified as inherited or acquired disorders
Pathophysiology	Majority of pulmonary emboli arise from propagation of lower limb thrombi. Hypoxia is more often seen in PE
Diagnosis	For DVT, Doppler ultrasonography is most commonly used, while contrast venography is considered the gold standard test (performed rarely)
	For PE, high-resolution CT angiography and pulmonary angiography are the gold standard tests to evaluate suspected PE and diagnose PE, respectively
Treatments	Treatment for DVT should include supportive care and anticoagulation Management for PE includes anticoagulation, possible fibrinolysis, thrombolysis, possible pulmonary embolectomy, and possibly placement of IVC filters
Prophylaxis	The best treatment for VTE is prevention. The methods of thromboprophylaxis proven to be clinically effective and relevant are low-dose anticoagulants and mechanical devices

VTE, venous thromboembolism; PE, pulmonary embolism; DVT, deep venous thrombosis; IVC, inferior vena cava.

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48

Part B: Immunology and Infectious Diseases

Pulmonary Hypertension and Perioperative Management Strategies

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Pulmonary hypertension (PH) presents significant perioperative management challenges for the clinical anesthesiologist. Patients with PH may have high perioperative morbidity and mortality associated with right ventricular (RV) failure, arrhythmias, ischemia, and hypoxia. A thorough understanding of the pathophysiology of the condition can improve perioperative management, as can careful preoperative assessment, prudent selection of anesthetic technique and drugs, and a familiarity of the appropriate intraoperative recommendations to prevent and treat pulmonary hypertension.

1) **Definition and etiology of pulmonary hypertension**
   PH is defined as mean pulmonary artery pressure (mPAP) >20 mm Hg at rest, measured by right heart catheterization (RHC).[1,2]
2) **Etiology and classification**
   a) **Etiology:** The principles underlying an elevated mPAP can be summarized by the following equation:

mPAP = ldeft atrial pressure + (cardiac output × pulmonary vascular resistance)

From the above equation, one should appreciate that a high mPAP value can be driven by an elevated left atrial pressure (which often measured indirectly using pulmonary arterial wedge pressure), cardiac output, and/or pulmonary vascular resistance. It is important to stress that the diagnosis of PH CANNOT be secured by echocardiogram alone. Performing an RHC is necessary not only to confirm the diagnosis of PH but also to fully characterize cardiopulmonary hemodynamic and help dictate a management approach. Causes of elevated mPAP from each component are summarized in Table 48-1.



Pulmonary hypertension is defined as mPAP of >20 mm Hg, and is driven by an abnormally high left atrial pressure, cardiac output, and/or pulmonary vascular resistance. An elevated pulmonary vascular resistance is the hallmark of PAH.

   b) **Classification:** Pulmonary hypertension is classified into five groups based on similar hemodynamic characteristics, similar pathophysiology, and therapeutic response[3] (Table 48-2).

TABLE 48-1 Causes of Elevated Pulmonary Arterial Pressure

Increased left atrial pressure	Left ventricular diastolic/systolic dysfunction, valvular disease
High cardiac output	Cirrhosis, anemia, fluid overload, hyperdynamic state, congenital heart disease with left-to-right shunt
Increased pulmonary vascular resistance	Pulmonary arterial vasculopathy, thromboembolic disease, pulmonary parenchymal disease, hypoxia, acidosis, hypercapnia, increased sympathetic tone

TABLE 48-2 Updated World Health Organization Clinical Classification of Pulmonary Hypertension

- **PAH**
  - Idiopathic PAH
  - Heritable PAH
  - Drug- and toxin-induced PAH
  - PAH associated with:
    - Connective tissue disease
    - HIV infection
    - Portal hypertension
    - Congenital heart disease
    - Schistosomiasis
  - PAH long-term responders to calcium channel blockers
  - PAH with overt features of venous/capillaries (PVOD/PCH) involvement
  - Persistent PH of the newborn syndrome
- **PH due to left heart disease**
  - PH due to heart failure with preserved LVEF
  - PH due to heart failure with reduced LVEF
  - Valvular heart disease
  - Congenital/acquired cardiovascular conditions leading to postcapillary PH
- **PH due to lung diseases and/or hypoxia**
  - Obstructive lung disease
  - Restrictive lung disease
  - Other lung disease with mixed restrictive/obstructive pattern
  - Hypoxia without lung disease
  - Developmental lung disorders
- **PH due to pulmonary artery obstructions**
  - Chronic thromboembolic PH
  - Other pulmonary artery obstructions
- **PH with unclear and/or multifactorial mechanisms**
  - Hematological disorders
  - Systemic and metabolic disorders
  - Others
  - Complex congenital heart disease

PVOD, pulmonary veno-occlusive disease; PCH, pulmonary capillary hemangiomatosis; LVEF, left ventricular ejection fraction.

Pulmonary arterial hypertension (PAH) is classified as WHO group 1 PH. Precapillary pulmonary arterioles in PAH develop plexiform arteriopathic lesions, characterized by excessive vasoconstriction, endothelial dysfunction, progressive vascular remodeling, and functional loss of the pulmonary vascular bed. This process eventually leads to a pathologic rise in pulmonary vascular resistance, resulting in progressive right heart failure and death.

3) **Signs and symptoms**
   a) Shortness of breath on exertion, fatigue, dizziness, and palpitations are nonspecific yet common symptoms of PH.
   b) Dyspnea at rest and presyncope or syncope are particularly ominous symptoms of advanced PH and right heart failure.
   c) A systolic murmur in the tricuspid valve position, loud pulmonic component to the second heart sound and right ventricular heave are typical signs of pulmonary hypertension on physical exam. Bilateral pedal edema, increased abdominal girth and elevated jugular venous pressure suggestive of right heart failure.
   d) The absence of these physical findings does not exclude the presence of PH.[4] Health care providers need to maintain a strong clinical suspicion for PH.



Symptoms of PH are unfortunately nonspecific. Signs of right heart failure on physical examination often suggest advanced disease.



Absence of clinical signs of right heart failure on exam does not exclude the presence of PH.

4) **Treatment strategies**
   a) Vasoreactivity is identified on RHC as a specific acute hemodynamic response to a vasodilator trial: mPAP must drop by 10 mm Hg to <40 mm Hg with a maintenance or increase in cardiac output.[5] Vasoreactive PAH is rare (7% of idiopathic PAH patients) but carries with it an excellent prognosis (5-year survival >90%).[6] Long-term non-centrally acting or dihydropyridine calcium channel blockers are recommended for vasoreactive PAH patients alone.
   b) The updated treatment algorithm for non-vasoreactive PAH supports the use of upfront combination therapy targeting:
      i) Endothelin
      ii) Nitric oxide
      iii) Prostacyclin pathways
   c) The principle of PAH therapy is to decrease RV afterload by way of pulmonary vascular vasodilatation and the stabilization of plexiform arteriopathic lesions. The augmentation of nitric oxide and prostacyclin effects and the inhibition of the endothelin pathway directly contribute to vasodilatation in the pulmonary vascular bed and plexiform lesion stabilization and hence underlie all 10 current FDA-approved PAH-specific therapies (see **Table 48-3**).
   d) These PAH-specific drugs are currently approved for WHO Group 1 PH as illustrated in **Table 48-3** and riociguat is approved for inoperable chronic thromboembolic PH (CTEPH).
5) **Perioperative assessment**
   Regardless of the etiology of PH, it is well accepted that patients with PH who undergo surgical procedures are at significantly higher risk of complications. Perioperative complications are attributable to many factors, including patient characteristics, the nature of surgery, and the quality of perioperative care. A multidisciplinary team approach utilizing anesthesiologists, pharmacists, surgeons, and PH experts is mandatory for risk stratification and patient management in order to achieve a successful outcome.
   a) History and physical
      i) Comorbidities such as pulmonary embolism, coronary disease, liver disease, and renal insufficiency should be reviewed and accounted for prior to surgery.

TABLE 48-3 Current FDA-Approved Treatments for PAH and Mode of Administration

Drug Class	Drug Name	Mode of Administration
Calcium Channel blockers[a]	Amlodipine, Nifedipine, Diltiazem	Oral
Phosphodiesterase type-5 inhibitors	Sildenafil Tadalafil	Oral
Endothelin receptor antagonists (ERAs)	Ambrisentan Bosentan Macitentan	Oral
Soluble guanylate cyclase (SGC) Stimulator	Riociguat	Oral
Prostacyclin analogue	Epoprostenol Iloprost Treprostinil	Intravenous Inhaled Intravenous, subcutaneous, inhaled, oral
Prostacyclin IP receptor agonist	Selexipag	Oral

[a]Reserved only for vasoreactive PAH

TABLE 48-4 New York Heart Association (NYHA) Functional Class Based on Symptoms

NYHA Functional Class	Description
I	Physical activity is not limited. Ordinary physical activity does not cause undue dyspnea, chest pain, or near syncope.
II	Comfortable at rest, but ordinary physical activity causes dyspnea, chest pain, or near syncope.
III	Comfortable at rest, but less than ordinary physical activity causes dyspnea, chest pain, or near syncope
IV	Dyspnea at rest, unable to perform any physical activity.

ii) A detailed assessment of prior surgical experiences, functional status, and a recent evaluation of RV function should be performed close to surgery. Functional capacity has been shown to correlate well with survival.[7] Functional clinical classifications are illustrated in **Table 48-4.**

iii) An acute to subacute worsening of functional status or findings of acute right heart failure on physical exam are critical pieces of information, which may lead to the necessary postponement or cancellation of the planned surgery.

iv) Re-evaluation of pulmonary hemodynamics via RHC might be indicated prior to surgery to confirm or obviate the need for additional medical therapy

b) Laboratory test

i) N-terminal pro-basic natriuretic peptide (NTproBNP) level that is highly prognostic of PAH long-term outcomes and can be used to predict treatment response. An acute elevation from baseline can suggest an acute exacerbation of right heart failure.

ii) A basic metabolic panel is useful to ensure stable sodium and bicarbonate levels and kidney function, all markers of adequate volume status and perfusion.

c) Imaging studies

i) A recent (within 3-4 months) transthoracic echocardiogram (TTE) is an essential step to assess RV function. TTE features such as a dilated right atrium (RA) or RV, hypertrophy of the RV wall, or septal bowing into the left ventricle during late systole or early diastole are suggestive of PH and poor RV function.

ii) The electrocardiogram (ECG) is an insensitive tool for detecting PH, but findings of right axis deviation or RV strain pattern are associated with increased morbidity.[8]

iii) The chest x-ray (CXR) may show signs of PH including RV hypertrophy, dilation of the pulmonary arteries as manifested by enlarged bilateral hila, and pruning of the pulmonary vasculature.

d) Management of PAH medication.

i) Many PAH medications require special consideration in the perioperative setting. Parenteral prostanoids, like epoprostenol and treprostinil for example, cannot be interrupted as their sudden discontinuation can lead to deadly rebound PH crises.[9,10]

ii) Lengthy procedures or procedures that disrupt GI absorption or require an extended preoperative fasting status may prevent the usual scheduled administration of oral or inhaled prostanoids. As such, transitions between oral, inhaled, and intravenous therapy classes require PH expert involvement and planned changes in medication should be implemented well in advance of surgery.

iii) Patients on parenteral prostanoid should have alternate routes of access for administration of other medications for blood draws as lines delivering prostanoid

therapies should never be interrupted under any circumstances. Staff well equipped to troubleshoot pulmonary vasodilator delivery system issues should be present in the operating room or procedure suite.



Patient optimization and multidisciplinary team involvement are mandatory during preoperative assessment. Transitions between classes of PAH medication may be required well in advance of surgery to ensure continuous therapy and avoid a PH crisis.

6) **Intraoperative management**

The most common and feared intraoperative complication in PH patients is acute RV failure.[8] Patients with PH are often unable to accommodate acute alterations in preload or afterload induced by various intraoperative dynamic states like fluid shifts, hypoxia, mechanical ventilation, induction of anesthesia, or acidosis. As such, when possible, one should consider the mode of anesthesia that has the least perturbation in hemodynamics. There are four tenets to the maintenance of RV function, namely (1) maintaining adequate RV preload; (2) minimizing RV afterload; (3) supporting RV contractility; and (4) maintaining sinus rhythm.[4,11,12]

a) **RV preload**

i) Maintaining adequate RV preload is necessary for optimal cardiac performance.

ii) Large fluid shifts can be poorly tolerated by the chronically stressed RV and may precipitate shock.

iii) Tracking the central venous pressure (CVP) or monitoring the trend of central venous saturation after each volume manipulation can help guide physicians in attaining the optimal volume status.

iv) If fluid replacement is necessary, a low volume repletion (eg, 250 mL) is preferred followed by close monitoring of the response in cardiac output or CVP and other markers of perfusion. These data allow one to manage hemodynamics by fine tuning the balance between inotropes/vasopressors and fluid replacement.



Maintaining adequate preload should be guided by invasive hemodynamic monitoring, with judicious replacement of fluid or administration of diuretic as necessary.

b) **RV afterload**

i) The right ventricle is exquisitely vulnerable to acute rises in pulmonary resistance. Acute elevations in PVR lead to acute RV strain, resulting in decreased RV stroke volume and low left ventricular (LV) output. Drop in LV preload impairs LV output, leading to ischemia and further RV compromise. As such, to avoid this catastrophic hemodynamic chain of events, every effort should be made to reduce PVR and/or avoid acute elevations in PVR.

ii) Triggers of PVR elevation include acidosis, hypothermia, hypercapnia, hypoxia, and increased sympathetic tone from noxious stimuli such as pain or anxiety. Avoiding or quickly counteracting these triggers are a mainstay of PH patient management in the perioperative setting.



Triggers of PVR elevation include acidosis, hypothermia, hypercapnia, hypoxia, and increased sympathetic tone from noxious stimuli such as pain or anxiety.

iii) Alveolar overdistention from over bagging peri-intubation, high tidal volumes, and the use of high positive end-expiratory pressures (PEEP) will increase PVR due to direct pressure on the pulmonary vascular bed.

iv) Traditional practices associated with lung protective ventilation may need to be modified in the PH patient as high PEEP and permissive hypercapnia can adversely affect PVR.

v) Inhalational agents such as nitric oxide, iloprost, and epoprostenol quickly reduce PVR, restoring and preserving ventilation-perfusion matching.[13] Their quick onset of action and lack of effect on systemic blood pressure favor their choice as primary agents in a PH crisis.



Strategies focused on the avoidance of PVR elevation triggers and the direct reduction of PVR via pulmonary vasodilators are imperative.

c) **RV contractility**
   i) Preserving coronary perfusion is crucial to avoiding RV ischemia and maintaining RV contractility. Maintaining systemic blood pressure while reducing RV pressure is a requisite strategy for preserving coronary blood flow to the RV. This can be achieved by using inotropes and vasopressors.
   ii) Dobutamine has minimal effects on PVR, but its beta-2 agonist effect may result in systemic hypotension.[14]
   iii) Milrinone has a favorable profile for PH patients in that it improves cardiac contractility, but hypotension is a well-known side effect. It also has a longer half-life (t½ ~ 2.5 hours), which may limit its use in the unstable patient.
   iv) Vasopressors such as vasopressin, phenylephrine, and norepinephrine improve blood pressure by increasing systemic vascular resistance. They can be used alone or as adjunct therapy with inotropic agents as their vasoconstrictor properties counteract the vasodilatory effects of dobutamine or milrinone. Many favor vasopressin over other vasopressors as it has little adverse effect on PVR.[14]
   v) Epinephrine or dopamine should be considered as alternative inotropic agents if hypotension is present. However, these adrenergic agonists are prone to induce tachyarrhythmias, which are poorly tolerated by the failing RV[14] and at higher doses are thought to increase PVR.

Right ventricular performance relies on the preservation of systemic blood pressure, which is vital to RV contractility. Inotrope and vasopressor selection should account for direct drug effects on both systemic and pulmonary vascular resistance.

d) **Maintain sinus rhythm**
   i) As RV contractile function deteriorates from long-standing PH, RV preload relies more on adequate right atrial contraction.
   ii) Supraventricular arrhythmia is poorly tolerated in the chronically failing, acutely stressed right ventricle. Hence, conditions that provoke arrhythmias should be avoided.
   iii) Therapeutic strategies should aim to restore sinus rhythm as well as rate control.
   iv) Standard ACLS protocol should be instituted in the unstable PH patient.
   v) Antiarrhythmic agents with negative inotropic properties such as beta blockers or calcium channel blockers should be avoided in the chronically stressed RV.
   vi) Amiodarone and digoxin are typically well tolerated in PH patients in the short term.

Restoring sinus rhythm is important to maintaining adequate RV output. Amiodarone is the preferred agent for the rate control and/or cardioversion in the patient with acute RV failure.

7) **Postoperative care**
   a) Regional analgesia may be offered to achieve adequate pain control.
   b) Systemic blood pressure should be maintained with inotropes or vasopressors and fluid or blood loss should be judiciously replaced if needed.
   c) Perioperative PAH-specific therapy should be continued or advanced until clinical stability is achieved, followed by gradual transition to the preoperative regime as necessary. Modification of PAH therapies requires close collaboration and involvement with PH experts.
   d) PH specialists should also be closely following PH patients in the postoperative setting.

Pain, arrhythmia, fluid shifts, and pulmonary thromboembolism may precipitate acute RV failure. Close monitoring in an intensive care unit with or without continuous hemodynamic monitoring should be employed postoperatively.

CHAPTER SUMMARY FOR PULMONARY HYPERTENSION AND PERIOPERATIVE MANAGEMENT STRATEGIES

Overview	Patients with PH may have high perioperative morbidity and mortality associated with right ventricular (RV) failure, arrhythmias, ischemia, and hypoxia.
Etiology	Elevated pulmonary arterial pressure can result from increased left atrial pressure, high cardiac output, or increased pulmonary vascular resistance. Each of these causes have many etiologies and dictates therapeutic strategy.
Classification	Pulmonary hypertension is classified into five groups based on hemodynamic characteristics, similar pathophysiology, and therapeutic response.
Preoperative Strategies	A thorough preoperative assessment including a history and physical, a detailed assessment of prior surgical experiences, functional status, and a recent evaluation of RV function (TTE) should be performed close to surgery.
Intraoperative Strategies	Strategies include maintaining adequate RV preload, reducing RV afterload, preserving RV contractility, and maintaining sinus rhythm.
Postoperative Strategies	Avoid factors that can precipitate acute RV failure postoperatively such as pain, arrhythmia, fluid shifts, and pulmonary thromboembolism.

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49 Allergy and Anaphylaxis

Christopher Szabo

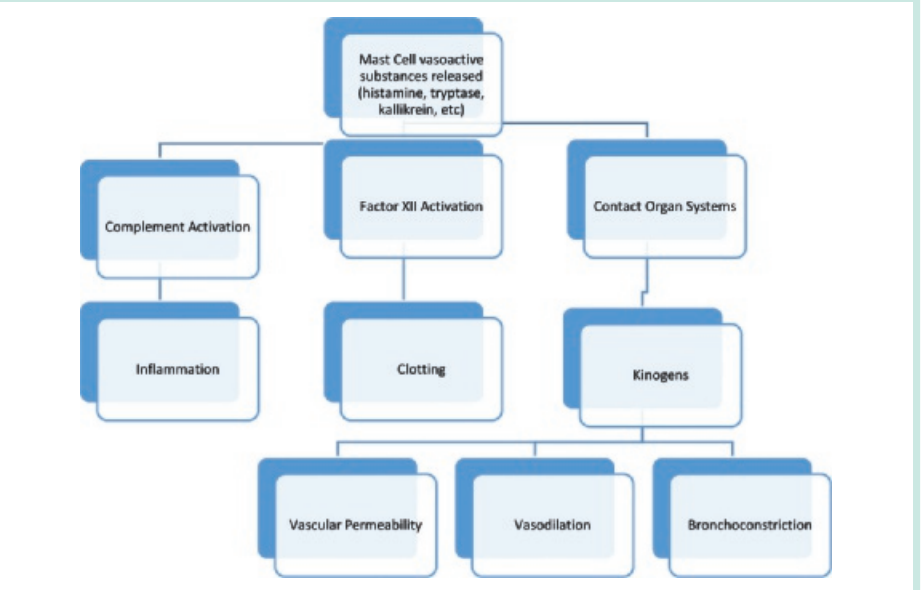
Every day, anesthesiologists encounter patients with a history of immune-mediated reactions. While there are many different types of immune-mediated reactions, anaphylaxis is a potentially fatal allergic reaction that can occur during anesthesia or in daily life and will be the focus of this chapter. Potentially, any drug or substance can cause anaphylaxis; the most commonly associated agents during anesthesia are the neuromuscular blocking drugs (NMBDs).

1) **Pathophysiology**
   a) **Immune-mediated reactions** (Fig. 49-1)[1]
      i) Type I hypersensitivity reactions[2]
         (1) Exposure to causative agent (antigen) results in production of IgE antibodies
         (2) When the individual is later reexposed to the same antigen, the IgE antibodies promote release of histamine and other vasoactive substances from mast cells and basophils.
         (3) Examples include anaphylaxis, asthma, and allergic rhinitis.
         (4) Anaphylaxis is the most severe Type I hypersensitivity reaction.
            (a) The severity of anaphylaxis is NOT dose dependent.
            (b) The incidence of perioperative anaphylaxis is estimated to be between 1:1250 and 1:20 000, with a mortality rate of 2%-7%.[3]
            (c) Women are at greater risk than men of developing anaphylaxis.[4]
      ii) Type II hypersensitivity reactions[2]
         (1) Antibody-dependent cell-mediated cytotoxic reactions.
         (2) Mediated by IgG or IgM antibodies.
         (3) Antigens are on the surface of foreign cells.
         (4) Examples include ABO-incompatible transfusion reactions and heparin-induced thrombocytopenia.
      iii) Type III hypersensitivity reactions[2]
         (1) Immune-complex reactions.
         (2) Antigens and antibodies bind in the bloodstream to form complexes.
         (3) Complexes deposit in the vasculature, which may result in tissue injury.
      iv) Type IV (delayed) hypersensitivity reactions[2]
         (1) Mediated by cytotoxic T cells.
            a) 2-4 days after exposure to antigen
         (2) Sensitized lymphocytes react with antigens.
         (3) Examples include tissue rejection and graft vs host reactions.
   b) **Nonimmunologic reactions (formerly anaphylactoid)**
      i) Clinically indistinguishable from anaphylaxis.
      ii) Causative agent directly stimulates the release of the vasoactive substances, without IgE mediation.
      iii) Severity of an nonimmunologic reaction is dose dependent.
      iv) Treatment is the same as for anaphylaxis (Table 49-1).



50%-70% of anaphylaxis events during anesthesia are caused by NMBDs.

FIGURE 49-1 Anaphylaxis Activation.



Adapted from Pattanaik D, Yataco JC, Lieberman P. Anaphylactic and anaphylactoid reactions. In: Hall JB, Schmidt GA, Kress JA. *Principles of Critical Care*. 4th ed. New York, NY: Mc-Graw Hill; 2014.1.[1]

2) **Agents responsible for Type I hypersensitivity reactions**
   a) 50%-70% of anaphylaxis events during anesthesia are caused by NMBDs.
      i) The order of frequency is rocuronium, succinylcholine, and vecuronium.
      ii) All NMBDs can cause anaphylaxis, and they can be cross-reactive with one another.
   b) Other causes of perioperative anaphylaxis in order of their frequency:
      i) Antibiotics > chlorhexidine > latex exposure > colloids > hypnotics > opioids

TABLE 49-1 Management of Anaphylaxis and Arrest Under General Anesthesia

Initial Therapy
• Fluids (volume expansion)
• Remove offending agent
• Epinephrine (10-50 µg IV with hypotension, 100 µg to 1 mg IV with cardiovascular collapse). Consider epinephrine infusion
• Steroids (50-150 mg of hydrocortisone or equivalent)
• Histamine blockers (H_1 and H_2 blocker)
• Airway (FiO_2 to 100%, may need to disconnect ventilator to treat Auto-PEEP)
• Initiate CPR if appropriate
• Check for serum tryptase
• Admit to ICU (24-h observation)
• Vasopressin (for hypotension refractory to epinephrine)
• ECMO evaluation

Adapted with permission from Perioperative cardiac arrest: focus on anaphylaxis. *Anesth Anal*. 2018;126(3):735.

c) **Allergic reactions to local anesthetics**
   i) Extremely rare
   ii) Ester-based local anesthetics are more likely than amides to cause a reaction.
   iii) Some patients react to the preservatives used in the preparation.

d) **Latex allergy**
   i) Incidence of latex allergy and anaphylaxis associated with latex products has been decreasing.
   ii) Populations at increased risk include children who have had numerous surgeries, patients with spina bifida, and health care workers.
   iii) Anaphylaxis to latex presents during the maintenance phase of anesthetic
   iv) Management of latex allergy
      (1) A patient with known hypersensitivity to latex should be scheduled as the first case of the day in a latex-free environment, with special latex-free equipment and carts.

e) Sugammadex allergy
   i) Uncommon source of intraoperative anaphylaxis
   ii) Estimated incidence of 1:33 000 although likely to see increase in incidence as it is used more often[5,6]

f) Chlorhexidine allergy
   i) Increasing in incidence
   ii) Utilized in antiseptic scrubs for surgery, ureteral lubricants, central venous catheters
   iii) Seen in over-the-counter bathing solutions, toothpaste, mouthwash, and lozenges

3) **Clinical manifestations**

a) Type I hypersensitivity reactions can present with pruritus, mild rash, or hives.
   i) Full-blown anaphylaxis may present with hypoxia, hemodynamic instability, or cardiovascular collapse.
   ii) The signs and symptoms often present rapidly following administration of the causative agent, which causes mast cell degranulation and release of vasoactive substances.
   iii) The most common feature is a rash or hives followed by cardiovascular collapse (hypotension and tachycardia or bradycardia), bronchospasm, and angioedema.
   iv) The patient may present with only a single manifestation of anaphylaxis or with multiple associated findings.
   v) One may only recognize anaphylaxis after a dramatic change in respiratory or hemodynamic parameters.

4) **Treatment of anaphylaxis (Table 49-1)**

a) The treatment is supportive with the goal of providing adequate oxygenation and maintaining adequate hemodynamic stability.

b) If you suspect a patient of having an anaphylaxis event, discontinue exposure to the suspected agent.
   i) Airway and ventilation
      (1) Place the patient on 100% oxygen.



Ester-based local anesthetics are more likely than amides to cause a reaction.



Anaphylaxis to latex presents during the maintenance phase of anesthetic



Full-blown anaphylaxis may present with hypoxia, cardiovascular collapse, and death

50%-70% of perioperative anaphylaxis events are caused by NMBAs



Anesthesia providers and other health care workers are at risk for developing latex allergy

When a patient with a latex allergy has surgery, it is important to check all equipment for the presence of latex



When treating a patient for anaphylaxis, **epinephrine** is the drug of choice

(2) If the patient is not already intubated, consider early airway intervention due to complications from angioedema.
(3) Treat bronchospasm with low-dose epinephrine in moderate to severe cases.
(a) Albuterol or other inhaled β_2 agonists can be used in milder cases.

ii) Circulatory support
(1) Administer epinephrine IV to treat hypotension.
(2) Epinephrine stabilizes mast cell degranulation, thus decreasing the amount of histamine circulating in the blood.
(3) Give an initial dose of 5-10 μg of epinephrine, increasing it (usually doubling) every 2 minutes, until you observe clinical improvement.
(a) If clinical improvement is observed, an epinephrine infusion should be considered and titrated to hemodynamic stability.
(b) Patients on β-blockade therapy may require large doses.
(4) Vasodilation may also be treated with IV fluids.
(a) Large volumes (2-4 L) are sometimes required.
(b) Consider central venous access to guide therapy.
(5) Consider administering vasopressin or norepinephrine if the patient continues to be hypotensive following treatment with epinephrine.[6]

iii) Secondary support measures
(1) H_1 antagonists such as diphenhydramine (0.5-1 mg/kg) should be administered.
(2) Although no data exists to support using H_2 blockers or corticosteroids in the acute-phase treatment of anaphylaxis, these agents might decrease the incidence of rebound or the return of symptoms that can occur after successful treatment.

iv) Consider obtaining a serum mast cell tryptase level.
(1) Tryptase is a marker for anaphylaxis and can be used as part of the differential diagnosis.
(2) Can be measured from 1 to 6 hours after the event.[7,8]
(3) Results are not available for several days.



Doses of epinephrine should initially be small (10-50 μg) and increased incrementally to avoid the possibility of severe hypertension and tachycardia

Patients treated for anaphylaxis are at risk of relapsing several hours after the initial event

5) **Postevent follow-up**
a) If successfully treated, most anaphylaxis events will resolve within 1 hour.
i) Depending on the severity of the reaction, the patient may require continuing treatment.
b) Monitor patients with moderate to severe anaphylaxis in an intensive care setting.
i) Continued intubation is usually warranted.
c) Update the patient's chart to reflect the most likely cause of anaphylaxis.
d) Inform the patient of his/her allergy status.
e) Perform postoperative allergy testing for all medications used prior to the event.[7]
i) If allergy to NMBD is suspected, recommend that the patient be tested for reactions to all NMBDs, and note which one(s) does not cause a reaction, for future reference when anesthetics will be used.
(1) However, it is important to bear in mind that if the patient has an allergy to a particular NMBD, there is still a risk of cross-reaction.

CHAPTER SUMMARY FOR ALLERGIC REACTIONS AND ANAPHYLAXIS

Pathophysiology	IgE-mediated histamine release in response to antigen exposure Nonimmunologic reactions are not IgE mediated, but signs and symptoms are clinically indistinguishable from anaphylaxis
Cause	Many drugs and environmental exposures
Symptoms	Rash, bronchospasm, airway edema, hypotension, tachycardia, and circulatory collapse
Treatment	Supportive care focusing on oxygenation, IV fluid management, and treatment with epinephrine

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50 Human Immunodeficiency Virus

Dan Hoang • Andrea J. Traynor

Human immunodeficiency virus (HIV) is a virus whose infection is responsible for acquired immunodeficiency syndrome (AIDS). In parts of sub-Saharan Africa, HIV infection is reaching epidemic proportions. In 2015, the CDC estimated 1.1 million people in the United States are infected with HIV, 15% of whom are unaware of their illness. Approximately 20%-25% of HIV-positive patients will require surgery throughout the course of their lifetime.[1]

1) **Pathophysiology**
   a) **HIV-1** is the most commonly encountered strain, which causes AIDS.
      i) HIV-2 predominates in western Africa.
   b) **Transmission**
      i) Through blood, saliva, sexual contact, and vertically (mother to infant)
         (1) Vertical transmission is substantially reduced by cesarean delivery and triple antiretroviral therapy (ART).
            (a) As low as <1% to 2.4% vs 9% to 22% in untreated women.[2]
   c) HIV-1 is a single-stranded RNA virus. Upon entering a cell, it produces double-stranded DNA via an enzyme called reverse transcriptase.
   d) CD4 T lymphocytes are predominantly infected.
2) **Diagnosis**
   a) **Testing for HIV**[2]
      i) Recommended screening in all individuals aged 15-65, in all pregnant women, and in younger or older patients at increased risk (injection drug use, unprotected sexual intercourse).
      ii) ELISA testing for anti-HIV antibody is the most common means of testing.
         (1) Because of the high sensitivity with ELISA testing, a second test with high specificity (Western blot) is usually performed after a positive result.
      iii) P24 antigen in combination with HIV antibody provides both screening and confirmation.
      iv) Rapid HIV testing from oral or finger-stick specimens is available but requires confirmatory testing with a positive result.


AIDS is defined as a CD4 count of <200 cells/µL.

   b) **Acquired immunodeficiency syndrome (AIDS)**
      i) Defined as a CD4 count of <200 cells/µL
      ii) High risk of opportunistic infections and malignancy (index conditions) such as Kaposi sarcoma, *Pneumocystis carinii* pneumonia, coccidioidomycosis, or recurrent bacterial infections
   c) **HIV RNA viral load** is a measurement of viral activity.
      i) Patients with a high viral load are at increased risk for disease progression and transmission to others.[2]
3) **Clinical manifestations**
   a) **Neurologic**
      i) Occur early in the disease and throughout its course
         (1) Early: Myelopathy, peripheral neuropathy, Guillain-Barré syndrome

(2) Latent: Autoimmune demyelinating neuropathies resembling Guillain-Barré syndrome
(3) Late: Opportunistic meningitis (cryptococcal most common), CNS lymphoma, AIDS dementia complex (cognitive, motor, and behavioral changes)


HIV-positive patients are at elevated risk for coronary artery disease.

b) **Pulmonary**
i) Opportunistic infections predominate. Thorough evaluation for residual pulmonary impairment is essential prior to formulating an anesthetic plan.
(1) Pneumocystis carinii, tuberculosis, aspergillosis, and other bacterial organisms are frequent pathogens.

c) **Cardiovascular**
i) Elevated risk for coronary artery disease due to chronic inflammation and immune activation.[3]
(1) Some ARTs are associated with elevated risk of MI, especially older generations of therapy.
ii) The incidence of cardiomyopathy, pulmonary hypertension, myocarditis, and pericardial effusions is higher in untreated HIV-positive patients compared to the general population.[1]

4) **Treatment**
a) It is recommended to begin antiretroviral therapy as soon as possible after diagnosis.[4]
b) Drugs used to treat HIV can be divided into five major classes. Common side effects and interactions are reported in **Table 50-1**.
i) Nucleoside analog reverse transcriptase inhibitors (NRTI)
ii) Nonnucleoside analog reverse transcriptase inhibitors (NNRTI)
iii) Protease inhibitors
(1) Potent inhibitors of cytochrome P-450 3A4 and 2D6[4]
(2) Can affect fentanyl and benzodiazepine metabolism
iv) Integrase strand transfer inhibitors (INSTI)
v) Fusion inhibitors
vi) Prophylactic agents are also used to prevent or treat opportunistic infections
c) Common starting treatment regimens include two NRTIs in combination with a drug from a different class.

5) **Anesthetic implications**
a) **Preoperative**
i) History should include a review of medications and side effects.
(1) A consultation with the primary care physician and/or HIV specialist may be warranted if the medications are unfamiliar.
(2) Include questions about the patient's general health including exercise tolerance, presence of pulmonary disease, and neurologic history.
ii) Physical examination
(1) A general physical with a thorough evaluation of the patient's cardiac, pulmonary, and neurologic systems is necessary.
iii) Labs and tests
(1) Should include CBC, liver enzymes, CD4 count, and a review of viral load. Some patients will require EKG, CXR, cardiac echo, PFTs, and ABGs depending on their overall health status.


All HIV-positive patients should be considered immunosuppressed.

b) **Intraoperative**
i) All HIV-positive patients should be considered immunosuppressed.

TABLE 50-1 Medications Used to Treat HIV-1 Infection

Drug Class	Mechanism of Action	Example	Side Effects	Special Concerns
Nucleoside analog reverse transcriptase inhibitor	Inhibits reverse transcriptase by preventing DNA elongation	• Zidovudine (AZT, Retrovir) • Emtricitabine (Emtriva) • Tenofovir (Viread) • Abacavir • (Ziagen)	• Bone marrow suppression, GI disturbances • Lactic acidosis • Renal insufficiency • Increased MI risk	• Few drug interactions • AZT not used in the United States as frequently as other medications
Protease inhibitors	Prevent HIV protease from making functional proteins	• Ritonavir (Norvir) • Darunavir • Atazanavir • Lopinavir	• Increased triglycerides and transaminases • GI effects (N/V/D) • Prolonged PR and QT	• Inhibitors of CYP3A4 • May alter metabolism of sedative-hypnotics, antiarrhythmics
Nonnucleoside reverse transcriptase inhibitors	Inhibit proper function of reverse transcriptase	• Nevirapine (Viramune) • Doravirine • Delavirdine (Rescriptor) • Efavirenz • Rilpivirine	• Severe skin rash, Nevirapine and Efavirenz induce CYP3A4 • Delavirdine inhibits CYP3A4 • QTc prolongation	• May alter serum concentrations of warfarin, calcium channel blockers, and antiarrhythmic drugs
Integrase strand transfer inhibitors	Prevents HIV from inserting viral DNA into host CD4 cell	• Bictegravir • Elvitegravir • Dolutegravir • Raltegravir	• Inhibits creatinine secretion; depression and suicidal ideation	• Elvitegravir is coadministered with a CYP3A4 inhibitor, may increase serum concentration of some drugs
Fusion inhibitors	Binds to HIV and prevents viral fusion with target cell membranes	• Enfuvirtide (Fuzeon)	• Injection site reactions	• Few drug interactions

Adapted from Hughes SC. HIV and anesthesia. *Anesth Clin North America*. 2004;22:379–404; United States Department of Health and Human Services. Available at: https://aidsinfo.nih.gov/contentfiles/lvguidelines/AA_Tables.pdf

ii) General anesthesia
   (1) There are no specific concerns with GA except those pertaining to the patient's underlying illness and drug interactions.

iii) Regional anesthesia
   (1) Epidural and spinal anesthesia are generally considered safe.
   (2) Viral infection of the CNS occurs early in the disease, so it is unlikely that neuraxial anesthesia will spread HIV infection to the CNS.[1]



Epidural and spinal anesthesia are considered safe in HIV-positive patients.

iv) Epidural blood patch is safe in this population and is reasonable to attempt when conservative measures fail to treat a postdural puncture headache.[1]

c) **Postoperative**
   i) Postoperative considerations are related to the underlying disease processes.

CHAPTER SUMMARY FOR HIV		
Definition	HIV is a retrovirus that infects the immune system and causes AIDS	
Laboratory Assessment	CD4 count is a measure of the immune system's strength CD4 <200 cells/μL indicates patient is at high risk for opportunistic infections	
Anesthetic Considerations	Preoperative	Thorough history and physical Review labs and medications Patients may be at increased risk for CAD
	Intraoperative	Protease inhibitors may slow fentanyl and benzodiazepine metabolism Regional anesthesia NOT contraindicated

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51 Part C: Respiratory Diseases
Chronic Obstructive Pulmonary Diseases§

Joyce Chang • Linda L. Liu

Chronic obstructive pulmonary disease (COPD) is characterized by persistent respiratory symptoms due to airflow limitation from airway and/or alveolar abnormalities and abnormal inflammatory responses to noxious particles or gases. COPD can include chronic obstructive bronchitis and/or emphysema and is distinguishable from asthma by the irreversibility of the obstructive pattern.

1) **Epidemiology**
   a) COPD is the fourth leading cause of chronic morbidity and mortality in the United States.[1] It is the fourth leading cause of death in the world and projected to be the third leading cause by 2020.[2]
   b) Prevalence can be as high as 5%-10% in the age-matched general surgery population and up to 40% of thoracic surgery patients.[3]



COPD is the fourth leading cause of chronic morbidity and mortality in the United States.[1,2]

2) **Pathophysiology**
   a) Pathological changes consist of chronic inflammation seen in the proximal and peripheral airways, lung parenchyma, and pulmonary vasculature.
      i) The gas exchange abnormalities of severe COPD depend on whether the inflammation leads to emphysema with alveolar wall and pulmonary capillary destruction or chronic bronchitis with airway obstruction and cough/sputum production.
   b) Emphysema is characterized by an inflammatory process that leads to destruction of respiratory bronchioles, alveolar ducts, and alveoli, and they tend to have $PaO_2 > 65$ mm Hg, with a normal to slightly decreased $PaCO_2$.
   c) Patients with chronic bronchitis have:
      i) Hypoxia from *V/Q* mismatch
      ii) Hypercapnia from increased dead space ventilation and impairment of ventilatory muscles[4]
3) **Risk factors**
   a) **Tobacco smoke is the primary cause.**
   b) However, indoor and outdoor air pollution and genetic predisposition also factor prominently.
4) **Clinical features**
   a) Patients often present with a history of smoking, presence of chronic cough, sputum production, and dyspnea on exertion.[2]
5) **Physical examination**
   a) Depending on severity of disease, patients can have
      i) Decreased breath sounds, wheezes, prolonged expiratory phase
      ii) Increased A-P diameter of the chest

iii) Evidence of hyperinflation on CXR
iv) Use of accessory muscles for respiration

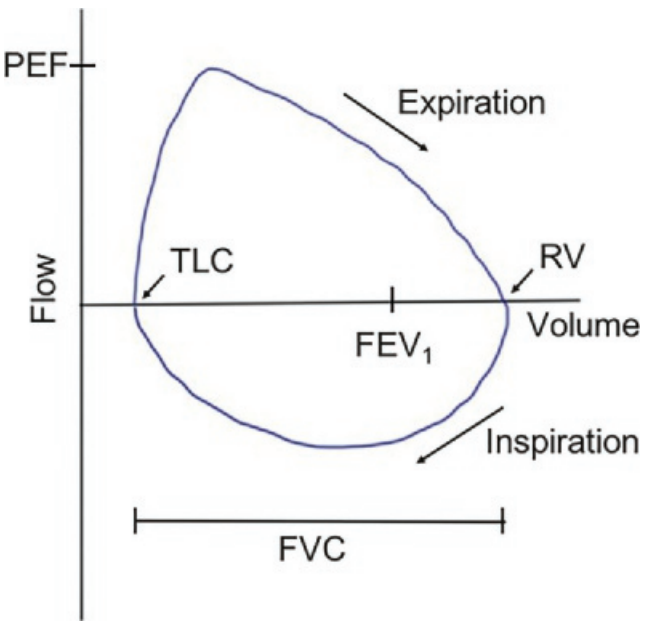
6) **Pulmonary function tests (PFTs) (Fig. 51-1)**
   a) **Narrowing of the small airways and decreased lung elastic recoil** lead to the airflow limitations in COPD. This translates into
      i) Reduced FEV_1 and FEV_1/FVC
      ii) Increased residual volume, functional residual capacity, and total lung volume



Fourteen percent of medical patients admitted for COPD exacerbation will die within 3 months.

7) **Prognosis**
   a) Fourteen percent of medical patients admitted for COPD exacerbation will die within 3 months of admission.
   b) In patients with COPD, the incidences of respiratory failure and pneumonia after noncardiac surgery are 1%-3% and 1%-5%, respectively.[3]
8) **Preoperative considerations**
   a) **Identification of high-risk patients is essential to improving their postoperative course (Table 51-1).**
      i) The odds ratio for postoperative pulmonary complications (PPC) is increased in patients with COPD (OR 1.79), with an unadjusted PPC rate of 18.2%.[5]
      ii) The incidence of PPCs often increases with increasing severity of disease, especially in the severe to very severe range (grades III-IV) or if patients have concomitant

FIGURE 51-1 Measurements Obtained From Pulmonary Function Tests as Shown on a Normal Flow Volume Loop



PEF, peak expiratory flow; TLC, total lung capacity; FEV1, forced expiratory volume at 1 second; RV, residual volume; FVC, forced vital capacity.

TABLE 51-1 High-Risk Patients

GOLD COPD Disease Grade[1]	Risk of Exacerbation/Symptom Burden
1. $FEV_1 \geq 80\%$ predicted	0-1 exacerbation without hospital admission, minimal symptomatic impact of disease
2. FEV_1 50%-79% predicted	0-1 exacerbation without hospital admission, high symptomatic impact of disease
3. FEV_1 30%-49% predicted	≥2 exacerbations or ≥1 leading to hospital admission, minimal symptomatic impact of disease
4. $FEV_1 < 30\%$	≥2 exacerbations or ≥1 leading to hospital admission, high symptomatic impact of disease

Identification of high-risk patients should be via a combination of GOLD COPD disease grade (left column) and risk of exacerbation/symptom burden (right column). FEV_1 loses precision and should not be used alone to assess risk. Progressive movement down each column indicates higher risk.

abnormal clinical findings (wheezes, rhonchi, prolonged expiration, decreased breath sounds).[3,6]

(1) Individual disease stratification can be done according to the GOLD criteria, a frequently updated evidence-based consensus report assembled by an expert panel on obstructive lung diseases.[2]

iii) PFTs are usually not necessary for preoperative screening because clinicians can diagnose and assess COPD severity with high accuracy based on history and physical examination alone.[2,3,6] Other patient-related risk factors include:

(1) Current smoking
(2) Acute COPD exacerbation
(3) Advanced age
(4) Poor general health

b) **Procedure-related risk factors** should also be kept in mind, with increased risk associated with

i) Operative site (incision closer to diaphragm)
ii) Surgery >3 hours
iii) Disruption of normal respiratory muscle function from anesthesia drugs, neuromuscular blocking drugs, and surgical trauma

c) Preoperative risk reduction strategies can include

i) Smoking cessation (preferably >2 months prior to surgery)
ii) Treatment of underlying COPD exacerbation (bronchodilators, glucocorticoids[7]
iii) Antibiotics for infections, if present
iv) Preoperative teaching and institution of lung expansion exercises[6]



Benzodiazepines can be used in COPD patients, but should be used cautiously or avoided in those with severe COPD due to respiratory depressant effects.

9) **Intraoperative considerations**

a) Care consists mainly of optimal airway, ventilator management, and postoperative pain management strategy (as described in **Table 51-2**) and the recognition that these patients are susceptible to PPCs.

b) **Preoperative anxiolytics** such as benzodiazepines can be used in COPD patients. However, because of their dose-dependent depressant effects on minute ventilation, they should be used with caution or avoided in patients with severe COPD.[9]

c) Multidisciplinary team approach works best in the care of COPD patients. Consideration should be given to use of minimally invasive surgery (laparoscopy) when possible. The mild CO_2 retention from the pneumoperitoneum in patients with mild/moderate COPD did not increase PPC or hospital stay.[10]

TABLE 51-2 Anesthetic Concerns for Patient With COPD

Airway management	• Face mask, LMA, tracheal intubation can all be appropriate: ○ If tracheal intubation—ensure adequate anesthetic depth, strongly consider inhaled beta-agonists, inhaled anticholinergic agents, inhaled or IV steroids, and IV lidocaine[3]
Ventilator management	• Warm and humidify fresh gas, especially in long surgeries • Reduce autoPEEP: low RR and increase I:E ratio—if suspect, autoPEEP, disconnect circuit, suction, and increase E time[3] • Low tidal volume ventilation: 6-8 mL/kg of ideal body weight • Avoid posthypercapnic alkalosis by avoiding hyperventilation • All volatile anesthetics are equipotent bronchodilators except desflurane which can cause airway hyperreactivity • Ensure full neuromuscular blockade reversal prior to extubation[2,3-5,7]
Regional anesthesia	• Lack of consensus if intraop neuraxial anesthetic vs GA lowers risk of PPC[2–5,11] • Unclear if intrathecal opioids lower PPC as compared to systemic opioids[3–5,11] • Regional anesthesia associated with lower morbidity and PPC but not mortality[8,12] • Postoperative maintenance of epidural local analgesia reduces PPC compared to systemic opioids and is recommended when appropriate[11]

d) Volatile anesthetics except for desflurane are good bronchodilators.
e) Ensure full reversal of neuromuscular blocking agents prior to extubation.
f) Consider regional anesthesia, although the idea that regional anesthesia has less risk of PPC compared to general anesthesia is controversial. For example, thoracic epidural analgesia may reduce risk of PPC in high-risk surgeries such as abdominal aortic surgery.[11]

10) **Postoperative considerations**
a) A primary postoperative goal is to minimize the incidence and severity of PPC.
b) Analgesia is important to maintain respiratory function and minimize splinting, which may permit early tracheal extubation or prevent reintubation.
c) Epidural analgesia may be helpful by preserving diaphragmatic function in addition to increasing tidal volume and vital capacity.[4]
d) PCA analgesia reduces PPC as compared to nurse-administered analgesia.[6]
e) Lung expansion techniques help to prevent/reopen atelectatic regions and to prevent postoperative reduction in lung volumes.
f) Incentive spirometry and deep breathing exercises each can reduce the incidence of PPC, but the combination of these techniques fails to confer additional benefits.
g) Noninvasive positive pressure ventilation (NIPPV) should be the first mode of ventilation used for acute respiratory failure from COPD exacerbation if no absolute contraindications.[2]
h) Respiratory rehabilitation and frequent pulmonary toileting improves secretion handling.
i) Head-up position is an effective way to decrease pulmonary aspiration.

CHAPTER SUMMARY FOR COPD	
Definition	COPD is characterized by chronic inflammation of the airways, lung parenchyma, and pulmonary vasculature. Patients exhibit obstructive respiratory physiology on pulmonary function testing.
Preoperative Issues	Identify those at risk: Severe COPD, acute exacerbation, active smoking, poor general health, advanced age, high-risk surgery PFT not usually needed Smoking cessation counseling, antibiotic, bronchodilator, glucocorticoid therapy as needed
Intraoperative Issues	Laparoscopic approach better if feasible Consider regional anesthesia and epidural analgesia Ensure adequate anesthetic depth if general anesthesia Avoid desflurane if possible
Postoperative Issues	Maximize analgesia: Epidural or PCA preferred Lung expansion techniques are crucial and include: Incentive spirometry Deep breathing exercises Noninvasive ventilation as needed Frequent pulmonary toileting Maintain head-up position

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52 Asthma and Reactive Airway Disease

Lisa Gramlich

Asthma is a chronic inflammatory disease of the airways and is the most common chronic disease of childhood in the United States. As the leading cause of absenteeism from school and the most prevalent cause of hospital admission, asthma wields a major impact on education and health care dollars. Asthma costs the U.S. economy more than $80 billion per year.[1] Asthma exacerbations show a linear increase in asthma costs as asthma control decreases.[2]

1) **Incidence**
   a) Worldwide the WHO estimates that asthma affects 235 million people.[3]
   b) 25 million people in the United States are affected; more than 6 million of these are children.
   c) The majority develop asthma in childhood.[3]
   d) The incidence has been rising steadily since 1970s.
   e) In 2016, the first decrease was seen in children with a reduction from 9.4% to 8.3%.[4]
   f) A higher incidence is seen in:
      i) Ex-premature infants in the preadolescent period.[3]
      ii) Those with a familial predisposition; two-thirds of affected patients have affected family members.[3]
      iii) African Americans and Puerto Ricans, which is the only group with an increased incidence from 2010 to 2016.[4]
      iv) Prepubertal males[3]
      v) Those with problem allergies, especially when associated with atopy.
      vi) Infants and children who develop lower respiratory tract viral infections, especially respiratory syncytial virus (RSV).[3]
      vii) Obese individuals[4]
2) **Pathophysiology**
   a) Inflamed airway is hyperresponsive to irritant stimuli. Eosinophils and mast cells are triggered in the airways and release cytokines, leukotrienes, and histamine. Airway obstruction ensues due to these mediators via:
      i) Smooth muscle spasm
      ii) Airway mucosal edema
      iii) Hypersecretion
      iv) Mucous plugging
3) **Precipitating factors**
   a) **Lower respiratory viral infections,** especially RSV, provoke hyperreactivity even in nonasthmatics for up to 6 weeks.[3]
   b) **Gastroesophageal reflux disease (GERD)**
      i) GERD in infants is part of the triad of the Sandifer syndrome, which is GERD, asthma, and opisthotonos.
      ii) Treatment of GERD with H_2 receptor antagonists and prokinetics decreases asthma exacerbations in patients with symptomatic GERD, but those with negative pH probe studies show no significant improvement in their symptoms.[5]

c) **Emotional stress**
d) **Inhaled allergens** such as dust mites, animal dander, mold, and dust
e) **Inhaled irritants** (cigarette smoke, caustic cleansers, inhaled anesthetics)
   i) All inhaled anesthetics are bronchodilators but initially act as an irritant, especially when the air is dry and cold. This effect can be primarily ablated by preoperative use of β_2-adrenergic agonists.[6]
   ii) Secondhand smoke increases severity and frequency of symptoms, but an increased incidence is not consistently shown in studies.[7]
f) **Mechanical irritation such as intubation**



If possible, intubation should be avoided in patients with reactive airways as it is associated with an increase in pulmonary complications.[8]

4) **Symptoms**
   a) Can follow circadian rhythms and worsen at night, especially around 04:00.[3]
   b) Dyspnea and tachypnea may lead to difficulty speaking.
   c) Chest tightness and tachycardia
   d) Wheezing, except when severe bronchospasm limits air movement.
   e) Coughing (chronic cough is the most common manifestation of asthma).[8]
   f) Pulsus paradoxus (a fall in BP > 10 mm Hg during spontaneous inspiration when BP should stay the same or slightly increase).
   g) Visible use of accessory muscles and pursed lip breathing.
   h) Prolonged expiration with I:E (inspiratory to expiratory time) ratio >1:3.
   i) Altered consciousness, especially when $SpO_2 < 90\%$ and $PaCO_2 >$ 45 mm Hg.



Patients at risk for imminent respiratory arrest have the following symptoms: drowsy, confused, no wheezing (little air movement), peak flow rate (PEF) <25% (though unlikely to perform test), bradycardia, and absence of pulsus paradoxus (due to muscle fatigue). These patients should be intubated urgently.

5) **Severity scoring (Table 52-1)**
   a) Included in **Table 52-1** are guidelines to aid the clinician in assessing asthma severity and control.
6) **Asthmatic wheezing** (See **Table 52-2** for differential diagnosis of wheezing.)
   a) Polyphonic
   b) Extends to the periphery
   c) Frequently accompanied by dry cough.
7) **Treatments (Table 52-3)**
   a) **β_2-Adrenergic agonists**
      i) The mainstay of initial asthma treatment.
      ii) Mechanism of action is an increase in cAMP resulting in smooth muscle relaxation and bronchiole dilation.
      iii) Greatest effect on medium and small airways[9]
      iv) Usually administered by metered dose inhaler (MDI) or nebulizer. If patient is moving very little air, then IV, IM, or SC epinephrine 0.01 mg/kg up to 0.5 mg should be given.
      v) Plasma levels in both children and adults are higher and achieved faster with lateral thigh IM injections vs SC injections.
      vi) Patients over 40 years old admitted to the ED in the midst of an asthma attack and treated with epinephrine had decreases in blood pressure, HR, and RR and an increase in PFRs after SC epinephrine 0.3-1.2 mg. No patient developed ischemic or serious ventricular arrhythmia.[10]
   b) **Anticholinergics**
      i) Ipratropium bromide nebulized or via MDI)
      ii) Slower onset but longer duration of action than β_2-adrenergic agonists
      iii) Slightly more effective on the larger conducting airways[9]
   c) **Leukotriene-modifying drugs**
      i) Two major categories
         (1) Leukotriene receptor antagonists (zafirlukast and montelukast)
         (2) Leukotriene synthesis inhibitors (zileuton)
      ii) Leukotrienes are 1000 times more potent bronchial constrictors than histamine, so for moderate to severe asthma, these have become a mainstay of treatment.

TABLE 52-1 Severity Assessment for Asthma (for the Average Week Over the Past 4 Weeks)

Symptoms	Classification		
	Well Controlled (Mild)	Not Well Controlled (Moderate)	Poorly Controlled (Severe)
Frequency of symptoms	≤2 d/wk AND ≤ 1 time/d	>2 d/wk OR ≥ 2 times/d	>2 times/d AND > 2 d/wk
Nighttime awakening	≤2 times/mo	1-3 times/wk	>4 times/wk
Interference with activity	No limitations	Some limitations	Extreme limitations
Shortness of breath	Occurs while walking or with exercise	At rest (infant with softer cry and difficulty feeding)	At rest (infant stops feeding)
Position when breathless	Is able to lie down	Prefers sitting	Sits upright
Able to speak in	Full sentences	Phrases	Words
Alertness	Alert	May be agitated	Usually agitated
Use of accessory muscles	Rare	Common	Usually
Wheezing	Moderate, usually end expiratory	Loud throughout exhalation	Usually loud in both inspiration and expiration
FEV_1, or PEF	>80%	60%-80%	<60%
No. of asthma meds	0	1-2	≥3
SABA use for symptoms	≤2 d/wk	>2 d/wk	Several times/day
No. of times PO steroids used	0-1/y	≥2/y	≥2/y
Treatment recommendations	Maintain tx	After verifying medication compliance, set up tx	Consider hospital admission for better control and assessment of adherence to medications

Adapted from 2007 NHLBI Guidelines for the Diagnosis and Treatment of Asthma Expert Panel Report 3.

TABLE 52-2 Differential Diagnosis of Wheezing

Differential	How It Is Different From Asthmatic Wheezing
Foreign body	Wheezing is usually fixed and central
Secretions	Moist cough with rhonchi frequently associated
Tracheomalacia	Onset is usually at birth and improves with age
Anaphylaxis	Will be associated with other symptoms, namely, flushing, urticaria, angioedema, and hemodynamic instability
Pneumothorax	Unilateral wheezing is noted verses bilateral wheezing in asthma
Congestive heart failure	Associated with cardiovascular symptoms and rales
Mainstem intubations or endotracheal tube (ETT) cuff herniations (over the end of the ETT) partially blocking the opening	Notable decreased inspiratory breath sounds
Acute tracheobronchiolitis	Similar wheezing; potential flulike prodrome
Mechanical obstruction—vascular rings or mediastinal masses would be extrinsic causes and laryngeal webs or bronchial stenosis intrinsic causes	More centrally located
Tuberculosis or other granulomatous disease	Similar wheezing

TABLE 52-3 Drugs for Asthma Treatment

	Maintenance	Emergency Treatment
β_2-Adrenergic Agonists		
• (SABA= short acting) • Albuterol (Proventil, Ventolin)	• MDI, 2 puffs q4-6 h • Nebulizer, 01-0.15 mg/kg	• Up to 10 puffs (1 mg) q20min • 2.5-5.0 mg q20min or continuous nebulization of 10 mg/h (requires special nebulizer)
Terbutaline		IV bolus: 15 µg/kg over 20 min followed by infusion 1-5 µg/kg/min
• (LABA = long acting) • Salmeterol (Serevent)	MDI, 1-2 puffs bid	Not indicated
Anti-inflammatory		
• Beclomethasone (Vanceril) • Budesonide (Pulmicort) • Flunisolide (AeroBid) • Fluticasone propionate (Flovent)	• MDI, 2-4 puffs bid • MDI, 1-2 inhalations bid • MDI, 1-2 puffs bid • MDI, 1 inhalation bid	• Methylprednisolone 1 mg/kg IV • Will not give immediate relief but should be given • Effect not usually seen for an hour • IV: 1.5 mg/kg hydrocortisone • *Dose per UpToDate Oct. 2018, pp. 1-31, Dobyns JB, Anesthesia for adult patients with asthma*
Prednisone or prednisolone (Prelone, Pediapred)	• 0.25-2 mg/kg qod • Pre-op 1 mg/kg/d × 3 d	Oral: 1-2 mg/kg prednisolone
Anticholinergics		
Ipratropium bromide (Atrovent)	MDI, 1-2 puffs qid	5-10 mg/kg nebulized q6h
Leukotriene Modifiers		
Montelukast (Singulair)	• ≥15 y/o, 10 mg qd • 6-14 y/o, 5 mg qd • 1-5 y/o, 4 mg qd	Peak plasma concentration achieved in 3-4 h; single preoperative dose may be beneficial
Nedocromil (Tilade)	2-4 puffs bid	Little added utility in acute setting
Methylxanthines		
Theophylline (therapeutic level 5-15 µg/mL)	5 mg/kg/d up to 200 mg	5 mg/kg over 10 min followed by 1 mg/kg/h
Magnesium		50 mg/kg over 10 min
Anesthetics		
Ketamine		1-2 mg/kg bolus followed by 15-45 µg/kg/min
Inhalational		0.5-1.5 MAC

MDI, metered-dose inhaler; MAC, minimum alveolar concentration.

d) **Inhaled corticosteroids (ICS)**
   i) Inflammation of the airways is a hallmark of asthma. Through suppression of airway, inflammation reduces airway hyperresponsiveness, and ICS are the most effective medications to control this inflammation.
   ii) ICS limit negative systemic effects while still providing potent anti-inflammatory effects on the airways.
   iii) ICS are now first-line therapy for all patients with persistent asthma, controlling symptoms and preventing exacerbations.[11]

iv) Inhaled β_2-adrenergic agonists and corticosteroids are frequently used together in the control of asthma, and it is now recognized that there are important molecular interactions between these two classes of drug.[11]
v) Oral or parenteral steroids are reserved for acute exacerbations of asthma unresponsive to maximal bronchodilator therapy.[11]
vi) Short courses of oral steroids affect the hypothalamic-pituitary-adrenal (HPA) axis for up to 10 days.[11]
vii) Large doses given over 3 weeks can affect PA axis for up to a year. Alternate days of treatment may decrease this.[11]

e) **Methylxanthines**
i) Inhibit adenosine-induced bronchoconstriction.
ii) Narrow therapeutic window requires routine blood level monitoring.
iii) These drugs are less commonly used but can be a helpful adjunct, especially for nighttime wheezing.

f) **Cromolyn sodium and nedocromil sodium**
i) Prophylactic only and not useful in the acute perioperative period.
ii) Stabilize mast cells to reduce IgE-mediated release of histamine and leukotrienes.

g) **Immunomodulators**
i) Namely omalizumab, are anti-IgE antibodies reserved for moderate to severe allergic asthmatics with elevated IgE levels who are uncontrolled despite treatment with ICS and long-acting β_2-adrenergic agonists. In these situations, it decreases asthma exacerbations, symptoms, and PO corticosteroid use and improves quality of life.[12]
ii) Administered subcutaneously every 2-4 weeks.

h) **Magnesium**
i) 50 mg/kg to treat acute presentation of moderate to severe asthma
ii) Is not effective for mild asthma or as a prophylactic agent

8) **Preoperative management**

a) Pulmonary function tests, namely, peak expiratory flow (PEF) rates, are commonly used to monitor asthmatics but are rarely required perioperatively for well-controlled asthmatics.
i) FEV_1 (forced expiratory volume) or PEF
(1) >75% predicted = good prognostic indicator
(2) 50%-75% predicted = fair to poor prognostic indicator
(3) <50% predicted = very poor prognostic indicator


In an asthmatic who has had a recent URI, strongly consider postponing elective surgery for 6 weeks.[8]

b) Patients should be queried about triggers, effective treatments, side effects to medications, and general respiratory well-being over the past month.
c) If the need for rescue medication has escalated or symptoms worsened and the surgery is elective, patients should be referred to their primary care physician to optimize their pulmonary status.
d) A recent URI (upper respiratory infection) should prompt postponing elective surgery for 6 weeks.[8]
i) In some patients, URIs are so frequent that a window of opportunity to operate becomes so narrow as to become impractical.
ii) Proven respiratory syncital virus (RSV) infections should elicit heightened concern due to their strong association with worsening bronchospasm.

READ MORE
Corticosteroids, Chapter 33

e) **Ascertain the need for steroid therapy**
i) Symptomatic asthmatics on maximal therapy may benefit from 3 to 5 days of oral steroids prior to surgery (prednisone 1 mg/kg up to 60 mg).
ii) Stress dose steroids
(1) Short courses of prednisolone can affect HPA function for up to 10 days.[11]

(2) High doses, treatment with steroids for >3 weeks, evening and daily dosing vs every other day dosing all increase the likelihood and duration of HPA suppression, which may last up to a year.[11]

(3) Inhaled steroids are usually not an indication for stress dose steroids perioperatively, but high doses (>440 μg/d) of fluticasone may reduce cortisol levels and lead to symptomatic adrenal suppression in children.[11]



A calm patient is important especially in patients with exercise-induced asthma to avoid triggering an attack.

9) **Intraoperative management**

a) **Optimizing a patient's preoperative status is crucial,** but patient compliance and the urgency of the case may make this difficult.

b) **Prophylactic use of a MDI or nebulizer prior to going to the operating room** is helpful at decreasing the bronchospastic response to airway manipulation. Most commonly a β_2-adrenergic agonist is used, but combination with an anticholinergic agent should be considered (see discussion of each drug above).

c) **Warm and humidify gases to avoid tracheal irritation**

i) HME (heat and moisture exchanger) with low flows on the ventilator

ii) Heated humidifiers attached in-line to the anesthesia circuit

d) **Induction**

i) If inhalation induction is indicated, a mask induction sevoflurane is a suitable choice.

ii) Propofol may be the best choice for IV induction because it provides bronchiole dilation.[13]

iii) As long as hemodynamically tolerated, larger doses of an induction agent, with the quick addition of an inhalational agent so the peak effect of the IV agent does not dissipate, are important.

iv) Ketamine is a consideration due to its bronchodilatory effects. A ketamine infusion may also be used for maintenance. Alternatively, inhalational agents may be used for maintenance. There are no comparative trials to argue for the choice of one agent (ketamine or inhalational anesthetics) over the other.

e) **Lidocaine may also blunt airway hyperresponsiveness.** Consider administration of 1 mg/kg, up to 100 mg, prior to induction in patients with active wheezing or on multimodal therapy.

f) **Avoid drugs that may cause histamine release** (eg, morphine and atracurium).

g) **Volatile technique** may be superior to a heavier narcotic technique due to the benefits of bronchiole dilatation.

i) Avoidance of an ETT whenever applicable is desirable to prevent triggering bronchospasm.

h) Deep extubation may be of great value in patients with hyperreactive airways.

i) **Consider avoidance of nonsteroidal anti-inflammatory drugs in the rhinitis prone patient who previously has not used nonsteroidals.**

i) Consider acetaminophen in asthmatics who have taken acetaminophen previously without sequelae.

ii) NSAIDs, especially aspirin, may cause a sudden exacerbation of asthma symptoms.[14]

iii) COX-2 (cyclooxygenase-2) inhibitors may be safely used. These anti-inflammatory drugs also do not cause significant antiplatelet effects.[15]

j) **In asthmatics, use of sugammadex vs with neostigmine combined with glycopyrrolate to reverse rocuronium is more rapid and may have a higher safety index.[16]**

10) **Ventilatory management for asthma**[17]

a) **Choose tidal volumes (TV) of 6-8 mL/kg.**

i) In the presence of bronchospasm, high peak pressures are not transmitted to the alveoli. If plateau pressures are >35 cm H_2O, then reduction in TV is prudent to prevent risk of pneumothorax.

b) Breath stacking occurs when obstruction to air flow does not allow full expiration.
   i) In order to avoid breath stacking, which leads to air-trapping and auto-PEEP, I:E ratios are changed from the usual 1:2 to 1:3 to 4.

c) Recruitment maneuvers every 30 minutes may prove helpful.

d) Permissive hypercapnia (up to $PaCO_2$ 80 mm Hg) keeping the pH > 7.2 is frequently adopted to minimize barotrauma during periods of sustained severe bronchospasm (status asthmaticus).

e) Intubation criteria:
   i) Cardiac arrest
   ii) Respiratory arrest or profound bradypnea
   iii) Physical exhaustion
   iv) Altered sensorium, such as lethargy or agitation
   v) $PaCO_2$ > 80 mm Hg
   vi) PaO_2 < 60 mm Hg on >60% FiO_2
   vii) pH < 7.2
   viii) Worsening symptoms despite escalating treatment should prompt preparation for intubation.

11) **Postoperative management**

a) **Extubation criteria/considerations**
   i) There are no well-defined postoperative extubation criteria and decisions must be made based on the severity of reactive airway disease (RAD), the surgical procedure, and the potential for difficult airway management.
   ii) Preparation for extubation
      (1) Intraoperative events should be reviewed
         (a) When anesthesia is lightened, signs of bronchospasm may be witnessed:
            (i) Increased airway pressures
            (ii) Upsloping CO_2 curves
            (iii) Desaturation
      (2) If any perioperative wheezing has occurred, preparations should be made to immediately administer nebulized β_2-adrenergic agonist and possibly an anticholinergic should bronchospasm occur postextubation.
      (3) Consideration for a deep extubation should be made if signs of bronchospasm are seen, which were readily relieved by a deepening of the anesthetic.
      (4) A deep extubation means the patient is without spontaneous reflexes, for example, gag, blink. A patient may be deeply extubated on inhalational agents or lightened and given a small dose (0.5-1 mg/kg) of propofol immediately before extubation.
      (5) Shortly before extubation, consider administering the same agents to decrease airway reactivity that may have been given at induction such as β_2-adrenergic agonists and/or IV lidocaine.
      (6) At least an hour before extubation, consider administration of methylprednisolone 2 mg/kg or hydrocortisone 4 mg/kg for patients who have active wheezing/bronchospasm preoperatively or intraoperatively.
      (7) If a patient is a difficult mask ventilation or reintubation, a deep extubation should not be considered.

(8) Postoperative pain control
   (a) Regional anesthesia may help to minimize opioids and crying/agitation, which is a known asthmatic trigger.
   (b) Care must be taken to spare blocking the diaphragm (phrenic paralysis) which has demonstrated a 25% reduction in FEV_1.
      (i) Interscalene block carries a 100% risk of phrenic nerve paralysis; supraclavicular 60%-70% risk.
      (ii) Infraclavicular and axillary blocks have a <1% risk of phrenic nerve paralysis.
   (c) Low-dose ketamine infusions (5-15 µg/kg/m) may decrease opioid usage while also providing bronchodilation. Ketamine is less likely to depress respiratory drive.

READ MORE
Atlas of peripheral nerve block procedures, Section 15

iii) Preoperative asthma drug management should be resumed after surgery.
iv) CPAP or BiPAP open up bronchioles to prevent alveolar air-trapping, improving symptoms more than air exchange. These modalities also improve delivery of nebulized drugs.
v) Vapotherm (high flow warmed, humidified O_2) with oxygen flows of 6-40 L/m has been shown to improve oxygenation and symptoms and also improves delivery of nebulized drugs.

12) **Less commonly used treatments**
   a) **Inhaled anesthetics in the ICU**
      i) Isoflurane or sevoflurane may be indicated for refractory status asthmaticus in the intubated patient already on multidrug therapy. Desflurane is associated with an increased risk of bronchospasm compared with sevoflurane or isoflurane, so it is usually avoided in asthma.[18] Stringent criteria for instituting and weaning need to be established prior to beginning this therapy.
      ii) Heliox may provide some initial decrease in feelings of dyspnea, but effects are controversial for moderate and severe asthma. For maximum effect the heliox concentration needs to be higher, decreases FiO_2 delivery, and limits its use.
         (1) In severe asthma, delivery of inhaled albuterol using heliox results in a greater improvement of PFTs compared with delivery utilizing oxygen alone. This has not been shown routinely with less severe exacerbations.[19]
      iii) ECMO use in refractory asthma is controversial. One study of 272 patients showed a survival rate of 83.5% with a complication rate of 65%, the most common being major hemorrhage which was associated with a significant increase in mortality and morbidity.[20]

CHAPTER SUMMARY FOR ASTHMA AND RAD

Preoperative Considerations	All routine medications should be administered the day of surgery Albuterol (β_2-adrenergic agonist) ± ipratropium bromide (anticholinergic) inhaler/nebulizer within an hour of induction Administer inhaled steroids, and consider IV steroids
Intraoperative Considerations	Ensure a deep level of anesthesia before instrumenting airway Consider lidocaine 1 mg/kg up to 100 mg maximum prior to instrumenting airway and before extubation Favor volatiles to opioids Repeat nebulizer treatments every 2 h if any signs of bronchospasm Avoid traditional NSAIDs. Acetaminophen and COX-2 inhibitors are generally tolerated Warm anesthesia gases For ventilation, TV of 6-8 mL/kg and I:E with greater than normal expiratory time Consider deep extubation if otherwise appropriate if bronchospasm has occurred
Postoperative Considerations	Consider leaving intubated and sedated (possibly with a ketamine infusion) if bronchospasm refractory to intraoperative measures Utilize BiPAP or Vapotherm when indicated postextubation Repeat nebulizers and steroids when indicated

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53 Restrictive Lung Disease

Anita K. Malhotra

Restrictive lung disease is an abnormality of ventilation that is due to a variety of causes such as disease of the lung parenchyma with interstitial lung disease being the most common, neuromuscular diseases, and other extrapulmonary processes such as heart failure. Because the causes are heterogeneous, the diagnosis, treatment, and prognosis are also variable. Diagnosis is made with pulmonary function tests, specifically, lung volumes in addition to spirometry. Every anesthesiologist should be aware of this disorder, as it is surprisingly as prevalent as obstructive lung disease.[1]

1) **Definition and diagnosis**
   a) Restrictive lung disease is defined as a reduction in total lung capacity (TLC) with a reduced FVC < 80% predicted and FEV1/FVC ratio >0.7.[2] Expiratory airflow is generally normal.[3]
   b) Diagnosis.
      i) Spirometry.
      ii) Lung volumes, including (TLC).
      iii) DLCO (diffusion capacity of lung for carbon monoxide) may help to diagnose etiology of restrictive disease.



Restrictive disease is diagnosed when **TLC < 80% predicted** and is suggested by **FVC < 80% predicted**.

2) **Pathophysiology**
   a) Underlying pathophysiology is reduced compliance of the lung, pleura, diaphragm, or chest wall as well as reduction in lung volumes.
      i) Increases the work of breathing, resulting in a characteristic rapid but shallow breathing.
      ii) This hyperventilation keeps the $PaCO_2$ at normal or depressed levels until the restrictive disorder is very severe.
   b) Gas exchange is usually maintained until the disease process is advanced.



Patients with restrictive lung disease tend to hyperventilate and will likely have a normal $PaCO_2$.

3) **Causes of restrictive lung disease**
   a) Can often be delineated by history, examination, PFTs, and radiographs
   b) **Parenchymal**
      i) DLCO is often altered.
      ii) Causes include:
         (1) Interstitial lung disease—sarcoidosis, connective tissue diseases
         (2) Idiopathic pulmonary fibrosis
         (3) ARDS
         (4) Environmental exposures
         (5) Medication induced/radiation induced
   c) **Pleura and chest wall**
      i) Usually no abnormalities of DLCO
      ii) Causes include:
         (1) Obesity
         (2) Pleural effusions/ascites
         (3) Kyphoscoliosis
         (4) Pregnancy

d) **Neuromuscular**
   i) Any pathology of the chest wall muscles or nerves that limits the ability of respiratory muscles to inflate the lung

4) **Symptoms**
   a) Dyspnea
   b) Cough
   c) Chest pain

5) **Management of restrictive lung disease**
   a) Medical therapy
   b) Oxygen
   c) Lung transplantation

6) **Anesthetic considerations**
   a) **Preoperative**
      i) Assessment of severity of lung disease—functional capacity, PFT results (see **Table 53-1**), comorbid conditions, current therapy, and smoking history.
      ii) Patients with restrictive lung disease have a higher prevalence of pulmonary hypertension.
      iii) Pulmonary function testing.
         (1) Not necessary in every case but consider in those with severe disease, those requiring oxygen therapy without known cause, and those who have not previously been evaluated, any time pulmonary hypertension is suspected, and to assess response to therapy



Ensuring optimal pulmonary function in the pre-op assessment is a major step in reducing post-op complications.

      iv) Preoperative smoking cessation.
         (1) Optimally 8 weeks prior to surgery, but shorter duration may also be beneficial by decreasing carboxyhemoglobin levels and improved mucociliary clearance.[4]
      v) Elective surgery should be delayed in patients experiencing an exacerbation of their restrictive lung disease.
   b) **Intraoperative considerations**
      i) Because all lung volumes are reduced, a lowered FRC leads to decreased time until hypoxia occurs when the patient is apneic.
      ii) Spinal or epidural anesthesia may provide some benefit vs general anesthesia as there is less impairment of pulmonary function.[5]
      iii) Peripheral nerve blocks may be advantageous; however, any block with the potential for respiratory muscle depression or complications such as pneumothorax should be undertaken with caution.
      iv) All volatile agents are bronchodilators and may be used safely.

TABLE 53-1 Degree of Severity of Restrictive Lung Disease

Mild	$FEV_1\% \geq 70$
Moderate	$FEV_1\%$ 60-69
Moderately severe	$FEV_1\%$ 50-59
Severe	$FEV_1\%$ 35-49
Very severe	$FEV_1\% < 35$

Reproduced with permission of the © ERS 2020: Pellegrino R, Viegi G, Brusasco V, et al. Interpretative strategies for lung function tests. *Eur Respir J*. 2005;26(5):948-968; doi: 10.1183/09031936.05.00035205.

TABLE 53-2 Risk Factors for Postoperative Pulmonary Complications in Patients With Restrictive Lung Disease

Risk Factors Preexisting Pulmonary Disease
Thoracic or upper abdominal surgery
Smoking
Obesity
Age >60 y
Prolonged general anesthesia >3 h

v) Nitrous oxide may raise pulmonary vascular resistance, and its use is not recommended.
vi) Recommendations to avoid ventilator induced lung injury.
(1) Lung protective low tidal volume ventilation (6 mL/kg).
(2) Maintain lower plateau pressures.
(3) Appropriate use of positive end expiratory pressure (PEEP)—that is, balance between beneficial effects of decreasing atelectasis and negative hemodynamic consequences and increased intrathoracic pressure.



Patients with restrictive lung disease are at high risk for barotrauma with positive pressure ventilation.

c) **Postoperative considerations (Tables 53-2 and 53-3)**
i) Patients with respiratory muscle weakness may require prolonged postoperative respiratory support to overcome normal decreases in respiratory function after surgery and anesthesia.
ii) Adequate analgesia to ensure full respiratory effort and ability to cough and deep breathe.

7) **Lung transplantation**
a) Restrictive lung disease is now the most common indication for lung transplantation
i) Mortality remains high with 5-year survival of 55%.[6]
ii) Lung transplant is covered extensively in Chapter 104.

READ MORE
Thoracic Organ Transplantation, Chapter 104

TABLE 53-3 Postoperative Factors That Exacerbate Restrictive Lung Disease

Post-op Factors	Pain
	Restrictive dressings
	Sedation from opioids
	Supine position further reducing FRC
	Residual anesthetic and neuromuscular blocking drug effects
	Operations near the diaphragm and anesthetics/neuraxial blockade causing diaphragmatic dysfunction

CHAPTER SUMMARY FOR RESTRICTIVE LUNG DISEASE

Definition	Reduction in FVC < 80% of predicted and FEV1/VC ratio >0.7
Pathophysiology	Acute or chronic reduction in pulmonary compliance from multiple etiologies
Causes	Parenchymal Pleural and chest wall Neuromuscular
Preoperative Considerations	Optimize pulmonary function Aggressively treat respiratory infections Encourage smoking cessation Selectively obtain PFT and assess severity of disease
Intraoperative Considerations	Consider neuraxial anesthesia/analgesia Lung protective ventilator settings Judicious avoidance of circumstances that result in increased pulmonary vascular pressures
Postoperative Considerations	Adequate analgesia Potential need for mechanical ventilation

FEV_1, forced expiratory volume; VC, vital capacity.

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54 Obstructive Sleep Apnea

Danielle B. Ludwin

Obstructive sleep apnea (OSA), a common and often undiagnosed medical condition, is caused by airway obstruction leading to pauses in breathing during sleep. OSA has significant perioperative implications including an increased risk of difficult ventilation and intubation, postoperative hypoxemia, and cardiopulmonary arrest.[1,2] Thus, several screening tools have been developed to assess for undiagnosed sleep apnea including the ASA guidelines, Berlin questionnaire, and the STOP-Bang, and a number of perioperative strategies have been suggested to minimize these complications.

1) **Prevalence**
   a) The prevalence of OSA is predicted to be 1 in 4 men and 1 in 10 women in the United States.
      i) Often OSA is undiagnosed. It is estimated that 80% of OSA patients presenting for surgery are undiagnosed.[3,4]
         (1) The majority of OSA patients are undiagnosed.



The majority of OSA patients are undiagnosed.

2) **Diagnosis**
   a) The gold standard for diagnosis is polysomnography (sleep study).
   b) An apnea-hypopnea index (AHI) is measured detailing the number of events per hour.
      i) Typically, apnea is defined as no breathing for ≥10 seconds.
      ii) Hypopnea is a decrease in oxygen saturation ≥4% or a decrease in airflow of 25%-50%.
      iii) There are no universally accepted cutoffs, but a general guide is AHI < 5 is normal, 5-15 is mild OSA, 16-30 is moderate OSA, and >30 is severe OSA.[5]



The cycle of OSA is an episode of apnea/hypopnea causing hypoxemia, hypercarbia, and acidosis, which is followed by CNS arousal and commencement of ventilation.

3) **Pathophysiology**
   a) Obesity leads to increase in adipose tissue in the pharynx reducing pharyngeal diameter.
   b) Other anatomical conditions that can decrease airway lumen diameter include craniofacial abnormalities, macroglossia, retrognathia, and connective tissue disorders (eg, Marfan's).[6]
   c) Decreased muscle tone leads to collapse of the pharyngeal tissues.
      i) During wakefulness, the pharyngeal muscle tone is adequate to maintain an airway, but during sleep, especially REM when muscle relaxation is most pronounced, obstruction occurs until sleep is interrupted and tone is restored.
   d) The cycle of OSA is an episode of apnea/hypopnea causing hypoxemia, hypercarbia and acidosis, which is followed by CNS arousal and commencement of ventilation.
      i) This leads to poor sleep hygiene, daytime sleepiness, hypertension, right heart failure, pulmonary hypertension, left heart failure, and arrhythmias.[5]
      ii) OSA patients have a blunted response to hypercarbia and hypoxia.
4) **Preoperative management for OSA patients**
   a) Since OSA is commonly undiagnosed, screening patients for OSA is important.[7,16]

TABLE 54-1 Adapted From ASA Guidelines

Physical Characteristics	History of Airway Obstruction
BMI > 35 kg/m^2	Snoring
Neck circumference >17 in (men), >16 in (women)	Observed pauses in breathing during sleep
Craniofacial abnormalities	Awakening with a feeling of choking
Anatomical nasal obstruction	Frequent arousals from sleep
Tonsils that touch/nearly touch	

b) There are a number of screening tools including the ASA guidelines (**Table 54-1**), the Berlin questionnaire, and the STOP-Bang[8–10] (www.stopbang.ca).
   i) The ASA guidelines for OSA include physical characteristics that suggest OSA, and the second category is a history of apparent airway obstruction during sleep.
      (1) If a patient meets the criteria by physical characteristics and has a history of obstruction, then there is a significant probability that the patient has OSA.
      (2) A grading scale is done with points awarded for the severity of OSA, the invasiveness of surgery and anesthesia, and the likely postoperative opioid requirement.[10]
      (3) It is controversial whether OSA patients can be safely treated in an ambulatory surgery setting.
         (a) Considerations include the severity of the OSA, the type of procedure, the likeliness of opioid requirement postoperatively, and the ability to transfer the patient to the inpatient setting as needed.[10]
         (b) If OSA patients are candidates for ambulatory surgery, they ideally should be scheduled as the first case of the day.
   ii) The Berlin questionnaire asks patients to quantify their snoring history including the loudness, quantity, if other people complain about their snoring, and presence of apnea.
      (1) There are questions about tiredness and fatigue after sleep, falling asleep while driving, and the presence of high blood pressure.[9]
   iii) The STOP-Bang screening tool (**Table 54-2**) stands for S, snoring; T, tiredness/fatigue during the day; O, observed apnea; P, high blood pressure; B, BMI > 35 kg/m^2; A, age >50; N, neck circumference >40 cm, and G, male gender.
      (1) Patients are considered at high risk for OSA if they answer yes to three or more of the above.[8]
c) For patients with diagnosed OSA, weight loss and avoidance of alcohol and sedatives can improve symptoms.
d) Patients on continuous positive airway pressure (CPAP) should bring their devices with them to the hospital.
   i) Since patients with OSA are prone to comorbidities, a thorough cardiopulmonary assessment should be done.

TABLE 54-2 STOP-Bang Screening Tool

S	Snoring	*B*	BMI > 35 kg/m^2
T	Tiredness/fatigue during the day	*A*	Age >50
O	Observed apnea	*N*	Neck circumference >40 cm
P	High blood pressure	*G*	male gender

Reprinted with permission from Chung F, Yesneswaran B, Liao P, et al. STOP questionnaire: a tool to screen patients for OSA. *Anesthesiology*. 2008;108:812-821.

e) OSA patients who undergo corrective airway surgery (eg, uvulopalatopharyngoplasty) are considered to still be at risk unless they have a normal sleep study and resolution of symptoms.[10]

5) **Intraoperative management**
   a) The ASA guidelines recommend:
      i) Local anesthetics and/or peripheral nerve blocks are preferred over general anesthesia for peripheral surgery.
      ii) A spinal or epidural technique is preferred over general anesthesia for peripheral surgery.
      iii) For intra-abdominal surgery, the recommendations are equivocal over whether a general or regional technique is superior.[10]
   b) The Society of Anesthesia and Sleep Medicine guidelines also recommend regional anesthesia over general anesthesia for OSA patients when feasible.[11]
   c) A semiupright or lateral position decreases airway obstruction compared to the supine position.
   d) For patients receiving general anesthesia, they should be extubated when fully awake and reversed if neuromuscular blockade has been given.
   e) For patients receiving monitored anesthetic care, $EtCO_2$ should be monitored to help assess adequate ventilation.
   f) Consider using CPAP/biPAP/oral appliances intraoperatively.
   g) Medications that do not cause respiratory depression are preferable including ketamine, dexmedetomidine, NSAIDs, and other adjuvants.[12]
   h) Minimize/avoid opioids if possible.



OSA patients can be exquisitely sensitive to opioids.

6) **Postoperative management**
   a) There is a risk of rebound rapid eye movement sleep postoperatively, so OSA patients may be at increased risk of apnea for 1 week postoperatively.[13]
      i) Epidural opioids are linked to respiratory depression in this patient population.[14]
   b) CPAP/BIPAP should be instituted in the recovery room for patients using these devices preoperatively.
      i) Patients with undiagnosed OSA who meet criteria and exhibit signs of OSA in the operating room should be considered for a respiratory consult postoperatively for use of CPAP.
   c) Avoid recovery in the supine position when possible.
   d) Supplemental O_2 should be given until the patient can maintain baseline SpO_2 on room air.[10]
   e) For patients with moderate to severe OSA, a monitored setting with continuous pulse oximetry and possibly capnography are recommended with possible ICU admission.[15]



OSA patients may be at increased risk of apnea for 1 week postoperatively.

READ MORE
Outpatient anesthesia, Chapter 115



OSA patients are more likely to obstruct in supine positions versus semiupright or lateral positions.

7) **Discharge criteria:** A panel of expert consultants convened by the ASA taskforce on perioperative management of patients with OSA developed monitoring indications. The following are based on the ASA guidelines:
   a) Patients should meet standard outpatient discharge criteria.
   b) Be able to maintain their baseline SpO_2 on room air.
   c) No hypoxemia or airway obstruction when left alone in an unstimulated environment.
   d) It is controversial if OSA patients with upper airway surgery should be admitted or discharged. The consultants disagree that these procedures can be done safely on an outpatient basis for patients at increased risk of OSA.

e) Additional factors to consider when assessing discharge readiness include severity of OSA, type of surgery, need for postoperative opioids, use of CPAP, and recovery room status (stable vs episodes of desaturation).[10]

CHAPTER SUMMARY FOR OSA

General Considerations	OSA is a common and often undiagnosed disease
Preoperative Considerations	Several screening tools are available to assess patients for undiagnosed OSA
Intraoperative Considerations	Increased risk of difficult ventilation and intubation When appropriate, use regional/local anesthesia, NSAIDs, and other adjuvants that do not cause respiratory depression If general anesthesia is given, patients should be fully awake and reversed prior to extubation
Postoperative Considerations	Increased risk of postoperative hypoxemia and cardiopulmonary arrest Avoid the supine position if possible. A lateral or head-up position is preferable

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55 Part D: Diseases of the Nervous System

Anesthesia and Diseases of the Nervous System

Divya Chander

Assessment of underlying neurological disease in a patient requiring anesthesia is critical and often overlooked. The nervous system controls all physiological systems. Most neuromuscular diseases predispose patients to respiratory failure and postoperative weakness. The autonomic branch controls compensatory mechanisms to anesthetic perturbations and all three branches contribute to maintenance of intact reflex systems, most notably, airway reflexes. Certain motor system diseases share susceptibility to malignant hyperthermia triggers. Anatomically, the nervous system is vulnerable to injury with delivery of anesthetics, especially with regional or neuraxial techniques. The nervous system itself is the target of anesthetics; cerebral vasoregulation, cerebral excitatory and inhibitory balance, traffic along peripheral nerves, and proper function of the neuromuscular junction are altered. Anesthesia also modulates the global arousal system and may have long-lasting cognitive effects in select, vulnerable patients.

1) **Motor system diseases**
   a) Motor system dominated diseases include myopathies and muscular dystrophies, motor end-plate denervation, motor neuron degeneration, spinal motor tract disorders, and disuse atrophy.
   b) Motor system diseases involving other branches of the nervous system include peripheral nerve disease, polyneuropathies, and spinal cord injury.
   c) **Amyotrophic lateral sclerosis (ALS), spinal muscle atrophy (SMA)**
      i) ALS is a progressive loss of both upper and lower motor neurons leading to fasciculations, flaccid paralysis, hyperreflexia, and spasticity.
      ii) SMA encompasses a number of genetic disorders, with four phenotypes based on onset and severity; it is also characterized by progressive muscular atrophy secondary to degeneration of cortical, brainstem, and spinal motor neurons. Similar anesthetic considerations hold.[1]
      iii) Some patients have bulbar symptoms (dysphagia, dysarthria, difficulty clearing secretions) or may have diaphragmatic or intercostal respiratory compromise.
      iv) Treatment
         (1) Riluzole (neuroprotective agent) and edaravone (antioxidant) are approved drugs to delay the course of ALS.
         (2) Riluzole results in a use-dependent decrease in presynaptic release of glutamate.[2] In doses higher than those used for neuroprotection, it can act as a general anesthetic (animal models). Therefore, it could theoretically potentiate the hypnotic effect of other anesthetic agents.

READ MORE
Muscular dystrophy, Chapter 87, Malignant hyperthermia, Chapter 150, Left ventricular assist device, Chapter 105.

(3) For amelioration of common ALS symptoms such as spasticity and cramping, patients may be on a number of GABAergic agents such as benzodiazepines and baclofen, which can interact with anesthetic agents.
(4) ALS patients may be on a number of other experimental treatments, many in active clinical trials.
(5) In 2011, the FDA approved diaphragmatic pacing for ALS under a humanitarian device exemption, but its efficacy has mixed reviews, showing both survival benefit and possible harm.[3] Respiratory sufficiency needs to be evaluated on an individual basis in these patients.
(6) Pulmonary function tests to assess functional vital capacity are valuable in this population. American Academy of Neurology practice guidelines suggest ALS patients in a nonsurgical setting with an FVC < 50% should be offered noninvasive positive pressure ventilation (NIPPV) outside the surgical setting.[4] This recommendation could be carried into the perioperative period.
(7) These patients are not at increased risk for malignant hyperthermia (MH) or rhabdomyolysis.
(8) Succinylcholine is strictly contraindicated due to an up-regulation of immature acetylcholine (ACh) receptors beyond the neuromuscular junction.
(9) Prolonged responses (peak and temporal response) to nondepolarizing neuromuscular blockers has been described.



When performing a history and physical in any patient with neurologic disease, rigorous documentation of preexisting deficits and a full risk-benefit discussion of anesthetic technique with patients is mandatory. The most important components are preexisting respiratory or bulbar involvement, cardiomyopathy or heart failure, autonomic dysfunction, and assessment of postoperative risk of weakness requiring ventilatory support.

d) **Duchenne muscular dystrophy (DMD), Becker muscular dystrophy (BMD)**
i) Both are X-linked disorders of the dystrophin gene leading to muscle wasting and weakness, dilated cardiomyopathy (in 50% of patients by age 15), ultimately resulting in early death.[10]
ii) Typically, the onset of BMD is later and is less severe than DMD.
iii) Scoliosis is common; this often leads to restrictive lung disease.
iv) Cardiomyopathy leading to left-sided heart failure is often treated with left ventricular assist device.
v) Administration of MH triggers can lead to perioperative, hypermetabolic complications reminiscent of MH.[5]

READ MORE
Ventricular assist devices, Chapter 105

e) **Central core disease (muscle channelopathy)/King-Denborough syndrome (myopathy)**
i) A group of rare muscle disorders directly linked to MH[5]
f) **Anesthetic management**
i) Preoperative considerations
(1) Risk of aspiration (bulbar symptoms) and requirement for postoperative ventilatory support should be rigorously assessed.
(a) Pulmonary function tests should be done in all patient populations, with special emphasis on forced vital capacity and FEV_1.
(b) In patients with muscular dystrophies, a cardiac workup including echocardiogram and/or cardiac MRI is also required.[6]
(2) Regional anesthesia is not contraindicated, though nervous system deficits should be meticulously documented and risks explained thoroughly so preexisting deficits are not confused with nerve injury.
(3) Neuraxial anesthesia can be used; exercise caution to ensure that block level has minimal impact on respiration.
(4) Prepare an anesthesia machine free of MH-triggering agents for those disorders that are MH-susceptible.[5]

ii) Induction
   (1) Succinylcholine should be avoided secondary to muscle denervation and up-regulation of extrajunctional receptors in all motor neuron diseases. Severe hyperkalemia may lead to intractable bradycardia and/or cardiac arrest.
   (2) Patients may be more sensitive to nondepolarizing muscle relaxants (peak response and duration). The lowest dose required for optimal intubation (eg, 0.3-0.5 mg/kg rocuronium), administered with active TOF monitoring, or use of alternate adjuncts for muscle relaxation such as remifentanil, is recommended. See also "Maintenance and emergence" below.
   (3) In the dystrophies and myopathies such as central core disease and King-Denborough syndrome, both succinylcholine and potent volatile anesthetics should be avoided because they may lead to rhabdomyolysis or hypermetabolic states reminiscent of MH.[5]



Succinylcholine should be avoided secondary to muscle denervation and up-regulation of extrajunctional receptors in all motor neuron diseases.



Neurologic diseases resulting in muscle denervation secondary to disuse atrophy or lower motor neuron denervation (eg, ALS, post-polio syndrome, tetanus, MS, or GBS with motor involvement) can cause a proliferation of extrajunctional receptors and an exaggerated release of extracellular potassium in response to subparalytic doses of succinylcholine.

iii) Maintenance and emergence
   (1) A total intravenous anesthetic (TIVA) technique and a "clean" machine are recommended for the dystrophies and myopathies.[5,6]
   (2) In cases where a nondepolarizing muscle relaxant has been given, monitoring of muscle twitch on an unaffected limb is imperative. Quantitative, rather than qualitative, monitoring is recommended, as recovery of a TOF ratio of 0.9 in these patients has been linked to inadequate recovery of clinical strength for extubation.[7–9]
   (3) The safety and efficacy of sugammadex has been reported, and it should be considered in all patients with motor neuron disease who receive muscle blockade at doses of 2-5 mg/kg.[9–11]
   (4) The autonomic nervous system (ANS) is typically not impaired in pure motor diseases, and hemodynamic responses are typically stable.
   (5) In peripheral nerve disease, polyneuropathies (see below), and spinal cord injury, the ANS may show variable impairment and could alter hemodynamics significantly.

iv) Postoperative considerations
   (1) Preparation for postoperative ventilatory support should be undertaken in select populations. Even in patients not requiring ventilation, consider postoperative admission to the ICU or step-down unit for increased monitoring.
   (2) Suspicion of hypermetabolic syndromes or rhabdomyolysis should be monitored appropriately in the PACU (eg, serial serum potassium and creatine kinase levels, urine myoglobin).



Demyelinating diseases can affect both peripheral and central nervous system and involve all three branches—motor, sensory, and autonomic. When multiple peripheral nerves are involved, the disease is considered a polyneuropathy.

2) **Peripheral demyelinating diseases: Guillain-Barré syndrome (GBS)**
   a) Demyelinating diseases typically have motor and sensory nervous system involvement; sometimes ANS involvement is also present.
   b) **GBS: Acute inflammatory demyelinating polyneuropathy (AIDP)**
      i) GBS is an infrequent, autoimmune response against peripheral nerves, triggered 60%-70% of the time by an acute viral infection.
      ii) Sequelae occur days to weeks after exposure to infection.

iii) Recently, with the use of immune checkpoint therapy to activate the immune system against certain neoplasms, a number of autoimmune neuromuscular disorders, including GBS, have been triggered.[12]
iv) GBS is characterized by an ascending paralysis, affecting limbs before trunk, and loss of deep tendon reflexes.
(1) Cranial nerves are involved 50% of the time.
v) Treatment for acute episodes is via IV-IG or plasmapheresis exchange. Steroids are not useful.

c) **Anesthetic management**
i) Preoperative considerations
(1) A focused understanding of involved systems is imperative to managing the patient safely—a thorough chart review and patient history must be performed. Clinical features with important anesthetic implications include the following:
(a) Autonomic/cardiac system
(i) BP lability in the absence of compensatory cardiovascular reflexes, requiring vasoactive treatment at various intraoperative time points
(ii) Dysrhythmias and possible cardiac arrest
(b) Brainstem
(i) Compromised pharyngeal reflexes, which may predispose to reflux or inability to protect the airway, necessitating unplanned intubation or tracheostomy
(ii) Disordered swallowing
(iii) Vocal cord paralysis
(c) Respiratory system
(i) Increased risk of pulmonary embolus
(ii) Diaphragmatic paralysis necessitating intubation or tracheostomy
(d) Secondary skeletal muscle denervation or atrophy
(2) Regional and neuraxial anesthesia are not contraindicated. This is especially important to consider in light of the overlapping demographics of patients susceptible to GBS and who can become pregnant. There are several case reports of safe neuraxial anesthesia undertaken in GBS patients, though one case report exists of worsening neurological symptoms in a parturient receiving an epidural.[13–15]
(a) There is an increased risk of complications due to exaggerated hemodynamic responses in patients with autonomic involvement.[16]
(b) Exaggerated receptor responses to local anesthetics may occur.
(c) Use of regional techniques should be balanced against the other risks and benefits.
(d) For example, regional techniques may have increased benefit in the GBS parturient, but increased risk in the patient with lung disease.
(e) A detailed discussion of risks should be undertaken with the patient and all preexisting neurological deficits meticulously documented so preexisting deficits are not confused for postanesthesia nerve injury.
(f) The choice to use regional must be made on a case-by-case basis.
(g) Ultrasound-guided blocks may improve the safety profile and reduce the risk of nerve damage.[17]
ii) Induction
(1) Skeletal muscle denervation, even subclinical, can lead to an up-regulation of extrajunctional receptors. Avoid succinylcholine because of the risk for hyperkalemic bradycardia or cardiac arrest.

(2) There are no known contraindications to use of nondepolarizing muscle relaxants, though there is the potential for increased sensitivity and delayed recovery. Sugammadex should strongly be considered in this population. Stress-dose steroids are only required in patients on long-term steroids.
(3) Autonomic involvement will lead to exaggerated responses to:
  (a) Induction agents, leading to hypotension, brady- or tachycardia. Anticipate and pretreat with vasoactive agents as necessary.
  (b) Direct laryngoscopy, leading to hypertension or tachycardia. Consider premedication (eg, 3-5 µg/kg fentanyl or 1.5 mg/kg lidocaine) prior to laryngoscopy to blunt autonomic responses.
(4) Bulbar involvement may increase risk of aspiration. Anticipate and use a modified rapid sequence technique with cricoid pressure if deemed necessary.
(5) Skeletal muscle denervation, even subclinical, can lead to an up-regulation of extrajunctional receptors. Avoid succinylcholine because of the risk for hyperkalemic bradycardia or cardiac arrest.
(6) There are no known contraindications to the use of nondepolarizing muscle relaxants, though there is the potential for increased sensitivity and delayed recovery. Have sugammadex available.



Patients with GBS are at high risk for autonomic instability, aspiration, and respiratory failure.

iii) Maintenance/emergence
(1) Autonomic instability will lead to exaggerated responses to:
  (a) Addition of PEEP (hypotension)
  (b) Surgical stimulation (hypertension)
  (c) Blood loss (hypotension)
  (d) Changes in posture (hypo- and hypertension)
(2) Invasive arterial monitoring can assist management in patients with autonomic instability.
(3) Indirect-acting vasopressors may elicit exaggerated responses secondary to up-regulation of postsynaptic receptors.
(4) Myocardial irritants or conditions that sensitize the myocardium should be avoided (eg, electrolyte imbalances; halothane).
(5) Wildly fluctuating BP responses during emergence should be anticipated and pretreated if possible (eg, β-adrenergic antagonists).
(6) If there is motor involvement, monitoring of muscle twitch with a nerve stimulator should preferably be on an unaffected limb.
(7) Careful attention to extubation criteria must be followed.
  (a) Appropriate respiratory mechanics[18]
  (b) Return of airway reflexes
  (c) Full recovery of neuromuscular tone
  (d) A warmed patient
  (e) Recovery of mental status
  (f) Normalized blood gases
  (g) Minimal intraoperative hemodynamic shifts
  (h) Normal electrolytes

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Post-op ventilation of the surgical patient, Chapter 131

iv) Postoperative
(1) Depending on severity of disease and involvement of the respiratory system, the GBS patient may require postsurgical

ventilatory support. Even in patients not requiring ventilation, consider postoperative ICU or step-down unit admission for increased monitoring.

(2) Patients with some baseline respiratory compromise, or who are already on preoperative NIPPV, should be extubated to NIPPV.

3) **Central demyelinating diseases: multiple sclerosis**

a) **Multiple sclerosis (MS)**

i) Autoimmune disorder against CNS myelin

ii) Has a relapsing and remitting course, with variable periods of latency in-between

iii) Disease severity and expression is based on the burden and distribution of CNS lesions.

iv) Sensory, motor, and ANS involvement is possible.

b) **Treatment includes the following:**

i) Chronic immunosuppressive therapies (eg, cyclophosphamide, azathioprine). Interferon-*B* can lead to abnormal blood counts (including thrombocytopenia, an important consideration for neuraxial techniques) or elevated LFTs.[19]

ii) Steroid treatment during acute flares

iii) Intravenous ACTH if afflicted with optic neuritis

iv) Antispasmodics if suffering severe spasticity or bladder involvement, which include GABAergic drugs or dantrolene

v) Neuropathic pain treatment, including antiepileptics, gabapentin, and THC/CBD.[19]

vi) Newer disease-modifying therapies, many of them based on immunomodulation, are being used.[20] A total of five new agents and one new dosage formulation were approved by the FDA from 2010 to 2015 alone.[21] While these agents do not seem to have direct interactions with anesthetic drugs, some have significant side effects, which must be considered in the preoperative anesthesia workup.



Patients with multiple sclerosis may be taking multiple immune-modulating medications or participating in clinical trials for new therapies.

c) **Anesthetic management**

i) Preoperative considerations

(1) Perform a focused but thorough chart review and patient history. Central white matter loss may affect targets with anesthetic implications.

(a) Autonomic/cardiac system resulting in BP lability with poor compensatory reflexes

(b) Brainstem nuclei affecting respiration, pharyngeal reflexes, swallowing, or vocal cord function

(c) Secondary skeletal muscle denervation or atrophy

(2) MS patients may have clinical or subclinical acute or chronic respiratory failure due to lesions in breathing centers or to nerves controlling respiratory muscles leading to ineffective cough, secretions, or deep breathing; even neurogenic pulmonary edema is a possibility.[22]

(3) PFTs should be considered, especially if wheelchair bound or bedridden.

(a) Lung volumes may be reduced.

(b) Direct assessment of upright and supine vital capacity (VC) as well as maximal inspiratory (MIEP) and expiratory pressure (MEIP) might be of great functional value.

(c) Respiratory compromise should lead to postextubation/postoperative planning, as well as a consideration of postoperative step-down or ICU care.

(4) Regional and neuraxial anesthesia are not contraindicated, though in some cases controversial. Retrospective studies have not found an increase in MS exacerbations or documented neurotoxicity with neuraxial anesthesia or nerve blocks.[23]

(a) There is a theoretical risk of increased sensitivity of nerve conduction to either single or repeated doses of local anesthetics, particularly with intrathecal administration, or concern for secondary crush injury to already compromised nerves.[24,25]
(b) There is the potential for exaggerated receptor responses to local anesthetics. Some studies suggest that local anesthetics could unmask subclinical demyelination or worsen negative symptoms (eg, paresis, hypoesthesia) in some MS patients.[26] Paradoxically, lidocaine can also treat positive symptoms (eg, spasticity) in this same patient population.[24]
(c) Use of regional techniques should be balanced against the other risks and benefits of such approaches in these patients. The benefits may be more strongly seen in the MS parturient when weighed against the risk of general anesthesia. Increased caution should be exercised in the patient with documented spinal cord (neuraxial) or peripheral nerve (regional) lesions. The decision to use regional techniques should be made on a case-by-case basis.
(d) A detailed discussion of risks should be undertaken, and all preexisting neurological deficits meticulously documented so preexisting deficits are not confused with nerve injury.
(e) If utilized, consider use of lower local anesthetic doses, supplemented with opioids.[26]

ii) Induction
(1) For patients treated with long-term steroids, the risk-benefit equation for stress-dose steroids has been questioned. A reasonable recommendation, in the absence of HPA testing, is to continue maintenance dosing during the perioperative period, and add rescue steroids to treat otherwise unresponsive or unexplained hypotension.[27]
(2) Autonomic involvement will lead to exaggerated responses to:
(a) Induction agents, leading to hypotension, brady- or tachycardia. Anticipate and pretreat with vasoactive agents as necessary.
(b) Direct laryngoscopy, leading to hypertension or tachycardia. Consider premedication (eg, 3-5 µg/kg fentanyl or 1.5 mg/kg lidocaine) prior to laryngoscopy to blunt autonomic responses.
(3) Stress can trigger MS exacerbations; treating pain adequately and blunting autonomic responses may help.
(4) Bulbar involvement may increase risk of aspiration. Anticipate and use a modified rapid sequence technique with cricoid pressure if deemed necessary.
(5) Skeletal muscle denervation, even subclinical, can lead to an up-regulation of extrajunctional receptors. Avoid succinylcholine because of the risk for hyperkalemic bradycardia or cardiac arrest.[28]
(6) There are no known contraindications to nondepolarizing muscle relaxants, though there is the potential for increased sensitivity and delayed recovery. Sugammadex has been used successfully for reversal in patients with MS.[29]

iii) Maintenance/emergence
(1) Autonomic instability can lead to exaggerated responses to:
(a) Addition of PEEP (hypotension)
(b) Surgical stimulation (hypertension)
(c) Blood loss (hypotension)
(d) Changes in posture (hypo- and hypertension)



Hyperpyrexia may be associated with MS exacerbations and should be avoided.

(2) Invasive arterial monitoring can assist management in patients with autonomic instability.
(3) Indirect-acting vasopressors may elicit exaggerated responses secondary to up-regulation of postsynaptic receptors.
(4) Labile BP responses during emergence should be anticipated and pretreated if possible (eg, β-adrenergic antagonists).
(5) If there is motor involvement, monitoring of muscle twitch with a nerve stimulator should preferably be on an unaffected limb.
(6) Careful attention to extubation criteria must be followed.
(a) Appropriate respiratory mechanics
(b) Return of airway reflexes
(c) Full recovery of neuromuscular tone
(d) A warmed patient
(e) Recovery of mental status
(f) Normalized blood gases
(g) Minimal intraoperative hemodynamic shifts
(h) Normal electrolytes
(7) Consider sugammadex for reversal of neuromuscular blockade.



Neuraxial and other regional techniques are generally safe in patients with peripheral nerve or neuromuscular involvement. The decision to perform these techniques should be made on a case-by-case basis with thorough discussion of risks and benefits.

iv) Postoperative
(1) Depending on severity of disease and involvement of the respiratory system, the MS patient may require postsurgical ventilatory support.
(2) Consider step-down care or ICU monitoring if preoperative respiratory involvement is present.
(3) Certain patients may be candidates for extubation to NIPPV.



Depending on severity of disease and involvement of the respiratory system, the MS patient may require postsurgical ventilatory support.

4) **Inflammatory neurologic disease**
a) **Lyme disease**
i) Polyinflammation of multiple organ systems (joints, heart, muscle, central and peripheral nervous system) is the hallmark of the disease.
ii) Early manifestations include erythema migrans and influenza-like symptoms.[30]
iii) Later systemic manifestations can masquerade as many other diseases.
iv) As many as 10% of cases can have significant cardiac involvement, complicating anesthetic management.[30]
(1) Most common problem is atrioventricular block; other dysrhythmias/conduction abnormalities are possible.
(2) Rarer cardiac manifestations include pericarditis, myocarditis, cardiomyopathy, and degenerative valvular disease.
(3) Unstable patients may require temporary pacing.[31]



Up to 10% of cases of Lyme disease have significant cardiac involvement.

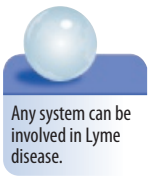
b) **Neurologic Lyme disease (*neuroborreliosis*)**
i) Occurs 1-4 weeks after initial infection in 10%-15% of cases
ii) Causes inflammation of the peripheral nerves, meningeal lining, or brain parenchyma
iii) Manifestations include meningitis, meningoradiculitis, cranial neuritis, encephalopathy, peripheral neuropathy, encephalitis, and encephalomyelitis.
c) **Treatment**
i) Nervous system infection responds well to parenteral high-dose penicillin, ceftriaxone, and cefotaxime in both adults and children.

ii) Oral doxycycline is often used in adults with meningitis, cranial neuritis, and radiculitis.

d) **Anesthetic management**

i) Preoperative considerations

(1) Anesthetic risk depends on the portion of the nervous system involved. An extremely thorough preoperative evaluation is needed to identify these affected systems to develop an anesthetic plan. Any system can be involved in Lyme disease.



(a) Cardiac involvement most prominently leads to dysrhythmias and can result in arrest.

(b) Myelitis can result in weakness, sensory loss, and dysautonomia, leading to autonomic lability.

(c) Denervation can affect the neuromuscular junction and respiratory muscles.

(d) Cranial neuritis may affect airway reflexes, cause vocal cord paralysis, and impact the ability to protect the airway.

(e) Management of peripheral neuritis depends on severity and distribution of lesions. It has maximal impact if autonomic fibers or muscles of respiration are affected.

(f) In rare cases, vasculitic involvement can lead to stroke (see Ischemic Vascular Disease/CVA).

(g) Parenchymal involvement can infrequently lead to seizures (see Epilepsy/Seizure Disorder).

(2) Consideration of use of neuraxial anesthesia in patients with *neuroborreliosis* with peripheral neuritis is similar to patients with ALS, GBS, and MS. A strict risk-benefit analysis and meticulous documentation of preexisting deficits should be performed.

ii) Induction

(1) Cardiac and autonomic involvement may lead to dysrhythmias or exaggerated responses to:

(a) Induction agents (hypotension, brady- or tachycardia)

(i) Anticipate and consider pretreatment with vasoactive agents as necessary.

(b) Direct laryngoscopy (hypertension, tachycardia)

(i) Consider premedication (eg, 3-5 µg/kg fentanyl, 1.5 mg/kg lidocaine) prior to laryngoscopy to blunt autonomic responses.

(2) Cranial neuritis and/or bulbar involvement may increase risk of aspiration—anticipate and consider using a modified rapid sequence technique with cricoid pressure.

(3) Myelitis leading to muscle denervation carries high risk of hyperkalemia-induced bradycardia or arrest. Motor compromise may be impossible to document, so it is safer to avoid succinylcholine.

(4) There are no contraindications to nondepolarizing muscle relaxants, though sensitivity to them may be slightly increased. Sugammadex availability increases safety.

iii) Maintenance/emergence

(1) Autonomic instability will lead to exaggerated responses to:

(a) Addition of PEEP (hypotension)

(b) Surgical stimulation (hypertension)

(c) Blood loss (hypotension)

(d) Changes in posture (hypo- and hypertension)

(2) Invasive arterial monitoring can assist management in patients with autonomic instability.
(3) Indirect-acting vasopressors may elicit exaggerated responses secondary to up-regulation of postsynaptic receptors.
(4) Myocardial irritants or sensitizers should be avoided (eg, halothane).
(5) Labile BP responses during emergence should be anticipated and pretreated if possible (eg, β-adrenergic antagonists).
(6) If there is motor involvement, monitoring of muscle twitch with a nerve stimulator should preferably be on an unaffected limb.
(7) Careful attention to extubation criteria must be followed.

iv) Postoperative considerations
(1) If myelitis or cranial neuritis affects the respiratory system or airway protection, the patient with Lyme disease may require postsurgical ventilatory support, postoperative step-down, or ICU monitoring.

5) **Cerebral disease: Seizure Disorder**

a) **Epilepsy/seizure disorder**
i) Seizures are paroxysmal, transient disturbances of brain function characterized by excessive or hypersynchronous discharge of neurons.
ii) The electrical signature of seizures can be measured by surface scalp electrodes (EEG).
iii) The clinical manifestations can be sensory, motor, or psychic and be accompanied by loss of consciousness.
iv) The hypermetabolic activity of brains undergoing seizure discharge necessitates supportive function, especially if seizure activity is not self-limited (*status epilepticus*).
v) Epilepsy is a chronic disorder characterized by recurrent seizures.

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b) **Pro- and anticonvulsant effects of anesthetic drugs**
i) Many anesthetic agents have dose-dependent paradoxical pro- and anticonvulsant effects.[32]
(1) Lower doses of IV anesthetics are more likely to be proconvulsant.
(2) Higher doses of infusions (eg, propofol, ketamine, thiopental, methohexital, etomidate) are more likely to be anticonvulsant and have been used to treat status epilepticus, while lower doses of volatiles (eg, sevoflurane) are more likely to be anticonvulsant.
ii) The opposing effects may be related to dose-dependent effects on differential recruitment of GABAergic circuits, effects on potassium-based hyperpolarization, and blockade of sodium and calcium currents.[33]
iii) Narcotics may show the reverse effect.[34]



Patients with seizure foci in a speech or other eloquent sensory center may require awake craniotomy for resection of seizure focus.

c) **Anesthetic management of the patient for intracranial resection of seizure focus**
i) Preoperative considerations
(1) May require an awake craniotomy if the seizure focus overlaps with speech (motor eloquent or receptive), sensory, or motor centers.
(a) Only motor centers can be tested with the patient asleep.
(2) Benzodiazepine premedication is usually avoided because they raise the seizure threshold.
(a) This may interfere with intraoperative assessment during awake craniotomies.

(3) IV access is obtained on the side of the patient not involved with (motor) seizures (ie, on the contralateral side).

ii) Induction

(1) Choose a method of sedation that:

(a) Provides patient comfort

(b) Is rapidly titratable or reversible

(c) Does not interfere with the seizure threshold

(d) Does not induce profound apnea

(i) Adjuncts often include propofol, remifentanil, and dexmedetomidine.

(2) Have a carefully thought out back-up plan in case of loss of airway, especially since the patient's head may be far from the anesthesiologist, fixed (ie, in pins), and difficult to access.

(a) Discuss this airway plan with the surgeon if necessary, especially in high-risk patients (eg, craniofacial compromise, obesity, obstructive sleep apnea).



For awake craniotomies requiring adjunct sedation, have a carefully thought out back-up plan in case of loss of airway, especially since the patient's head may be far from the anesthesiologist, fixed (ie, pins), and difficult to access. Discuss plan with the surgeon if necessary, especially if the patient has predictable airway difficulties (eg, craniofacial compromise, obesity).

iii) Maintenance

(1) Intraoperative brain mapping is common.

(a) May be done with electrocorticography (ECoG), electrodes placed directly on the cortical surface overlying the focus, or with intracranial depth electrodes in the region of interest.

(b) Less commonly, the neurosurgeon may ask for pharmacological adjuncts for stimulation of the epileptogenic focus.

(i) Methohexital: 10-50 mg IV

(ii) Propofol: 10-20 mg IV

(iii) Thiopental 25-50 mg IV

(iv) Etomidate 2-8 mg IV

d) **Anesthetic management of the epileptic patient undergoing nonepilepsy surgery**

i) Preoperative evaluation

(1) Thoroughly assess comorbidities, including psychiatric illnesses, head trauma, or associated syndromes, some more common in childhood.

(a) West syndrome, Lennox-Gastaut syndrome, mitochondrial syndromes such as MERRF and MELAS, neurofibromatosis, tuberous sclerosis, and multiple endocrine adenomatosis are childhood seizure-associated syndromes.

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(2) Knowledge of the patient's anticonvulsant therapy and associated complications is imperative.

(a) Phenytoin: gum disease/poor dentition, airway management issues

(b) Valproate: altered platelet function, thrombocytopenia

(c) Carbamazepine: bone marrow suppression, cardiac toxicity



Patients on antiseizure medication may require increased dose and frequency of medications.

ii) Maintenance

(1) Avoid anesthetic agents that potentiate the seizure threshold such as etomidate and methohexital.

(2) Note that most anesthetics have pro- and anticonvulsant properties and patients with baseline epilepsy disorder are more likely to respond to anesthetic agents by seizing.[33] Some of the cortical proconvulsant activity is subclinical.

(3) It is valuable to monitor these patients with ECoG or EEG to detect seizure activity, so it can be immediately aborted. At a minimum, patients with documented epilepsy who are having nonepilepsy surgery should be preloaded with their regular antiseizure medication. Benzodiazepines and high-dose propofol demonstrate anticonvulsant properties, should an undesired seizure occur.



In any setting where a seizure takes place, the first goal is to ensure a patent airway, administer supplemental O_2, and maintain adequate perfusion (ABCs).

(4) Avoid hypocapnia (through hyperventilation, by controlling minute ventilation).
   (a) Hypocapnia potentiates the seizure threshold and decreases cerebral blood flow through vasoconstriction.

(5) For long intraoperative procedures, ongoing anticonvulsant therapy may need to be administered. At the very least, baseline dosing should be continued throughout the perioperative period. Consult with the surgical team to determine the patient's dosing schedule.

(6) Most anticonvulsants (especially phenytoin and phenobarbital) undergo hepatic transformation and up-regulate the P450 enzyme system.
   (a) This usually necessitates an increased amount or frequency of dosing of many anesthetic drugs, especially muscle relaxants.



Hypoglycemia should always be considered and treated in a patient who is seizing.

e) **Anesthetic management of the epileptic patient in status epilepticus**

i) Status epilepticus is ongoing, intractable seizure activity, either convulsive or nonconvulsive.
   (1) Status epilepticus is a neurological emergency

ii) Treatment
   (1) Primary goal is prevention of brain damage.
   (2) Secondary goal is prevention of injury due to involuntary convulsions.
   (3) Brain damage prevention includes the following:
      (a) ABC: Secure the airway, provide supplemental O_2, and maintain adequate perfusion/circulation.
      (b) Decrease or silence neuronal activity, which decreases neuronal metabolism and secondary glutamate excitotoxicity.
      (c) If hypoglycemia has not been ruled out and cannot be ruled out quickly, supplement with D50 as a slow IV push.
         (i) Dextrose 50% = 25 g D-glucose in 50 mL water.
      (d) Commonly used medications to decrease convulsive activity include the following:
         (i) Benzodiazepines
         (ii) Barbiturates
         (iii) Propofol (at higher doses)
         (iv) Phenytoin
      (e) Most intravenous anesthetics have anticonvulsant properties when used at adequate dosages as infusions.
         (i) Anticonvulsant therapy can be titrated to a simultaneously monitored EEG.

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6) **Cerebral disease: ischemia**

a) **Ischemic vascular disease/cerebrovascular accident (CVA)**

i) Rupture or obstruction of a blood vessel feeding the brain parenchyma that results in sudden impairment of consciousness, sensory, or motor function.

b) **Treatment**
   i) Medical
      (1) Multifactorial treatment
         (a) Anticoagulation (if ischemic disease)
         (b) Decrease the risk of the associated comorbidities
            (i) Hypertension, dyslipidemia, atrial fibrillation, tobacco use, diabetes
   ii) Surgical
      (1) Open or endovascular carotid endarterectomy (CEA)
         (a) Performed to correct stenosis of the carotid artery in ischemic disease
      (2) Open clipping or endovascular coiling treatment of aneurysm or AVMs is performed for hemorrhagic strokes.

c) **Anesthetic implications**
   i) Anesthetic management of the patient with ischemic vascular disease undergoing non-CEA surgery.
      (1) Preoperative considerations
         (a) Meticulous attention should be paid to multiple comorbidities. These patients are generally have small and large vessel atherosclerosis. They present with associated proinflammatory conditions including:
            (i) Hypertension
            (ii) Diabetes
            (iii) Coronary artery disease
            (iv) Obesity
            (v) Obstructive-restrictive lung disease
         (b) History should emphasize exercise tolerance, use of home O_2, symptoms of chest pain, orthopnea, dyspnea on exertion, and those consistent with obstructive sleep apnea.
            (i) Preexisting neurological deficits should be documented.
         (c) Physical examination should focus on thorough airway and cardiopulmonary evaluation, including carotid auscultation.
         (d) Medications, electrolyte abnormalities (especially glucose, potassium, bicarbonate), coagulopathies, and anemia should be noted.
         (e) The ECG should be examined and compared to prior ECGs if available. Echocardiogram or stress testing should be performed in select patients. The cardiac evaluation should occur according to the most recent guidelines for the management of such patients undergoing noncardiac surgery.

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      (2) Intraoperative management
         (a) Induction
            (i) Minimize significant hemodynamic swings during induction and direct laryngoscopy.
               1. β-Adrenergic antagonists, opioids (eg, fentanyl 2-3 μg/kg), or lidocaine (1.5 mg/kg) may blunt tachycardia associated with direct laryngoscopy.
            (ii) A slow, controlled induction is preferred (either etomidate or propofol, supplemented by pressors such as phenylephrine or norepinephrine) as necessary.
            (iii) If significant cardiac comorbidities or autonomic instability exist, preinduction invasive arterial monitoring may be necessary to ensure rapid responses to hemodynamic shifts.
         (b) Heart rate (HR) is the most significant driver of myocardial O_2 consumption.

(i) Strict HR control (<80-85 bpm) is important to prevent transient myocardial ischemia by balancing O_2 supply and demand.

(c) BP should be kept within 20% of baseline mean arterial pressure (MAP) or systolic pressure to prevent further cerebral ischemia.

(i) The cerebral autoregulation curve is generally shifted to the right; higher MAPs are required to maintain cerebral perfusion.

(ii) Choice of pressor is based on baseline HR and other comorbid conditions.

(iii) Note: If these patients have associated elevations in intracranial pressure (ICP), elevated MAPs are required to maintain cerebral perfusion pressure (CPP). The surgeon may provide MAP or systolic BP targets, and this should be discussed with the surgeon, and updated throughout the case.

1. CPP = MAP − ICP

(d) Hypocapnia/hyperventilation should be avoided except to acutely decrease brain swelling.

(i) Hypocapnia may cause cerebral vasculature constriction and contribute to cerebral ischemia.

(ii) $PaCO_2$ should be kept within 5 mm Hg of baseline.

1. This can be determined by comparing the $ETCO_2$ with a $PaCO_2$ from an arterial blood gas.
2. The $PaCO_2$ gradient will allow the anesthesiologist to optimize ventilation to maintain cerebral perfusion.

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(3) Emergence/postoperative management:

(a) Avoid sympathetic surges (hypertension, tachycardia) associated with emergence by having β-adrenergic antagonists readily available.

(b) This elevated sympathetic drive may continue into the postoperative period, in recovery, and up to 24 hours following the procedure. The perioperative risk for myocardial events and elevated ICP also continues well into this period.

(c) IV or tracheal lidocaine can be used to blunt the response to extubation. Intraoperative TIVA will also blunt autonomic responses to extubation.

(d) Adequate pain control is imperative to blunt sympathetic overdrive.

7) **Cerebellar and basal ganglia diseases: dementia and cognitive decline**

a) **Dementia and cognitive decline refer to a mixed group of disorders that represent a global decline in cognitive processing and may result from global cerebral insult or neurodegenerative process.**

i) Well-known neurodegenerative dementias include Parkinson disease (PD) and Alzheimer disease.

ii) Global ischemic or hypoglycemic insults may occur during development (eg, cerebral palsy) or in adulthood (eg, following a seizure or secondary to traumatic brain injury).

b) **Anesthetic management**

i) Preoperative

(1) Document preexisting neurological and cognitive deficits.

(2) A large subset of these patients is older; a thorough preoperative evaluation to assess for comorbidities is imperative.

(3) The patient's targeted medication list may include drugs that alter cholinergic (eg, donepezil, a reversible acetylcholinesterase inhibitor) and dopaminergic (L-DOPA) balance.

(a) Side effects should be anticipated.

(b) Interactions with anesthetic medications that affect the cholinergic system (eg, neostigmine) or dopaminergic system (eg, metoclopramide) must be considered.



Diphenhydramine and other oral anticholinergics can be used to decrease tremor in awake patients with PD and may ease IV placement if given as preoperative medication.

(4) Regional anesthesia may be preferred in this population (where appropriate) to decrease exposure to general anesthesia.[35]

ii) Induction

(1) Hemodynamic swings to induction and laryngoscopy should be anticipated and managed appropriately (see Cerebral disease: ischemia).

iii) Maintenance/emergence

(1) There is increasing evidence that preexisting cognitive decline may predispose to delayed emergence and postoperative cognitive worsening. The evidence is not conclusive but suggests that those with preexisting, even mild cognitive dysfunction may be at higher risk for postoperative delirium, while their long-term cognitive trajectory is relatively unaffected or very slightly decreased.[36–38]

(2) One obvious approach would be to minimize depth of anesthesia and length of procedure/anesthetic exposure time for elderly patients with preexisting cognitive vulnerabilities; the evidence for this approach is suggestive but not conclusive.[35]

(a) Autonomic responses and pain should be appropriately blunted by pharmacological intervention, judicious use of regional anesthesia, etc.



Patients on L-DOPA for PD are at increased risk of catecholamine-induced tachyarrhythmias.

iv) Postoperative

(1) Cognitive engagement/enrichment during this period may hasten recovery and increase the likelihood that the patient will return to their preoperative baseline.

(2) Postoperative delirium has an increased risk of morbidity and mortality; testing should be used to more immediately identify it in the PACU (or ICU) and should be treated upon identification.

(3) Restart any medications on the patient's outpatient list that protect or preserve cognitive function.

8) **Cerebellar and basal ganglia diseases: Parkinson disease**

a) **Parkinson disease**

i) PD is a neurodegenerative disease that causes a relative deficiency of dopamine (DA) relative to acetylcholine (ACh) in the basal ganglia. In addition to bradykinetic manifestations, it may also result in dementia (see Cerebral disease: dementia and cognitive decline).

ii) Treatment

(1) Patients are usually on medications that increase DA delivery (eg, levodopa) or prevent its degradation.

(2) Chronic increase in DA delivery can result in myocardial irritability, decreased intravascular volume, orthostatic hypotension, or nausea and vomiting.

(3) Anticholinergic therapy may be added to decrease dyskinesias from dopaminergic therapy.

(4) Some patients refractory to medication have deep-brain stimulation (DBS) electrode implants.

(5) A new approach to treating PD through disease-modifying therapies and repurposed drugs is evolving rapidly, with small molecules, immunotherapies, anti-inflammatory, and antibody-based therapies being evaluated.[39]
A Parkinsonian patient may be participating in a current clinical trial. Experimental agents the patient may be on should be well documented and understood.

b) **Anesthetic management**
   i) Preoperative considerations
      (1) Therapeutic agents should be continued through the morning of surgery.
      (2) Patients with moderate to severe PD may have swallowing difficulties, which increases aspiration risk.
      (3) These patients may have coexisting restrictive lung disease secondary to respiratory muscle rigidity and bradykinesia, which increases risk or postoperative pulmonary complications. Kyphosis, pharyngeal dysfunction, and sialorrhea may further compound the restrictive dysfunction. Consider plans for extubation and postoperative management as previously described.
      (4) IV access may be more difficult secondary to tremor. Use of preoperative oral anticholinergics (eg, Benadryl) may assist in decreasing tremor to assist with peripheral access.
      (5) Baseline neurological deficits and associated dementia should be documented (see Cerebral disease: dementia and cognitive decline).
   ii) Induction
      (1) Hypotension may occur secondary to relative hypovolemia.
         (a) Consider fluid loading prior to administration of an induction agent.
         (b) Treat hypotension with direct-acting pressors (phenylephrine rather than ephedrine).
      (2) Succinylcholine and nondepolarizing neuromuscular blockers are generally considered acceptable.
         (a) If muscle denervation secondary to disuse atrophy is suspected, succinylcholine should be avoided because of the potential for hyperkalemic bradycardia and arrest.
   iii) Maintenance/emergence
      (1) Maintain euvolemia to prevent hypotension.
      (2) Avoid medications that can precipitate extrapyramidal symptoms (typically first- and second-generation antipsychotics and some antinausea medications that are antidopaminergic). The stronger the D2 receptor affinity, the more likely the effect. Some examples
         (a) Phenothiazines (typically end in "-azine")
            (i) Chlorpromazine, promethazine, prochlorperazine
         (b) Butyrophenones (typically end in "-dol")
            (i) Droperidol, haloperidol, benperidol
         (c) Benzamides
            (i) Metoclopramide
         (d) Of the second-generation antipsychotics, risperidone, olanzapine, ziprasidone, lurasidone, and paliperidone are associated with a higher risk of parkinsonism; quetiapine and clozapine have a lower risk.
         (e) Central dopaminergic-depleting agents include reserpine (treatment of schizophrenia), tetrabenazine (treatment of hyperkinetic movement disorders like Huntington's chorea), deutetrabenazine, and valbenazine.
      (3) There is an increased potential for catecholamine-induced tachyarrhythmias with L-DOPA.
         (a) Use epinephrine with extreme caution in these patients.
         (b) Also use caution with myocardial irritants or sensitizers (eg, halothane, digitalis, glycosides).
      (4) Although these medications are generally safe in PD, there are isolated case reports suggesting:
         (a) Exaggerated hypertensive responses to ketamine
         (b) Increased rigidity to fentanyl and inhaled anesthetics
         (c) Increased predisposition toward dyskinesias with high-dose morphine

iv) Postoperative considerations
(1) Propofol may block PD tremors in the postoperative period.
(2) Resume the patient's therapeutic medications as soon as possible.

9) **Brain death**[40]
a) **Brain death is defined as the absence of cerebral function (unresponsiveness/coma), absence of brainstem function, and loss of respiratory drive (apnea).**
b) **Diagnosis**
i) Standards for diagnosis and determination differ by state and globally. Criteria are more likely to be standardized in locales with organ donation networks.
ii) Absence of cerebral function can only be diagnosed when the cause of coma is identified, and conditions that mimic brain death have been ruled out.
(1) Brain death mimicking conditions
(a) Drug intoxication or overdose (includes drugs of abuse, anesthetic agents)
(b) Neuromuscular paralysis (eg, severe, acute polyneuropathies or neuromuscular blockade)
(c) Hypothermia
(d) Locked-in syndrome or stroke variants that may prevent a patient from responding and/or producing motor output
(e) Shock/hypotension
(f) Guillain-Barré syndrome
(g) Encephalitis/encephalopathy
iii) Brainstem reflexes (sequential cranial nerve testing) (**Table 55-1**)
iv) Apnea test
(1) Absence of respiratory drive with a $PaCO_2 \geq 60$ or 20 mm Hg above normal baseline
(2) Connect pulse oximeter.
(3) Disconnect the patient from the ventilator.
(4) Deliver apneic oxygenation (6 L/min flow) into trachea.
(a) Can be done via catheter through the endotracheal tube to the level of the carina
(5) Observe for spontaneous ventilation (abdominal, chest excursions).
(6) A $PaCO_2$ rise of 3 mm Hg/min of apnea is expected.
(a) This means that 8-10 minutes is usually sufficient for assessment of the return of respiratory drive.
(b) Reconnect the patient to the ventilator after obtaining arterial blood gas for assessment of $PaCO_2$, PaO_2, and pH.
v) Ancillary testing for diagnosing brain death (if complete brainstem evaluation not possible)
(1) Isoelectric EEG for at least 30 minutes
(2) Absence of cerebral perfusion as demonstrated by angiography (gold standard), 99mTc scan, or transcranial Doppler ultrasound (usually of the middle cerebral artery)
(3) Absence of somatosensory and brainstem auditory evoked potentials
c) **Organ donation**[41]
i) If designated for organ donation, the brain dead patient must be optimally cared for to preserve remaining organ function.
ii) Brain death is often accompanied by dysautonomia.
(1) Reflected in a loss of systemic sympathetic tone and relative or absolute hypovolemia
(2) Dysautonomia causes fast deterioration of donor organ viability.

TABLE 55-1 Anesthetic Risk Associated With Disorders of the Nervous System

Nervous System Division	Associated Disorders	Anesthetic Risk
Sensory	Includes peripheral nerve disease, myelin disorders, polyneuropathies, cord insult: • Guillain-Barré syndrome (GBS) • Multiple sclerosis (MS) • Lyme disease • Spinal cord injury • Burn injury	• Nerve injury with regional techniques • Increased sensitivity to local anesthetics • Impaired airway reflexes, sensory arc (eg, gag)
Motor	Includes myopathies, motor end-plate denervation, motor neuron disorders, spinal motor tract disorders, peripheral nerve disease, polyneuropathies, cord injury: • Amyotrophic lateral sclerosis (ALS) • Spinal muscular atrophy (SMA) • Spinal cord injury • Guillain-Barré syndrome (GBS) • Multiple sclerosis (MS) • Lyme disease • Parkinson disease (PD) • Duchenne muscular dystrophy (DMD) • Trauma • Burn injury • Disuse atrophy	• Aspiration • Respiratory muscle weakness necessitating perioperative ventilatory support • Muscle denervation increasing the risk of hyperkalemic arrest with succinylcholine administration • Impaired airway reflexes, motor arc (eg, cough)
Autonomic	Includes peripheral nerve disease, myelin disorders, polyneuropathies, cord injury: • Guillain-Barré syndrome (GBS) • Multiple sclerosis (MS) • Lyme disease • Parkinson disease (PD) • Familial dysautonomia • Spinal cord injury (upper thoracic, cervical) • Diabetes • Alcoholism • Burn injury	• Baseline hypotension • Blood pressure lability with absence of appropriate compensatory reflexes • Lack of response to indirect-acting pressors • Increased catecholamine sensitivity • Increased myocardial irritability • Exaggerated responses to fluid shifts (eg, blood loss) • Exaggerated responses to sympathetic stimulation (eg, laryngoscopy, surgical stimulation) • Exaggerated responses to changes in intrathoracic pressure (eg, PEEP, CO_2 insufflation, Trendelenburg position)

iii) Perioperative monitoring requires
   (1) Standard ASA monitors
   (2) Temperature
   (3) Urine output
   (4) Invasive arterial pressure
   (5) Central venous/pulmonary artery pressure

iv) Physiological goals are geared toward maintaining normal parameters to preserve the viability of the organs to be harvested and transplanted.
   (1) Oxygen saturation 97%-100%
   (2) HR 60-100 beats per minute, sinus rhythm
   (3) MAP 60-80 mm Hg

TABLE 55-2 Criteria for Diagnosis of Brain Death in Adults and Children

Diagnosis	Absence of cerebral function	• Cause for coma must be sought • Conditions mimicking brain death must be ruled-out
	Absence of brainstem reflexes	• Pupillary light response • Corneal, jaw reflex • Oculocephalic reflex (doll's eyes) • Oculovestibular reflex (cold water calorics) • Gag, cough reflex • Sucking, rooting reflex (infants)
	Absence of respiratory drive	• Apnea test: no attempt to breathe at $PaCO_2 > 60$ or 20 mm Hg above baseline
Evaluation Interval by Patient Age	• Adult (>18 y) • Child, 1-18 y • Toddler, 2 mo-1 y • Infant, term to 2 mo	• 2 exams, usually 6 h apart • 2 exams, 12 h apart • 2 exams, 24 h apart • 2 exams, 48 h apart
Confirmatory Tests	• EEG • Cerebral perfusion testing • Cerebral somatosensory evoked responses • Brainstem auditory evoked responses	• Optional in adults • Required in children

(4) CVP 8-12 mm Hg
(5) Pulmonary artery wedge 10-15 mm Hg
(6) CI > 2.1 L/min/m^2
(7) Urine output 1-2 mL/kg/h
(8) Volume replacement ~50 mL/h > urine output
(9) Temperature >35°C

v) Heparin anticoagulation will be required several minutes before aortic cross-clamp, which precedes infusion of the organ preservative fluid.

10) **Summary considerations**

a) **A systematic approach can assist the anesthesiologist in management of patients with neurological diseases and can simplify the understanding of how to care for the patient with uncommon nervous system disorders.**
   i) Which branches of the nervous system are involved: sensory, motor, or autonomic? (**Table 55-2**).
   ii) What treatment or drug therapy is the patient on that can interact with anesthetic management and anesthetic drugs?
   iii) Is this patient at risk for residual weakness after anesthesia or prolonged respiratory support?
   iv) How can baseline nervous system function be preserved?
   v) How can baseline organ function be preserved?

b) Disuse atrophy is often overlooked in bedridden (eg, ICU), wheelchair bound, or young, healthy, but casted patients who have been unable to use limbs for longer than 3 days. These conditions predispose toward succinylcholine-induced hyperkalemia and cardiac arrest.

c) Rigorous documentation of preexisting deficits and attention to the full risk-benefit discussion with patients must be undertaken.
   i) The decision to use epidural, spinal, or nerve block anesthetics should be made on a case-by-case basis.

CHAPTER SUMMARY FOR ANESTHESIA AND DISEASES OF THE NERVOUS SYSTEM

General Principles	• Neurologic diseases are extremely variable in their presentation and clinical severity.
Preoperative	• Assess and document degree of neurologic dysfunction. • Assess risk for postoperative weakness and respiratory support. • The anesthetic plan, including the possibility of regional anesthesia, should be made on a case-by-case basis depending on degree of bulbar involvement, autonomic dysfunction, and sensory/motor deficits.
Intraoperative	• Succinylcholine is usually contraindicated. • Patients may have heightened sensitivity to nondepolarizing neuromuscular blockers (dose or time-course). • Rigorous attention to recovery of muscle strength is required; sugammadex is recommended for reversal of paralysis. • Hemodynamic instability may occur due to autonomic dysfunction. • Medications may alter anesthetic drug metabolism. • Extubation may not be easily achieved if there is a high degree of respiratory muscle involvement.
Postoperative	• Pain management may be difficult due to coexisting pain syndromes. • Neurologic assessment should be performed. • May require ICU care. • May require postoperative respiratory support (NIPPV, intubation).

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56 Part E: Diseases of the Gastrointestinal and Urologic Systems

Diseases of the Liver

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Although comprising only 2% of the adult body weight, the liver serves many vital functions. Its synthetic and metabolic activities allow for physiologic homeostasis and any disruption results in numerous physiologic derangements. These derangements can be acute or chronic and are best categorized as either parenchymal liver disease or cholestatic disease. This chapter will primarily discuss liver anatomy, physiology, and parenchymal liver disease.

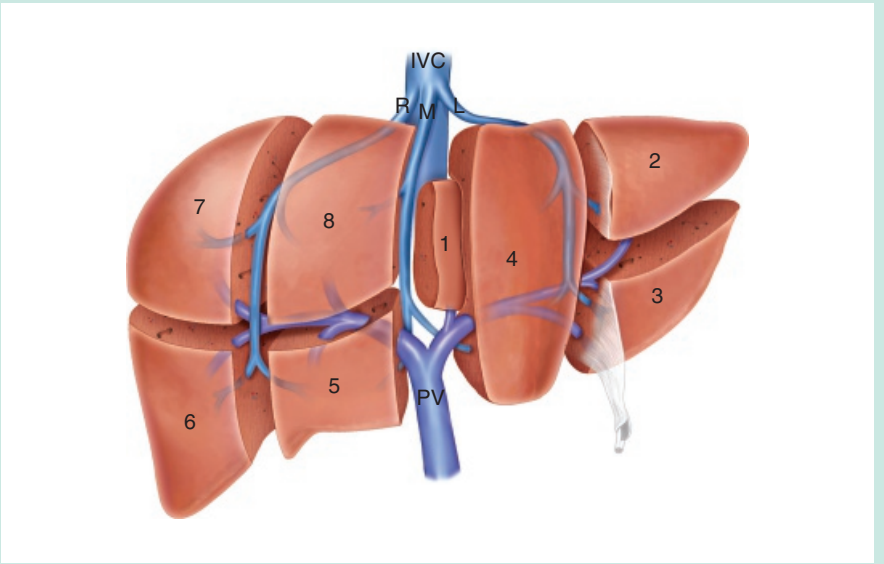
1) **Functional hepatic anatomy and circulation**
   a) **Surgical anatomy of the liver**[1] **(Fig. 56-1)**
      i) Surgically, the liver is divided into eight segments based primarily upon hepatic vein architecture.
      ii) Three hepatic veins—right, middle, and left:
         (1) The hepatic veins drain into the inferior vena cava (IVC).
      iii) The middle hepatic vein is the dividing point for right and left hemiliver, which has separate blood inflow and biliary drainage.
      iv) The left hemiliver is to the left of the IVC and divided into medial and lateral portions. Segment 4 is the left medial segment, whereas segments 2 and 3 are the left lateral segment.
      v) The right hemiliver is divided into anterior and posterior portions. Segments 5 and 8 are anterior, and segments 6 and 7 are posterior.
      vi) Overlying the IVC is segment 1, the caudate lobe.



Mortality of nonhepatic surgery in patients with decompensated liver disease depends upon the type of surgery and the level of hepatic dysfunction.[2]

   b) **Microscopic anatomy**
      i) The liver acinus is a functional anatomic unit representing the distance of a hepatocyte to a portal triad (hepatic arteriole, portal venule, and bile ductule).
         (1) The acinus possesses three zones.
            (a) Hepatocytes in zone 1 are located closest to the portal triad and receive more blood flow and nutrients.
            (b) Hepatocytes in zone 3 receive less oxygenated blood and are prone to ischemic injury.[3]
   c) **Circulation**
      i) The liver receives ~30% of the cardiac output and possesses 10%-15% of the total blood volume.
         (1) The proper hepatic artery, a branch of the celiac axis, provides 25% of the liver's blood flow and 50% of its O_2 requirements.
            (a) Hepatic artery flow is regulated by:
               (i) α_1, α_2, and β_2 sympathetic nervous system receptors
                  1. α_1 Stimulation vasoconstricts the hepatic artery, whereas β_2 stimulation vasodilates it.[4]
               (ii) Local metabolic factors such as adenosine and nitric oxide

FIGURE 56-1 Segments 2 and 3, Left Lateral Segment; Segment 4, Left Medial Segment; Segments 6 and 7, Right Posterior; Segments 5 and 8, Right Anterior; Segment 1, The Caudate Lobe.



(2) The portal vein (draining the intestines, spleen, pancreas, and stomach) provides 75% of the liver's blood flow and 50% of its O_2 requirements.
(3) Hepatic blood is returned to the IVC via hepatic veins.[4]

2) **Hepatic function**
   a) **Protein synthesis and degradation**
      i) Hepatocytes synthesize almost all plasma proteins (excluding hemoglobin and gamma globulins).
      ii) Through transamination and oxidative deamination, the liver contributes to protein metabolism.
         (1) Amino acids are degraded to ammonia, which is eliminated as urea.[5]
      iii) Albumin accounts for 15% of the protein made by the liver and 50% of all circulating proteins.
         (1) Albumin binds to drugs, hormones, and bile and dictates plasma oncotic pressure.
      iv) α_1-Acid glycoprotein binds amide-type local anesthetics, opioids, and propranolol.
      v) Butyrylcholinesterase, or pseudocholinesterase, is an enzyme which metabolizes succinylcholine and ester-type local anesthetics.
      vi) With the exception of factors III (tissue thromboplastin), IV (calcium), and VIII–von Willebrand complex—all coagulation factors are synthesized by the liver.
      vii) Fibrinolytic factors such as plasminogen activator inhibitor, protein C, protein S, and antithrombin III are also produced in the liver.
         (1) Vitamin K–dependent factors include II, VII, IX, and X and proteins C and S.[6]



Administration of vitamin K to a patient with cirrhosis **does not** improve coagulation.[7]

Spider angiomata, testicular atrophy, and gynecomastia are sequelae of chronic liver disease which result from estrogen excess.

b) **Carbohydrate metabolism**
   i) In the fed state, the liver converts glucose to glycogen.
   ii) During starvation, the liver stimulates gluconeogenesis from lactate and amino acid substrates.
   iii) Hepatic glycogen stores can provide glucose for a starvation period of 12-24 hours.[8]



Acute intermittent porphyria is the most common porphyria and presents as abdominal pain and neurologic dysfunction.[11]

c) **Lipid metabolism**
   i) When glycogen stores are full, hepatocytes convert proteins and carbohydrates to fats.
   ii) Other hepatic sources of lipids include plasma lipoproteins, exogenous fatty acids, and cytoplasmic triglycerides.
      (1) The acetyl-CoA portion of these lipids produce ATP, the body's main source of fuel.
   iii) Cholesterol is formed and metabolized in the liver.
      (1) Steroid hormones are a byproduct of cholesterol.
      (2) In the presence of hepatic disease, diminished breakdown of aldosterone results in sodium retention, hypokalemia, and volume overload.



Thiopental and etomidate are two anesthetic drugs which can porphyria in susceptible individuals.

d) **Bile metabolism**
   i) The liver synthesizes 200-500 mg of bile salts per day.[9]
   ii) Bile salts, the primary component of bile, are chiefly composed of cholesterol and bilirubin.
   iii) Bile is stored in the gallbladder, and its release is governed by the enzyme cholecystokinin.
   iv) Bile carries waste to the intestine and promotes fat micelle formation, which is needed for intestinal uptake of fats (ie, vitamins A, D, E, and K).



The liver serves as the main organ of drug metabolism.

e) **Erythropoiesis**
   i) The liver is responsible for 20% of heme production.[10]
   ii) Accumulation of heme substrates from abnormal heme synthesis results in porphyria.
   iii) Hepatocytes convert heme to biliverdin and bilirubin.
      (1) Albumin binds bilirubin in the plasma.
      (2) The liver extracts bilirubin from the blood and conjugates it with glucuronic acid. It is then secreted as bile into the intestines and excreted in the urine or feces.

3) **Endocrine functions of the liver**
   a) Degradation of approximately half of the insulin produced by the pancreas before it reaches the systemic circulation[12]
   b) Conversion of thyroxine (T_4) to triiodothyronine (T_3)
   c) Production of hormones such as angiotensinogen and insulin-like growth factor
   d) Degradation of aldosterone, estrogens, and antidiuretic hormone
4) **Immune function**
   a) Kupffer cells filter splanchnic venous blood. These cells scavenge and phagocytize bacteria, protecting the body from systemic bacterial exposure and infection.[13]
5) **Drug metabolism**
   a) Drug elimination is governed by hepatic blood flow, hepatic clearance, and protein binding.
      i) The extraction ratio measures the liver's ability to eliminate a drug (ER = hepatic clearance/hepatic blood flow).
         (1) Drugs such as lidocaine, morphine, and metoprolol have a high extraction ratio. They are rapidly metabolized and elimination is blood flow dependent.[14]
      ii) Conversely, hepatic clearance is the product of hepatic blood flow and extraction ratio.
         (1) Drugs with a low extraction ratio, such as ethanol, acetaminophen, and anticonvulsants, are more dependent on hepatic clearance and have a slow metabolism.[14]

b) Only drugs that are unbound to a carrier protein ("free" drug fraction) are clinically active and undergo metabolism.

c) **Drug metabolism is governed by three hepatic reactions.**

i) Phase 1 reactions

(1) Utilize the cytochrome P450 family of proteins.

(2) Consist of oxidation, reduction, or hydrolysis

(3) Resulting metabolites may be clinically active.

(4) Because of the newly acquired polar moiety, these metabolites are more hydrophilic and easily excreted in the urine or bile.

ii) Phase 2 reactions

(1) Drugs undergo conjugation with substances such as glucuronic acid and amino acids.

(2) Metabolites are more hydrophilic and readily excreted in the urine or bile.

iii) Phase 3 reactions

(1) Involve elimination reactions.

(2) Endogenous hepatic proteins utilize ATP to excrete various substances.[15]

6) **Hepatic pathophysiology**

a) **Acute parenchymal liver disease**

i) Most commonly secondary to viral infection but can also be caused by drugs and toxins

(1) Viral hepatitis is usually due to hepatitises A, B, C, D, and E.

(a) Traditionally, hepatitis B virus (HBV) was the most common cause of acute viral hepatitis.

(b) With the advent of the HBV vaccine, hepatitises A and C are accounting for a larger percent of cases worldwide.

(2) Cytomegalovirus and Epstein-Barr virus can also cause acute hepatitis.[16]



DILI causing an elevation in bilirubin of more than twice the upper limit carries a 10% mortality rate.[17]

ii) Drug-induced liver injury (DILI) clinically resembles acute viral hepatitis. It is typically idiosyncratic but can be toxic or cholestatic in etiology.

(1) The most common causes of DILI are alcohol, antibiotics, acetaminophen, and NSAIDs.

(2) Other causes include analgesics, antihypertensives, anticonvulsants, statins, and antiretroviral therapy.

(3) Acetaminophen can cause hepatic dysfunction ranging from acute hepatitis to massive hepatic necrosis and failure. The maximum daily dose of acetaminophen is 4 g for a healthy 75-kg adult.

iii) Symptoms

(1) Dark urine, fatigue, anorexia, and nausea.

(2) In some instances, acute hepatitis can progress to hepatic failure requiring transplant.

b) **Chronic parenchymal liver disease**

i) Chronic hepatitis is defined as elevated liver chemistries and hepatocyte inflammation, for >6 months.

ii) Signs and symptoms

(1) Fatigue, malaise, jaundice, and abdominal pain

iii) The most common causes are HBV and HCV.

(1) Chronic hepatitis is seen in ~2%-10% of patients with acute HBV cases and 50%-85% of HCV cases.

(2) Other causes include drugs, Wilson disease, hemochromatosis, nonalcoholic steatohepatitis, and α_1-antitrypsin deficiency.[16]

c) **Hepatic cirrhosis**

i) The most serious sequelae of chronic hepatitis represents end-stage liver disease.

(1) Histologically characterized by liver fibrosis and parenchymal nodular regeneration

(2) HBV, HCV, steatosis, and alcohol are common causes of end-stage liver disease.[18]

d) **Cholestatic liver disease**
   i) Signs and symptoms
      (1) Pruritus, hyperbilirubinemia (with jaundice), and right upper quadrant pain
      (2) A rise in alkaline phosphatase and gamma-glutamyl transpeptidase is common.
   ii) Although often benign, cholestatic liver disease can progress to liver failure and/or cirrhosis.[19]
   iii) Intrahepatic
      (1) Causes include drugs, alcoholism, pregnancy, sepsis, primary biliary cirrhosis, and primary sclerosing cholangitis.
   iv) Extrahepatic
      (1) Most common cause is cholelithiasis. Other causes include chronic bile duct stricture or bile duct/pancreatic tumor.

7) **Perioperative risk assessment in patients with cirrhosis**

a) **Child-Turcotte-Pugh (CTP) score (Table 56-1)**
   i) Places patients in three different classes: A (low risk), B (medium risk), and C (high risk)
   ii) Both type of surgery and CTP score should be taken in to consideration. For example, appendectomy for a patient with a CTP score of C is still reasonable risk, but major abdominal surgery carries a mortality rate around 80% and cardiac surgery a mortality rate of 100%.[2]



Independent risk factors such as emergent surgery, open abdominal procedures, ascites, azotemia, and perioperative infection increase mortality in cirrhotics undergoing surgery.[23]

b) **The model for end-stage liver disease (MELD) and MELD-Na (Table 56-2)**
   i) MELD was initially created to predict 3-month mortality in patients who had undergone the TIPS procedure. It was later found to be a useful predictor of mortality in cirrhotic patients having surgery.
   ii) In 2002, the MELD score became the ranking system for liver transplant graft allocation.
   iii) MELD scores of <10, 10-14, and >14 correlate well with CTP classes A, B, and C.[20]
   iv) MELD-Na replaced MELD for liver graft allocation in 2016 after it was shown that use of MELD-Na resulted in lower wait list mortality than MELD alone.
   v) MELD-Na scores over 19 have been associated with a higher 30-day mortality in patients undergoing emergent noncardiac surgery.[21]
   vi) The MELD formula is composed of INR, total bilirubin, and serum creatinine. The MELD-Na is comprised of the MELD and sodium.

TABLE 56-1 Modified Child-Turcotte-Pugh Score[22]

Presentation	Points		
	1	2	3
Albumin (g/dL)	>3.5	2.8-3.5	
INR	<1.7	1.7-2.3	>2.3
Bilirubin (mg/dL)	<2	2-3	>3
Ascites	Absent	Moderate	Tense
Encephalopathy	None	Grades I-II	Grades III-IV

Class A, 5-6 points; Class B, 7-9 points; Class C, 10-15 points.
Reprinted with permission from Barash P, Cullen B, Stoelting R. *Clinical Anesthesia*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2006:1103. Ref. [11]

TABLE 56-2 Model for End-Stage Liver Disease (MELD) and MELD-Na

MELD score = (0.957 × Ln(serum Cr) + 0.378 × Ln(serum Bilirubin) + 1.120 × Ln(INR) + 0.643) × 10
MELD-Na = MELD + 1.32 × (137 − [serum Na]) − [0.033 × MELD × (137 − [serum Na])]

Ln, natural logarithm; INR, international normalized ratio; Cr, creatinine; Na, sodium.

8) **Physiologic derangements of cirrhosis (Table 56-3)**
   a) **Central nervous system (CNS)**
      i) Hepatic encephalopathy represents CNS dysfunction
         (1) Presentation ranges from confusion to coma and may be secondary to increased dietary protein consumption or GI bleeding.
         (2) Increased ammonia levels do not correlate with the degree of encephalopathy.
         (3) Treatment is lactulose, rifaximin, or neomycin.
      ii) Acute liver failure may present with cerebral edema and increased intracranial pressure.[23]



The response to vasopressors is decreased with cirrhosis.[16]

   b) **Pulmonary**
      i) Patients may have hypoxemia secondary to atelectasis from ascites/hydrothorax or hepatopulmonary syndrome (HPS).
         (1) HPS is caused by intrapulmonary arteriovenous shunting of blood.
         (2) Clinical features include orthodeoxia and platypnea.[24]
      ii) Portopulmonary hypertension
         (1) Defined as mean pulmonary artery pressure >25 mm Hg is seen in ~5% of patients with a CPT score >7.[25]
         (2) Depending upon the center, a mean pulmonary artery pressure >45-50 mm Hg (consistent with severe pulmonary hypertension) is a contraindication to liver transplantation.



Cardiomyopathy should always be considered in patients with a history of alcoholic cirrhosis.

   c) **Cardiovascular**
      i) Patients with cirrhosis may have a hyperdynamic state consisting of increased cardiac output and decreased systemic vascular resistance (SVR).
      ii) It is postulated that numerous systemic arteriovenous shunts, as well as an increase in endogenous vasodilators, result in the decreased SVR. Cardiac output increases to compensate.
      iii) Patients typically have a low-normal BP.

TABLE 56-3 Physiologic Derangements in Patients With Cirrhosis

Organ System	Physiologic Derangement
Central nervous system	Encephalopathy, confusion, coma
Pulmonary	Hypoxemia resulting from atelectasis or HPS; portopulmonary hypertension
Cardiovascular	Hyperdynamic state/increased cardiac output, decreased SVR, decreased arterial blood volume, decreased response to vasoconstrictors, cirrhotic cardiomyopathy
Gastrointestinal	Portal hypertension with resulting esophagogastric varices, splanchnic hypervolemia
Renal	Renal hypoperfusion with resulting AKI/acute tubular necrosis, hepatorenal syndrome
Hematologic	Anemia, thrombocytopenia, coagulopathy

HPS, hepatopulmonary syndrome; SVR, systemic vascular resistance; AKI, acute kidney injury.

iv) Hypoalbuminemia results in decreased plasma oncotic pressure and decreased effective arterial blood volume (with increased total body volume).
v) Development of portal hypertension leads to splanchnic hypervolemia.
vi) Cirrhotic cardiomyopathy (CCM) may also develop. CCM is defined as "a chronic cardiac dysfunction characterized by impaired contractile responsiveness to stress stimuli and/or impaired diastolic relaxation and electrophysiological abnormalities with prolonged QT interval, in the absence of other known cardiac disease."[26]

d) **Gastrointestinal**
i) Complications result from portal hypertension (>10 mm Hg).
(1) Leads to the development of portosystemic venous collaterals, including esophagogastric varices
(2) Ruptured varices with hemorrhage account for one third of the mortality in patients with cirrhosis.[27]
(3) Treatment of bleeding varices consists of sclerotherapy, endoscopic ligation, β-blockade, vasopressin, somatostatin, and transjugular intrahepatic portosystemic shunt (TIPS).
(4) Portal hypertension also leads to the development of ascites, splenomegaly, and hemorrhoids.

e) **Renal dysfunction**
i) Common in advanced liver disease and is almost always secondary to hypoperfusion
ii) It manifests as either acute kidney injury or hepatorenal syndrome (HRS).
iii) Acute kidney injury (AKI)
(1) Due to decreased blood flow to the kidneys
(2) May be caused by hemorrhage (ruptured varices), splanchnic sequestration of blood, ascites formation, or dehydration.
(3) Restoring intravascular volume typically corrects AKI, but it can progress to acute tubular necrosis.
iv) Hepatorenal syndrome (HRS) is seen with cirrhosis and portal hypertension.
(1) Although unknown, the etiology is thought to be renal hypoperfusion from a decrease in vasodilating prostaglandins or profound splanchnic sequestration of blood.
(2) HRS is defined as a plasma creatinine >1.5 mg/dL in the absence of other causes of renal disease.
(3) Type I HRS progresses rapidly and may require hemodialysis and/or liver transplantation.
(4) Type II HRS is more stable and responsive to conservative treatments, including terlipressin, midodrine, and TIPS procedure.

f) **Hematologic dysfunction**
i) Anemia
(1) May be secondary to bleeding, malnutrition, or bone marrow suppression
ii) Thrombocytopenia
(1) Caused by splenic sequestration, bone marrow depression, and immune-mediated destruction
iii) Coagulopathy
(1) Due to decreased synthesis of vitamin K–dependent clotting factors and accelerated fibrinolysis
(2) Cirrhotics often present with chronic disseminated intravascular coagulation.
(3) Standardized lab tests may not represent an accurate clotting picture (see below).

g) **Metabolic abnormalities**
   i) Include susceptibility to hypoglycemia and hyponatremia
   ii) Hyponatremia is typically dilutional and is treated with volume restriction.



Patients with cirrhosis are at risk for hypoglycemia because the injured liver cannot use glycogen stores to create glucose.

9) **Anesthetic implications of liver disease**
   a) **Preoperative considerations**
      i) Due to the high perioperative morbidity and mortality associated with liver disease, a thorough preoperative evaluation is necessary.
      ii) Assessment of the type of liver disease and preoperative fluid and coagulation optimization may improve postoperative outcome.
      iii) Identification of risk factors
         (1) History of blood transfusions, IV drug abuse, sexual promiscuity, alcoholism, and a family history of liver disease should increase your suspicion for liver disease.
         (2) History of jaundice, pruritus, and anorexia may indicate liver dysfunction.
      iv) Physical exam may yield stigmata of chronic liver disease including ascites, hepatosplenomegaly, caput medusae, spider angiomata, and gynecomastia.
   b) **Determine the type and severity of liver disease.**
      i) Perioperative mortality depends upon both the severity of the liver disease and the type of surgery being performed. In general, patients with an MELD > 14 and a CTP class of C should not have elective surgery.
      ii) Child's class A (MELD < 10) cirrhotics can safely undergo elective surgery.
      iii) Patients requiring emergency surgery should be warned of their high perioperative risk of morbidity and mortality.
      iv) Acute hepatitis and fulminant liver diseases are contraindications to surgery other than liver transplantation.[28]
      v) Patients with CTP class C disease have been reported in some series to have a mortality rate of 100% with surgery requiring cardiopulmonary bypass.[28]
   c) **Organ system review** should be performed specifically looking for encephalopathy, ascites, portal hypertension, and renal insufficiency.
   d) **A complete laboratory evaluation** should be performed.
      i) Elevation of aminotransferases (AST and ALT) reflects hepatocellular injury.
      ii) Increased serum bilirubin indicates decreased hepatic excretory function, increased hemolysis, or hepatocellular injury.
      iii) Elevated INR or decreased serum albumin reflects decreased hepatic synthetic capacity.



In general, patients with an MELD > 14 and a CTP class of C should not have elective surgery.

   e) **Correct volume status and electrolyte imbalances** prior to surgery if possible.
      i) Hyponatremia should be corrected slowly with an increase of no more than 8 mEq in 24 hours.
   f) Ideal management of preoperative coagulopathy is unknown.[29]
      i) PTT, PT, and INR are not thought to accurately reflect bleeding risk secondary to rebalancing of the coagulation system.
      ii) Patients are often thrombocytopenic but may have adequate platelet function due to increased expression of von Willebrand factor.
      iii) Fibrinogen levels are often low in severe liver disease, but the fibrin clot may be relatively normal in strength.
      iv) Viscoelastic testing may provide a more accurate picture of coagulopathy in these patients and may be a better way to determine if and what type of preoperative coagulation system resuscitation is needed.



Intraoperative hypoxemia, hemorrhage, and hypotension in a patient with liver disease may further reduce hepatic blood flow.

v) It is unknown if the use of synthetic agents such as Kcentra, activated factor 7, and fibrinogen concentrate result in different outcomes than conventional blood component therapy.
vi) Traditional teaching advocates correcting INR, hypofibrinogenemia and thrombocytopenia prior to major surgery.

g) **Operating room preparations**
i) Include transducers for invasive monitoring, a fluid warming/rapid infusion device, and increased room temperature.

10) **Intraoperative considerations**
a) Management goals include preserving hepatic blood flow and existing hepatic function.
b) Drug selection and dosing
i) IV induction agents
(1) Have a modest effect on hepatic blood flow
(2) Thiopental and etomidate decrease hepatic blood flow.
(3) Propofol and ketamine increase hepatic blood flow.
(4) Awakening from short-acting induction agents is not prolonged by liver disease, likely due to redistribution and a larger volume of distribution.
(5) Hypoalbuminemia can cause an exaggerated induction response with both etomidate and thiopental.
(6) Propofol, etomidate, and ketamine possess a high hepatic extraction ratio, and their pharmacokinetic profile is relatively unchanged in mild to moderate cirrhosis.
ii) Neuromuscular blocking agents (NMBs)
(1) Should be titrated and monitored appropriately
(2) Although pseudocholinesterase levels may be decreased, clinical prolongation of succinylcholine is not significant.
(3) Succinylcholine is the neuromuscular blocking induction agent of choice as patients with ascites are at risk for aspiration and rapid sequence induction is indicated. Patients with end-stage liver disease have a variable response to rocuronium and may take longer to achieve adequate neuromuscular blockade.[30]
(4) Intermediate acting nondepolarizing agents, such as rocuronium and vecuronium, have a prolonged effect in the presence of liver disease, which is accentuated with repeated dosing.
(5) Pharmacokinetic-pharmacodynamic modeling shows that there is prolonged time for recovery from neuromuscular blockade in patients with end-stage liver disease compared to healthy controls when sugammadex is dosed at 4 mg/kg 15 minutes after 1.2 mg/kg of rocuronium and when dosed at 2 mg/kg after reappearance of T2. Interestingly, a dose of 16 mg/kg of sugammadex 3 minutes after rocuronium showed no significant delay in recovery.[30]
(6) Benzyl isoquinoline neuromuscular blockers, such as atracurium and cisatracurium, undergo organ-independent elimination via ester hydrolysis and Hoffman degradation.
iii) Volatile anesthetics
(1) The newer volatile anesthetics do not significantly alter hepatic blood flow or oxygenation.
(2) At levels below 1 MAC, sevoflurane, desflurane, and isoflurane minimally decrease hepatic blood flow and undergo minimal hepatic metabolism (<2%) with no significant clinical sequelae.
iv) Other commonly used medications



All benzodiazepines should be dosed cautiously in patients with liver disease due to the potential for worsening preexisting encephalopathy.

(1) Benzodiazepines
  (a) Midazolam and diazepam are metabolized by phase I oxidative reactions and have a prolonged duration of action.
  (b) Lorazepam is cleared by phase II reactions via glucuronidation and undergoes normal metabolism.
(2) Opioids
  (a) Metabolism is decreased in liver disease.
  (b) Morphine and meperidine have a prolonged half-life and can precipitate hepatic encephalopathy.
  (c) Fentanyl, although completely metabolized by the liver, does not show a prolonged effect in cirrhosis.
  (d) Remifentanil, metabolized solely by blood esterases, is not affected by liver disease.
  (e) All opioid agonists can increase sphincter of Oddi pressure.
    (i) Agonist/antagonists (nalbuphine, tramadol) seem to have less of an effect.
(3) Intravascular volume status
  (a) Can be very difficult to assess in patients with liver disease as they are usually "arterially underfilled" and clinically hypovolemic.
  (b) Arterial blood volume should be monitored via arterial pH, urine output, and central venous pressure.
  (c) A transesophageal echocardiogram may also be useful, but one must be cautious with varices and coagulopathy.



Fentanyl and remifentanil are the opioids of choice in patients with liver disease.

c) **Postoperative considerations**

i) Postoperative liver dysfunction is common in patients with liver disease.
  (1) With mild disease, the dysfunction may be limited to elevation of transaminases or postoperative jaundice.
  (2) In patients with class C (MELD > 14) cirrhosis, 1-month mortality ranges from 60% to 100%.
  (3) The most common postoperative complications include new-onset ascites, volume overload, worsening hepatic encephalopathy, upper gastrointestinal hemorrhage, sepsis, renal failure, and decreased liver function with coagulopathy.
  (4) Poor outcomes are common in emergency or major surgery especially with coexisting renal failure.
  (5) Preoperative jaundice is a risk factor for postoperative renal failure.



Patients with liver disease are usually clinically hypovolemic.

ii) Manifestations of alcohol withdrawal should be monitored for 48-72 hours in patients with suspected alcohol abuse.

iii) All postoperative patients with preexisting liver disease should be monitored closely for 24 hours in a step-down or ICU setting as morbidity and mortality are extremely high.

Read More
Alcohol and substance abuse, Chapter 73

CHAPTER SUMMARY FOR THE PATIENT WITH LIVER DISEASE

Preoperative Evaluation	Assessment and optimization of coagulation, electrolyte, and intravascular volume status; warm room and fluids; warn patient of high perioperative risk.
Induction of Anesthesia	RSI for presumed full stomach, expect exaggerated desaturation due to decreased FRC from ascites, minimize preoperative benzodiazepines, and use short-acting induction agents (propofol).
Maintenance of Anesthesia	Maintain hepatic/renal blood flow, volatile anesthetics at <1 MAC, use short-acting opioids (fentanyl and remifentanil), and invasive monitoring may be used to assess/restore intravascular volume, acid/base, and electrolyte status.
Postoperative Concerns	Monitor closely in a step-down/ICU setting for signs of volume overload, worsening renal/liver dysfunction, sepsis, encephalopathy.

FRC, functional residual capacity; ICU, intensive care unit; INR, international normalized ratio; MAC, minimum alveolar concentration; RSI, rapid sequence induction.

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57 Diseases of the Gastrointestinal System

Cara Crouch • Matthew J. Fiegel • Barbara J. Wilkey

The gastrointestinal (GI) tract's main function is to absorb water, electrolytes, and nutrients. Derangement can adversely affect many other organ systems. It is the responsibility of the anesthesiologist to ensure preoperative optimization of fluids, electrolytes, and nutrition. One must also search for other organ involvement (eg, anemia from B12 deficiency) and treat these systems accordingly. Intraoperatively, manipulation of the GI tract can lead to bacteremia and sepsis. This should always be anticipated and treated as necessary with volume, antibiotics, and vasopressor agents.

1) **Esophageal diseases**
   a) **Gastroesophageal reflux disease (GERD)** (Fig. 57-1)
      i) One of the more common problems that anesthesiologists encounter
      ii) Patients are at risk for aspiration of gastric contents upon induction of anesthesia, which can result in a life-threatening aspiration pneumonitis.
      iii) Pathophysiology
         (1) Risk factors for GERD and/or delayed gastric emptying include
            (a) Obesity
            (b) Diabetes
            (c) Obstructive sleep apnea
            (d) Collagen vascular diseases
         (2) Signs and symptoms
            (a) Dysphagia
            (b) Retrosternal chest pain
            (c) Acidic taste
      iv) Pharmacologic treatment
         (1) Of the prophylactic medications, proton pump inhibitors (PPIs) appear to raise the gastric pH the most.[1]
            (a) Allow 45-60 minutes for full onset of action.
         (2) Metoclopramide, H_2 receptor antagonists, and oral antacids may also be used.
      v) Perioperative considerations
         (1) Preoperative considerations
            (a) There is a paucity of evidence-based recommendations. Therefore, pharmacologic prophylaxis is at the discretion of the individual clinician.
            (b) Measures that lessen the likelihood of significant aspiration include
               (i) Removal of gastric contents (ie, preinduction NG tube)
               (ii) Proper nil per os (NPO) status
               (iii) Head-up position (reverse Trendelenburg)
            (c) The ASA does not recommend preoperative treatment with an antireflux medication as the evidence is equivocal.[1]
            (d) Patients on chronic antireflux therapy should take their medication on the day of surgery.



Approximately 25 mL of a gastric aspirate with a pH of 2.5 or 50 mL of a gastric aspirate with a pH of 3.5 is necessary to cause clinically significant chemical pneumonitis. See Table 57-1 for aspiration risk factors.

TABLE 57-1 Risk Factors for Aspiration

Patient Factors	Surgical Factors	Anesthesia Factors
• Full stomach • Delayed gastric emptying • Morbidly obese patients • Incompetent LES tone	• Emergency surgery • Upper abdominal surgery • Lithotomy or Trendelenburg positioning • Laparoscopic surgery • Cholecystectomy	• "Light" anesthesia • Supraglottic airway devices • Positive pressure ventilation • Surgery length >2 h

From Asai T. Who is at risk of pulmonary aspiration? *Br J Anaesth.* 2004;93:497-500; Robinson M, Davidson A. Aspiration under anaesthesia: risk assessment and decision-making. *Contin Educ Anaesth Crit Care Pain.* 2014;14(4):171-175. Ref.[2,3]

(2) Intraoperative considerations
(a) There are no definitive recommendations regarding which patient population should undergo rapid sequence induction (RSI). Each clinician should use his/her best judgment.
(b) Patients at high risk for aspiration include:
(i) Full stomach by NPO guidelines
(ii) Bowel obstruction
(iii) Multiple risk factors for delayed gastric emptying (listed above)
(c) RSI with succinylcholine or high-dose rocuronium should always be considered for patients at high risk for aspiration.
(i) Succinylcholine is not contraindicated. It increases both intragastric pressure and lower esophageal sphincter (LES) tone.[4]
(3) Postoperative considerations
(a) Patients at risk for aspiration on induction are still at risk for aspiration with emergence and should be treated with caution.
(b) Awake extubation with intact airway reflexes is recommended.

b) **Esophageal motility disorders:** esophageal motility disorders can be associated with regurgitation of esophageal contents.
i) Diffuse esophageal spasm
(1) Esophageal spasm does not present many anesthetic-related concerns. It can mimic angina and must therefore be considered in a patient with chest pain.
ii) Achalasia
(1) Achalasia is characterized by failure of the LES to relax as well as loss of peristalsis in the distal esophagus. Achalasia is commonly associated with regurgitation of partially digested or undigested food.[4]
(2) These patients are at high risk for aspiration; RSI should be considered.

c) **Hiatal hernia**
i) These are often asymptomatic if LES tone remains intact. Regardless, these patients remain at higher risk of aspiration.

d) **Esophageal bleeding**
i) Causes
(1) Esophageal varices
(2) Mallory-Weiss tears
(3) Erosive esophagitis
ii) Acute variceal bleeds
(1) Typically caused by portal hypertension
(2) Treatment
(a) Medical management
(i) Vasopressin or octreotide are common medications for treatment of varices.

(b) Sclerotherapy
(c) Transjugular intrahepatic portosystemic shunt (TIPS) procedure
(d) Surgical treatment (including banding)

(3) Perioperative considerations
(a) If a patient is presenting for control of active bleeding, resuscitation should be anticipated.
(b) The airway should be secured with endotracheal intubation.
(c) Adequate IV access should be obtained and blood products available.
(d) If varices are chronic and stable, manipulation of the esophagus should be kept to a minimum.
(i) Placement of an oro/nasogastric tube or transesophageal echocardiography probe should be done with caution.

e) **Esophageal perforation**
i) Pathophysiology
(1) Multiple causes including spontaneous perforation following forceful vomiting (Boerhaave syndrome), iatrogenic/procedural, and traumatic
(2) May be associated with intrinsic esophageal disease
ii) Signs and symptoms
(1) Fever
(2) Substernal pain
(3) Perforation will be seen on imaging.
iii) Treatment frequently includes emergent surgical intervention.
iv) Perioperative considerations
(1) These patients can develop mediastinitis and sepsis and may be hemodynamically unstable upon presentation to the operating room. If hemodynamically unstable, consider invasive monitoring.
(2) Adequate IV access should be obtained and invasive blood pressure monitoring considered given the possibility of hemodynamic instability. If expected to require vasopressor support, consider central line placement.
(3) If perforation is secondary to vomiting, volume resuscitation is often necessary.
(4) Lung isolation is often required for surgical intervention (double-lumen ETT placement or bronchial blocker).



Patients with esophageal perforation can be septic. Be prepared for hypotension after induction.

f) **Esophageal cancer**
i) Pathophysiology
(1) Squamous cell carcinoma
(a) Alcohol consumption and smoking are the two most common risk factors.
(2) Adenocarcinoma
(a) Usually related to long-term GERD symptoms resulting in Barrett esophagus
ii) Signs and symptoms
(1) Dysphagia
(2) Weight loss
(3) Malnutrition is often seen.
iii) Perioperative considerations
(1) Esophagectomy is frequently the treatment of choice once cancer is identified.[5]
(a) There are several variations of the surgical approach to esophagectomy, including traditional and minimally invasive techniques. Traditional surgical approaches include transhiatal (cervical incision and laparotomy), Ivor Lewis (right thoracotomy and laparotomy), and left thoracoabdominal (includes a left thoracotomy). Minimally invasive approaches involve

utilizing video-assisted thoracoscopic (VATs) techniques. Any approach that involves a thoracotomy or VATs will require one lung ventilation; bronchial blockers or double-lumen ETT should be available. Patients with an open approach may benefit from thoracic epidural placement; this is considered standard at many institutions.[6]



If reintubation is required, consider video scope to avoid unintentional esophageal conduit intubation.

(2) Patients are at high risk for aspiration.

(3) These patients often have other comorbidities from the underlying risk factors associated with esophageal cancer (alcohol use, smoking, GERD).

(a) Liver disease

(b) Chronic obstructive pulmonary disease (COPD)

(4) It is important that noninvasive positive pressure ventilation (CPAP/BIPAP) be avoided postoperatively after esophagectomy. This should be considered when determining extubation plans at the end of surgery.



Absolute contraindications to transesophageal echocardiography probe placement include prior esophagectomy or esophagogastrectomy. Relative contraindications: esophageal stricture, tracheoesophageal fistula, post esophageal surgery, and esophageal trauma.[7]

2) **Gastric diseases**

a) **Peptic ulcer disease (PUD)**

i) Pathophysiology

(1) PUD is seen in ~10% of men and 4% of women.[8]

(2) Represented by focal ulceration in the esophagus, stomach, or duodenum

(3) Causes

(a) *Helicobacter pylori* (*H pylori*)

(b) Nonsteroidal anti-inflammatory drugs (NSAIDs)

(c) Stress

(d) Excessive gastric secretion (Zollinger-Ellison syndrome—covered below)

(e) Smoking and steroid use are also implicated in the development of PUD.

ii) Chronic PUD

(1) Does not warrant any major anesthetic alterations

(2) Treatment includes

(a) Eradication of *H pylori*

(b) PPIs

(c) H_2 receptor antagonists

(d) Cessation of any other offending agents

iii) Acute bleeding ulcers

(1) As with esophageal variceal bleeding, resuscitation must be anticipated.

(a) Adequate IV access should be obtained.

(b) Volume replacement in the form of crystalloid, colloid, and/or blood products

(2) Intraoperative considerations

(a) Airway management

(i) If the patient is unable to maintain his/her airway, an RSI and intubation should be performed.

(b) After assessment of volume status and airway stability, endoscopy should be performed to treat the bleeding.

(c) Consider a PPI infusion as it may decrease the risk of recurrent bleeding.



Patients with bleeding gastric ulcers are at risk for aspiration.

iv) Perforated ulcers

(1) Typically secondary to NSAID use or cancer

(2) Signs and symptoms
(a) Presents as fever, abdominal pain, and free air on abdominal imaging
(b) Patients can deteriorate quickly.
(3) 30-day mortality is ~10% but can be as high as 38% in patients with advanced age, multiorgan failure, and prolonged diffuse peritonitis.[9]
(4) Perioperative considerations
(a) Electrolyte and volume status should be corrected prior to surgery.
(b) Vasopressor agents may be necessary if peritonitis and sepsis are present.
(c) Broad-spectrum antibiotics should be initiated.

b) Gastric cancer
i) Initial signs and symptoms are often similar to PUD.[4]
ii) Malnutrition and weight loss can be associated with electrolyte abnormalities.

3) **Small intestinal diseases**

a) **Zollinger-Ellison syndrome**
i) Pathophysiology
(1) Patients have duodenal ulcers resulting from a gastrinoma.
(2) Usually treated with PPIs and H_2 blockers
(3) Although not an acute process, ZE syndrome sometimes necessitates surgery.
ii) Perioperative considerations
(1) Preoperative liver function tests and electrolytes should be obtained.
(2) Malabsorption may be present.
(a) Results from secretion of large volumes of gastric fluid
(b) Can influence clotting factors and electrolytes[10]
(3) Preoperative nasogastric tube is often necessary to remove the large gastric volumes.
(4) All patients should undergo RSI.

b) **Inflammatory bowel disease (IBD):** IBD, which includes Crohn disease and ulcerative colitis, is the second most common inflammatory disease in the body. Both are autoimmune mediated and, therefore, may affect many different organ systems.
i) Crohn disease
(1) Can affect the entire GI tract but is typically confined to the ileum and the colon.
(2) Chronic disease results in rectal fissures, fistulas, and abscesses.
(3) Anemia, with accompanying malnutrition, is often present.
(4) Immunosuppressive agents are the treatment of choice.
(a) Includes steroids and anti–tumor necrosis factor (TNF) antibodies
(5) Patients often require total parenteral nutrition (TPN) and bowel rest to treat the fistulas.
(a) If medical therapy failures, surgery is required.[11]
ii) Ulcerative colitis
(1) Signs and symptoms
(a) Presents as intermittent diarrhea, the disease process is confined to the colon.
(b) Can progress to toxic megacolon, bowel stricture/perforation, and colon cancer.
(c) Extraintestinal manifestations
(i) Iritis
(ii) Ankylosing arthritis
(iii) Primary sclerosing cholangitis (PSC)—discussed below
1. If PSC is present, patients typically develop cirrhosis of the liver.

Read More
Arthritis, Chapter 69
Diseases of the liver, Chapter 56

(2) Treatment
(a) Initial treatment consists of sulfasalazine and mesalamine.
(i) If these are ineffective, more potent immunomodulating agents may be necessary (steroids, anti-TNF antibodies, and cyclosporine).
(b) Patients require surveillance colonoscopies and often a colectomy for prevention/treatment of colon cancer.[11]

iii) Anesthetic management of patients with IBD
(1) Preoperative considerations
(a) Assess volume and electrolyte status.
(b) Determine the extent of extraintestinal manifestations.
(c) If cirrhosis is present from PSC/UC, blood products and adequate IV access should be available.
(2) Intraoperative considerations
(a) RSI should be considered in anyone with intestinal obstruction from IBD.
(b) The choice of muscle relaxant may be influenced by the presence of liver disease.
(c) If ankylosing spondylitis is present, a fiberoptic intubation may be necessary.
(d) Stress-dose steroids may be required in patients with long-term steroid use.
(e) Avoidance of nitrous oxide is prudent as it may distend an already scarred bowel lumen.
(f) NSAIDs should be avoided as they can exacerbate inflammatory bowel disease by increasing mucosal inflammation.[11]


Look for evidence of cirrhosis in patients with primary sclerosing cholangitis.

c) **Carcinoid tumors**
i) Pathophysiology
(1) Carcinoid tumors arise from enterochromaffin cells and reside in the GI tract 75% of the time.
(a) The most common location for benign tumors is the appendix.
(b) The ileum and cecum are the most common sites for malignant tumors.
(2) Cells of these tumors secrete a variety of vasoactive hormones including[12]:
(a) Serotonin
(b) Histamine
(c) Bradykinin
(d) Prostaglandins

ii) Carcinoid syndrome
(1) Results from metastasis of the tumor to the liver
(a) The affected liver cannot metabolize the secreted hormones and carcinoid syndrome ensues.
(2) Although most tumors secrete hormones, only about 7%-18% of patients will develop carcinoid syndrome.
(3) Signs and symptoms[12]
(a) Cutaneous flushing
(b) Diarrhea
(c) Right-sided cardiac symptoms
(i) Tricuspid and pulmonic valve dysfunction
(d) Bronchoconstriction
(e) Hypotension
(4) Treatment
(a) The mainstay of treatment of carcinoid syndrome is the somatostatin analog, octreotide (Sandostatin).[10,12]

(i) Inhibits release of vasoactive hormones from the tumor
(ii) Dosing ranges from 50 to 100 µg 2-3 times daily. There is also a long-acting depot formulation.

iii) Perioperative considerations
(1) Preoperative considerations
(a) Extraintestinal manifestations should be evaluated.
(b) A preoperative echocardiogram may diagnose right-sided heart disease.[13]
(c) Patients should be on octreotide prior to surgery.
(2) Intraoperative considerations
(a) Octreotide should be available for potential intraoperative release of hormones.
(b) If refractory hypotension ensues, vasopressin should be used.
(c) Steroids and H2 blockers have both been studied in carcinoid syndrome. However, neither has been shown to be effective in decreasing any of the carcinoid symptoms.[12]



Evaluate for tricuspid and pulmonic valve dysfunction in patients with carcinoid syndrome.

d) **Small bowel obstruction**
i) Pathophysiology
(1) The degree of obstruction and duration are important.
(2) Obstruction can cause bowel necrosis, which can present with sepsis.
ii) Signs and symptoms
(1) Abdominal pain
(2) Nausea and vomiting
iii) Perioperative considerations
(1) These patients are at very high risk for aspiration. They will often have a nasogastric tube placed preoperatively.
(2) RSI is recommended.
(3) Adequate IV access should be available.
(4) Resuscitation requirements should be expected, these patients are typically intravascularly depleted.
(5) If patient is febrile or showing signs of a systemic inflammatory response, invasive monitoring may be considered and vasopressors should be available.



Any disease that affects the intestines can interfere with absorption of nutrients, which can have many downstream effects. Always review preoperative labs for any uncorrected electrolyte abnormalities or coagulopathy that may be present.

4) **Large intestinal diseases**
a) **Diverticulosis**
i) Causes
(1) Diverticulosis results from herniations through the colonic muscular layer.
(2) Patients who consume low-fiber diets are at highest risk.
ii) Signs and symptoms
(1) Symptoms are infrequent but can include abdominal pain and lower GI bleeding.
iii) Perioperative considerations
(1) If surgery is required to control bleeding, preoperative volume status should be assessed.
(2) Adequate IV access is necessary and blood should be available.

b) **Diverticulitis**
i) Approximately 10%-25% of patients with diverticulosis progress to diverticulitis.[14]
ii) Signs and symptoms
(1) Patients present with fever, nausea, vomiting, and right-sided abdominal pain.

(2) Abscess and fistula formation may necessitate surgical intervention.

iii) Perioperative considerations

(1) RSI should be considered.

(2) Patients are at high risk for sepsis; therefore, invasive hemodynamic monitoring and vasopressor therapy should be considered.



Approximately 10%-20% of the population older than 50 years of age are known to be affected by diverticular disease.[14]

Duodenal ulcers are responsible for the majority of upper GI bleeding, whereas diverticula are responsible for the majority of lower GI blood loss.

c) **Appendicitis**

i) Often mimicking diverticulitis, inflammation of the appendix occurs in 7%-9% of the population during their lifetime.[14]

ii) Signs and symptoms

(1) Patients present with anorexia, fever, and periumbilical/right lower quadrant pain.

(2) If the patient is febrile, appendiceal rupture with peritonitis should be suspected.

iii) Perioperative considerations

(1) RSI should be considered.

(2) Vital signs should be monitored for signs of sepsis.

d) **Colon cancer**

i) Pathophysiology

(1) Typically arise from adenomatous polyps

(2) Risk factors[4]

(a) Family history

(b) IBD

(c) Smoking history

ii) Signs and symptoms

(1) Melena

(2) Anemia from chronic ongoing blood loss

iii) Perioperative considerations

(1) Patient should be evaluated for preoperative anemia.

(2) Electrolyte abnormalities may be present.

(3) If bowel obstruction is present, evaluate for increased aspiration risk.

5) **Pancreatic diseases**

a) **Acute pancreatitis**



Patients with acute pancreatitis may become critically ill very quickly and require ICU care.

i) Pathophysiology

(1) Gallstones obstructing the ampulla of Vater or alcohol consumption (70% of cases)[15]

(2) May also be secondary to trauma, hypercalcemia, or an endoscopic retrograde cholangiopancreatography (ERCP)[16]

ii) Signs and symptoms

(1) Patients can present with nausea, vomiting, fever, and abdominal pain.

(2) Can progress to systemic inflammatory response syndrome/shock, acute respiratory distress syndrome (ARDS), acute kidney injury (AKI), and pancreatic infection

iii) Treatment

(1) In the acute form of the disease, cessation of the offending agent will allow the pancreas to return to its normal function.

(2) Supportive care includes NPO status, IV hydration, occasional antibiotics, cessation of alcohol, and/or removal of gallstones.

(3) Surgery is only required if infection is confirmed.

(a) For gallstone pancreatitis, cholecystectomy should be considered once recovered from acute episode.

(4) Patients often undergo therapeutic ERCP in the acute phase of illness.[17]
(a) Are often symptomatic and may decompensate acutely.
(b) Often require GETA, and RSI should be considered.

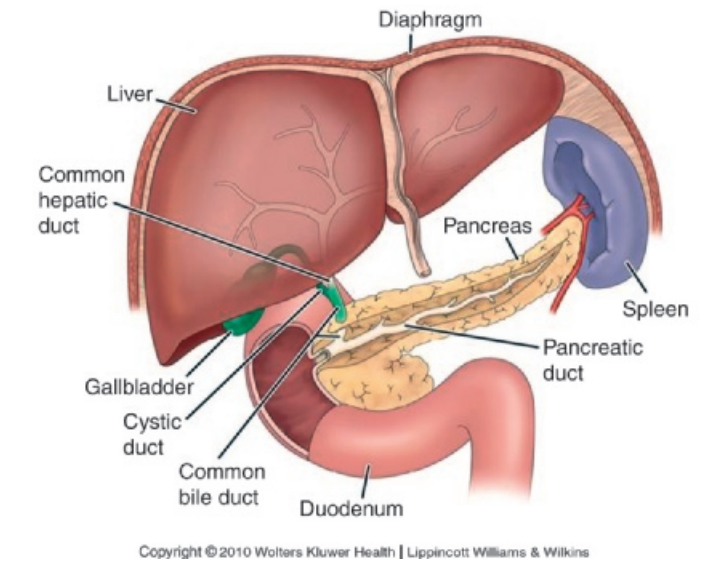
iv) Perioperative considerations
(1) When surgery is required, the anesthesiologist must assess the degree of organ dysfunction and treat accordingly.
(2) Vasopressor agents and fluid resuscitation may be necessary for septic shock.
(3) Lung-protective ventilation strategies and/or an ICU ventilator may be necessary for ARDS.
(4) Meticulous volume management and/or hemodialysis may be necessary for AKI.
(5) Coagulation status should be assessed as pancreatitis can progress to disseminated intravascular coagulation (DIC).

b) **Chronic pancreatitis**
i) Pathophysiology
(1) Defined as repeated injury to the pancreas resulting in loss of normal function
(2) Chronic pancreatitis is almost always secondary to alcohol use.[16]
ii) Signs and symptoms
(1) Loss of exocrine function leads to lack of digestive enzymes and chronic malabsorption.
(2) Loss of endocrine function can lead to diabetes.
(3) Chronic pain is often present.
iii) Perioperative considerations
(1) If a patient with chronic pancreatitis presents for surgery: intravascular volume, coagulation, and metabolic and electrolyte status should be evaluated.
(2) A perioperative, multimodal plan for pain control should be instituted as these patients are often taking large amounts of opioids.

c) **Pancreatic cancer**
i) Pathophysiology
(1) The majority of pancreatic cancers are ductal adenocarcinomas that are found within the head of the pancreas.[4]
ii) Signs and symptoms
(1) These patients will often present with abdominal pain and weight loss.
(2) Can develop jaundice from biliary obstruction by the tumor
(3) As with other types of GI tract cancer, these patients are at risk for malnutrition and electrolyte abnormalities.
iii) Perioperative considerations
(1) Surgical resection (pancreaticoduodenectomy) is the primary treatment.[4] This is a very common operation worldwide, it is also known as a Whipple resection.[18]
(2) These cases often require large-bore IV access and invasive arterial line monitoring.
(3) Thoracic epidural analgesia has been shown to reduce postoperative pain and reduce postoperative pulmonary complications. It is routinely performed for this operation (**Fig. 57-1**).[19]

6) **Biliary diseases**
a) **Acute cholecystitis**
i) Pathophysiology
(1) Inflammation caused by acute obstruction of the cystic or common bile duct by a gallstone

FIGURE 57-1 Normal Anatomy of Liver, Pancreas and Biliary System



ii) Signs and symptoms
  (1) Nausea and vomiting
  (2) Abdominal pain
  (3) Right upper quadrant tenderness
  (4) Dark urine
  (5) Scleral icterus
iii) Treatment
  (1) IV hydration
  (2) Pain management
  (3) Surgical treatment
iv) Perioperative considerations
  (1) These patients should be assumed to have delayed gastric emptying from an inflammatory response. They are often actively experiencing nausea/vomiting and RSI is recommended.

b) **Primary sclerosing cholangitis (PSC)**
i) Pathophysiology
  (1) Chronic inflammation of the intrahepatic or extrahepatic biliary ducts[4]
  (2) Often associated with IBD (ulcerative colitis)[20]
  (3) Eventually leads to cholestasis and liver failure
ii) Signs and symptoms
  (1) Fatigue
  (2) Pruritus
  (3) Jaundice, hepatomegaly, and splenomegaly

iii) Perioperative considerations
(1) The progression of disease can lead to cirrhosis and portal hypertension; the implications to anesthetic management depend on the degree of subsequent hepatic disease.
(2) These patients often require liver transplantation as definitive treatment.
(3) Patients are frequently on corticosteroids or other immunosuppressive agents.[20] Medications should be reviewed prior to surgery.

c) **Primary biliary cirrhosis/primary biliary cholangitis (PBC)**
i) Pathophysiology
(1) Immunologic attack on intralobular biliary ducts
(2) Untreated, it will result in cholestasis, cirrhosis, and liver failure.
ii) Signs and symptoms
(1) Fatigue
(2) Pruritus
(3) Skin manifestations (hyperpigmentation, xanthomas, xanthelasmas)
(4) Hepatosplenomegaly
iii) Treatment
(1) Ursodeoxycholic acid
(2) Liver transplantation may sometimes be required.
iv) Perioperative considerations
(1) These patients may present with malabsorption.
(2) The degree of liver failure is an important factor in anesthetic management.



Biliary tract disease can eventually lead to hepatic failure, evaluating the degree of hepatic impairment is essential to providing appropriate anesthetic care.

CHAPTER SUMMARY FOR DISEASES OF THE GI SYSTEM

Definition	Diseases of the GI tract are varied and include esophageal, gastric, pancreatic, intestinal, and appendiceal processes.	
GI Diseases	Esophageal	GERD, esophageal motility disorders, hiatal hernia, esophageal bleeding, esophageal perforation, esophageal cancer
	Gastric	Peptic ulcer disease, gastric carcinoma
	Intestinal	ZE syndrome, IBD, carcinoid tumors, small bowel obstruction, diverticular disease, acute appendicitis, colon cancer
	Pancreatic	Acute pancreatitis, chronic pancreatitis, pancreatic cancer
	Biliary	Acute cholecystitis, PSC, PBC
Anesthetic Implications	The anesthetic implications of almost every GI process include assessment of aspiration risk, nutrition, volume, electrolyte, and hemodynamic status.	

GI, gastrointestinal; GERD, gastroesophageal reflux disease; ZE, Zollinger-Ellison; IBD, inflammatory bowel disease; PSC, primary sclerosing cholangitis; PBC, primary biliary cirrhosis/primary biliary cholangitis.

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58 Kidney Disease§

Claudia R. Fernandez Robles

The kidneys perform the major functions of volume regulation, electrolyte homeostasis, and drug/toxin excretion. They also produce hormones involved in blood pressure regulation (renin) and red blood cell production (erythropoietin). Despite being 0.5% of the body weight, the kidneys normally receive 20%-25% of the cardiac output.[1] Renal blood flow is normally autoregulated between mean arterial pressures of 60 and 150 mm Hg providing primary protection against hypotensive or hypertensive injury. Inadequate renal perfusion under anesthesia can lead to renal ischemia and the development of postoperative acute kidney injury with an incidence of 1% after general surgery and up to 30% after cardiovascular procedures.[2,3]

1) **Nomenclature**
   a) Acute renal failure => Acute kidney injury (AKI).
   b) Chronic renal failure => Chronic kidney disease (CKD).
   c) The nomenclature has been modified to ensure more accurate definition of kidney injury, better predictive measures of mortality, and recognition of early kidney injury before onset of failure.[4]
   d) The nomenclature AKI and CKD will be used in this chapter.



The duration of impairment in renal function determines the classification of the disease.

2) **Epidemiology**
   a) Kidney disease is the ninth leading cause of death in the United States.[5]
   b) Perioperative AKI is the leading cause of morbidity and mortality, with an eightfold increase in mortality in patients who exhibit AKI.[6]

In the United States, diabetes is the leading cause of CKD, followed by high blood pressure.

3) **Pathophysiology**
   a) Renal dysfunction is classified based on the glomerular filtration rate (GFR) and the rapidity of onset of the dysfunction.
   b) GFR is the volume of fluid filtered from the renal glomerular capillaries per unit time.
      i) Varies with age, gender, and body size, and declines with age.
      ii) GFR > 40% of normal is usually asymptomatic.
      iii) GFR between 10% and 40% of normal.
         (1) In the elderly, decreased GFR is an independent predictor of adverse outcomes, such as death and cardiovascular disease.
         (2) Doses of medications need to be adjusted to avoid complications from accumulation of metabolites or further kidney damage.[7]
      iv) GFR < 10% of normal is associated with end-stage renal disease (ESRD) and usually requires hemodialysis (HD).



GFR should be checked when administering renal excreted medications.

   c) **AKI vs CKD**
      i) Acute kidney injury (AKI)
         (1) Defined as an abrupt decrease in kidney function, characterized by increased serum creatinine levels and/or a decrease in urine output.
         (2) There are different criteria for categorizing the severity of the disease:
            (a) KDIGO (Kidney Disease: Improving Global Outcomes) guidelines

TABLE 58-1 KDIGO Criteria[3]

Stage of Kidney Disease	Serum Creatinine Concentration	Urine Output (UO)
1	Increase of ≥0.3 mg/dL or 50%-99%	UO < 0.5 mL/kg/h for 6-12 h
2	Increase of 100%-199%	UO < 0.5 mL/kg/h for 12-24 h
3	Increase of ≥200% OR ≥0.3 mg/dL to >4 mg/dL	UO < 0.3 mL/kg/h for ≥24 h OR anuria for ≥12 h OR initiation of RRT

(b) RIFLE (Risk, Injury, Failure, Loss of kidney function, and End-stage kidney disease)
(c) AKIN (Acute Kidney Injury Network). KDIGO is the most recent and widely used **(Tables 58-1 to 58-3).**

(3) Acute on chronic failure refers to AKI that develops in patient with preexisting CKD.

4) **Acute kidney injury (AKI)**
a) Clinical signs and symptoms
i) Volume overload characterized by peripheral and pulmonary edema
ii) Pericarditis, hyperkalemia, metabolic acidosis, coagulopathy
iii) Nausea/vomiting, anorexia, pruritus, fatigue, altered mental status, shortness of breath, decreased urine output

b) **Etiologies of AKI**
i) Risk factors include
(1) Critical illness, sepsis, burns, trauma, cardiac surgery, major noncardiac surgery, or exposure to nephrotoxic drugs
(2) Advanced age, volume depletion, female gender, black race, chronic disease, diabetes, obesity

ii) Prerenal AKI
(1) Decrease in renal perfusion pressure without damage to the renal parenchyma
(2) Hypoperfusion from hypovolemia, decreased cardiac output, increased intra-abdominal pressure, decreased systemic vascular resistance
(3) NSAIDs can provoke acute prerenal failure in patients with other risk factors for developing AKI.[6]

iii) Renal (acute tubular necrosis)
(1) There are three causes of acute tubular necrosis (ATN): renal ischemia, sepsis, and nephrotoxins.
(2) In ATN, renal tubular epithelial cells are injured, leading to apoptosis and necrosis, followed by tubular obstruction and finally affecting the glomerular filtrate.

iv) Postrenal
(1) Obstruction to urine flow from the kidneys leading to renal vasoconstriction and progressive renal fibrosis.

TABLE 58-2 AKIN Criteria[3]

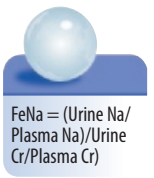
Stage of Kidney Disease	Serum Creatinine Concentration	Urine Output (UO)
1	Increase of ≥0.3 mg/dL or 50%-100%	UO < 0.5 mL/kg/h for 6-12 h
2	Increase of >100%-200%	UO < 0.5 mL/kg/h for 12-24 h
3	Increase of >200% OR by >0.5 mg/dL to ≥4.0 mg/dL	UO < 0.3 mL/kg/h for >24 h OR anuria for >12 h OR Initiation of RRT

TABLE 58-3 RIFLE Criteria

Class	Serum Creatinine or GFR	Urine Output (UO)
Risk	Increase Cr × 1.5 or GFR decrease >25%	UO < 0.5 mL/kg/h for 6-12 h
Injury	Increase Cr × 2 or GFR decreased >50%	UO < 0.5 mL/kg/h for 12-24 h
Failure	Increase Cr × 3, or >4 mg/dL with an acute rise >0.5 mg/dL or GFR decreased >75%	UO < 0.3 mL/kg/h for ≥24 h OR anuria for ≥12 h OR initiation of RRT
Loss	Complete loss of kidney function >4 wk	
ESRD	Complete loss of kidney function >3 mo	

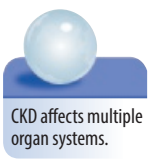
(2) Causes include prostatic hypertrophy, abdominal or pelvic mass impinging on ureters, calculi, and blood clots.

c) Differentiation between the different causes of AKI can be aided by
   i) Measuring urine and serum sodium and creatinine
   ii) Calculating Fractional Excretion of Na (FENa) = (Urine Na/Plasma Na)/(Urine Cr/Plasma Cr)



   iii) FeNa may help distinguish between the possible causes of AKI (prerenal causes vs oliguric acute tubular necrosis states). However, the results may be affected by several factors such as preexisting renal disease, electrolytes abnormalities, and use of diuretics.
   iv) Analysis of FeNa, serum creatinine, and FeUrea may aid toward a better understanding of the underlying cause by defining prerenal, intrarenal, or postrenal disease.[7,8] Patients with prerenal causes should have a low fractional excretion of sodium (FeNa <1%). Oliguric patients with intrinsic renal dysfunction, typically have FeNa >1%-2%.

5) **Chronic kidney disease (CKD) (Table 58-4)**
   a) Abnormality of the kidney structure or function present for more than 3 months
   b) **Multiple systems are affected in patients with CKI.**
      i) Hematologic
         (1) Anemia—due to decreased production of erythropoietin
         (2) Coagulation system
            (a) PT/INR, PTT, and platelet counts are usually normal.
            (b) Patients have poor platelet adhesion from defective interaction of the von Willebrand factor and platelet glycoprotein IIb/IIIa receptors.



   c) Hypercoagulation
      i) Despite platelet dysfunction, thromboelastographic indices show tendency toward clotting.
      ii) Electrolyte disturbances (Table 58-5)

TABLE 58-4 Chronic Kidney Disease (CKD) Criteria

Either of the following present for more than 3 months.	
Markers of kidney damage (one or more)	• Albuminuria (AER ≥30 mg/24 h; ACR ≥30 mg/g) • Urine sediment abnormalities • Electrolyte and other abnormalities due to tubular disorders • Abnormalities detected by histology • Structural abnormalities detected by imaging history of kidney transplantation
Decrease GFR	• GFR < 60 mL/min/1.73 m^2

TABLE 58-5 Common Electrolyte Disturbances in Renal Failure

Electrolyte Abnormality	Pathophysiology	Signs/Symptoms	Treatment
Hyperkalemia	Poor excretion, transcellular shifts due to acidemia and insulin deficiency	• Weakness, paresthesias • Peaked T waves, ST depression, prolonged PR, QRS widening, sine wave, ventricular fibrillation	• IV calcium stabilizes myocardium • Sodium bicarbonate, insulin/dextrose, hyperventilation shifts K intracellularly, HD
Hypocalcemia	Elevated parathyroid hormone	Usually well tolerated, can cause tetany, seizures if severe or sudden	Vitamin D, calcium supplements
Hypermagnesemia	Poor excretion	Usually well tolerated, can cause hypoventilation, altered mental status, prolongation of neuromuscular blocking drugs if severe	Avoid magnesium-containing medications, HD
Metabolic acidosis	Inadequate hydrogen ion excretion	Respiratory distress, hypotension, cardiac arrhythmias, hyperkalemia	Hyperventilation, sodium bicarbonate, hemodialysis

iii) Cardiovascular
  (1) Hypertension
    (a) Present in 80% of patients
    (b) May be primary cause or as a result of kidney disease
  (2) Left ventricular hypertrophy due to volume and pressure overload
  (3) Accelerated atherosclerosis due to dyslipidemia and inflammation
  (4) Vascular and valvular disease due to abnormal calcium deposition
  (5) Predisposition to arrhythmias due to conduction system abnormalities
  (6) Pericarditis due to uremia

iv) Neurologic
  (1) Autonomic neuropathy
    (a) Present in up to 90% of patients with CKD on HD[9]
    (b) Characterized by reduced baroreceptor sensitivity, sympathetic overactivity, and parasympathetic dysfunction
  (2) Peripheral sensory neuropathy is also evident.

v) Gastrointestinal
  (1) Delayed gastric emptying seen in up to 69% of patients

vi) Immunologic
  (1) Patients are immunocompromised and have a higher prevalence of systemic and superficial infections.



A large number of patients with CKD on HD have autonomic neuropathy.



Preoperative evaluation of the patient on HD includes HD history, assessment of volume status, and laboratory results.

6) **Anesthetic considerations**

a) **Preoperative considerations**

i) If patient is not on HD
  (1) Identify risk factors.
  (2) Assess renal function using most recent creatinine and urine output.
  (3) In hospitalized patients, assess whether renal function is stable, worsening, or improving by noting creatinine and urine output trends.



Review when patient last received HD and HD record if available.

ii) If patient is on HD
  (1) Assess volume status using current vital signs and weight as compared to dry weight.
  (2) Review when patient last received HD and HD record if available.
iii) Vascular access can be difficult due to the presence of arteriovenous fistulas.
iv) Consider invasive monitoring or echocardiography to aid in volume assessment, in patients with uncontrolled hypertension, decreased ejection fraction, severe electrolyte abnormalities, and surgical procedures with anticipated major volume shifts.
v) Carefully review labs, specifically potassium levels, creatinine values, and pH.



Potassium levels should be checked before rapid sequence intubation.

b) **Intraoperative considerations**
  i) Induction of anesthesia
    (1) Oral nonparticulate antacid should be used if appropriate.
    (2) Rapid sequence induction (RSI) should be considered as delayed gastric emptying is seen in the majority of patients.[10–12]
  ii) Maintenance of anesthesia
    (1) Patients will have decreased excretion of drugs cleared by the kidneys.
    (2) Sensitivity to drugs that suppress the central nervous system is increased.
    (3) Muscle relaxation
      (a) Succinylcholine is an acceptable choice for muscle relaxation if there is no preexisting hyperkalemia.
      (b) **Vecuronium and rocuronium** are renally excreted, and patients may exhibit prolong muscle relaxation.
      (c) **Cisatracurium is the muscle relaxant of choice** as its duration of action is unaffected by renal disease (metabolized by Hoffman reaction).
      (d) **Neostigmine** has prolonged duration of action, therefore recurarization is unlikely.
      (e) Sugammadex, although cleared by the kidneys, has been use safely and effectively in patients with renal disease.[13,14]
    (4) Volatile anesthetics can be used.
      (a) Sevoflurane in the setting of low flows and dry CO_2 absorbent may result in accumulation of the nephrotoxic agent, compound A.
    (5) Pain medication
      (a) Morphine
        (i) Leads to accumulation of active and toxic metabolites, especially morphine-6-glucuronide, which may cause delayed respiratory depression.
        (ii) Lowers seizure threshold
        (iii) Doses should be reduced in patients with altered renal function.
      (b) Meperidine
        (i) Metabolite normeperidine accumulates
          1. Associated with seizures, myoclonus, and altered mental status. Should not be used in patients with renal failure and acute renal dysfunction
      (c) Fentanyl
        (i) Has no active metabolites, but clearance is reduced



Morphine should be avoided in patients with renal failure. Its active metabolite can accumulate in patients with renal failure leading to respiratory depression.



Intraoperative urine output in adults 0.5-1 mL/kg/h

(d) Intravenous fluids
(i) Balanced crystalloid solutions are preferred.
(ii) Avoid diuretics unless volume overload is present.

iii) Intraoperative low urine output (<0.5 mL/kg/h, if not oliguric at baseline)
(1) Rule out new postrenal cause.
(a) Check urinary catheter patency, flush if necessary.
(b) Discuss the possibility of surgical ureteral obstruction in gynecologic and pelvic procedures.
(2) Rule out prerenal cause.
(a) Ensure adequate intravascular volume.
(b) Maintain renal perfusion by optimizing cardiac output.
(3) Consider intrarenal cause.
(a) Preexisting CKD
(b) Recent use of nephrotoxic agents such as IV contrast



Laparoscopic surgery often causes transient low urine output due to renal hypoperfusion and ureteral compression.

c) **Postoperative considerations**
i) Hypertension is common.
(1) If volume overload is suspected, urgent HD should be considered.
ii) Fentanyl, remifentanil, alfentanil, and hydromorphone can be used at reduced dosages for pain management.
iii) Consider emergent hemodialysis in patients with refractory volume overload, potassium levels >6.5 mEq/L, signs of uremia, uremic pericarditis, and severe metabolic acidosis.

CHAPTER SUMMARY FOR KIDNEY DISEASE

	Non-HD Dependent	HD Dependent
Preoperative Evaluation	Assess volume status, urine output, and recent laboratory results (potassium, pH, BUN/Cr, serum bicarbonate) Initiate preoperative HD if needed	Assess volume status. Verify timing of last HD Assess recent laboratory results (potassium, pH, BUN/Cr) Check location of AVF if present and patent (check for thrill)
Induction of Anesthesia	Assume full stomach, use RSI if appropriate, avoid hypoventilation, succinylcholine only if preoperative K+ normal, and adjust medications doses	
Maintenance of Anesthesia	Inhaled agents, cisatracurium NMB of choice, reduce dose of opioids, monitor for signs of cardiac ischemia, if AVF present—palpate intermittently for thrill	
Postoperative Concerns	May need to initiate HD	May need urgent HD

AVF, arteriovenous fistula; BUN, blood urea nitrogen; Cr, creatinine, HD, hemodialysis; K+, potassium; RSI, rapid sequence induction.

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59 Part F: Endocrine Diseases
Diabetes Mellitus§

Stacy L. Fairbanks

Diabetes mellitus (diabetes) is a common metabolic disorder which presents with hyperglycemia, with the long-term risk for organ damage and the possibility of acute metabolic derangements. Anesthesia providers must understand the treatment and complications of this disease to provide adequate perioperative care.

1) **Overview**
   a) **Epidemiology**
      i) The overall incidence in the United States in 2015 was estimated to be 30.3 million people (9.4% of the population).[1]
      ii) Diabetes is the seventh leading cause of death in the United States.[2]
2) **Classification**[3]
   a) **Type I diabetes**
      i) Also called immune-mediated diabetes (previously called insulin-dependent diabetes)
      ii) Due to autoimmune destruction of beta-cell, usually leading to absolute insulin deficiency
      iii) Patients must receive exogenous insulin in order to survive.
      iv) Occurs most frequently in those <20 years of age, but it has been reported in persons in their 50s.
      v) Patients are at risk for diabetic ketoacidosis (DKA) and may rarely present with hyperosmolar, hyperglycemic state (HHS—previously called hyperosmolar, hyperglycemic nonketotic coma [HHNC]).



Patients with IDDM are at much higher risk for DKA than those with NIDDM.

   b) **Type 2 diabetes**
      i) Previously referred to as non–insulin-dependent diabetes
      ii) Patients have relative insulin deficiency and have peripheral insulin resistance.
      iii) Patients with fasting glucose levels of >126 mg/dL are considered diabetic. Patients with abnormal 2-hour plasma glucose level (>200 mg/dL during a standard glucose tolerance test) or symptoms of diabetes (polyuria, polydipsia, and unexplained weight loss) plus a random glucose >200 mg/dL are also considered diabetics.
      iv) Most common form of diabetes in the United States. Risk of diabetes increases with age, obesity, and lack of physical activity.
      v) Occurs more frequently in women with prior gestational diabetes mellitus, patients with hypertension, or dyslipidemia and those of African American, American Indian, Hispanic/Latino, and Asian American descent
      vi) Patients are at risk for HHS, but very rarely present with DKA.
   c) **Prediabetes**
      i) Describes individuals whose glucose levels do not meet criteria for type 2 diabetes, typically fasting glucose levels between 101 and 125 mg/dL

ii) Should be viewed as an increased risk for diabetes and cardiovascular disease but not diagnosed as diabetics

d) **Gestational diabetes mellitus (GDM)**

i) Diabetes that is first diagnosed in the second or third trimester of pregnancy and do not have either preexisting type 1 or type 2 diabetes

ii) These individuals are at increased risk of developing type 2 diabetes after delivery and should receive lifelong diabetes screening.



Atherosclerotic cardiovascular disease (coronary artery disease, cerebrovascular disease, or peripheral arterial disease) is the leading cause of morbidity and mortality of people with diabetes.

3) **Long-term complications from poorly controlled diabetes**

a) Patients are at higher risk for heart failure and coronary artery disease, peripheral vascular disease, stroke, kidney disease, peripheral neuropathy, vision impairment, joint mobility restrictions, delayed gastric emptying, and autonomic dysfunction.

b) **Heart disease**

i) Patients with diabetes have two to three times the risk for coronary artery disease than the general population.

ii) Diabetic patients are at high risk for silent myocardial ischemia or atypical chest pain, because diabetes-related neuropathy can interfere with pain signals in the body.

c) **Autonomic dysfunction**

i) Results from neuropathy of the autonomic nerves, which control vascular tone responses as well as other autonomic functions such as gastric and bladder emptying

ii) Patients are at risk for hypotension and hypothermia and may have blunted responses to changes in heart rate as seen with β-adrenergic antagonists and atropine.[4]



Patients with autonomic dysfunction due to diabetic neuropathy may have blunted responses to β-blockers and atropine.

d) **Delayed gastric emptying**

i) Patients may have impaired motility and delayed gastric emptying.

ii) May be at higher risk for aspiration events

4) **Diabetic ketoacidosis**[5]

a) A serious, potentially fatal, complication of diabetes

b) Most common in type 1 diabetics, but it has been reported in type 2 diabetics

c) It is characterized by ketone formation (from a lack of insulin), an anion gap acidosis, profound dehydration (5-8 L), and electrolyte abnormalities (especially hypokalemia) (Table 59-1).

TABLE 59-1 Signs and Symptoms of DKA[5]

Clinical Signs and Symptoms	Laboratory Abnormalities
Nausea and vomiting	Hyperglycemia
Dehydration	Metabolic acidosis
Hypotension	Arterial pH <7.3
Tachycardia	Bicarbonate <15 mEq/L
Kussmaul respirations	Ketones present in urine and blood
Abdominal pain	Hyperosmolarity
Ileus	Hypokalemia
Somnolence or coma	Increased BUN and/or creatinine

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5) **Treatment of DKA**[6]
   a) If severe CNS depression is present, intubate the trachea to protect the airway.
   b) Administer regular insulin intravenously to facilitate glucose-based cellular metabolism.
      i) Start with 0.1 unit/kg IV insulin bolus, followed by an infusion at 0.1 units/kg/h.
      ii) Goal is to decrease blood glucose level by 50-70 mg/dL in the first hour.
   c) Restore intravascular volume.
   d) If patient needs to go emergently to OR, consider 20 mL/kg bolus(es) of normal saline to restore intravascular volume prior to induction.
   e) **Once hemodynamically stable, or if time permits, replace volume with normal saline 5-10 mL/kg/h.**
      i) Add 5% glucose when serum glucose level is <250 mg/dL.
   f) Restore total body potassium deficit (0.3-0.5 mEq/kg/h).
   g) **Closely monitor blood glucose, serum potassium, and pH.**
   h) **Patients with DKA have large total body potassium deficits.**
      i) Yet, they often have normal or elevated serum potassium levels due to insulin deficiency and metabolic acidosis shifting potassium out of cells.
         (1) When insulin is administered, the potassium is shifted back into cells, resulting in a precipitous fall in the serum potassium level.
            (a) Potassium repletion should begin once the serum level is below ~5.0 mEq/L and diuresis has been established.


Patients with DKA may have severe intravascular volume depletion and may require 5-8 L of intravenous fluid.

6) **Hyperosmolar, hyperglycemic nonketotic syndrome (HHNS)**[5]
   a) **It is a serious complication of diabetes with mortality rates reported to be 10%-20%.**
      i) Characterized by severe hyperglycemia (>600 mg/dL)
      ii) Profound dehydration
      iii) Hyperosmolarity (>350 mOsm/kg)
      iv) Electrolyte abnormalities (especially hypokalemia)
      v) A normal pH
         (1) Most prevalent in elderly patients in their 70s who have type 2 diabetes
   b) **Signs and symptoms**
      i) Similar to DKA
         (1) However, there is a lack of ketoacidosis.
         (2) CNS symptoms tend to predominate.
         (3) Unlike DKA where symptoms arise rapidly, in HHS, symptoms are gradual in onset due to gradual volume depletion and dehydration, usually progressing over a few days.
   c) **Treatment of HHS**[6]
      i) Administer normal saline or lactated Ringer's to correct severe hyperosmolarity, and restore intravascular volume.
         (1) Begin with 1 L/h in patients with shock. Use caution in those with heart failure or renal insufficiency. Continue to administer based on patient's hemodynamic status.
      ii) Administering insulin before adequate fluid replacement can result in cardiovascular collapse. This is because insulin mediated glucose uptake by the cells and also leads to water moving from intravascular space into the cells, causing intravascular hypovolemia.
      iii) Insulin infusions are useful to help lower the blood glucose below 300 mg/dL.
         (1) Begin with regular insulin 0.15 units/kg bolus. Then 0.1 units/kg/h infusion


Administering insulin before adequate fluid replacement can result in cardiovascular collapse in patients with HHS.

(2) Goal is to decrease glucose by 50 mg/dL first hour then 50-70 mg/dL hourly thereafter.

iv) Potassium replacement is often needed to replace losses from the osmotic diuresis.

v) As with DKA, frequent laboratory monitoring is indicated.



Perioperative glucose control is extremely important because hyperglycemia and hypoglycemia are associated with poor outcomes.

7) **Perioperative insulin management**[4,7]

a) **The guiding principle should be to maintain the patient as close to normal as possible, while avoiding certain conditions.**

i) Hypoglycemia

ii) Hyperglycemia

iii) Ketoacidosis

iv) Electrolyte abnormalities

b) **Goal is to maintain a blood glucose (BG) level <180 mg/dL without causing hypoglycemia.**

i) The easiest method is via a continuous IV insulin infusion (**Table 59-2**).

ii) Initiate insulin infusion when BG 140-180 mg/dL.

iii) If BG < 150 mg/dL, initial infusion rate should be BG divided by 70 (rounded to nearest 0.5 units), no bolus.

iv) If BG > 150 mg/dL, give bolus. Calculate bolus amount by dividing initial BG by 70 (rounded to nearest 0.5 units). Then start infusion at same hourly rate as bolus.



Perioperative patients who have taken their diabetes medications are at high risk for hypoglycemia and should be monitored carefully.

8) **Perioperative approach to the diabetic patient**[4,7]

a) **Preoperative considerations**

i) If possible, diabetic patients should be scheduled as first case to minimize disruption to their blood glucose management and hasten return to their normal meal plan.

ii) Determine what type of diabetes the patient has, how long they have had the disease, and how well it is controlled.

(1) Hemoglobin A1c is the best marker of diabetic control in the last 3 months.

iii) Determine what medications they have taken on the morning of surgery.

(1) Be aware of the risk of hypoglycemia in NPO patients who have taken either insulin or oral drugs that stimulate insulin secretion (**Tables 59-3 and 59-4**).

TABLE 59-2 Example of Continuous IV Insulin Infusion Protocol for Use With Continuous Pump Delivery Systems[4,7]

1. Mix regular insulin to a concentration of 1 unit/mL.
2. If BG > 150 mg/dL, give insulin bolus (initial BG divided by 70).
3. Initiate infusion at calculated rate (initial BG divided by 70 [rounded to nearest 0.5 units])
4. Measure blood glucose concentrations as necessary (every 30-60 min), and adjust infusion rate accordingly

Blood Glucose Value	Action to Be Taken
<80 mg/dL	• Turn infusion off for 30 min • Administer 25 mL of 50% glucose • Measure blood glucose in 30 min
80-150 mg/dL	Stop insulin, recheck in 30 min, when BG is >150 mg/dL, and restart rate with 50% of previous rate
150-180 mg/dL	If BG decreasing since last check, keep infusion the same. If BG stable or increasing, increase infusion rate
>180 mg/dL	If BG decreasing >40 mg/dL since last check, keep infusion the same. If BG stable or increasing, increase infusion rate

Wall, RT. Endocrine Disease in Hines RL & Marschall KE (eds.). *Stoelting's Anesthesia and Co-Existing Disease*. 7th ed. Philadelphia, PA: Churchill Livingston, 2018:449-476; Joshi GP, Chung F, Vann MA, et al. Society for ambulatory anesthesia consensus statement on perioperative blood glucose management in diabetic patients undergoing ambulatory surgery. *Anesth & Analg*. 2010;111(6):1378-1387.

TABLE 59-3 Pharmacologic Properties of Commonly Available Insulin[7]

Name	Type	Onset	Peak	Duration
Humalog (lispro), Apidra (glulisine), NovoLog (aspart)	Rapid	5-15 min	30-90 min	4-6 h
Regular insulin (R)	Short-acting	30-60 min	2-4 h	6-8 h
NPH (Novolin N, Humulin N-NF)	Intermediate	2-4 h	4-10 h	10-16 h
Lente (L)	Intermediate	2-4 h	4-10 h	12-20 h
Ultralente (U)	Intermediate- to long-acting	6-10 h	10-16 h	18-24 h
Glargine (Lantus), detemir (Levemir)	Very long-acting	2-4 h	Evenly for 24 h	20-24 h
NPH and regular mixed in either 50/50 mix or 70/30 mix	Premixed	30-90 min	Dual	10-16 h

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TABLE 59-4 Common Oral Diabetes Medications[7]

Drug Class	Mechanism of Action	Brand and Generic Names	Half-Life	Perioperative Considerations
Sulfonylurea	Stimulates pancreatic islet beta-cell insulin secretion, decreases insulin resistance	Amaryl (glimepiride) Diabeta, Micronase, Glynase (glyburide) Glucotrol (glipizide)	2-10 h	Risk of hypoglycemia, GI disturbance
Meglitinides	Stimulates pancreatic islet beta-cell insulin secretion	Prandin (repaglinide) Starlix (nateglinide)	1 h	Risk of hypoglycemia
Biguanides	Decreases hepatic glucose production; increases insulin sensitivity	Glucophage (metformin)	6-18 h	Diarrhea, nausea, black box warning: risk of lactic acidosis
Alpha-Glucosidase Inhibitors	Delays/reduces glucose absorption from the intestines	Precose (acarbose) Glyset (miglitol)	2-4 h	Risk of ileus
Thiazolidinediones	Increases insulin sensitivity, decreases hepatic glucose production	Avandia (rosiglitazone) Actos (pioglitazone)	3-8 h	Fluid retention, hepatotoxicity, black box warning: may cause or exacerbate CHF
DPP-4 inhibitors	Slows incretin metabolism, increases insulin release, decreases glucagon levels	Januvia (sitagliptin) Onglyza (saxagliptin)	8-14 h	Risk of angioedema and pancreatitis, infection
Incretin mimetics (noninsulin injectable)	Suppresses glucagon secretion, decreases gluconeogenesis, reduces incretin metabolism	Byetta (exenatide)	6-10 h	Nausea, vomiting, weight loss, hypoglycemia if combined with sulfonylureas

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(2) If patient have an insulin pump, diabetes specialist should be contacted prior to elective surgery, and institutional protocol needs to be followed. If insulin infusion is going to continue during surgery, blood glucose should be monitored every hour intraoperatively and postoperatively until the patient is awake and back to their functional baseline state.

iv) Check blood glucose level in the preoperative area.
(1) If it is significantly elevated, consider starting an insulin infusion before going to the OR.

v) Be very alert to the possibility of DKA in the following cases.
(1) The patient is an insulin-dependent diabetic who is acutely ill.
(2) The patient has not been taking their insulin.

vi) Attempt to ascertain the extent of end-organ damage that has occurred.
(1) Keep in mind that not only are patients with diabetes at higher risk of cardiovascular disease but are at risk for perioperative silent myocardial ischemia, injury, and infarction.
(a) Evaluate patient for functional capacity, and ensure elective patients are optimized regarding coexisting diseases.
(b) Have high index of suspicion for coronary artery disease. Patients with multiple risk factors and/or poor functional capacity should undergo stress testing.

b) **Intraoperative considerations**[4,7]

i) In long-standing diabetic patients and those with neuropathy, gastric emptying may be significantly delayed. Consider rapid sequence induction to prevent aspiration.

ii) The autonomic dysfunction in diabetes leads to abnormal vascular tone responses.
(1) Patient may be less sensitive to beta-blockers and alpha-agonists.
(2) Normothermia and adequate analgesia is important in order to limit excessive stress and resultant increases in blood glucose.

iii) For patients who are felt to be at high risk for myocardial ischemia, consider using 5-lead EKG monitoring in the form of precordial V-lead monitoring.

iv) Pay close attention to hydration status in order to protect kidneys from injury.

v) Monitor blood glucose at regular intervals during the case with blood glucose goal <180 mg/dL (as detailed in perioperative insulin management).
(1) Autonomic neuropathy plus effects of sedatives and analgesics can mask symptoms of hypoglycemia.
(2) Patient on continuous insulin infusion or insulin pump should have their blood glucose checked every hour in the perioperative period.



In any patient with postoperative confusion, delayed emergence, or seizures, RULE OUT HYPOGLYCEMIA.

c) **Postoperative considerations**

i) For patients with delayed emergence or who are confused in the recovery area, always rule out hypoglycemia as a cause.

ii) If the patient required an insulin infusion in the operating room, consider continuing it in the recovery area.

iii) Patients with diabetes are at risk for many postoperative complications.
(1) Poor wound healing
(2) Infection
(3) Pressure ulcers

CHAPTER SUMMARY FOR DIABETES MELLITUS

Diabetes	Prevalent disease that is the seventh leading cause of death in the United States
Long-Term Complications	Heart failure, coronary artery disease, peripheral vascular disease, stroke, kidney disease, peripheral neuropathy, vision impairment, joint mobility problems, delayed gastric emptying, and autonomic dysfunction
Type 1 Diabetes	Characterized by lack of endogenous insulin. Patients are at risk for DKA, which can be life-threatening
Type 2 Diabetes	Characterized by resistance to insulin. Most common form of diabetes in the United States
Diabetic Ketoacidosis (DKA)	Potentially deadly complication characterized by ketone formation, an anion gap acidosis, profound dehydration (5-8 L), and electrolyte abnormalities (especially hypokalemia)
Intraoperative Care	Maintain the blood glucose under 180 mg/dL while simultaneously avoiding hypoglycemia, ketoacidosis, and electrolyte abnormalities. Monitor glucose every hour if the patient is on insulin infusion or insulin pump

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60 Thyroid Disease

Yefim Vilnits • Jessica Spellman

The thyroid is one of the largest endocrine glands in the body. It produces hormones that regulate cellular metabolism, growth, function of body systems, and regulation of calcium levels. This chapter will discuss the implications of diseases of the thyroid on perioperative anesthetic management.

1) **Overview**
   a) The thyroid gland consists of two lobes connected by the isthmus. It is located anteriorly in the neck below the cricoid cartilage.
   b) The thyroid gland contains large stores of thyroid hormone in the form of thyroglobulin. Two-thirds of iodine that is absorbed from the follicle binds to thyroglobulin to become the thyroid hormones thyroxine (T4) and triiodothyronine (T3).
      i) The thyroidal secretion is predominantly the prohormone T4. T3:T4 hormones are secreted in a ratio of 1:10. About 80% of plasma T3 comes from deiodination of T4 by peripheral tissues.
      ii) Local activation of T4 also occurs in target tissues (eg, brain and pituitary) and is increasingly recognized as an important regulatory step in thyroid hormone action.
      iii) Local deactivation of T3 is an important regulatory step. Serum concentrations of thyroid hormones are precisely regulated by the pituitary hormone thyrotropin stimulating hormone (TSH) in a negative-feedback system.
      iv) T3 is three to four times more potent than T4. Elimination half-time for T4 is 7 days and for T3 is 1.5 days.
      v) T3 and T4 are mostly inactive and bound to thyroxine-binding globulin (80%), albumin (5%-10%), and prealbumin (10%-15%). Biologic active free T4 (fT4) represents 0.03% of total T4 plasma levels, while free T3 (fT3) represents 0.3% of total T3.
   c) **T3 and T4:**
      i) Promote gene transcription and basal cell metabolism, body growth, bone maturation, and formation.
      ii) Increase intestinal absorption of glucose and insulin-dependent intracellular transport of glucose.
      iii) Enhance hepatic gluconeogenesis and glycogenolysis.
      iv) Increase lipid utilization from the adipose tissue, increase plasma free fatty acids levels, and decrease of plasma cholesterol, phospholipids, and triglycerides.
      v) Increase the number of β-adrenergic receptors and the sensitivity of those receptors to catecholamines, thus increasing myocardial contractility, reducing systemic vascular resistance, and increasing intravascular volume. Patients who are hyperthyroid can have a cardiac output 50%-300% higher than euthyroid patients.[1]
      vi) Promote brain development in the intrauterine and neonatal periods. Manifestations of cretinism (congenital hypothyroidism) include intellectual disability, short stature, gait abnormalities, deaf mutism, and spasticity.



Thyroid hormones increase the number of beta-adrenergic receptors and the sensitivity of those receptors to catecholamines, thus increasing myocardial contractility.

vii) Thermogenesis: Thyroid hormone is necessary for both obligatory thermogenesis (the heat resulting from vital processes) and adaptive thermogenesis. Thermogenesis is highly sensitive to thyroid hormone around the physiologic range, and small changes in levothyroxine replacement doses may significantly alter resting energy expenditure in the hypothyroid patient.

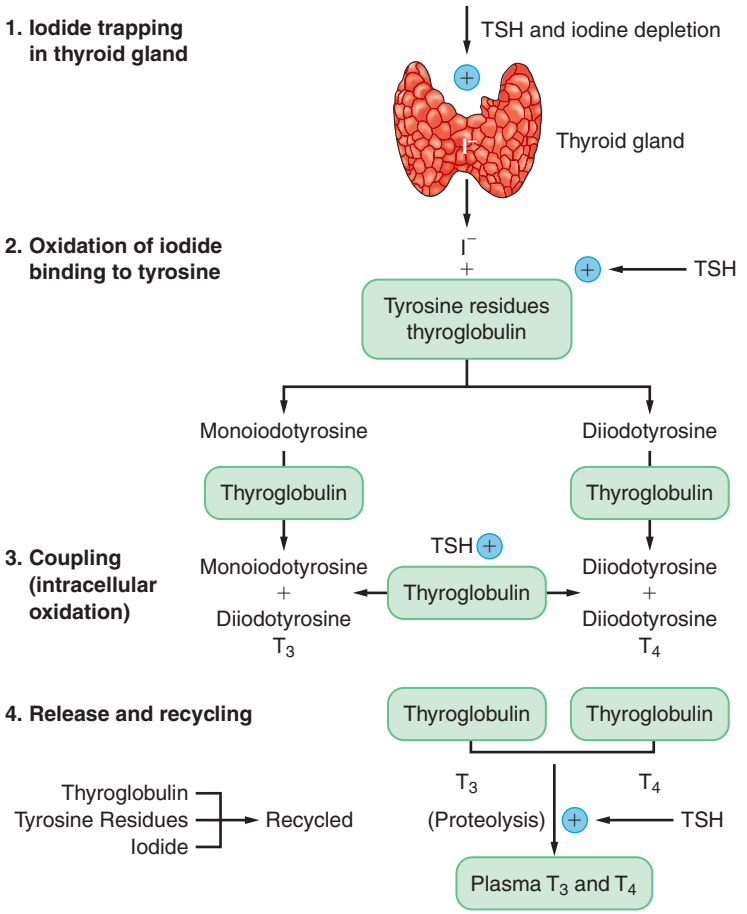


Thyroid hormone is necessary for both obligatory thermogenesis (the heat resulting from vital processes) and adaptive thermogenesis.

d) Thyroid function is regulated through thyrotropin (TSH), thyrotropin-releasing hormone (TRH) T3, T4, and iodine plasma levels (**Fig. 60-1**).

i) TSH is an anterior pituitary gland hormone, which enhances iodine uptake, T3/T4 production, and secretion by the thyroid gland.

FIGURE 60-1 Thyroid hormone biosynthesis consists of four stages: (1) organification, (2) binding, (3) coupling, and (4) release. TSH, thyroid-stimulating hormone; T3, triiodothyronine; T4, thyroxine.



From Schwartz JJ , Akhtar S , Rosenbaum SH . Endocrine function. In: Barash PG , Cahalan MK, Cullen BF , Stock MC, et al., eds. Clinical Anesthesia. 8th ed. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2018:1328 , with permission.

ii) TRH is a hormone produced by the hypothalamus, which promotes TSH secretion.
iii) High (or low) plasma levels of T3/T4 down (or up)-regulate the secretion of both TRH and TSH through down (or up)-regulation of T3/T4 receptors in the hypothalamus and anterior pituitary gland.
iv) High plasma iodine levels down-regulate T3/T4 production through down-regulation of iodine transport and sensitivity to TSH. The opposite occurs with low plasma iodine levels.



Hyperthyroidism is a state of thyroid gland hyperfunction, which presents with signs of hypermetabolism and sympathetic overstimulation.

e) Scattered between the follicles are parafollicular cells, which secrete calcitonin as a response to increased calcium plasma levels. Calcitonin decreases the levels of calcium and phosphorus by inhibiting osteoclast activity and by inhibiting renal reabsorption of calcium and phosphorus.

2) **Hyperthyroidism**
a) **Etiology**
i) Hyperthyroidism is a state of thyroid gland hyperfunction (goiter) with excessive secretion of thyroid hormones. Thyroid hyperfunction causes suppression of TSH levels.
b) **Causes**
i) Graves disease
(1) Multinodular diffuse autoimmune disease commonly affecting females between 20 and 40 years due to circulating stimulating IgG antibodies to the TSH thyroid receptor.
ii) Thyroid single adenoma or multinodular goiter
(1) Autonomic functioning thyroid tissue that is not down-regulated by increased thyroid hormone levels
iii) Subacute (de Quervain) thyroiditis
(1) Inflammation of the thyroid with flulike symptoms and thyroid pain that develops usually after an upper respiratory infection and is treated with anti-inflammatory medications.
iv) Ectopic thyroid tissue/neoplasm
v) Exogenous, usually iatrogenic, administration of thyroid hormone or iodine/iodine-rich medications (eg, amiodarone) in patients with chronically low iodine intake or nodular goiters[2,3]
c) **Prevalence**
i) Five to seven times more common in females.
ii) About 60%-80% of patients with hyperthyroidism have Graves disease.[4–6]
d) **Signs and symptoms (Table 60-1)**
i) Hypermetabolism and sympathetic overstimulation
e) **Associated findings**
i) Graves disease is associated with myasthenia gravis and exophthalmos ophthalmopathy.[3,4,6,7]
f) **Treatment**[3,6,8] can be pharmacologic, with radioactive iodine, or with surgical intervention. The goal of treatment is to improve the patient's hypermetabolic state (thyroid hyperfunction, sympathetic stimulation, anxiety) while limiting the incidence of hypothyroidism and other side effects.
i) Pharmacologic
(1) This is recommended for children and pregnant/breast-feeding women.
(2) β-Adrenergic antagonists (propranolol, metoprolol, esmolol). Control of adrenergic manifestations. Propranolol additionally reduces peripheral conversion of T4 to T3.
(3) Antithyroid agents
(a) Decrease thyroid hormone production and circulation.
(b) Thionamides (propylthiouracil [PTU] or methimazole). Block the production of T3, T4. PTU additionally prevents peripheral conversion of T4 to T3.

TABLE 60-1 Hyperthyroidism Signs

Symptoms	• Tachycardia • Palpitations/ectopic heart beats • Atrial fibrillation • Heat intolerance • Emotional lability • Exophthalmos ophthalmopathy: ○ Upper lid retraction ○ Increased intraocular pressure ○ Proptosis and ocular muscle weakness due to retro-orbital tissue inflammation and edema • Fine hand tremor
Signs	• Patients may have sympathetic overstimulation: ○ ↑HR ○ ↑Systemic blood pressure (SBP) ○ ↑Ejection fraction ○ ↑Left ventricular contractility ○ Atrial fibrillation, ectopic heart beats • Thrombocytopenia • Anemia • Hypercalcemia • Weight loss • Diarrhea • Skeletal muscle weakness • Anxiety

(c) Nonradioactive iodines (Lugol iodine, potassium iodine, sodium iodine). Reduce thyroid gland vascularity and block release of T3, T4. They are iodine substrates and should be given 2-3 hours after administration of thionamides to avoid a thyrotoxic crisis in a hyperthyroid patient.

(d) Glucocorticoids (dexamethasone, hydrocortisone). Inhibit peripheral conversion of T4 to T3 and correct subsequent adrenal suppression.

ii) Radioactive iodine reduces thyroid hormone production through thyroid gland destruction. It is not used for pregnant/breast-feeding women or patients with Graves ophthalmopathy (may deteriorate ophthalmopathy). A thionamide should be administered prior to radioactive iodine to avoid thyrotoxic crisis.

iii) Surgical thyroidectomy is performed in patients who are refractory to medication treatment, retrosternal or large goiter, thyroid cancer or lymphoma, and obstructive symptoms.

g) **Preoperative anesthesia evaluation**

i) Goals are to reduce thyroid hyperfunction, sympathetic stimulation, anxiety, and pain.

ii) All patients undergoing elective procedures should be euthyroid (normal thyroid function tests). This is usually clinically represented by a heart rate <85 bpm and no hand tremor before the procedure.

iii) Usually takes 1-8 weeks of treatment to become euthyroid.[6,7]

iv) Patients undergoing emergency surgery, the goal of treatment is to prevent thyroid storm.

(1) β-Adrenergic antagonist (usually propranolol), thionamide, and a stress dose of glucocorticoid should be given.

(2) Inorganic iodine should be given 2-3 hours after the thionamide.[6]



All patients with thyroid abnormalities undergoing elective procedures should be euthyroid (normal thyroid function tests). This is typically clinically represented by a heart rate <85 bpm and no hand tremor before the procedure.

v) Perioperative testing
   (1) Thyroid function tests (TSH and free T4 and T3), CBC, comprehensive metabolic panel.
   (2) An ECG to evaluate for ischemia, left ventricular hypertrophy, tachycardia, atrial fibrillation.
   (3) Chest x-ray (CXR) to evaluate for tracheal compression/deviation and for retrosternal thyroid extension should be ordered in large goiters.
      (a) A CT or MRI should be done to further evaluate the airway if there is >50% narrowing of the trachea, a substernal thyroid mass, or any signs/symptoms of airway compression.
   (4) An ENT consult or fiberoptic evaluation may be needed to document for vocal cord dysfunction or paralysis due to laryngeal nerve damage and pharyngeal displacement in patients with neoplasms or previous neck surgeries, especially in patients with a nonreassuring airway exam or obstruction symptoms.[7]



Patients undergoing emergency surgery should be given a β-blocker (usually propranolol), thionamide, and a stress dose of glucocorticoid.



Enlarged substernal thyroid is an anterior mediastinal mass.

vi) Preoperative medications[2,6,7]
   (1) All antithyroid and β-adrenergic antagonist medications should be continued through the morning of surgery.
      (a) Consider an esmolol IV infusion perioperatively to control tachycardia to <90 bpm.
      (b) Benzodiazepines can be useful as anxiolytics.
   (2) Regional anesthesia with a superficial and deep cervical block may be used for intraoperative sympathetic blockade and for postoperative analgesia.
   (3) Consider omitting epinephrine to avoid stimulation of an up-regulated sympathetic system.
   (4) If a patient has signs of tracheal compression, an awake fiberoptic bronchoscopy (FOB) or an inhalational induction, while maintaining spontaneous ventilation, may be indicated.



A substernal goiter can present with airway or cardiovascular compression, especially with cessation of spontaneous ventilation and muscle relaxation.

h) **Intraoperative treatment**[2,5,7,9]
   i) Maintain adequate anesthetic depth and pain control.
   ii) Complications to be prepared for include
      (1) Difficult ventilation/intubation
      (2) Cardiac arrhythmias
      (3) Sympathetic hyperactivity (hypertension and tachycardia)
      (4) Hyperthermia requiring active cooling and excessive airway pressures during surgical manipulations



Be sure to evaluate for signs of tracheal compression in patients with a thyroid mass.

   iii) Patient's head and neck should be positioned in a slight head-up tilt position.
      (1) Careful eye protection in patients with exophthalmos
   iv) Medications
      (1) Avoid adrenergic stimulation or parasympathetic blockade to avoid exacerbated responses from an up-regulated sympathetic system. Medications that cause hypertension or tachycardia are best avoided and when needed smaller doses should be used to avoid exacerbated elevations of blood pressure and heart rate.
         (a) Avoid ketamine, halothane, pancuronium, anticholinergics, indirect-acting vasopressors (eg, ephedrine), and epinephrine.
         (b) For hypotension, a decreased dose of a direct-acting vasopressor (eg, phenylephrine) can be used.
         (c) Esmolol and diltiazem can be used for tachycardia or supraventricular arrhythmias.



Consider awake FOB or inhalational induction for patients with signs of tracheal compression.

(2) Patients are often hypovolemic and vasodilated.
(a) Give all IV medications slowly and titrate to effect.
(3) Minimal alveolar concentration (MAC) of volatile anesthetics is not directly affected by thyroid state but is increased in hyperthermic patients (5% MAC increase for every degree above 37°C). Anesthetic requirements for hemodynamic control may also be higher due to sympathetic overstimulation.
(4) Muscle relaxants: Initial and maintenance doses of muscle relaxants should be reduced since subclinical myopathy may be present and reversal with an anticholinergic medication may be unwanted. Sugammadex use may be considered. If recurrent laryngeal nerve monitoring is employed, nondepolarizing neuromuscular blockers should be avoided.
(5) Recurrent laryngeal nerve monitoring can be achieved by regional technique allowing phonation or with a nerve-monitoring endotracheal tube. Video laryngoscopy is a useful tool to visualize alignment of a nerve-monitoring tube.
(6) Avoid salicylates, NSAIDs, or furosemide, which can increase levels of thyroid hormones by increasing fT3 and fT4.

v) Postoperative treatment
(1) Supplemental O_2
(2) Monitor patient's vital signs including temperature
(3) Provide adequate pain control

i) **Thyroid storm** is a life-threatening condition that develops rapidly in patients with poorly treated hyperthyroidism.

i) Thyroid storm is due to marked hypersensitivity to increased catecholamine secretion.
ii) Due to acute emotional or physical stress from conditions such as:
(1) Emergent surgery
(2) Surgical manipulations of an overactive thyroid gland
(3) Cardiovascular stress
(4) Trauma
(5) Infection
(6) Diabetic ketoacidosis
(7) Pregnancy-induced hypertension

iii) Symptoms that may present
(1) Anxiety
(2) Agitation
(3) Delirium
(4) Hyperthermia
(5) Hypertension
(6) Tachycardia
(7) Arrhythmias
(8) Nausea/vomiting
(9) Diffuse abdominal pain/obstruction
(10) Myocardial ischemia
(11) Congestive heart failure

iv) Thyroid storm most commonly presents 6-18 hours postoperatively.
v) Mortality rates is 10%-75%.
(1) Immediate diagnosis of thyroid storm is critical.
vi) Treatment and resuscitation
(1) Treatment should focus on ventilation and cardiovascular support.
(a) Supplemental O_2 to maximize oxygen delivery
(b) Maintaining a heart rate <100 bpm
(c) Decreasing temperature
(d) Treating the precipitating factor

(2) Since drug metabolism is slowed, consider reducing drug dosages.
(3) The following should be given promptly:
  (a) β-Adrenergic antagonist (esmolol infusion or propranolol 10-40 mg PO q4-6h)
  (b) Corticosteroids (hydrocortisone 50-100 mg IV q6h) to reduce peripheral T4 to T3 conversion and because relative adrenal deficiency may be present
  (c) Antithyroid medications (PTU 200-400 mg enterally q6h)
  (d) Sodium iodide 250 mg PO or IV q6h
(4) Invasive monitors such as arterial line and Foley catheter should be established. Central venous and pulmonary artery catheterization can be helpful with managing volume status, cardiac output, and continuous blood temperature but are usually not necessary.
(5) Consider ice packs or hypothermic blankets for cooling. Give acetaminophen for temperature control.[9,10] Meperidine may be administered to prevent shivering.
(6) Correct fluid deficits and metabolic abnormalities. Rehydrate with IV saline and glucose.
(7) Avoid salicylates or furosemide, which can increase levels of thyroid hormone.
(8) Thyroid hormone levels return to normal after 24-48 hours and recovery occurs after 1 week.
(9) If conventional therapy is not successful, consider direct removal of circulating thyroid hormones with cholestyramine,[11] plasmapheresis, and peritoneal dialysis.

3) **Hypothyroidism**
  a) **Etiology**
    i) Hypothyroidism is a state of thyroid gland hypofunction with decreased production of thyroid hormones.
    ii) Most common disorder of thyroid function.
  b) **Primary hypothyroidism**
    i) TSH is increased above the upper limit of normal.
    ii) The most common causes are iatrogenic.
      (1) Surgical resection
      (2) Iodine or amiodarone treatment
        (a) High iodine content causes a reduction in thyroid hormone synthesis and release.
      (3) Lithium use
        (a) Lithium blocks thyroid hormone synthesis and release.
      (4) Hashimoto disease
        (a) Autoimmune disease with lymphocytic infiltration of the thyroid gland
      (5) Other causes are thyroiditis (eg, de Quervain thyroiditis that initially can cause hyperthyroidism)
  c) **Secondary hypothyroidism (pituitary)**
    i) Thyroid hormone level is decreased due to decreased TSH
    ii) Due to any pituitary abnormality
      (1) Postpartum pituitary necrosis (Sheehan syndrome)
      (2) Expansive pituitary mass
      (3) Surgical resection
      (4) Intracranial radiation
  d) **Tertiary hypothyroidism (hypothalamic)**
    i) Thyroid hormone and TSH levels are decreased due to decreased levels of TRH.
    ii) Due to neoplasms and surgical resection.[8,12]
  e) Endemic goiter due to iodine deficiency
    i) Patients may be hypo, hyper, or euthyroid
    ii) May present with large neck mass, which may complicate airway management.

f) **Prevalence**
  i) Hypothyroidism is seen in 1%-2% of patients.
  ii) More common in females and older patients.[12]

g) **Signs and symptoms**
  i) Often insidious in onset
  ii) Decreased metabolism
  iii) Lethargy/fatigue
  iv) Depressed cardiac and respiratory function
    (1) Bradycardia
    (2) Reduced cardiac output
    (3) Reduced ventilatory response to hypoxia and hypercapnia
  v) Cold intolerance[5,6,13]

h) **Associated findings in untreated hypothyroidism**
  i) Mental slowness
  ii) Depression
  iii) Obesity
  iv) Obstructive sleep apnea
  v) Hyperlipidemia
  vi) Hypothermia
  vii) Delayed gastric emptying
  viii) Congestive heart failure
  ix) Pericardial/pleural effusions
  x) Nonpitting peripheral edema
  xi) Anemia
  xii) Adrenal insufficiency
  xiii) Hyponatremia
  xiv) Hashimoto disease
    (1) Associated with other autoimmune diseases including primary adrenal insufficiency and diabetes mellitus[5,6,8,12]

i) **Treatment**
  i) Pharmacologic
    (1) Thyroid replacement
      (a) Levothyroxine is most commonly used at doses of 1.7 µg/kg/d.
      (b) 6-8 weeks after initiation of treatment, T4 and TSH levels should be checked and doses modified accordingly.[8,12]
    (2) Drug interactions are common.
      (a) Iron, antacids, proton-pump inhibitors, cholestyramine, sevelamer, and orlistat interfere with levothyroxine absorption.
      (b) Phenytoin, phenobarbital, and carbamazepine decrease levothyroxine levels.[14]
    (3) Endemic goiter is treated with iodine supplementation

j) **Preoperative anesthesia evaluation**
  i) For patients with mild-moderate hypothyroidism, elective surgery is safe.
  ii) For patients with severe hypothyroidism, a euthyroid state (HR > 60) should be established prior to elective surgery.[6,7,9]
  iii) Hypothyroid patients with coronary artery disease (CAD) are at increased risk of myocardial ischemia during establishment of a euthyroid state due to a sudden increase in metabolic O_2 demands.
  iv) For patients undergoing emergency surgery with severe hypothyroidism, aggressiveness of therapy should be based on the patient's clinical status.
  v) The same labs should be obtained as for patients with hyperthyroidism.



For patients with hypothyroidism, the goals are to protect from respiratory depression, hypovolemia, hypoglycemia, hyponatremia, and hypothermia.

vi) Preoperative medications
(1) Treatment goals are to protect from respiratory depression, hypovolemia, hypoglycemia, hyponatremia, and hypothermia.
(2) Levothyroxine has a $t_{1/2}$ of 5-7 days but ideally should be continued perioperatively.
(3) Thyroid hormone medications are associated with wrong dose safety issues with conversions from µg to mg and adjusting IV to PO dosing.
(a) Oral dosing has 50%-80% bioavailability vs IV dosing with 100% bioavailability.[3]
(4) Patients may have heightened sensitivity to respiratory depression from sedatives.
(5) Since patients may have delayed gastric emptying, consider histamine H_2 antagonists, metoclopramide, rapid sequence induction, and nonparticulate antacid.[2,12,15]



Patient's with hypothyroidism may be more sensitive to respiratory depression from sedatives.

k) **Intraoperative treatment**[2,5–7]
i) Anticipate the potential for difficult intubation (generalized edema, tracheal compression/deviation) and anterior mediastinal mass effect in patients with goiter.
ii) Other complications include
(1) Exaggerated effects of cardio-depressant drugs
(2) Prolonged muscle relaxation with neuromuscular blockade
(3) Hypocarbia
(4) Hyponatremia
(5) Hypoglycemia
(6) Congestive heart failure
iii) If monitoring of the recurrent laryngeal nerve is employed, use video laryngoscopy to confirm endotracheal tube placement and avoid nondepolarizing neuromuscular blockers.
iv) For patients with refractory hypotension, consider adrenal insufficiency.
v) For patients with severe hypothyroidism, consider invasive monitoring.
vi) Preventing hypothermia
(1) Increase ambient OR temperature
(2) Warm IV fluids
(3) Use forced air warming devices



Myxedema coma is a life-threatening condition of severe hypothyroidism, which can be precipitated by sepsis, omission of thyroid replacement therapy, trauma, or surgery.

l) **Postoperative treatment**
i) Anesthesia recovery may be delayed.
ii) Ensure patients have adequate muscle strength and are normothermic prior to extubation.
iii) Nonopioid analgesics like ketorolac and peripheral and or central neuraxial blockade are preferable for postoperative pain management.[2,6,8,10]

4) **Myxedema coma**[6,9,10,12]
a) A life-threatening condition of severe hypothyroidism.
b) Sepsis, omission of thyroid replacement therapy, trauma, or surgery can precipitate myxedema in hypothyroid patients.
c) Mortality can be as high as 15%-60%.
d) Strong mortality predictors include
i) Low mean arterial pressure (MAP)
ii) Need for mechanical ventilation
iii) Sepsis

e) The diagnosis can be made by very low blood levels of free T4, T3 with or without TSH elevation.

f) **Signs and Symptoms**
   i) Impaired mental status
   ii) Seizures
   iii) Loss of deep tendon reflexes
   iv) Hypoventilation
   v) Hypothermia
   vi) Hyponatremia
      (1) Due to inappropriate antidiuretic hormone (ADH) secretion
   vii) Hypoglycemia
      (1) Due to concomitant adrenal insufficiency
   viii) Ileus
   ix) Effusions
   x) ECG abnormalities
      (1) Sinus bradycardia
      (2) Low voltage
      (3) Nonspecific ST and T wave changes
   xi) Congestive heart failure
      (1) Dilated cardiomyopathy
      (2) Pericardial effusion

g) **Treatment and resuscitation**
   i) Patients should be in an ICU setting.
      (1) Mechanical ventilation
      (2) Cardiovascular support
      (3) IV thyroid hormone
      (4) Steroids
      (5) Saline
      (6) Glucose for gradual correction of hyponatremia and hypoglycemia
   ii) A loading dose of levothyroxine 200-300 μg is given, followed up with a maintenance dose of 100 μg IV q24h.
   iii) Continuous ECG monitoring should be done.
   iv) Steroid replacement with hydrocortisone 100 mg every 8 hours should be given to prevent adrenal insufficiency.
   v) Patients become euthyroid after 3-5 days.
   vi) External warming for correction of hypothermia should be done gradually to avoid cardiovascular collapse due to sudden peripheral vasodilatation of a hypovolemic patient.



Complications after thyroid surgery include superior laryngeal nerve damage, recurrent laryngeal nerve damage, tracheal compression, and hypoparathyroidism.

5) **Complications after thyroid surgery**[6,9]

a) **Superior laryngeal nerve damage**
   i) Hoarseness
   ii) Loss of sensation above the vocals cords, which can place the patient at risk for aspiration.

b) **Recurrent laryngeal nerve damage**
   i) Unilateral nerve damage can present with hoarseness.
   ii) Bilateral can present with airway obstruction and aphonia.
   iii) To assess vocal cord function postoperatively, ask the patient to say "e."

c) **Tracheal compression**
   i) Can be secondary to hematoma or tracheomalacia.
   ii) May require surgical intervention and/or reintubation.

d) **Hypoparathyroidism**
   i) Due to inadvertent removal of the parathyroid glands.
   ii) Typically presents with hypocalcemia 24-96 hours postoperatively.
   iii) Patients may present with laryngospasm.
   iv) Treatment is with calcium carbonate/gluconate.
      (1) Magnesium levels should also be followed.

CHAPTER SUMMARY FOR THYROID DISEASE

Hyperthyroidism Perioperative Considerations	Defined as ↑T_4 and ↑T_3, ↓TSH. Presents with signs and symptoms of hypermetabolism and increased sympathetic activity. Treat with medications (β-blockers, thionamides, glucocorticoids, iodine) or surgical treatment. Patients should be euthyroid (HR < 85) prior to elective surgery. Thyroid storm can be life threatening.
Hypothyroidism Perioperative Considerations	Defined as ↓T4 and ↓T3, ↑TSH. Presents with signs and symptoms of hypothermia, respiratory depression, and lethargy. Treat with thyroid replacement. Severe hypothyroidism should be treated prior to elective surgery. Myxedema coma can be life threatening.
Thyroid Surgery Perioperative Considerations	Assess for tracheal compression and vocal cord dysfunction. Postoperatively patients can have superior laryngeal nerve damage (hoarseness, aspiration risk), recurrent laryngeal nerve damage (hoarseness, risk of complete obstruction), tracheal compression (hematoma), and hypoparathyroidism (low calcium).

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61 Adrenal Disorders

Teresa A. Mulaikal • Jessica Spellman

The adrenal gland is responsible for catecholamine, mineralocorticoid, glucocorticoid, and androgen production and secretion. Excess catecholamines from pheochromocytomas, excess aldosterone, or derangements in cortisol secretion can result in physiologically significant diseases with impact on perioperative management.

1) **Overview**
   a) **The adrenal glands** are small (4-5 g) triangular-shaped endocrine glands that are located at the superior portion of both kidneys.
      i) Synthesize, store, and release hormones that are essential for homeostasis.
      ii) Release hormones in conjunction with stress.
      iii) Divisions of the adrenal glands
         (1) Adrenal cortex produces three hormones.
            (a) Mineralocorticoids (aldosterone) are responsible for regulation of extracellular volume via urinary sodium and potassium exchange in the renin-angiotensin-aldosterone system.
            (b) Glucocorticoids (cortisol) are responsible for gluconeogenesis, elevation of blood glucose, promotion of glycogen storage, modulation of protein, lipid and carbohydrate metabolism, anti-inflammatory actions, and blood pressure maintenance.
            (c) Androgens: Adrenal androgens are responsible for sex organ development and changes that manifest during puberty.
         (2) Adrenal medulla produces catecholamines that function as hormones throughout the body.
            (a) Embryologically derived from neuroectodermal cells
            (b) Catecholamine release is under sympathetic nervous system control.
            (c) Secrete epinephrine (in greater amount) and norepinephrine (in smaller amount).
   b) **Disorders of hyperadrenalism or hypoadrenalism (Table 61-1)**
      i) Hyperadrenalism: Disorders that cause increased production of adrenal hormones
         (1) Increased catecholamine production
            (a) Pheochromocytoma
            (b) Paragangliomas
         (2) Increased aldosterone production
            (a) Primary aldosteronism (Conn syndrome)
            (b) Secondary aldosteronism: due to increased renin production
         (3) Increased cortisol production
            (a) Cushing syndrome: excess cortisol secretion
            (b) Cushing disease: excess cortisol production from anterior pituitary secretion of adrenocorticotropic hormone (ACTH)
      ii) Hypoadrenalism: Disorders that cause decreased production of adrenal hormones
         (1) Addison disease or primary adrenal insufficiency
         (2) Secondary adrenal insufficiency due to decreased ACTH production
         (3) Tertiary adrenal insufficiency

TABLE 61-1 Adrenal Disorders

Hyperadrenalism	Catecholamines	Pheochromocytoma Paragangliomas
	Aldosterone	Primary (Conn's) Secondary
	Cortisol	Cushing syndrome Cushing disease
Hypoadrenalism	Primary (Addison's)	
	Secondary	
	Tertiary	

2) **Hyperadrenalism**
   a) **Pheochromocytoma**
      i) Rare but significant disease
         (1) Incidence is <0.2% of patients with hypertension.
         (2) Sometimes associated with familial syndromes such as multiple endocrine neoplasia (MEN), von Hippel-Lindau, neurofibromatosis-1, and familial paragangliomas[1]
         (3) High perioperative mortality
            (a) Without preoperative treatment, mortality can be as high as 45%.
            (b) With appropriate preoperative treatment, mortality is <3%.
         (4) Surgical excision of the tumor is curative in >90% of patients.



"Classic" signs of pheochromocytoma: headache, palpitations, sweating, hypertension.

      ii) Pathophysiology
         (1) Pheochromocytomas are tumors that arise from chromaffin cells of the adrenal medulla.
         (2) Secrete epinephrine and norepinephrine, which leads to clinical manifestations.
         (3) Paragangliomas are extra-adrenal pheochromocytomas.
      iii) Signs and symptoms result from catecholamine release.
         (1) Hypertension, headache, palpitations, tremor, sweating, anxiety, hyperglycemia, and catecholamine-induced cardiomyopathy[2]
         (2) Signs and symptoms may be sustained or paroxysmal as a result of episodic increased secretion of catecholamines.
         (3) Paroxysmal catecholamine release may be precipitated by
            (a) Stimulation, including laryngoscopy, displacement of abdominal contents, pneumoperitoneum, tumor manipulation
            (b) Tyramine containing foods
            (c) Certain drugs such as tricyclic antidepressants and metoclopramide
            (d) Micturition, in the case of urinary bladder tumors[1]
      iv) Diagnosis
         (1) Excess catecholamines or metabolites (metanephrine, normetanephrine, or VMA) found in urine or plasma testing
            (a) Plasma-free metanephrines or urinary-fractionated metanephrines are the most sensitive tests for diagnosis and exclusion of pheochromocytoma.[1]
            (b) False positive elevated catecholamine levels may occur with physiologic stimuli.
            (c) True positive elevated catecholamines can be distinguished by the magnitude of elevation often two to three times the upper reference limit.[1,3]

(2) Imaging allows localization of the tumor.[1]
(a) CT imaging with and without contrast
(i) Contrast may have an effect on plasma catecholamine release
(ii) Prophylactic alpha and beta blockade (see below) prior to contrast administration is recommended.
(b) MRI is the preferred diagnostic modality for extra-adrenal tumors or tumors found in pregnant patients, children, or patients allergic to CT contrast.
(c) MIBG scanning allows for functional and anatomic coupling, which may be required for metastatic or multifocal tumors.



For patients with pheochromocytoma, α-blockade is initiated for 10-14 days before surgery along with intravascular volume expansion prior to beta blockade initiation.

v) Perioperative management
(1) Preoperative goals are to prevent the effects of catecholamines released by the tumor.
(a) α-Blockade
(i) Prevents the hypertensive effects of catecholamines[3]
(ii) α-Blockade is initiated for 10-14 days before surgery along with intravascular volume expansion to avoid excessive orthostatic hypotension.
(iii) α-Antagonists
1. Long-acting α-antagonists (irreversible, noncompetitive)
a. Phenoxybenzamine 10 mg BID, increasing every 2-3 days by 10-20 mg to maximum dose of 1 mg/kg
2. Short-acting α-antagonists (competitive)
a. Doxazosin increasing from 1 to 16 mg/d
b. Prazosin 2-5 mg two to three times a day
c. Terazosin 2-5 mg/d
(b) β-Blockade
(i) Patients with dysrhythmias or persistent tachycardia require β-blockade.
(ii) β-Blockade should only be added after several days of α-blockade to avoid the possibility of unopposed α-constriction resulting in severe hypertension, ischemia, and heart failure as well as other hypertension-related end-organ dysfunction.[1]
(iii) β-Antagonists
1. Propranolol 20-80 mg one to three times a day
2. Atenolol 12.5-25 mg two to three times a day
3. Metoprolol 25-50 mg three to four times a day[3]
(c) Other agents
(i) Labetalol is a nonselective α- and β-antagonist with predominant effects at β-receptors.
(ii) Calcium channel antagonists do not completely prevent hemodynamic instability.
(iii) α-Methyl-paratyrosine or metyrosine can be used to block catecholamine synthesis.
(d) Preoperative treatment endpoints
(i) Blood pressure control of <160/90 for more than 24 hours
(ii) Presence of orthostatic hypotension (not <80/45 standing)
(iii) Less than one premature ventricular contraction every 5 minutes



β-Blockade should only be added after several days of α-blockade to avoid the possibility of unopposed α-constriction resulting in severe hypertension, ischemia, and heart failure as well as other hypertension-related end-organ dysfunction.

(iv) Absence of ST segment changes and T wave inversions on ECG for 1 week[1]
(v) Hematocrit decrease of 5% to suggest the adequacy of intravascular volume expansion and satisfactory α-blockade[4]

vi) Intraoperative considerations
(1) Arterial blood pressure monitoring should be initiated prior to induction and intubation.
(2) Large bore IV access and/or central venous access should be established for administration of fluids and vasoactive infusions.
(3) Adequate monitoring for ischemia from catecholamine-induced acute, severe hypertension should be used.
(a) ECG
(b) Transesophageal echocardiography (TEE) in patients with cardiomyopathy or uninterpretable ECG
(4) Adequate depth of anesthesia should be achieved prior to laryngoscopy to minimize catecholamine responses. Consider use of antihypertensives to stabilize hemodynamics during induction.
(5) Acute increases in blood pressure can be treated with short-acting agents such as sodium nitroprusside, phentolamine, or nicardipine.
(6) Tachycardia can be treated with esmolol.
(7) Short-acting agents are preferred as hypotension may ensue following ligation of the venous drainage of the tumor.
(8) Magnesium infusions can be used intraoperatively though the data regarding stabilization of hemodynamics is controversial. Magnesium is a vasodilator that inhibits catecholamine release form the adrenal medulla and also has alpha blocking properties.
(9) If hypotension occurs, IV fluids and phenylephrine or norepinephrine infusions are the initial treatment.



Pheochromocytoma patients should be watched for hypotension and hypoglycemia postoperatively.

vii) Postoperative considerations
(1) Hypotension may result from an abrupt fall in circulating catecholamine levels combined with the residual effects of preoperative adrenergic blockade.
(2) Hypoglycemia may occur from hyperinsulinemia due to rebound insulin release after tumor excision.[1]
(3) Patients may need to be admitted to an intensive care unit postoperatively for hemodynamic monitoring.

b) **Adrenal pathology with increased aldosterone production**[2]
i) Two types
(1) Primary aldosteronism (Conn syndrome) from adrenal hyperplasia
(2) Secondary aldosteronism due to increased renin production
ii) Pathophysiology
(1) Increased aldosterone causes increased urinary sodium and potassium exchange that leads to sodium and water retention and loss of urinary potassium causing hypokalemia.
iii) Signs/symptoms of increased aldosterone
(1) Hypertension, extracellular volume expansion, weakness
iv) Perioperative management
(1) Normalization of intravascular fluid volume and electrolytes
(2) Preoperative sodium restriction
(3) Aldosterone antagonists (spironolactone)
(4) Potassium repletion

c) **Adrenal pathology with increased cortisol production (Cushing syndrome)**[2]
   i) Cortisol production is regulated by the hypothalamic-pituitary-adrenal (HPA) axis
      (1) Hypothalamic corticotropin-releasing hormone (CRH) stimulates anterior pituitary ACTH release, which stimulates the adrenal cortex to release cortisol.
      (2) Normal daily production of cortisol is 20 mg, with up to 300 mg produced in times of stress.
   ii) Excess cortisol production is caused by adrenocortical overproduction (pituitary microadenoma or neuroendocrine tumors producing excess ACTH, adrenal neoplasm) or exogenous administration of cortisol.
   iii) Signs/symptoms
      (1) Truncal obesity, hypertension, hyperglycemia, increased intravascular fluid volume, hypokalemia, osteoporosis, and muscle weakness
   iv) Perioperative management
      (1) Management of hypertension
      (2) Management of blood glucose
      (3) Normalization of intravascular fluid volume and electrolytes
      (4) When adrenalectomy is performed, glucocorticoid replacement must be initiated (see below).

3) **Hypoadrenalism**
   a) **Adrenal pathology with decreased cortisol production**
      i) Adrenal Insufficiency (AI)[5]
         (1) Three types
            (a) Primary adrenal insufficiency: Addison disease results from decreased production of adrenocortical hormones by adrenal cortex
               (i) Loss of adrenal tissue or atrophy
               (ii) Results from autoimmune destruction, infection (tuberculosis, advanced HIV, sepsis), malignancy, or hemorrhage
               (iii) Patients are deficient in both mineralocorticoids and glucocorticoids.
               (iv) Mineralocorticoid deficiency manifests as hyponatremia, hyperkalemia, acidosis.
               (v) Glucocorticoid deficiency manifests with hypoglycemia and depressed metabolic function.
            (b) Secondary adrenal insufficiency
               (i) Decreased anterior pituitary ACTH production
               (ii) Patients usually have intact mineralocorticoid function via the renin-angiotensin system.
            (c) Tertiary adrenal insufficiency
               (i) Most common form of AI
               (ii) Results from chronic exogenous corticosteroid therapy followed by withdrawal or decreased production of CRH by the hypothalamus due to tumor or radiation therapy
               (iii) Patients usually have intact mineralocorticoid function via the renin-angiotensin system.
         (2) Relative or functional adrenal insufficiency may be seen in critically ill patients or with surgical stress.
         (3) Signs/symptoms
            (a) Chronic AI is characterized by fatigue, weakness, anorexia, weight loss, and hyperkalemia.
            (b) Acute AI may result in refractory distributive or vasodilatory shock.



Adrenal insufficiency may be seen in critically ill patients.

Chronic adrenal insufficiency usually presents with fatigue, weakness, anorexia, weight loss, hypotension, and/or hyperkalemia.

(4) Treatment of AI
  (a) Mineralocorticoid and glucocorticoid administration
    (i) Patients suspected of acute AI should receive immediate steroid replacement with hydrocortisone (50 mg IV every 6 hours continued for 24 hours). Consider the addition of enteral fludrocortisone 50 µg daily.[6]
    (ii) Electrolyte correction
(5) Perioperative management[5]
  (a) All patients with AI who undergo a procedure should receive their normal daily corticosteroid dose orally or intravenously.
  (b) Patients with primary or secondary AI are often unable to increase endogenous cortisol production during stress and should receive supplemental corticosteroid therapy.
  (c) Patients on hydrocortisone doses <50 mg/d or equivalent do not require additional mineralocorticoid supplementation (see **Table 61-2** for steroid equivalencies among different glucocorticoid preparations).
  (d) Patients with tertiary AI who receive prednisone ≤5 mg/d (or equivalent) may have adequate HPA response to stress and usually do not require additional supplementation other than their normal daily dose.
  (e) Patients with tertiary AI on higher doses should receive supplemental corticosteroid therapy.
  (f) Preoperative testing of adrenal function in patients with tertiary AI is not required as it does not predict clinical outcome or development of adrenal crisis.[5,7]
  (g) Supplemental corticosteroid dosing
    (i) Corticosteroid doses should be administered based on the degree of surgical stress:

TABLE 61-2 Equivalent Doses of Glucocorticoid Preparations

Generic Name	Anti-inflammatory	Mineralocorticoid	Approximate Equivalent Dose (mg)
Short Acting			
Hydrocortisone	1.0	1.0	20.0
Cortisone	0.8	0.8	25.0
Prednisone	4.0	0.25	5.0
Prednisolone	4.0	0.25	5.0
Methylprednisolone	5.0	—	4.0
Intermediate Acting			
Triamcinolone	5.0	—	4.0
Long Acting			
Dexamethasone	30.0	—	0.75

Relative milligram comparisons with cortisol. The glucocorticoid and mineralocorticoid properties of cortisol are set as 1.0.
Adapted from Ogunnaike BO, Jones SB, Jones DB, et al. Anesthetic considerations for bariatric surgery. *Anesth Analg*. 2002;95:1793.

1. Minor procedures (hernia repair): hydrocortisone 25 mg or methylprednisolone 5 mg IV on the day of surgery only
2. Moderate procedures (open cholecystectomy): hydrocortisone 50-75 mg or methylprednisolone 10-15 mg IV on the day of surgery
3. Major procedures: hydrocortisone 100 or methylprednisolone 20 mg IV on the day of procedure

(ii) Excessive doses (>200-300 mg) or prolonged duration of glucocorticoids is of no benefit, and deleterious effects may include hyperglycemia, immunosuppression, accelerated protein catabolism, altered wound healing, volume overload, and acute psychosis.[5]

CHAPTER SUMMARY FOR ADRENAL DISORDERS

Adrenal Gland Functions	Synthesis and secretion of hormones including aldosterone, cortisol, catecholamines, and androgens
Adrenal Gland Disorders	May involve increased hormone production (pheochromocytoma, hyperaldosteronism, Cushing syndrome) or decreased hormone production (AI)
Pheochromocytoma	Marked catecholamine release resulting in severe hypertensive disease. Careful preoperative preparation, intraoperative monitoring and treatment, and postoperative monitoring of blood pressure and glucose are required
Adrenal Insufficiency (AI)	Most commonly results from exogenous steroid administration and suppression of the hypothalamic-pituitary-adrenal axis. During periods of septic shock or acute surgical stress, patients may require supplemental glucocorticoid and mineralocorticoid treatment[7]

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62 Pituitary Disorders

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The pituitary gland is responsible for the secretion of many different endocrine hormones. Disorders may be due to under- or oversecretion of one or more hormones, each with its characteristic clinical signs and symptoms. Pituitary surgery is most commonly performed for a pituitary adenoma, and the anesthesiologist must be aware of the intraoperative management of patients with specific pituitary disorders.

1) **Anatomy**
   a) The pituitary gland is located in the sella turcica at the base of the brain.
      i) Consists of two lobes with separate functions
   b) The gland sits outside of the blood-brain barrier.
   c) The lateral border of the sella turcica is the cavernous sinus, in which the internal carotid artery, and cranial nerves III, VI, V (maxillary and ophthalmic divisions), and VI traverse.
   d) Anterior to the pituitary is the optic chiasm.
2) **Physiology:** The pituitary gland is under the control of the hypothalamus and secretes many different hormones.[1]
   a) **Anterior pituitary (adenohypophysis)**
      i) Thyroid-stimulating hormone (TSH)
         (1) Stimulates the thyroid gland to release thyroid hormone
      ii) Follicle-stimulating hormone (FSH)
         (1) Stimulates estradiol production in females
      iii) Adrenocorticotropic hormone (ACTH)
         (1) Stimulates the secretion of cortisol and androgens
      iv) Prolactin
         (1) Stimulates lactation
      v) Growth hormone
         (1) Acts throughout the body and has a variety of metabolic and endocrine functions
         (2) Exerts much of its effects via insulinlike growth factors
      vi) Luteinizing hormone
         (1) Stimulates progesterone production and ovulation in females.
         (2) In males, it results in testosterone production and spermatogenesis.
   b) **Posterior pituitary (neurohypophysis):** The hormones released by the posterior pituitary are synthesized in the hypothalamus and released by the posterior pituitary.
      i) Vasopressin (ADH or antidiuretic hormone)[2]
         (1) Controls water retention in the kidney.
         (2) Causes systemic vasoconstriction in higher doses.
         (3) Increases the levels of plasma von Willebrand factor and factor VIII.
         (4) Synthetic analog is desmopressin (DDAVP).
      ii) Oxytocin
         (1) Contracts the uterus during parturition and aids in expressing milk during lactation

3) **Pituitary disorders**[2]
   a) **Hyposecretory**
      i) Involves decreased hormone secretion.
      ii) Causes include postpartum necrosis (Sheehan syndrome), trauma, radiation therapy, and compression from adjacent tumor.
      iii) Treatment is hormone replacement therapy, including cortisol, thyroid hormone, and possibly DDAVP.
   b) **Hypersecretory**
      i) Involve excess hormone secretion, most commonly caused by a pituitary adenoma (see below for specific hypersecretory disorders).
      ii) Treatment is surgical excision.
   c) **Mass effect**[3]
      i) More likely in nonfunctioning macroadenomas (>1 cm).
      ii) Commonly causes compression of optic nerves/chiasm leading to bitemporal hemianopia. Larger tumors can cause hydrocephalus and elevated ICP.



Endocrine abnormalities caused by a pituitary adenoma should be normalized prior to surgery whenever possible.

4) **Hypersecretory disorders of concern for anesthesiologists:**[2,4]
   a) **Signs/symptoms**
      i) Visual field disturbances, headaches, papilledema, nausea, vomiting, and physiologic changes due to hormone hypersecretion.
      ii) Laboratory evaluation should include ECG, electrolytes, and blood glucose.
   b) **Prolactinoma**
      i) Causes galactorrhea and amenorrhea but has few other physiologic implications
   c) **Cushing disease**
      i) Excess cortisol secretion by the adrenal gland due to excess secretion of ACTH by the pituitary[2]
      ii) Signs/symptoms
         (1) Weight gain accompanied by thickened facial fat (moon faces), hyperglycemia, hypertension, skeletal muscle wasting, and hypokalemia
   d) **Acromegaly**
      i) Results from excess secretion of growth hormone[2,4]
      ii) Signs/symptoms
         (1) Distorted facial anatomy including mandibular expansion and enlarged tongue
         (2) Airway changes such as enlarged epiglottis, overgrowth of cricoarytenoid joints, vocal cord dysfunction, enlargement of nasal turbinates, and subglottic narrowing
            (a) Airway management can be difficult in patients with acromegaly and appropriate precautions should be taken.
               (i) Signs concerning for difficult airway include a history of stridor, hoarseness, and dyspnea.
               (ii) Often necessitate smaller than expected endotracheal tubes due to subglottic narrowing.
               (iii) Patients are at risk for postoperative respiratory failure or airway obstruction.
         (3) Glucose intolerance
         (4) Abnormal collateral blood flow in the ulnar artery[4]
         (5) Systemic hypertension
         (6) Ischemic heart disease
         (7) Skeletal muscle weakness



Patients with acromegaly are at high risk for difficult airway.

5) **Pituitary surgery and anesthetic considerations**
   a) The most common surgical approach is transsphenoidal, but an open transcranial approach can be used.[3,4]
   b) **Preoperative considerations**
      i) A thorough history and physical should be performed, concentrating on possible signs/symptoms of hypersecretion or mass effect.
      ii) Laboratory tests should be reviewed, including serum glucose, electrolyte panels, and hormone levels. A type and cross should be performed.
      iii) Imaging studies should be performed and reviewed with the radiologist or surgeon to determine the extent of tumor invasion.
      iv) Cardiac considerations
         (1) ECG should be reviewed, looking for signs of left ventricular hypertrophy, right ventricular hypertrophy, and arrhythmias (especially in the presence of acromegaly).
         (2) If the hormone imbalance has been long-standing and/or the patient has symptoms of cardiac dysfunction, an echocardiogram may be helpful in determining possible ventricular dysfunction and/or valvular anomalies.
         (3) Cardiac function should be optimized prior to surgery.
   c) **Intraoperative considerations**
      i) Invasive monitoring
         (1) An arterial line is usually placed for monitoring BP and laboratory values.
         (2) A central venous catheter may be required if significant hemodynamic changes will require vasoactive infusions.
      ii) Difficult airway equipment should be available in all acromegalic patients and any patient whose exam is concerning for difficult ventilation or intubation.
      iii) A lumbar drain may be placed by the surgeon. Saline may be injected to aid in the visualization of the pituitary gland during surgery.
      iv) Intraoperative hypotension
         (1) May be due to inadequate cortisol production, especially in patients with Cushing disease or impaired cortisol production.
            (a) Cortisol should be replaced with hydrocortisone 100 mg IV in this situation.[5]
      v) Blood loss
         (1) Usually minimal although there is the potential for massive blood loss if the cavernous sinus is entered inadvertently
      vi) Venous air embolism is very uncommon but possible due to the semiseated position required for the procedure.
   d) **Postoperative**
      i) Diabetes insipidus (DI)
         (1) Characterized by excessive urine production and high plasma osmolality.
         (2) May occur intra- or postoperatively.
         (3) Usually due to reversible trauma to the posterior pituitary resulting in insufficient ADH production.
         (4) If DI is suspected, the diagnosis can be made by measuring serum electrolytes, plasma osmolality, and urine osmolality.[2]
            (a) Patients will have a low urine osmolality, high plasma osmolality, and hypernatremia.
         (5) Treatment
            (a) Monitor urine output and electrolytes.



Consider cortisol deficiency when faced with refractory intraoperative hypotension during pituitary surgery.

(b) Ensure adequate fluid replacement.
(c) Neurogenic ADH deficiency can be replaced with intranasal, intramuscular, or IV DDAVP.[2]
(d) Nephrogenic ADH deficiency can be ameliorated with chlorpropamide.[4]

CHAPTER SUMMARY FOR PITUITARY SURGERY

Pituitary Gland Function	Responsible for initiating secretion of many hormones
Pituitary Surgery	Typically transsphenoidal approach
Preoperative Considerations	Evaluate for visual field defects, cardiac disease, glucose intolerance, and difficult airway (especially in acromegaly)
Intraoperative Considerations	Blood loss usually not high, closely monitor glucose, consider cortisol deficiency with refractory hypotension
Postoperative Considerations	Diabetes insipidus possible

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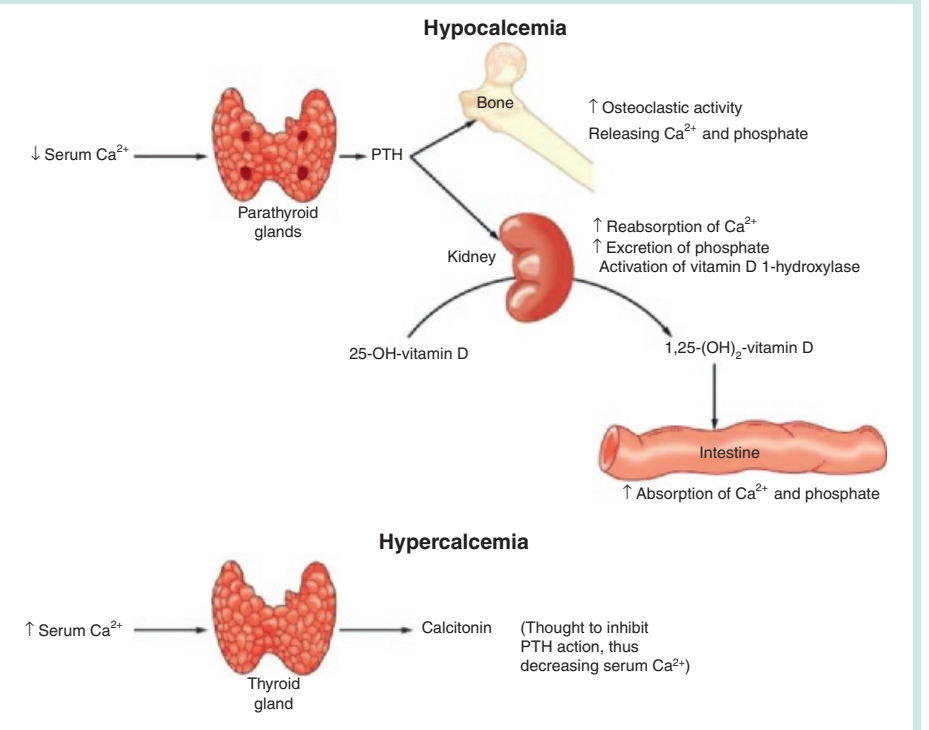
63 Parathyroid Disorders§

Janette D. McVey

The parathyroid glands regulate intravascular calcium concentrations. One common disorder of these glands is hyperparathyroidism, usually caused by an adenoma. Removal of the parathyroid glands can lead to hypocalcemia. Patients with parathyroid disease and calcium abnormalities must be properly assessed before administering anesthesia.

1) **Anatomy/normal function of the parathyroid glands**
   a) **Anatomy**
      i) Parathyroid glands are in the neck posterior to the thyroid gland.
      ii) Most people have four parathyroid glands.
   b) **Normal function**
      i) Parathyroid glands secrete parathyroid hormone (PTH), which regulates intravascular calcium concentrations (**Fig. 63-1**). PTH plays roles in bone resorption, renal distal tubular resorption, and synthesis of 1,25-dihydroxyvitamin D.
      ii) Calcium
         (1) Provides the foundation for bone and plays a role in skeletal muscle contraction.
         (2) It is a key neurotransmitter and plays a role in intracellular signaling and coagulation.
         (3) Exists as protein bound (to albumin) or ionized divalent cations.
            (a) The free, ionized calcium (Ca^{2+}) exerts physiologic effects.[1]
         (4) Extracellular (intravascular) calcium concentration depends on the albumin concentration.
            (a) Normal calcium levels = 8.6-10.4 mg/dL[2]
2) **Disorders of the parathyroid glands**
   a) **Hyperparathyroidism**
      i) Primary hyperparathyroidism
         (1) Most commonly caused by an adenoma.
         (2) Other causes include hyperplasia and parathyroid carcinoma (rare).
         (3) May also be associated with multiple endocrine neoplasia syndrome (MEN).
      ii) Secondary hyperparathyroidism
         (1) Causes
            (a) Chronic renal failure
            (b) Sarcoidosis
            (c) Multiple myeloma
            (d) Vitamin D intoxication
            (e) Other malignancy
      iii) Diagnosis of hyperparathyroidism
         (1) High serum calcium or elevated serum ionized calcium in the presence of high serum PTH.

FIGURE 63-1 Parathyroid Hormone (PTH) and Vitamin D Metabolism and Action



Reprinted with permission from McClatchey KD. *Clinical Laboratory Medicine*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2002 and reprinted with permission from Barash PG, Cullen BF, Stoelting RK, et al., eds. *Clinical Anesthesia*. 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2013:1332.

(a) Increased circulating PTH causes hypercalcemia (>10.4 mg/dL) (**Table 63-1**).

(2) Other laboratory abnormalities include:
- (a) Hypophosphatemia
- (b) Phosphaturia
- (c) Hyperchloremia
- (d) Hypercalciuria
- (e) Increased serum alkaline phosphatase

TABLE 63-1 Signs and Symptoms of Hypercalcemia

Organ System	Manifestation
Musculoskeletal	Profound muscle weakness, bone pain
Gastrointestinal	Nausea, decreased appetite, vomiting, constipation, increased gastric ulcer formation, pancreatitis
Neurologic	Depression, lethargy, impaired memory, and confusion
Cardiovascular	Decreased intravascular volume, hypertension, shortened QT intervals, myocardial calcification (long-term)
Renal	Polyuria, nephrolithiasis, proximal renal tubular acidosis

iv) Clinical manifestations of hypercalcemia
  (1) Nephrolithiasis
  (2) Polyuria/polydipsia
  (3) Hypovolemia
  (4) Hypertension
  (5) Confusion/psychosis
  (6) Peptic ulcer disease
  (7) Muscle weakness and atrophy



The cardiovascular manifestations of hypercalcemia include hypertension and shortened QT intervals; hypocalcemia may manifest as hypotension and a prolonged QT interval.

v) Medical treatment of hypercalcemia
  (1) Bisphosphonates (pamidronate, etidronate, clodronate)
    (a) Prevent calcium reabsorption
    (b) Rapid onset with long-lasting effects
  (2) Calcitonin
    (a) Decreases skeletal release of calcium.
    (b) Short duration limits use
  (3) Emergency correction of hypercalcemia
    (a) Necessary with serum concentrations >15 mg/dL[1]
    (b) Volume expansion with intravenous fluid (isotonic saline)
      (i) 4-6 L may be required in 24 hours.
  (4) Increased Ca^{2+} excretion with loop diuretics (not recommended in the absence of renal failure or heart failure).
    (a) Risks include:
      (i) Cardiac decompensation
      (ii) Hypophosphatemia
      (iii) Hypokalemia
      (iv) Hypomagnesemia
  (5) Dialysis (for patients with renal failure)
  (6) Glucocorticoids (useful for hypercalcemia associated with the excess administration or ingestion of vitamin D or with the endogenous overproduction of calcitriol)[2]
  (7) Denosumab: This is an option for patients with hypercalcemia that is refractory to bisphosphonates or in whom bisphosphonates are contraindicated due to severe renal impairment.[3]
    (a) Denosumab is a human monoclonal antibody for the treatment of osteoporosis that causes hypocalcemia as a side effect of the medication.
    (b) Most useful in treating hypercalcemia associated with malignancy.
  (8) Mithramycin
    (a) Inhibits osteoclast activity; can lower Ca^{2+} levels in 24-48 hours[4]
    (b) May have toxic side effects



At calcium levels >15 mg/dL, prolongation of the PR and QRS segments occurs and can result in heart block or bundle branch block. Bradycardia may also result.



Emergency treatment of hypercalcemia must be commenced before surgery if the level exceeds 15 mg/dL; hypokalemia and hypomagnesemia may result from this treatment.

vi) Surgical treatment for primary hyperparathyroidism
  (1) Surgical excision of the abnormal tissue is the definitive treatment.[5]
    (a) May be approached by bilateral cervical exploration or a more directed, unilateral approach.
    (b) PTH levels may need to be checked intraoperatively though peripheral blood draws to assess surgical resection.
  (2) Minimally invasive radiation guided
    (a) Involves localizing the parathyroid adenoma through injection of Tc99m sestamibi, localization with gamma probes, and removal through a small incision.[6]

(3) Open neck exploration
(a) Involves removal of the adenoma but potentially removal of most parathyroid tissue

vii) Anesthetic management of patient with primary hyperparathyroidism
(1) Most patients presenting with primary hyperparathyroidism will be undergoing parathyroidectomy.
(a) Preoperative
(i) Attention should be paid to potential dysfunction of the cardiac, neurologic, and renal systems.
(ii) Evaluation should include:
1. Electrolyte levels
2. ECG
3. Neurologic exam
(iii) Medical correction of extremely high serum Ca^{2+} levels may be necessary prior to elective surgery.
(b) Intraoperative
(i) No induction or maintenance agents are preferred over any others.
(ii) Careful titration of muscle relaxants with a nerve stimulator is necessary as patients may be more sensitive to their effects.[7]
(iii) Intraoperative PTH levels may need to be drawn.
(c) Postoperative
(i) Serum Ca^{2+} levels return to normal 1-3 days postoperatively.
1. Acute hypocalcemia (see above) may occur immediately postoperatively.
a. Occurs more commonly in those with severe bone deficits, leading to "hungry bone syndrome," a severe and prolonged hypocalcemia despite normal or even elevated levels of PTH due to a rapid increase of bone formation to bone resorption ratio which causes augmented requirements of calcium.[4]
2. If suspected, serum Ca^{2+} should be checked.



Postoperative hypocalcemia may occur after parathyroidectomy and manifest as neuromuscular irritability and laryngospasm, possibly leading to the need for reintubation.

(ii) Recurrent laryngeal nerve injury
1. The recurrent laryngeal nerve innervates the intrinsic muscles of the larynx.
a. Partial injury is more likely to affect abductor fibers.
2. Unilateral recurrent laryngeal nerve palsy.
a. Vocal cord is in paramedian position and cannot abduct or adduct.
b. Presents as hoarseness and stridor.
3. Bilateral recurrent laryngeal nerve palsy[2]
a. Results in inability to abduct or adduct the vocal cords, with both vocal cords in the paramedian position.
b. May be life threatening and require reintubation.

b) **Hypoparathyroidism**
i) Most commonly caused by removal of parathyroid during thyroid or parathyroid surgery, resulting in hypocalcemia (Ca^{2+} <8 mg/dL)[4]
ii) Other causes of hypocalcemia
(1) Hypoalbuminemia
(2) Chronic renal failure
(3) Rapid blood transfusion
(4) Acute pancreatitis
(5) Hypomagnesemia

iii) Clinical manifestations of hypocalcemia
  (1) Acute
    (a) Perioral paresthesia
    (b) Restlessness
    (c) Neuromuscular irritability
    (d) Laryngospasm
    (e) Stridor/apnea
    (f) Hypotension
  (2) Chronic
    (a) Fatigue.
    (b) Skeletal muscle cramps.
    (c) Decreased mentation.
    (d) QT interval may be prolonged.
    (e) Decreased inotropy.
  (3) Trousseau sign
    (a) Carpopedal spasm after 3 minutes of tourniquet-induced limb ischemia
  (4) Chvostek sign
    (a) Facial muscle twitching elicited by tapping the facial nerve at the angle of the mandible
  (5) Treatment of hypocalcemia
  (6) Acute treatment with infusion of calcium chloride or calcium gluconate
  (7) Chronic treatment with calcium supplements, vitamin D, and phosphate binders
  (8) Replacement of other electrolyte deficits



Position osteopenic patients carefully to avoid pathologic bone fractures that may occur during patient transfers.

CHAPTER SUMMARY FOR PARATHYROID DISORDERS

Causes of Hyperparathyroidism	Parathyroid adenoma, rarely associated with MEN syndrome
Manifestations	Hypercalcemia
Anesthetic Management	
Preoperative	May need to correct hypercalcemia with IV fluid and/or loop diuretics Assess cardiac, neurologic, and renal function
Intraoperative	May be sensitive to neuromuscular blockade
Postoperative	Recurrent laryngeal nerve injury possible and may result in airway compromise Acute hypocalcemia can present as laryngospasm, apnea, restlessness, and neuromuscular irritability

MEN, multiple endocrine neoplasia; IV, intravenous.

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64 Obesity

Amanda Kirzner • Andrea J. Traynor

The growing obesity epidemic represents a clinical challenge for anesthesiologists. According to the CDC, the obesity rate in America was 39.8% in 2016 and is still sharply rising. Obesity is associated with many pathophysiological changes that affect the outcome as well as the morbidity and mortality of anesthesia and surgery. Obesity increases the risk of many diseases, including diabetes mellitus (DM), coronary artery disease (CAD), obstructive sleep apnea (OSA), hypertension (HTN), and degenerative joint disease. The anesthesiologist must understand the unique challenges, both practical and physiologic, faced when taking care of these patients.

1) **Introduction**
   a) **Ideal body weight (IBW)**
      i) The weight associated with the lowest mortality for a given height
      ii) IBW can be calculated by the formula[1]

      IBW (kg) = height (cm) − *x*, where *x* = 100 for men and 105 for woman

   b) **Body mass index (BMI)**
      i) The BMI is the weight in kg divided by the height in meters squared (wt/ht^2).
      ii) The CDC classifies BMI as represented in **Table 64-1**.
   c) **Epidemiology**
      i) The prevalence of obesity in 2015-2016 was 35.7% among young adults aged 20-39 years, 42.8% among middle-aged adults aged 40-59 years, and 41.0% among older adults aged 60 and older.[2]
      ii) The rate of obesity varies with geographic region, with the Southern United States having the highest prevalence.
2) **Coexisting diseases**: Obesity is associated with many physiologic derangements and can lead to multiple coexisting diseases, all of which should be optimized prior to elective surgery and managed appropriately intraoperatively.
   a) **Diabetes mellitus**
      i) The incidence of DM is 21% in patients weighing >300 lb.[3]
      ii) Patients with DM have a higher incidence of CAD, kidney disease, and cerebrovascular disease.
      iii) Patients with DM have decreased gastric emptying and are at a higher risk for aspiration during anesthesia.
      iv) DM increases the risk of surgical complications such as poor wound healing and infection.
   b) **Arterial HTN**
      i) The incidence of HTN in patients weighing >300 lb is 44%.[3]
      ii) HTN in the morbidly obese is due to increased blood volume, which in turn increases preload, afterload, and cardiac output eventually causing left ventricular hypertrophy and failure.[4]



The American Association of Clinical Endocrinologists (AACE) and the American College of Endocrinology (ACE) have replaced the word "obesity" with "adiposity-based chronic disease (ABCD)" because the term "obesity" has a negative connotation in American society.

READ MORE
Diabetes mellitus, Chapter 59
Essential hypertension, Chapter 45
Obstructive sleep apnea, Chapter 54

TABLE 64-1 Centers for Disease Control Classification of Body Mass Index (BMI)

Classification	BMI Low	BMI High
Underweight	—	18.5
Normal	18.5	25
Overweight	25.0	30
Obese Class 1	30.0	35
Obese Class 2	35	40
Obese Class 3[a]	40	—

[a]Class 3 obesity is sometimes categorized as "extreme" or "severe" obesity. From Adult Obesity Facts, Overweight & Obesity, CDC. *Centers for Disease Control and Prevention*. www.cdc.gov/obesity/data/adult.html. Accessed April 22, 2019.

c) **Coronary artery disease**
   i) Obesity accelerates atherosclerosis, which can increase the incidence of ischemia and the risk of a perioperative or postoperative MI.
   ii) Obesity also increases the incidence of intraoperative arrhythmias due to ischemic heart disease, electrolyte imbalances, and sleep apnea.[4]

d) **Obstructive sleep apnea (OSA)**
   i) Pharyngeal airway tissue enlargement is extremely common in the obese population and can cause OSA.
   ii) It is estimated that 60%-90% of patients with OSA are obese and most cases are undiagnosed.[5]
   iii) A history of snoring is highly suspicious for OSA.[5]
   iv) The STOP BANG questionnaire should be used as a screening tool in this high-risk population.
   v) OSA is characterized by repetitive episodes of upper airway obstruction which inevitably result in chronic hypoxemia and desaturation.
   vi) Careful precaution with sedatives, ventilation, and airway technique is advised.
   vii) Regional anesthesia should be considered if surgery permits, as it decreases incidence of morbidity and mortality when compared to general anesthesia in the OSA patient.[4]
   viii) Obesity hypoventilation syndrome (OHS)[6]
      (1) 90% of people with OHS also have OSA.
      (2) In contrast to obese individuals, who have an increased central respiratory drive to compensate for increased ventilator requirements, OHS patients have a blunted central respiratory drive to both hypoxia and hypercapnia.[4]
      (3) Characterized by hypercarbia, hypoxemia, polycythemia, hypersomnolence, pulmonary HTN, and biventricular failure.

e) **Gastroesophageal reflux disease**
   i) The increased intra-abdominal pressure due to the upward displacement of the stomach in morbidly obese individuals is often considered to increase the risk for gastroesophageal reflux although this is controversial.
   ii) Aspiration precautions, such as administration of a nonparticulate antacid, or a rapid sequence induction should be strongly considered.



Patients with obesity hypoventilation syndrome are at extremely high risk for apnea, acidosis, respiratory failure, and potentially right heart failure either postoperatively or with minimal sedation (eg, monitored anesthesia care cases).

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Gastrointestinal and hepatobiliary surgery, Chapter 116
Arthritis, Chapter 69

f) **Osteoarthritis**
   i) Increased weight places stress on joints and leads to the development of osteoarthritis. Many patients have chronic back and/or joint pain.
   ii) Proper padding of pressure points and positioning should be done in order to avoid exacerbation of symptoms after surgical procedures.

g) **Thromboembolic disease**
   i) Obesity increases the risk of thromboembolic events.
   ii) The increased incidence is likely due to polycythemia, increased abdominal pressure, and venous stasis from immobility.[6]
   iii) Early ambulation and thromboprophylaxis are paramount in the obese surgical patient.

3) **Physiologic changes (Table 64-2)**

a) **Increased airway tissue and neck circumference**
   i) The anatomy of the obese patient greatly increases the odds of difficult ventilation and intubation.
   ii) Emergency airway equipment, including LMAs, oral airways, bougies, glidescopes, and fiberoptic scopes, must be available, and a thorough history of prior intubations should be obtained.
   iii) If there is any history of a difficult intubation, or great concern about airway management, an awake fiberoptic intubation should be considered.[8]

b) **Increased blood flow to adipose tissues**
   i) In the obese patient, blood flow increases significantly, with 20-30 mL blood flow/1 kg adipose tissue.[3]
   ii) This leads to increased cardiac output and blood volume, which increases myocardial oxygen demand and metabolic rate, increasing CO_2 and potentially leading to acidosis.[8]



Obese patients may not tolerate the supine position due to extreme decreases in FRC.

c) **Respiratory**
   i) Respiratory physiology changes dramatically in the obese population, making these patients at high risk for hypoxemia and possibly death.
   ii) Functional residual capacity (FRC) is reduced.[3]
      (1) The reduction in lung volumes, from the upward pressure of the abdominal contents, may be so severe that normal breathing approaches closing capacity and leads to ventilation-perfusion (V/Q) mismatch.[3]
      (2) The supine position is often not well tolerated due to extreme *V/Q* mismatch.
   iii) Obese patients have decreased respiratory compliance due to chest wall adipose tissue and often exhibit a restrictive pattern of breathing.
   iv) Chronic hypoxemia can lead to pulmonary HTN and right ventricular failure.
   v) Most respiratory issues, such as v/q mismatch, resolve with weight loss.

d) **Gastrointestinal (see Table 64-2)**

e) **Endocrine**
   i) Severe obesity leads to the "metabolic syndrome," which includes abdominal obesity, atherogenic dyslipidemia, hypertension, insulin resistance, a proinflammatory state, and a prothrombotic state.[8]

4) **The perioperative approach to the obese patient**

a) **Preoperative consideration**
   i) Surgical procedures
      (1) Obese patients can present to the operating room for any type of surgical procedure, both elective and emergent.
      (2) Procedures performed to treat obesity include bariatric procedures such as gastric bypass, gastric banding, and sleeve gastrectomies.[9]

TABLE 64-2 Physiologic Derangements in Obese Patients and Their Anesthetic Implications

Organ System	Physiologic Derangements	Anesthetic Implications
Respiratory	↑ oxygen consumption	Rapid arterial desaturation
	↑ pharyngeal tissue	↑ risk of OSA
	possible ↓ ventilatory response to CO_2	↑ sensitivity to narcotics
	↓ FRC and ERV	• Rapid arterial desaturation, ventilation-perfusion (*VQ*) abnormalities, possibly causing right to left shunt and pulmonary HTN • Assumption of the supine position can worsen *VQ* mismatch
Cardiovascular	↑ blood volume and cardiac output	LVH
	↑ risk for CAD	May need cardiac evaluation to determine presence of CAD
	↑ risk for arterial HTN	
Endocrine	Impaired glucose tolerance leading to DM	Require intraoperative monitoring and control of glucose
Gastrointestinal	↑ intra-abdominal pressure	↑ Risk for acid aspiration
	↑ gastric volume	Consider premedication to reduce risk of pulmonary injury if aspiration occurs
Airway	↑ neck circumference	Possible increased risk of difficult airway
	↑ Mallampati score	

OSA, obstructive sleep apnea; LVH, left ventricular hypertrophy; CAD, coronary artery disease; HTN, hypertension; DM, diabetes mellitus.

(3) Other common procedures include uvulopalatopharyngoplasty or orthopedic surgeries due to joint degeneration.

ii) Preoperative assessment
   (1) Should focus on comorbid conditions, with special attention to respiration, OSA, and airway assessment.
   (2) Preoperative laboratory evaluations will vary with the patient and procedure but may include:
      (a) Chemistry panel to evaluate electrolytes, blood glucose, and kidney function.
      (b) ECG to evaluate for ventricular (left or right) hypertrophy and evidence of ischemia.
      (c) CBC to evaluate for polycythemia.
      (d) A room air ABG to detect chronic hypoxemia.
      (e) Other studies such as cardiac stress tests and more extensive laboratory analysis may be indicated but are not routine.

iii) Airway assessment
   (1) It is controversial whether airway management is more difficult in obese patients.[7,8]
   (2) Risk factors for difficult intubation in obese patients are large neck circumference and high Mallampati score.[7,8]
   (3) With a neck circumference of 60 cm or greater, the incidence of problematic intubation in one study was 35%.[7]
   (4) Equipment to aid in management of a difficult airway should always be available, and plans for difficult intubation should be made in high-risk patients.

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Epidural and combined spinal epidural anesthesia, Chapter 42

b) **Regional anesthesia**
   i) Obese patients may benefit from regional anesthesia due to minimal airway intervention, the avoidance of cardiopulmonary depressant agents, improved postoperative pain control, and decreased narcotic requirements.[4]
   ii) A recent study showed that the odds ratio of failed regional anesthesia in obese patients was 1.6, presumably due to technical difficulties.[4]
   iii) Despite the fact that the regional failure rate is low, the anesthesiologist must always be prepared for failure and a potentially difficult airway in the obese patient, so should have emergency airway equipment on standby.
   iv) Decreased cerebral spinal fluid volume, high intra-abdominal pressure, and altered respiratory physiology may increase the risk of high spinal anesthesia.
      (1) Careful monitoring is necessary in obese patients when spinal anesthesia is administered.
      (2) A combined spinal epidural with a lower than average spinal dose may be considered when the optimal spinal anesthetic dose is unknown.



While most modern OR beds and equipment are designed with a high weight limit, it is important to verify the weight limits of the OR table prior to caring for a morbidly obese patient.

Intubation in obese individuals can be facilitated by specially designed elevation pillows or placement of blankets under the head and shoulders until the sternal notch and external auditory meatus are aligned.[10]

c) **Preparing the OR for a morbidly obese patient**
   i) OR table and gurneys
      (1) Check the weight limits of the operating room table and gurneys.
      (2) Ensure that the table is wide enough to accommodate the patient. Table extensions or a bariatric table may be necessary.[9]
      (3) If special positions (eg, Trendelenburg position) will be required, one must ensure that the table is able to accommodate this position with the increased weight of the patient and that the patient is strapped on well, since they tend to slip during position changes.
      (4) Use of a bean bag should be considered, in order to facilitate moving the patient off the operating table after surgery.[10]

d) **Intraoperative management**
   i) Positioning
      (1) Airway management
         (a) Intubation is often easier when the patient is placed on a "ramp" to align the sternal notch and external auditory meatus.[8]
         (b) If a "ramp," often made from a stack of towels or blankets, is used to facilitate airway management, one must ensure that there are not pressure-inducing bumps in the bedding.
         (c) The arms must be at the same level as the patient's shoulders to avoid stretch injury, which is caused by extreme abduction of the arms, which stretches the roots of the brachial plexus.
         (d) For long cases, it is prudent to remove the material used for the ramp after intubation is achieved.
      (2) Padding
         (a) Obesity places patients at risk of developing decubitus ulcers.
         (b) The heels, buttocks, and shoulders are at particular risk and should be checked and padded.[3]
   ii) Preoxygenation
      (1) FRC approaches closing volume in obese individuals, making them an increased risk for hypoxemia on assumption of the supine position and induction of anesthesia.

READ MORE
Endotracheal intubation, Chapter 162

(2) Adequate preoxygenation to adequately denitrogenate the lungs is required and can be invaluable in cases of difficult airway.

iii) Aspiration precautions
  (1) The obese patient is at risk for gastric acid aspiration and appropriate precautions, including premedication with a nonparticulate antacid and H_2 receptor antagonist are usually necessary.
  (2) A rapid sequence induction should be strongly considered but may need to be modified if the patient experiences hypoxia on induction of anesthesia.



When caring for an obese patient, ensure that the blood pressure cuff is adequately sized. Too small a cuff will overestimate blood pressure.

iv) IV access
  (1) IV access can be difficult in the presence of excess adipose tissue.
  (2) If adequate peripheral IV access is not possible, the patient may require a central venous catheter.

v) Monitoring
  (1) BP management
    (a) Many patients present with arterial HTN, which must be managed intraoperatively.
    (b) A large blood pressure cuff should be used. The measurement can be overestimated with a cuff that is too small for the patient.
    (c) If noninvasive blood pressure measurement is inaccurate or impossible, an arterial line should be placed.
  (2) Cardiac considerations
    (a) LVH and CAD may be present.
    (b) Invasive monitoring of CVP or use of TEE is not routine but may be considered based on the patient's underlying cardiac disease and the surgical procedure.

READ MORE
Ischemic heart disease, Chapter 98

vi) Pharmacology[10]
  (1) Can be complex due to changes in elimination and enzymes (eg, pseudocholinesterase) which occur as body weight increases.
  (2) Obese patients have an increased volume of distribution for lipid-soluble drugs (eg, benzodiazepines).
    (a) Initial dosing should be based on actual body weight.
    (b) Clearance may be the same or increased with obesity, but elimination may be prolonged due to the large volume of distribution.[10]
      (i) Extreme caution should be taken with any lipid-soluble sedative/hypnotic drug administered to an obese individual at risk for hypoventilation (ie, those with OSA).



Obese patients have both an increased risk of hypoventilation and decreased elimination of benzodiazepine drugs, and extreme care should be taken with their administration.

  (3) For hydrophilic drugs (eg, muscle relaxants), the volume of distribution is unchanged, and dosing should be based on IBW.[10]
    (a) Pseudocholinesterase is increased, and therefore, succinylcholine should be dosed closer to total body weight.
  (4) Volatile anesthetics
    (a) The newer, more water-soluble volatile anesthetics (sevoflurane, desflurane) have very negligible differences in emergence times for obese vs lean individuals.[10]
    (b) More lipid-soluble anesthetics have much greater differences in emergence between obese and lean patients.

(5) Drugs with extensive extrahepatic metabolism (eg, remifentanil) appear to have similar pharmacokinetics in obese and lean individuals.[10]

e) **Anesthetic emergence**

i) Obese patients are at high risk of airway obstruction on emergence and should be extubated awake with intact airway reflexes.

ii) Due to altered respiratory mechanics, it may be helpful to place the patient semiupright prior to extubation.

iii) Careful postoperative respiratory monitoring is required.

iv) Patients with OSA often require 3+ hours of observation for apnea episodes in the PACU. If these patients are on CPAP at home, they are encouraged to bring it and use it postoperatively.



The pharmacokinetics of remifentanil are not significantly altered in obese patients.

5) **Postoperative pain management**

a) **Risk of airway obstruction**

i) Any patient with a history suspicious for OSA is at risk for postoperative airway obstruction.

ii) At the end of the procedure, patients should be awake with muscle relaxants fully reversed on extubation.

iii) An oro- or nasopharyngeal airway is helpful to prevent airway obstruction.

iv) The patient should be transported with oxygen, preferably via a face mask.

v) Retaining the endotracheal tube for postoperative ventilation may be preferable to extubation at the end of the procedure, depending on the risk of airway difficulty, length of surgery, and fluid shifts.

b) **Sensitivity to opioid medications**

i) Any patient with a history suspicious for OSA is at risk for a decreased ventilatory response to increasing CO_2.

ii) Respiratory monitoring should be considered when postoperative pain control includes use of opioids.[5]

c) **Neuraxial analgesia**

i) Obese patients may benefit from neuraxial opioid analgesia.

ii) However, careful monitoring is imperative due to the risk of delayed respiratory depression.

CHAPTER SUMMARY FOR OBESITY

Definition	Obesity class 1 = BMI 30-35; extreme obesity = BMI > 40.
Coexisting Diseases	Diabetes, HTN, OSA, GERD, osteoarthritis, thromboembolic disease, cardiac disease. All should be optimized prior to elective surgery.
OR Preparation	May require special beds and other equipment. Noninvasive BP may be inaccurate. Pressure points must be padded.
Anesthetic Considerations	Airway management may be improved by aligning the sternal notch with the external auditory meatus. Patients are at risk for hypoxemia in the supine position. IV access may be difficult. Coexisting diseases may necessitate additional monitoring. Patients are at risk for airway obstruction on extubation and in PACU.
Drug Dosing	Patients may be sensitive to lipid-soluble sedative-hypnotic drugs (such as benzodiazepines).

BMI, body mass index; HTN, hypertension; OSA, obstructive sleep apnea; GERD, gastroesophageal reflux disease.

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65 Part G: Hematology and Oncology
Red Blood Cell Diseases

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Anemia is a deficiency in red blood cell (RBC) concentration caused by decreased production or peripheral loss. Perioperative anemia is associated with increased morbidity and mortality.[1] Preoperative hemoglobin (Hgb) levels of <8 g/dL are associated with a 16-fold increase in mortality rate as compared with nonanemic patients.[2,3] Blood transfusions due to perioperative anemia increase health care resource utilization and expose patients to increased length of stay, need for ICU admission, infection risk, and immunosuppressive effects.[2] Hemoglobinopathies include sickle cell disease (SCD), thalassemias, and methemoglobinemia. SCD is caused by an inherited structural disorder of the hemoglobin β-globin chain with a wide range of severity and variable speed of progression.

Anemia

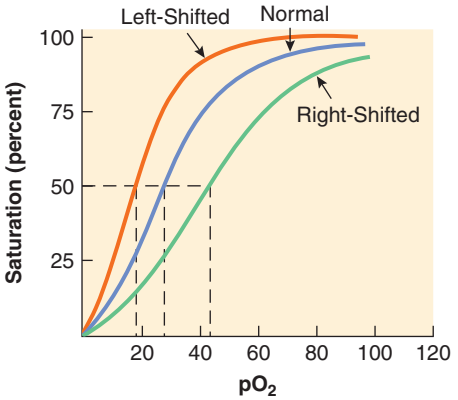
1) **Definition**
   a) The key characteristic of anemia is low oxygen transport capacity of the blood, either due to decreased RBC number or function.
   b) There is no absolute value to define anemia because the RBC number may vary with different physiologic states.
   c) However, anemia is generally defined in adults as a hemoglobin level <12.5 g/dL in males and <11.5 g/dL in females.[4]



↑ 2,3-DPG, acidosis, and hyperthermia shift the oxyhemoglobin dissociation curve to the right, which facilitates tissue oxygen delivery.

2) **The oxyhemoglobin dissociation curve**
   a) The relationship between the arterial oxygen tension and the saturation of hemoglobin molecules with oxygen is described by the oxyhemoglobin dissociation curve (**Fig. 65-1**).
   b) The P50 value is the arterial oxygen concentration at which hemoglobin is 50% saturated with oxygen.
   c) A shift to the left occurs in the presence of alkalosis, hypothermia, decreased 2,3-diphosphoglycerate (DPG), and high concentrations of fetal hemoglobin. It indicates that oxygen is more tightly bound and there is less unloading of oxygen in the tissues.
   d) A right shift occurs in the presence of acidosis, hyperthermia, and increased 2,3-DPG. Hemoglobin is less tightly bound to oxygen, which facilitates tissue oxygen delivery.
3) **Causes of anemia**
   a) **Blood loss**
      i) May be chronic or acute
      ii) If blood loss is the suspected cause of anemia, the source of bleeding should be ascertained.
   b) **Bone marrow failure**
      i) Drug toxicity from medications such as myelosuppressive drugs, chloramphenicol, and sulfonamides
      ii) Bone marrow suppression due to neoplasms, infections, irradiation, toxins, or idiopathic causes

FIGURE 65-1 The Oxyhemoglobin Dissociation Curve



The relationship between arterial saturation of hemoglobin and oxygen tension is represented by the sigmoid-shaped oxyhemoglobin dissociation curve. When the curve is left shifted, the hemoglobin molecule binds oxygen more tightly.
Reproduced with permission from Greenberg SB, Murphy GS, Vender JS. Standard monitoring techniques. In: Barash PG, Cullen BF, Stoelting RK, et al., eds. *Clinical Anesthesia*. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009:701.

c) **Hematopoietic input deficiencies**
   i) Deficiencies of the building blocks of hemoglobin such as iron, folate, and B_{12}
   ii) Thalassemias
      (1) Genetic diseases that result in deficiency in the synthesis of normal hemoglobin, discussed later in the chapter
   iii) Anemia of chronic disease
      (1) Occurs with chronic inflammation, tissue injury, infections, and cancer
      (2) Due to inadequate iron delivery to the bone marrow despite normal or increased iron stores

d) **Peripheral RBC destruction**
   i) Autoimmune/antibody mediated
      (1) Causes include drugs and diseases such as systemic lupus erythematosus, non-Hodgkin lymphoma, chronic lymphocytic disease leukemia
   ii) Nonimmune mediated
      (1) Causes include microangiopathic hemolytic anemia, hypersplenism, prosthetic heart valves, infections, burns, drugs, toxins.
   iii) Hemolysis due to congenital defects
      (1) Thalassemias, sickle cell disease, hereditary spherocytosis, elliptocytosis, enzymopathies (G6PD/G6PDH deficiency)



Anemia decreases oxygen-carrying capacity and the physiologic response is to preserve oxygen delivery to the tissues.

e) **Defects in RBC membrane proteins or components**
   i) Causes include paroxysmal nocturnal hemoglobinemia, Wilson disease

f) Hemodilution due to fluid resuscitation

4) **Physiologic changes associated with anemia**
   a) **Severity and clinical manifestations of anemia are related to the speed of onset.**

TABLE 65-1 Compensatory Response to Anemia

Anemia	Compensatory Response
Acute	↑HR, ↑SV, ↑O_2 extraction
Chronic	↑2,3-DPG, ↑CO, ↑PV, ↑P-50, ↓blood viscosity, rightward shift O_2-Hgb curve

HR, heart rate; SV, stroke volume; DPG, diphosphoglycerate; CO, cardiac output; PV, plasma volume; Hgb, hemoglobin.
Reprinted with permission from Practice guidelines for perioperative blood transfusion and adjuvant therapies: an updated report by the American Society of Anesthesiologists Task Force on Perioperative Blood Transfusion and Adjuvant Therapies. *Anesthesiology.* 2006;105:198-208.

b) **Acute anemia (Table 65-1)**
   i) Sudden decrease in blood O_2 content and decreased O_2 delivery lead to a brisk sympathetic response.
   ii) The response to acute blood loss is multifactorial. It depends on the volume and timing of blood loss, as well as patient's age, comorbidities, preexisting volume status, and starting hemoglobin level.
   iii) There is an inverse relationship between hemoglobin levels and cardiac output.[5]
   iv) Most patients begin to experience symptoms of tissue hypoxia, such as fatigue, headache, lightheadedness, dyspnea, or angina when hemoglobin levels reach 7 g/dL or lower.

c) **Chronic anemia (Table 65-1)**
   i) Compensation includes
      (1) Increased production of 2,3-diphosphoglycerate, which increases the P50 of hemoglobin binding, resulting in a right shift in the oxyhemoglobin dissociation curve to facilitate easier unloading of oxygen to tissues
      (2) Increased cardiac output and plasma volume
      (3) Decreased blood viscosity

5) **Treatment**
   a) Treatment of anemia is based upon the underlying cause.
   b) Correction of perioperative anemia has the potential to decrease the need for blood transfusions perioperatively.
   c) Slow correction of hemoglobin levels, such as with oral supplementation or supplement injections, is generally favored.
   d) Anemia from nutritional deficiencies (iron, folate, B_{12}) should be treated with appropriate replacement therapy.
   e) In cases of anemia of chronic disease (eg, chronic renal insufficiency, malignancy, infections, inflammatory conditions, autoimmune disorders), treatment often involves erythropoietic agents (including recombinant human erythropoietin) and supplemental iron.

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Hematologic agents, Chapter 35

6) **Perioperative approach to the patient with anemia**
   a) **Preoperative considerations**
      i) History should focus on potential causes for anemia.
         (1) Inquire about prior history of anemia or other hematologic, hepatic, renal, or endocrine disorders that may be associated with anemia.
         (2) A thorough blood transfusion history is important.
         (3) Poor diet can lead to B_{12}, folate, and iron deficiency.
         (4) Medications such as aspirin and NSAIDs
         (5) History of alcohol and/or drug abuse may lead to nutritional deficiencies and/or chronic disease.
         (6) Family history may reveal sickle cell anemia or thalassemia.
         (7) History of splenectomy

ii) Symptoms may include fatigue, headaches, palpitations, shortness of breath, or angina.
iii) Patients with B_{12} deficiency may have dementia and neuropathy.
iv) If symptoms of myocardial ischemia are present, further cardiac workup is indicated.

b) **Physical exam**
i) Patients may present with jaundice, mucous membrane pallor, hepatosplenomegaly, glossitis, petechia, purpura, koilonychia, neurologic abnormalities, tachycardia, or orthostatic hypotension.

c) **Laboratory and other studies**
i) A complete blood count (CBC) should be obtained and if anemia is present, further workup should be done.
ii) Other tests may include
(1) Reticulocyte count measures the amount of RBC production by the bone marrow
(a) Increases reflect increased production by the bone marrow in response to acute blood loss or hemolysis.
(b) Decreases reflect inability of the bone marrow to mount an appropriate response to decreased O_2 delivery.
(2) Peripheral blood smear will aid in the diagnosis of anemias with abnormal RBC morphology.
(3) Iron studies
(a) Include ferritin, transferrin, and total iron binding capacity (TIBC)
(b) Low ferritin, high transferrin, high TIBC, and low serum iron are seen with iron deficiency anemia.
(4) B_{12} and folate levels
(5) Liver and renal function tests
(6) Stool guaiac to evaluate for chronic gastrointestinal bleeding
iii) For patients with known preoperative anemia who are scheduled for elective surgery, workup and treatment of anemia is ideally started at least 1-2 months prior to the date of surgery for maximal optimization.
iv) Anemic patients undergoing emergency surgery may require blood transfusion prior to surgery, depending on the symptoms and underlying cause.
v) Careful consideration should be given to the patient's intravascular volume status and hypovolemia should be corrected if possible prior to proceeding with surgery.
vi) Patients with anemia may have thrombocytopenia or coagulopathy, which may contraindicate neuraxial anesthesia.
vii) Preoperative autologous blood donation can be considered for nonanemic patients undergoing procedures with high possibility of blood loss.

d) **Intraoperative considerations**
i) The decision to transfuse RBC should be made based on the patient's symptoms, coexisting disease, and the surgical procedure.
ii) The current guidelines outlined by the American College of Physicians, the American Society of Anesthesiologists, and the Canadian Medical Association include a transfusion threshold Hgb level of 6-8 g/dL.[3,6–8]

Iron deficiency anemia and anemia of chronic disease are the two most common causes of anemia.

Increased mean cell volume (MCV) is seen in B_{12} deficiency anemia, while decreased MCV is seen with iron deficiency anemia.

Workup and treatment of anemia is ideally started at least 1-2 months prior to the date of surgery for maximal optimization.

Preoperative assessment of the anemic patient should focus on the underlying cause and intravascular volume status.

The current guidelines outlined by the American College of Physicians, the American Society of Anesthesiologists, and the Canadian Medical Association include a *transfusion threshold Hgb level of 6-8 g/dL*.[3,6–8]

(1) Patients with a Hgb > 10 are unlikely to benefit from blood transfusion.

(2) Other potential indications for transfusion include otherwise stable patients with Hgb < 7 g/dL, patients with Hgb < 9 or 10 g/dL and preexisting cardiovascular disease, patients with acute bleeding (with anticipated blood loss resulting in hemoglobin levels falling below recommended transfusion thresholds), and patients exhibiting symptomatic anemia.[3,5,6,9]

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Alternatives to blood product replacement, Chapter 39

(3) In patients with preoperative chronic anemia, the decision to transfuse may be made earlier than in patients without anemia.

iii) Monitoring

(1) In addition to Standard ASA monitors, invasive monitors such as arterial BP monitoring and central venous pressure may be helpful.

iv) Induction

(1) The goal at induction should be to maintain tissue O_2 delivery.

(2) In euvolemic anemic patients, there is no preferred induction agent provided hemodynamic stability is maintained.

v) Efforts should be made to maintain O_2 delivery during the anesthetic.

vi) Conditions that would shift the oxyhemoglobin dissociation curve to the left should be avoided (eg, alkalosis from hyperventilation, hypothermia).[4]

vii) Prevention strategies to decrease perioperative blood loss should be considered.

(1) This includes minimizing blood sampling frequency, using lower volume sampling tubes, employing surgical techniques that reduce intraoperative blood loss, intraoperative cell salvage, autotransfusion, and use of tranexamic acid.

(a) Recent evidence regarding use of tranexamic acid in patients with ongoing bleeding has made it standard of care in such patients.[10,11]



Consider administration of tranexamic acid in patients with anemia with ongoing bleeding.

e) **Postoperative considerations**

i) Careful postoperative monitoring of hemodynamics, volume status, and laboratory values is necessary.

ii) Transfusion may be required.

Sickle cell disease

1) **Etiology**

a) Sickle cell disease (SCD) is caused by an inherited structural disorder of the hemoglobin β-globin chain with a wide range of severity and variable speed of progression.

2) **Pathophysiology**

a) **Normally adult RBCs contain three types of hemoglobin:**

i) HbA: α and β chains, 96%-98% of total hemoglobin

ii) HbA_2: α and δ chains, 1.5%-3.2% of total hemoglobin

iii) HbF: α and γ chains, 0.5%-0.8%; 90% of total hemoglobin during the first 10 weeks of life

(1) As HbF levels decrease in the first months of life, patients with homozygous (SS) disease almost exclusively produce HbS.

(2) Deoxygenation with a PaO_2 of <40 mm Hg is likely to result in formation of sickle cells in a homozygous HbSS patient, but sickle polymer can also be found in well-oxygenated SS cells.

(3) Acidosis, dehydration, and hypothermia with vasoconstriction also favor the formation of sickle cells.

3) **Prevalence**

a) The sickle cell trait occurs in 10%-30% of people in Equatorial Africa and is also found in populations in the Mediterranean, Middle East, and India.

b) Approximately 8% of African Americans carry the sickle cell trait and 0.3%-1.3% have SCD.[12]

4) **Diagnosis**
   a) Early diagnosis is by neonatal screening with Hb electrophoresis.
   b) Later in life, patients present with anemia, (Hb: 6-9 g/dL) elevated mean corpuscular volume, and leukocytosis (10 000/mm^3-20 000/mm^3). Sickle-shaped erythrocytes are seen on peripheral blood smear.



Vasoocclusive crises (VOCs) are treated with rest, warming, analgesia, and fluid replacement therapy.

5) **Signs and symptoms**
   a) Sickling results in decreased survival of RBCs and a chronic hemolysis with baseline anemia.
   b) Acute exacerbations can manifest as hemolytic, aplastic, or splenic sequestration crises, all resulting in a rapid fall in hemoglobin and are treated with blood transfusions.
   c) VOCs are treated with rest, warming, analgesia, and fluid replacement.
   d) Hydroxyurea has been found to increase levels of HbF and decrease the incidence of VOC.
   e) Acute chest syndrome (ACS) is responsible for up to 25% of sickle cell–related deaths and has an incidence as high as 10% postoperatively, on average developing 3 days after surgery. Patients with ACS present with fever, cough, tachypnea, pleuritic chest pain, hypoxemia, pulmonary hypertension, and radiologic evidence of lung infiltrates.
6) **Physiologic changes**
   a) Biventricular hypertrophy and a hyperdynamic circulation are frequently found in sickle cell patients.
   b) Patients may show signs of pulmonary fibrosis and restrictive lung disease from past episodes of ACS with 30% of adults developing pulmonary hypertension.[13]
   c) CVAs and renal insults are common with renal damage causing a decrease in urine concentrating ability, and if severe, progressing to renal failure.
   d) Decreased or absent splenic function predisposes patients to infections.
   e) Patients are at risk for priapism.
7) **Perioperative considerations**
   a) **Preoperative considerations**
      i) Knowledge of a patient's history of VOC, severity of illness, and chronic organ damage allows for assessment of an individual's perioperative risk.
      ii) Pregnant women tend to have greater perioperative morbidity, with increased age and infection also generating additional risk.[14]
      iii) Preoperative medications that can depress spontaneous ventilation should be used with caution and supplemental oxygen should be considered.
      iv) Transfusions
         (1) Routine use of preoperative blood transfusions is controversial.
         (2) Transfusion regimens range from aggressive (lowering the HbS to <30%), to conservative (only correcting the anemia), to not transfusing at all.
         (3) Two RCTs evaluating the effects of transfusion in low-to-moderate risk procedures found no worse outcome with a conservative technique of maintaining a Hb of 10 g/dL regardless of HbS level,[15] while another compared transfusion to a Hb of ~10 g/dL with no preoperative transfusions and found an increased risk of serious adverse events with no transfusions.[16]



Consultation with a hematologist is recommended for complex patients with SCD.



Hypoxia, acidosis, dehydration, and hypothermia favor formation of sickle cells.

   b) **Intraoperative concerns**
      i) To avoid an acute occlusive crisis, maintain hydration, oxygenation, normothermia, and avoid acidosis and hypovolemia.

ii) End-organ function assessment should focus on cardiac, pulmonary, renal function, and neurologic deficits.
iii) Controversy exists over the use of general vs regional anesthesia with some studies showing worsened outcomes with regional anesthesia[17] while others show no difference.[18]
iv) The use of tourniquets for orthopedic surgery is controversial and the risk of precipitating an acute event with the benefit of decreased bleeding must be assessed.
v) In cases requiring contrast dye, the use of hypertonic dye is associated with increased sickle cell formation, and isotonic contrast is a safer alternative.[19]
vi) Induced hypothermia for CPB can potentially precipitate VOC but has been performed safely.

c) **Postoperative concerns**
i) Patients should be observed for postoperative pain, pulmonary compromise, and hypovolemia.
ii) Early mobilization with aggressive chest physiotherapy is recommended.
iii) Supplemental oxygen should be weaned as abrupt withdrawal has been anecdotally associated with VOC.

Variant sickle cell syndromes

1) **Hemoglobin SC**

a) **Etiology**
i) HbSC disease involves the presence of HbC, which is a variant hemoglobin that does not polymerize with HbS, but can cause red cell dehydration and increased sickling.

b) **Signs and symptoms**
i) HbSC patients exhibit symptoms that are more severe than sickle cell trait and less severe than SCD.
ii) Patients usually have a mild anemia (Hb > 9).
iii) Patients who have homozygous HbC usually have a mild hemolytic anemia.

Other hemoglobinopathies

1) **Thalassemia**

a) **Etiology**
i) The thalassemias are inherited diseases characterized by the inability to produce structurally normal hemoglobin.
(1) β-Thalassemia—no β chains, excess α chains
(2) α-Thalassemia—no α chains, excess β chains

b) **Signs and symptoms**
i) β-Thalassemia major patients develop severe anemia, splenomegaly, and bone changes from marrow hypertrophy.
ii) Facial bones can be involved, resulting in difficult intubation.
iii) These patients are dependent on blood transfusion and have complications from iron overload.
iv) A high cardiac output state is often seen due to the anemia as well as a dilutional coagulopathy due to frequent transfusions.
v) Cardiac failure and ventricular arrhythmias are frequent causes of death.
vi) β-Thalassemia minor patients are generally asymptomatic with a microcytic anemia (Hb > 10 g/dL).
vii) Thalassemia intermedia patients have intermediate clinical severity (Hb: 7-10 g/dL) and are typically not transfusion dependent.
viii) Homozygous forms of α-thalassemia are incompatible with life and the surviving heterozygous patients seldom require transfusions or splenectomy.

2) **Methemoglobinemia**
   a) **Mechanism**
      i) Methemoglobin (Met-Hb) occurs when the iron in HbA is oxidized from a ferrous (Fe^{2+}) to ferric (Fe^{3+}) state, resulting in an inability to bind oxygen.
      ii) The remaining nonoxidized heme groups develop an increased affinity to oxygen (a leftward shift in the O_2-Hb dissociation curve) resulting in more difficulty with release of bound oxygen. This severely limits oxygen delivery and can cause tissue hypoxia.
      iii) Acquired methemoglobinemia can be induced by oxidizing agents such as nitrites, nitroprusside, nitroglycerin, nitric oxide, dapsone, chloroquine, sulfonamides, chlorates, aniline dyes, metoclopramide, and local anesthetics such as benzocaine (Hurricane spray), lidocaine, and prilocaine.



Methemoglobinemia patients will have an SPO_2 of 85% regardless of PaO_2.

   b) **Diagnosis**
      i) Patients are cyanotic in the presence of normal PaO_2 levels, but a low measured SaO_2 concentration.
      ii) SpO_2 measured by pulse oximetry shows a value of 85% regardless of PaO_2.
      iii) Cyanosis and blood with a chocolate-colored appearance occur when methemoglobin levels reach 15%, but healthy patients without anemia may be asymptomatic.
      iv) Mental status changes, dizziness, and headache occur at 20%-30%, while levels of 50% can cause dysrhythmias, seizures, coma, and death.
   c) **Treatment**
      i) Cyanosis is treated with methylene blue (1 mg/kg IV over 5 minutes), which acts as an electron donor reducing oxidized hemoglobin.
      ii) This dose can be repeated every 60 minutes, but doses above 7 mg/kg and rapid administration can paradoxically cause oxidization of Hb to Met-Hb.
      iii) Methylene blue should not be given to patients with G6PD deficiency as it can cause hemolysis.
3) **Sulfhemoglobinemia**
   a) **Etiology**
      i) Caused by the oxidation of iron in hemoglobin and by similar drugs that cause methemoglobinemia. It is unknown why some patients develop sulfhemoglobinemia over methemoglobinemia.
      ii) Patients have a normal PaO_2 value with decreased measured SpO_2.
      iii) Sulfhemoglobin cannot bind oxygen, but contrary to Met-Hb, it causes a rightward shift in the oxyhemoglobin dissociation curve aiding in release of bound oxygen, so even high concentrations are well tolerated.
      iv) No pharmacologic treatment is known.

Polycythemia vera

1) **Pathophysiology**
   a) Polycythemia vera (PV) is a chronic myeloproliferative disorder characterized by the proliferation of red cells, white cells, and platelets.
2) **Signs and symptoms**
   a) Associated with increased blood viscosity resulting in decreased cerebral blood flow (headache, cognitive dysfunction) and thrombotic complications (ischemic stroke, myocardial infarction, peripheral arterial and deep vein thrombosis).
3) **Treatment**
   a) Chronic treatment involves phlebotomy with a target hematocrit of <45%, myelosuppressive drugs, low-dose aspirin, and possible splenectomy.[20]



Patients with polycythemia vera are at higher risk for thrombotic complications such as deep venous thrombosis.

4) **Perioperative considerations**
   a) **Preoperative considerations**
      i) Assess any coexisting bleeding disorder or coagulopathy and patients should be optimized with phlebotomy and myelosuppressive agents.
      ii) Aspirin and perioperative anticoagulation discontinuation should be discussed with the surgery and hematology services.
   b) **Intraoperative and postoperative considerations**
      i) Patients have an increased risk of thrombosis, hemorrhage, and infection.
      ii) Bleeding complications arise in 10%-14% of patients.
      iii) DVT after major surgery occurs at a fivefold greater rate in PV patients.[21]

CHAPTER SUMMARY FOR ANEMIA

Definition	Deficiency in O_2-carrying capacity of the blood due to decreased RBC. Generally defined as Hgb <12 g/dL in women and <13 g/dL in men.
Causes	May be acute or chronic and include blood loss, nutritional deficiencies, chronic disease, bone marrow failure, hemolysis, and congenital abnormalities.
Acute Anemia	Sudden decrease in blood O_2 content and decreased O_2 delivery leads to a brisk sympathetic response.
Chronic Anemia	↑ 2,3-diphosphoglycerate produces a right shift in the oxyhemoglobin dissociation curve to facilitate tissue O_2 delivery. ↑ cardiac output and plasma volume. ↓ blood viscosity.
Preoperative Considerations	Patients with preoperative anemia scheduled for elective surgery should ideally have workup and treatment 1-2 mo prior surgery for maximal optimization. Attempt to ascertain cause of anemia if unknown. Treatment is based on underlying cause. Consider preoperative blood transfusion if patient is symptomatic and/or indicated based on surgical procedure.
Intraoperative Considerations	Maintain hemodynamic stability, O_2 delivery, and euvolemia. Invasive monitoring may be required. Transfusion may be indicated bearing in mind that there is no universal or absolute transfusion threshold. Avoid conditions that will shift O_2-Hgb dissociation curve to the left such as alkalosis and hypothermia.
Postoperative Considerations	Carefully monitor for blood loss, myocardial ischemia, and hemodynamic instability.
Sickle Cell Disease, Hemoglobinopathies, and Polycythemia Vera	
Sickle Cell Disease	Caused by a disorder of the hemoglobin β-globin chain with a wide range of severity and variable speed of progression. Patients can have chronic hemolytic anemia and recurrent VOC causing pain, tissue injury, and organ failure. To avoid a VOC, maintain hydration, oxygenation, normothermia, and avoid acidosis. VOC are treated with rest, warming, analgesia, and fluid replacement. Complicated patients can benefit from a hematology consult.

Methemoglobinemia	Methemoglobin has an increased affinity for oxygen resulting in more difficulty with release of bound O_2. Patients are cyanotic in the presence of normal PaO_2 levels, but a low measured SaO_2 concentration. Methemoglobinemia is treated with methylene blue.
Polycythemia Vera	A myeloproliferative disorder of red cells, white cells, and platelets. Patients have increased blood viscosity leading to decreased cerebral blood flow. Patients are at increased risk for thrombosis, hemorrhage, and infection.

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66 Coagulopathies§

Gregory J. Moy • Larry F. Chu

A coagulopathy is any condition that impairs the blood's ability to clot. Impaired hemostasis requires careful evaluation and timely treatment. It can be congenital or acquired and may lead to significant perioperative morbidity and mortality.

1) **Pathophysiology**
   a) Bleeding disorders typically involve the coagulation cascade, platelet function, or both (**Fig. 66-1**).[1]
   b) **Congenital coagulation factor defects**
      i) Any coagulation factor may be deficient or have reduced activity, in isolation or in combination.
      ii) Hemophilia A (factor VIII deficiency) is inherited in an X-linked recessive pattern.
      iii) Hemophilia B (factor IX deficiency) is less common, also inherited in an X-linked recessive pattern, and clinically indistinguishable from hemophilia A (**Table 66-1**).[4]
   c) **Acquired coagulation factor defects**
      i) Immune-mediated factor defects
         (1) Autoantibodies may cause an acquired hemophilia.
         (2) Acquired hemophilia A, the most common type, is typically seen in older patients and may cause severe bleeding. About one-third of patients will have underlying disorders (autoimmune diseases such as SLE or rheumatoid arthritis) or lymphoproliferative disorders.
         (3) Alloantibody factor inhibitors in hereditary hemophilia patients occur in 4%-30% of hemophilia A and 5% of hemophilia B patients receiving factor replacement.[4]
         (4) Alloantibody factor inhibitors neutralize the infused factor and may cross-react with endogenous factor, sometimes increasing the severity of the clinical disease.
      ii) Non–immune-mediated factor defects
         (1) Decreased factor production such as in liver disease and newborn infants
         (2) Abnormal production
            (a) Warfarin treatment interferes with the ability of vitamin K–dependent coagulation factors to bind the surfaces where coagulation reactions occur.
         (3) Vit K deficiency
         (4) Factor loss and sequestration (eg, nephrotic syndrome)
         (5) Increased factor destruction
            (a) Destruction of fibrinogen in disseminated intravascular coagulation (DIC)
         (6) Inactivation by drugs used for thrombolytic therapy (eg, tissue plasminogen activator) and direct thrombin inhibitor use (eg, hirudin, argatroban)[4]



The functional activity of the factor determines the clinical presentation of a factor deficiency.

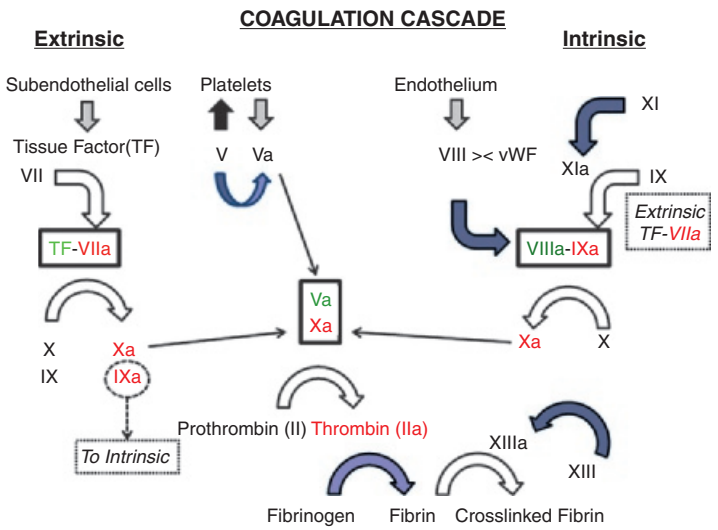
Acquired coagulation factor deficiencies are common and may be immune or nonimmune mediated.[2,3]

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Warfarin treatment is the most common cause of non–immune-mediated acquired coagulation factor defects.

FIGURE 66-1 Coagulation Cascade



Schematic diagram of extrinsic, intrinsic, and common pathways. von Willebrand factor (VWF) and factor VIII circulate together (><). *Gray arrows:* secretion. *Red:* vitamin K–dependent factors. *Green:* cofactors. *Purple arrows:* thrombin activation. *Black arrow:* uptake. *Dotted lines:* intrinsic-extrinsic crossover. *Solid boxes:* also Ca++ and platelet phospholipid. Reproduced with permission from Carabini LM, Ramsey G. Hemostasis and transfusion medicine. In: Barash PG, Cullen BF, Stoelting RK, et al., eds. *Clinical Anesthesia*. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2017:421.

d) **Abnormalities of platelet function**

i) von Willebrand disease (VWD) (an abnormality of von Willebrand factor) is the most common congenital bleeding disorder, having a prevalence of ~1%. There are three main types.

ii) Difficult to diagnose because of variable inheritance and clinical presentation.

iii) Should be suspected in patients with a history of menorrhagia or postpartum hemorrhage.

(1) Type 1, which has reduced VWF levels, is a mild disease, inherited in an autosomal dominant manner. These patients usually respond with a two- to

TABLE 66-1 Coagulation Factor Defects[3]

Factor Deficiency	Incidence	Inheritance	Bleeding Severity
F8	1:10 000	X-linked recessive Xq28	Mild <5%, mod 1%-5%, severe <1%
F9	1, 30 000	X-linked recessive Xq27	Mild-severe
F11	Rare, Ashkenazi population	Autosomal recessive, 4q32q3	Mild-severe
F2, 5, 7, 10	Rare	Variable inheritance	Mild-severe

Afibrinogenemia, dysfibrinogenemia, and hypofibrinogenemia are rare abnormalities with variable clinical presentations. In addition, there are multiple congenital combinations of coagulation factor deficiencies and decreased activity.
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fivefold increase in factor VIII-VWF within 30 minutes of desmopressin (DDAVP) treatment (0.3 µg/kg in 50 mL infused over 30 minutes, consult hematologist for dosing).[5]

(2) Type 2 is caused by abnormal VWF and has several subtypes including 2A, 2B, and 2N. DDAVP has variable effects on factor VIII and VWF release in these patients; consult a hematologist.

(3) Type 3 (severe VWD), these patients have very little or no plasma or platelet VWF and are not responsive to DDAVP. Consider factor VIII-VWF concentrate (50 IU/daily major surgery, 40 IU/daily or every other day minor surgery, consult hematologist for dosing).[5]

e) **Abnormal or deceased platelet production**
   i) Hypocellular bone marrow, for example, aplastic anemia, drug, alcohol, or viral-induced aplasia
   ii) Hypercellular bone marrow—B_{12} or folate deficiency
   iii) Myelodysplastic diseases
   iv) Myelofibrosis
   v) Metastatic disease

f) **Increased peripheral platelet destruction** (Table 66-2)

g) **Platelet dysfunction** (Table 66-3)

h) **Dilutional** coagulopathy due to massive blood transfusion

i) **Sequestration** due to liver disease, splenic disease

2) **Evaluation and treatment**

a) **Family history**
   i) A family history of bleeding disorder may be seen in hemophilia patients and patients with inherited platelet disorders.
   ii) Up to 30% of cases of hemophilia A are spontaneous mutations with no family history.
   iii) VWD, the commonest bleeding abnormality, is difficult to diagnose because of the variability in inheritance and clinical presentation.

b) **Personal history**
   i) Personal history of surgical, dental bleeding, or easy bruising.
   ii) If abnormal bleeding occurs, note the frequency and the duration.
   iii) Location of abnormal bleeding
      (1) Bleeding from skin and mucous membranes tends to occur with platelet disorders.
      (2) Bleeding in joints and muscles tends to occur with the hemophilias.

c) **Medical diseases associated with bleeding abnormalities** (Table 66-4)



VWD should be suspected in patients with a personal or a family history of menorrhagia, postpartum hemorrhage, or excessive bleeding after minor surgical procedures.

VWD is highly variable with many different subtypes. Patients with type I VWD respond to desmopressin treatment, others may not. Consultation with a hematologist to determine the type of patients VWD is suggested.



VWD, type 3 patients are not responsive to DDAVP.



A bleeding abnormality typically manifests as moderate bleeding over a prolonged period, not as bleeding at an excessive rate.

TABLE 66-2 Causes of Increased Platelet Destruction

Sepsis	Platelet Antibodies
Disseminated intravascular coagulation	• Drug induced (eg, penicillin) • Posttransfusion HLA directed
Thrombotic thrombocytopenic purpura (TTP/HUS)	• Collagen vascular disease • Idiopathic thrombocytopenic purpura (ITP)
Cardiopulmonary bypass (CPB)	Mechanical heart valves

TABLE 66-3 Causes of Platelet Dysfunction

Cause	Clinical Example
Disorders of platelet adhesion	von Willebrand disease
Abnormalities of platelet receptors	• Glanzmann thrombasthenia • α-Adrenergic defects
Abnormalities of platelet granules	Quebec platelet disorder
Defects in platelet surface receptor agonists	Collagen defects
Platelet cytoskeletal defects	Wiskott-Aldrich
Drug induced	Aspirin, clopidogrel, fish oil

d) **Medication history**
   i) Aspirin, warfarin, heparin, clopidogrel.
   ii) Herbal/dietary supplements that may interfere with clotting include fish oil, flax seed oil, garlic, ginger, gingko, saw palmetto.
   iii) In the case of known bleeding disorders, a full history of the severity of the disease and required medications/blood products and previous surgical experience should be elicited.

e) **Physical examination**
   i) Assess the patient for bruising or petechiae. In particular, note if petechiae appear after application of BP cuff or tourniquet. Be aware of increased bleeding at IV cannula placement or blood testing.

f) **Laboratory testing**
   i) Various laboratory tests may be available at your institution. Consultation with a hematologist will best aid you in test selection and interpretation (**Table 66-5**).
   ii) Blood tests of coagulation
      (1) Full blood count and examination, including cell count and morphology
      (2) Whole blood clotting time (rarely performed)
         (a) A clot should occur in 5-15 minutes. A weak friable clot suggests hypofibrinogenemia. Early dissolution of the clot suggests enhanced fibrinolysis.
      (3) aPTT (activated partial thromboplastin time)
         (a) Measures the intrinsic and common coagulation pathways.
         (b) Normal PTT times require the presence of the following coagulation factors: I, II, III, IV, V, VI, VIII, IX, X, XI, and XII.
         (c) Normal: 25-39 seconds
      (4) PT, INR (prothrombin time, international normalized ratio)
         (a) PT and its derived measure, INR, are measures of the extrinsic pathway.
         (b) Normal PT: 12-15 seconds, INR: 0.8-1.2.
         (c) PT measures factors I, II, V, VII, and X.



Deficiencies in factor VII or XIII will not be detected with aPTT test.

TABLE 66-4 Medical Diseases Associated With Abnormal Bleeding

- Liver disease
- Hematologic malignancy (eg, leukemia or myeloproliferative disease)
- Solid organ malignancies (eg, prostate, lung, colon)
- Vitamin K or C deficiency
- Kidney disease

TABLE 66-5 Test Abnormalities Seen in Common Coagulopathies

Disorder	Platelet No.	Bleeding	PT	APTT
Hemophilia A	⇔	⇔	⇔	⇑
Hemophilia B	⇔	⇔	⇔	⇑
Vit K def	⇔	⇔	⇑	⇑
DIC	⇓	⇑	⇑	⇑
von Willebrand	⇔	⇑	⇔	⇑/⇔
Bernard-Soulier	⇓	⇑	⇔	⇔
Glanzmann thrombasthenia	⇔	⇑	⇔	⇔

(5) TT (thrombin time)
   (a) Thrombin time compares a patient's rate of clot formation to that of a sample of normal pooled plasma.
   (b) Reflects fibrinogen activity.
   (c) Normal: 10-15 seconds.
(6) ACT (activated clotting time)
   (a) Activated clotting time is a point-of-care test, used to monitor the effect of heparin.
   (b) A sample of whole blood is added to a tube containing negatively charged particles and time for the formation of a clot.
   (c) Normal values are kaolin: 90-150 seconds, Celite: 100-170 seconds, glass: 110-190 seconds.
   (d) Platelet dysfunction and factor deficiencies may prolong the ACT.

iii) Coagulation factor assays
(1) Coagulation factors are defined by their functional activity rather than absolute value (ie, the functional activity of the factor in the patient's plasma compared with that of a calibrator or standard plasma, the latter with a defined assayed activity of 100%).[4]

iv) Factor inhibitor assay
(1) Measures antibodies to factors, which may reduce the activity of both intrinsic and administered factors

v) Platelet function tests
(1) CBC gives platelet number and size.
(2) Blood film shows platelet morphology.

vi) Assessment of platelet function
(1) Bleeding time
   (a) Involves making a standardized incision or lance on the ventral forearm. A BP cuff is inflated above the wound. The time it takes for bleeding to stop is measured.
   (b) Normal bleeding time is 2-9 minutes.
   (c) Rarely performed.
(2) Aggregometry
   (a) Addition of an agonist (thrombin, ADP, epinephrine, collagen, ristocetin) to platelet-rich plasma to assess platelet aggregation
(3) Flow cytometry
   (a) Assesses platelet membrane receptor functional status

vii) Thromboelastography
(1) Thromboelastography is a method of testing the coagulation system by measuring clot formation.

TABLE 66-6 Thromboelastogram Parameters and Suggested Therapies

Parameter	Measures	Suggested Therapy, If Abnormal
R time	Coagulation factors	FFP
K time	Fibrinogen	Cryoprecipitate
Alpha angle	Fibrinogen	Cryoprecipitate
MA	Platelet number and function	Platelets
LY30	Fibrinolysis	Antifibrinolytic (eg, TXA)

(2) It involves either oscillating or rotating a blood sample to initiate clot formation. It is available as a point-of-care test and is particularly suited for managing transfusion therapy in the operative setting.[6,7]
(3) The thromboelastogram (TEG) measures a number of parameters and can be used to direct therapy (**Table 66-6**).
(a) R value or reaction time—the time in minutes from the start of a sample run until the first detectable levels of fibrin clot formation.
(i) Reaction time generally reflects coagulation factor levels.
(b) K value—the measure of time from the beginning of clot formation until the amplitude of the TEG reaches 20 mm.
(i) K factor in conjunction with the alpha angle reflects coagulation factor amplification.
(c) Alpha angle—the size in degrees of the angle formed by the tangent line to TEG tracing measure at the reaction time.
(i) The angle reflects the acceleration of fibrin build up and cross-linking.
(d) Maximum amplitude (MA)—the greatest amplitude of the TEG tracing. It reflects the maximum strength of the final hemostatic plug. MA assesses the combination of platelet count and function plus fibrinogen activity.
Amplitude at 60 minutes is the amplitude of the TEG tracing 60 minutes after the MA is recorded and reflects the stability of the clot.
(e) Clot lysis index—the amplitude at 30 or 60 minutes expressed as a percentage of the maximal amplitude.

3) **Anesthetic concerns**
a) **Preoperative**
i) The involvement of the patient's hematologist is imperative in the creation of a perioperative management plan. In the case of patients receiving anticoagulants or antiplatelet drugs, particularly in the setting of a recent revascularization, consultation should be made with their cardiologist.
ii) Thorough history and physical exam should be elicited.
iii) Appropriate laboratory tests for the patient depending on their history should be performed.
iv) Many patients with bleeding abnormalities may have had multiple blood product transfusions in the past and may have developed antibodies. Be sure you know the status of your patient and the length of time it may take to cross-match blood.
v) A comprehensive care plan should be constructed between the clinicians involved, such as surgeons, anesthesiologist, hematologist, and intensivist as appropriate.
vi) The type of procedure, location it is performed (eg, suitability of surgery center, ICU facilities, blood bank capabilities) should be considered.
vii) The patient and the family must be counseled regarding potential complications.
viii) Consider the appropriate anesthetic technique for the procedure in the setting of the patient's coagulopathy: regional, general anesthesia, monitored anesthesia care, or neuraxial blockade.

(1) Regional anesthesia, particularly neuraxial blockade, is controversial because of the risk of developing hematoma. In some instances, if coagulation tests are normal or if prophylactic treatment is planned, regional block may be possible. Consultation with a hematologist may be necessary.

ix) Consider the need for preemptive therapy with coagulation factors, platelets, or FFP.

x) Evaluate the patient's medications to determine if any need to be stopped prior to surgery (eg, aspirin).

xi) Consider the need for additional monitoring, for example, arterial line, CVP, PA catheter.

xii) Ensure adequate IV access and plan for the possibility of large volume resuscitation in the event uncontrolled bleeding.

xiii) Communicate with the Blood Bank to ensure appropriate resources.

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b) **Intraoperative management**

i) Monitor blood loss

(1) Visual cues, swabs, suction, surgical field.

(2) Communicate with the surgical team to get an accurate idea of ongoing blood loss; venous/arterial, speed of bleeding, the likelihood of controlling bleeding.

(3) Laboratory monitoring for coagulopathy should include determination of ACT, platelet count, PT or INR, and aPTT. Other tests may include fibrinogen level, assessment of platelet function, thromboelastogram, D-dimers, and thrombin time as indicated.[7]



Be vigilant—bleeding disorders may become apparent or develop at any point during the case.

ii) Monitor for adequate perfusion and oxygenation of vital organs.

iii) Monitor for transfusion indications relative to patient status and intraoperative course.

iv) Therapies[8]

(1) Know the transfusion policies in your institution, in particular whether a "massive transfusion" policy exists. Know the location of the blood transfusion services.

(2) Maintain adequate intravascular volume and BP with crystalloids or colloids. When appropriate, intraoperative or postoperative blood recovery and other means to decrease blood loss (eg, deliberate hypotension) may be beneficial.

(3) Transfusion of allogeneic red blood cells or autologous blood.

(4) Transfusion of platelets

(a) If possible, a platelet count should be obtained before transfusion of platelets in a bleeding patient, and a test of platelet function should be done in patients with suspected or drug-induced platelet dysfunction (eg, clopidogrel).

(b) Rarely indicated if the count is known to be >100 000/μL and is usually indicated when <50 000 μL in the presence of excessive bleeding.

(c) May be indicated despite apparently adequate count or in absence of a result if there is known or suspected platelet dysfunction (eg, presence of potent antiplatelet agents, cardiopulmonary bypass, congenital platelet dysfunction, and bleeding).

(d) Typical dose of platelets is six units of pooled donor platelets and is expected to increase platelet count by 30 000-60 000/μL.

(5) Transfusion of fresh frozen plasma

(a) If possible, coagulation tests (ie, PT or INR and aPTT) should be obtained before the administration of FFP.

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Alternatives to blood product replacement, Chapter 39
Massive transfusion and resuscitation, Chapter 40

(b) Is indicated for coagulopathy in the presence of INR > 2.0 and for coagulation factor deficiency in patients transfused more than one blood volume and when coagulation studies cannot be obtained in a timely fashion.
(c) Is not indicated solely for augmentation of plasma volume.

(6) Single coagulation factor administration
(a) Factor replacement is dosed relative to the activity of the factor and its half-life (eg, fibrinogen $T_{1/2}$ ~96 hours, 50% activity required. Factor VIII, $T_{1/2}$ is ~12 hours, 50% activity required, factor IX, $T_{1/2}$ is ~24 hours, 30% activity required.)

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(7) Transfusion of cryoprecipitate
(a) If possible, a fibrinogen concentration and/or viscoelastic study should be obtained before the administration of cryoprecipitate in a bleeding patient.
(b) Is indicated when a test indicates fibrinolysis or a concentration <80-100 mg/dL with excess bleeding.
(c) Transfusion of cryoprecipitate is rarely indicated if fibrinogen concentration is >150 mg/dL.
(d) May be used as an adjunct in massively transfused patients when concentrations cannot be obtained in timely fashion.

(8) Multiple coagulation factor deficiency therapy
(a) FFP and platelets are indicated when there are demonstrable multifactor deficiencies associated with severe bleeding and/or DIC.

(9) Drugs to treat excessive bleeding
(a) DDAVP
(i) Increases factor VIII and von Willebrand factor. Useful for uremia-induced platelet dysfunction, aspirin, VWD.
b) Antifibrinolytic agents—e-aminocaproic acid, tranexamic acid

(10) Supportive measures are extremely important in maintaining organ function and preventing further exacerbation of the coagulopathy and may include
(a) Maintenance of normothermia
(b) Use of blood warmers
(c) Ensure adequate acid-base balance
(d) Replacement of calcium and other electrolytes
(e) Maintenance of hemodynamic stability and oxygenation

c) **Postoperative management**
i) Postoperative considerations are related to the underlying disease processes and surgical procedure.
ii) Consider the appropriate facilities for postoperative care, including regular ward, step down unit, ICU, or coronary care unit.
iii) Consider postoperative intubation if large volumes of fluid and blood products have been given and airway edema is suspected.
iv) A thorough handover to PACU staff is essential in order to inform them of possible issues such as rebleeding, transfusion of remaining blood products, and monitoring of the patient's vital signs.
v) A pain management plan should be formulated and discussed with the surgical or acute pain management services, especially with regard to neuraxial catheters left *in situ*.
vi) Follow-up with your patient. Postoperative rounds on coagulopathic patients are important to insure optimal postoperative patient management.

CHAPTER SUMMARY FOR COAGULOPATHIES

Definition	A coagulopathy is any condition that impairs the blood's ability to clot.
Pathophysiology	Bleeding abnormalities are typically coagulation factor or platelet related. Acquired coagulation abnormalities are more common than congenital. A significant number of congenital abnormalities occur as spontaneous mutations and thus have no family history.
Management Strategies	1. Bleeding abnormalities can develop/become apparent at any time in patient care. 2. Be aware of available tests at your institution and their interpretation. 3. Be aware of the processes and protocols regarding blood product availability. 4. Ensure consultation with a hematologist. 5. Have a multidisciplinary plan for the patient's care.

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67 Part H: Skin and Musculoskeletal Diseases

Malignant Hyperthermia§

Karim T. Rafaat

Malignant hyperthermia (MH) is a rare and potentially lethal condition associated with the administration of volatile anesthetics and/or depolarizing paralytic agents (succinylcholine). With appropriate treatment, the mortality from MH has decreased from >70% to <5%.

1) **Pathophysiology**
   a) **Molecular mechanism**[1]
      i) Genetic mutation is often undetected but can be secondary to:
         (1) RYR1 (the ryanodine receptor), a mutation that increases calcium release from skeletal muscle sarcoplasmic reticulum
         (2) CACNA1S (calcium voltage-gated channel subunit alpha 1 subunit S), mutations that suppress the calcium voltage-gated channels' regulatory effect on RYR1, also increasing calcium release
         (3) STAC3 (SH3 and cysteine-rich domain 3 protein), mutations of which increase the amount of calcium stored in the sarcoplasmic reticulum
      ii) Individuals with variations in one or more of the above receptors/proteins, when exposed to either volatile anesthetics or succinylcholine, will experience:
         (1) Uncontrolled calcium release, which produces a hypermetabolic state within the skeletal muscle.
         (2) This hypermetabolic state produces the signs and symptoms associated with malignant hyperthermia (MH).
   b) **Prevalence**
      i) The prevalence of MH has been reported to be 1:30 000 for pediatric anesthetics and 1:100 000 for adult anesthetics.[2]
      ii) MH is most commonly a condition of children and young adults.
         (1) However, cases of MH have been reported for all ages from infants to the elderly.
         (2) Although two-thirds of MH-susceptible individuals will have an MH event during their first anesthetic, it can occur after several uneventful prior anesthetics.



The earliest sign of MH is increased CO_2 production. This manifests as an increase in end-tidal CO_2 ($ETCO_2$) that is resistant to increasing minute ventilation.

2) **Signs and symptoms**
   a) **Early signs and symptoms**[3]
      i) The signs and symptoms associated with MH are related to the hypermetabolic state that occurs in MH.
      ii) The earliest signs are:
         (1) Increased CO_2 production (present in 92% of adults and 68% of children <18 years old) with a resultant increase in respiratory rate in the spontaneously breathing patient or a rising end-tidal CO_2 ($ETCO_2$) in a mechanically ventilated patient.

(a) Patients with less muscle mass will manifest this sign later, often after tachycardia develops.

(2) Tachycardia (present in ~73% of both adults and children).

(3) Rapidly increasing temperature (present in 65% of adults and 50% of children <18 years of age). As with increased $ETCO_2$, this sign is present more frequently in those with greater muscle mass.

b) **Later signs and symptoms**

i) Secondary to the sustained contractile activity of skeletal muscles, cellular metabolism soon exceeds oxygen supply and metabolic acidosis develops.

ii) With continued cellular hypoxia and acidosis, rhabdomyolysis occurs, which results in a massive release of intracellular potassium.

iii) Cardiac arrhythmias can develop secondary to hyperkalemia and acidosis.

(1) A muscular build increases the odds of a life-threatening MH event more than 10-fold.[4]



Cardiac arrhythmias can develop due to acidosis and hyperkalemia.

iv) Myoglobin released from the damaged muscle will result in red urine (myoglobinuria) and can result in acute renal failure and disseminated intravascular coagulation.

v) The signs and symptoms of MH can occur quickly following a trigger or take hours to fully develop.

(1) In the absence of succinylcholine use, the presenting symptoms tend to be of a more gradual onset.[5]

c) **Associated findings**

i) An associated finding with MH is masseter muscle spasm (MMS).

ii) Although mild jaw stiffness can be detected in all patients receiving succinylcholine, if rigidity approaches the oft-used term "jaws of steel," then true masseter spasm is likely.

iii) When MMS occurs, it may be both a harbinger of acute MH and associated with clinically significant rhabdomyolysis.[6]



When masseter muscle spasm occurs, it may be both a harbinger of acute MH and associated with clinically significant rhabdomyolysis.

iv) Following the detection of MMS, attention should be given to the appearance of hypercarbia, tachycardia, and other signs of hypermetabolism and hyperkalemia.

(1) These signs may not appear immediately following the appearance of MMS.

v) Isolated MMS is not enough to warrant treatment of MH without further signs of hypermetabolism, but it is prudent to cancel elective cases. If the case is emergent, then switching to a nontriggering anesthetic is advisable and remaining vigilant for presence of any ensuing signs of hypermetabolism.[7]

vi) All patients who have experienced MMS should be sent for testing and be treated as MH susceptible, even if MH did not follow its first occurrence.

3) **Treatment**

a) **Initial treatment and resuscitation**

i) Mobilize help

(1) Call for help.

(2) Once a presumptive diagnosis of MH has been made, one should quickly mobilize additional help and have an MH cart brought to the room.

(3) Consult MH cognitive aid

Read More
Malignant hyperthermia, Chapter 150

ii) Stop triggering agent(s)

(1) After calling for help and turning off the volatile, one should increase the flow rates on the anesthesia machine to at least 10 L/min of 100% O_2.

(2) The anesthesia machine and the circuit need not be changed. The surgeon should be notified to complete the surgery as quickly as possible.

iii) Administer dantrolene

(1) The most important treatment step in treating MH, after turning off the triggering agent, is the rapid administration of dantrolene.

(a) Dantrolene is poorly soluble and difficult to mix. An extra pair of hands is advisable; a 20-mg vial of dantrolene should be mixed in 60 mL of sterile water.

(b) Ryanodex is far more soluble; each 250-mg vial should be mixed with 5 mL sterile water.

The most important management step after turning off triggering agent is rapidly administering dantrolene. If dantrolene administration is delayed beyond 50 minutes, complication rates are 100%.[9]

(2) Assign one team member the sole task of mixing the dantrolene as quickly as possible.

(3) The loading dose is 2.5 mg/kg and should be repeated up to 10 mg/kg until clinical signs of improvement are noted.

iv) Monitor and treat signs and symptoms

(1) An ABG should be sent to monitor the acid/base and electrolyte status. Consider placing an arterial line to facilitate frequent follow-up tests.

(a) pH, electrolytes (particularly potassium), and CK (creatine kinase) should all be sent and serially followed.

Ask for help mixing dantrolene. It should be mixed in sterile water and is poorly soluble.

(2) Cardiac arrhythmias should be treated as being secondary to hyperkalemia; accordingly, treatment should include CaCl, insulin-glucose, and sodium bicarbonate.

(3) Institute hyperventilation

(a) Increase minute ventilation to eliminate excess CO_2 caused by hypermetabolism.

(i) Monitor patient's peak inspiratory pressures for signs of air trapping due to insufficient expiratory time because of increased respiratory rate.

Efforts should center on the administration of dantrolene and the treatment of hypercarbia and acidosis BEFORE cooling is considered.

(4) Cooling should be instituted if possible

(a) The patient's core body temperature may rise significantly and can result in central nervous system injury.

(b) Efforts should center on the administration of dantrolene and the treatment of hypercarbia and acidosis BEFORE cooling is considered.

(c) Consider the use of:

(i) Chilled saline for the intravenous infusion

1. 40 cc/kg of 4°C and 20°C fluid decreases core temperature by 2.5°C and 1.4°C, respectively.

(ii) Remove patient's clothing, and use a convective forced air-cooling device set to ambient temperature.

(iii) Gastric, peritoneal, and bladder lavage with cold saline may also help decrease the patient's body temperature but is invasive and associated with considerable risk. Packing the patient in ice can also be an effective way to cool the patient, but prolonged exposure can predispose patient to tissue injury.

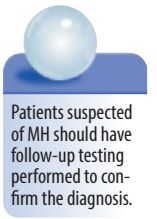
1. Cooling measures should stop at 38°C.

An ABG should be sent to monitor the acid/base and electrolyte status. Consider placing an arterial line to facilitate frequent follow-up tests.

v) Maintenance of anesthesia

(1) The patient's anesthetic should be maintained via total IV anesthesia.

(2) Urinary output should be maintained at >1 mL/kg/h with fluids, mannitol, and Lasix to prevent acute renal failure from the myoglobin release secondary to rhabdomyolysis.
(a) If CK or K rises, assume myoglobinuria and treat with a $NaHCO_3$ infusion at 1 meq/kg/h.


Patients suspected of MH should have follow-up testing performed to confirm the diagnosis.

b) **Postoperative care**[8]
i) The frequency of recrudescence ranges between 14% and 20%, so all patients should be admitted to the ICU for continuation of care.
(1) The patient should continue to receive treatment for MH for 48-72 hours with a dose of 1 mg/kg of dantrolene every 6 hours in an ICU setting.
ii) In addition, urinary output should be maintained with aggressive IV fluids and diuretics. Urine should be tested for presence of myoglobin.
iii) The patient should have frequent arterial blood gases including electrolytes and creatine kinase levels every 6 hours in addition to being monitored for possible DIC.

c) **Follow-up**
i) Further diagnostic testing
(1) Patients who have had a suspected MH event should be referred for a caffeine-halothane muscle biopsy test at one of the regional MH testing centers.
(2) Contact the Malignant Hyperthermia Association of United States (MHAUS) for more information on test sites.

4) **Future anesthetic care**
a) All MH-susceptible patients should receive a nontriggering anesthetic.
b) All volatile agents and succinylcholine should be avoided. All other anesthetic agents are ok for use.
c) The anesthesia machine should be appropriately prepared[10]:
i) The case should be the first of the day.
ii) The absorbent and circuit should be fresh.
iii) Vaporizers should be removed from the machine, if possible.
iv) The machine should be flushed for at least 30 minutes with 100% O_2 at >10 L/min.
v) During the anesthetic, the O_2 flow should not be decreased below 10 L/min in order to avoid a rebound increase in detectable volatile agent.

CHAPTER SUMMARY FOR MALIGNANT HYPERTHERMIA

Definition	A rare and potentially lethal condition due to a genetic defect in one of several receptors that, upon exposure to succinylcholine or volatile anesthetics, leads to an uncontrolled calcium release from skeletal muscle and a resulting hypermetabolic state.
Triggering Anesthetic Agents	Depolarizing neuromuscular blocking agent (succinylcholine) Volatile anesthetic gases.
Signs	Early: ↑CO_2 production, tachycardia, temperature increase. Late: metabolic acidosis, hyperkalemia, cardiac arrhythmia, renal failure.
Treatment	Call for help! Stop triggering anesthetic agent. Administer dantrolene immediately. Monitor and treat associated signs and symptoms.
Postoperative Care	Continue dantrolene in a monitored clinical setting (ICU). Aggressive IV fluids and diuretics. Refer for further testing (caffeine-halothane muscle biopsy).

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68 Burn Injuries

Alma N. Juels

Burn injuries continue to be the leading causes of unintentional death and injury in the United States. One civilian fire death occurs every 2 hours and 35 minutes. The majority of burns are from fire flames, scalds, and contact with hot objects, electrical, and chemical. Almost one-quarter of all burn injuries occur in children under 15 years of age. Children under 5 are 2.4 times more likely to suffer a burn that requires emergency medical treatment compared to the rest of the population. 96.7% of burn injuries treated at a burn center will survive, but unfortunately these survivors will have permanent scarring and disabilities.[1]

1) **Classification of thermal burns**
   a) **Superficial**
      i) Upper layers of the dermis.
      ii) The skin appears red and slightly edematous.
   b) **Superficial partial thickness**
      i) Damage extends into the superficial layer of the dermis.
      ii) Blisters develop, and the skin is red or whitish.
   c) **Deep partial thickness**
      i) Extends into the deep layer of the dermis
      ii) Marked edema and altered skin sensation
   d) **Full thickness**
      i) Extends through the epidermis, dermis, and subcutaneous tissue layer.
      ii) Every system may be affected.
   e) **Subdermal**
      i) Damages muscle, bone, and interstitial tissue
2) **Pathophysiology:** Burns can affect all physiologic functions and organs.
   a) **Skin**: all skin functions are disrupted by burns.
      i) Loss of sensory function of the skin.
      ii) Hypothermia can occur due to extensive evaporative heat and water losses and loss of thermoregulation.
      iii) Increased risk of infection and sepsis.
   b) **Cardiovascular**[2]
      i) Acute phase
         (1) Cardiac output decreases as much as 50%, causing decreased organ and tissue perfusion.
         (2) An increase in capillary permeability occurs with loss of fluid and protein and is greatest in the first 12 hours.
      ii) Second phase (metabolic phase)
         (1) Begins 48 hours after the burn and involves increased blood flow.
         (2) Older patients may have a delayed or nonexistent second phase.
         (3) Profound hypertension is common.
   c) **Respiratory**[3]
      i) Early complications (up to 24 hours):

(1) Carbon monoxide poisoning
(2) Airway obstruction
(3) Pulmonary edema

ii) 2-5 days postinjury:
(1) Patients may develop adult respiratory distress syndrome.

iii) Late complications (days to weeks later):
(1) May include pneumonia, atelectasis, and pulmonary emboli



The most common respiratory sequelae after thermal injury are pneumonia and respiratory failure.

d) **Gastrointestinal**[4]
i) Acute complications
(1) Ileus
(2) Curling ulcer (acute peptic ulcer of the duodenum)
(3) Acute necrotizing enterocolitis
ii) 2-3 weeks postinjury
(1) Acalculous cholecystitis

e) **Renal**[4]
i) Diminished blood flow activates the renin-angiotensin-aldosterone system
(1) Antidiuretic hormone is released, which results in retention of sodium and water with loss of potassium, calcium, and magnesium.
ii) Acute renal failure can develop and has a very high mortality.
iii) Acute tubular necrosis can develop secondary to hemoglobinuria and myoglobinuria.

f) **Hepatic**[3]
i) Decreased hepatic function occurs due to hypoperfusion.

g) **Endocrine**[3]
i) Massive release of catecholamines, glucagon, adrenocorticotropic hormone, antidiuretic hormone, renin, angiotensin, and aldosterone.
ii) Patients can develop nonketotic hyperosmolar coma and adrenal insufficiency.

h) **Hematologic**[3]
i) Anemia
ii) Consumption of coagulation factors
iii) Thrombocytopenia
iv) Platelet dysfunction
v) Antithrombin deficiency

i) **Immunologic**[1]
i) Increased susceptibility to infection, causing most morbidity and mortality



Thermal injury patients with facial burns, singed facial hair hoarseness, or swallowing difficulties are at increased risk for airway swelling and obstruction, and endotracheal intubation should be considered early.

3) **Electrical burn patients may have more extensive injuries.**[5]
a) Areas of damaged skin may be present under "normal-looking" skin, causing fluid requirements to be underestimated.
b) **Complications include:**
i) Myoglobinuria.
ii) Peripheral neuropathies.
iii) Spinal cord damage.
iv) Apnea.
v) Late cataract formation.
vi) Cardiac dysrhythmias may occur up to 48 hours postinjury.



Small amounts of airway edema may occlude a pediatric airway. Early endotracheal intubation should be considered in patients with inhalational injury even in the absence of respiratory distress.

4) **Treatment**
a) The primary goal is to resuscitate the patient, using the "ABCs."[3]
b) **Airway/breathing**
i) Management is generally supportive.
ii) Includes administration of supplemental O_2, inhaled β-agonist, and pulmonary toilet.

TABLE 68-1 Total Body Surface Area Burned as Calculated by the "Rule of Nines"[2]

	Percentage of Body Surface Area
Head and neck	9%
Upper extremities	9% each
Chest (anterior and posterior)	9% each
Abdomen	9% each
Lower back	9% each
Lower extremities	18% each
Perineum	1%

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iii) Intubation or tracheotomy may be necessary and should be considered early; it might be very difficult later due to edema and excessive secretions.

c) **Circulation**[6]

i) Aggressive fluid resuscitation corrects hypovolemia and organ hypoperfusion.

ii) Therapy can be guided by urine output.

(1) Should be maintained *between* 0.5 and 1 mL/kg/h.

(2) In the presence of myoglobinuria, urine output should be at ~2 mL/kg/h.

iii) Respiratory variation in the pulse oximetry or arterial waveforms can be a good guide to volume therapy.[7]

iv) The Parkland formula is the most common formula used to guide[5] fluid requirements.

(1) The Parkland formula involves giving 4 mL of lactated Ringer/kg/total body surface area (TBSA) burned **(**Tables 68-1 and 68-2**)**. This is the most common guide used in the United States.

(2) Half is given during the first 8 hours and the remainder over the next 16 hours.

(a) This fluid requirement is in addition to daily maintenance.

v) Another formula is the Brooke formula, which uses *1.5 mL/kg/% TBSA burn of lactated Ringer's and 0.5 mL/kg/% TBSA burn of colloid.*[2]

vi) Bicarbonate may be used to alkalinize the urine in the setting of pigment-related acute tubular necrosis.[2]

vii) Colloid administration[6]

(1) Controversy exists on the ideal time to initiate colloid therapy in burn resuscitation.

(2) Parkland formula adds colloids 24 hours after the burn[3]

(a) 0%-30% TBSA—no colloid

(b) 30%-50% TBSA—0.3 mL/kg/%/24 h

(c) 50%-70% TBSA—0.4 mL/kg/%/24 h

(d) 70%-100% TBSA burned—0.5 mL/kg/%/24 h

TABLE 68-2 Rule of Nines for Children[2]

	Percentage of Body Surface Area According to Age		
	Newborn (%)	3 y (%)	6 y (%)
Head	18	15	12
Trunk	40	40	40
Arms	16	16	16
Legs	26	29	32

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(3) Colloid should be started earlier in pediatric patients and anyone that not responding to crystalloid fluids. Some institutions start colloids immediately following the Brooke formula.[6]



Electrical burns may result in more extensive tissue damage than is appreciated from physical exam.

5) **Wound care**
   a) Early surgical intervention is crucial to improving outcome.
   b) Procedures usually involve decompression, such as:
      i) Escharotomies
      ii) Fasciotomies
      iii) Excision
      iv) Reconstruction
   c) Supportive surgical procedures include[1]:
      i) Tracheostomy
      ii) Gastrostomy
      iii) Cholecystectomy
      iv) Bronchoscopy
      v) Vascular access



Pulse oximetry may overestimate the oxygen saturation in a patient with carbon monoxide poisoning.[5]

6) **Anesthetic considerations**[3]
   a) **Preoperative considerations**
      i) History
         (1) Inquire about the time of burn and extent of burn for fluid replacement, type of burn, airway involvement, and associated injuries.
         (2) A standard preoperative history is obtained including current medical conditions, medications, and allergies.
      ii) Physical exam
         (1) Assess airway damage, wheezing, and decreased breath sounds.
         (2) Include cardiac exam, vital signs, urine output, and neurologic exam.
      iii) Lab tests
         (1) ABG, electrolytes, and carboxyhemoglobin levels to determine the presence of carbon monoxide poisoning.[5]
   b) **Monitors**[3]
      i) Adequate monitoring can be difficult depending on the extent of skin damage.
      ii) Needle electrodes or ECG pads sewn on may be needed.
      iii) BP cuff may be effective on a burned area, but usually an arterial line is preferred.
      iv) Temperature monitoring is essential.
   c) **Large-bore IVs or central lines are needed for fluid and blood intraoperative considerations.**[2]
      i) Induction
         (1) Propofol is used if the patient is hemodynamically stable and euvolemic.
         (2) Ketamine may be considered if hemodynamics are not normal. It is also the drug of choice for sedation for dressing changes due to its analgesic properties.
         (3) Etomidate is a good choice for induction in an unstable patient and offers the advantage of not having the dysphoric reaction associated with ketamine.
         (4) All induction agents could cause hypotension due to hypovolemia or depleted catecholamine administration.



Succinylcholine should be avoided after the first 24 hours in patients with thermal injuries due to the risk of hyperkalemia.

   d) **Muscle relaxants**[3]
      i) Succinylcholine is not to be used after the first 48 hours.[6]
         (1) Hyperkalemia may result after administration of succinylcholine due to proliferation of extrajunctional acetylcholine receptors.

(2) Succinylcholine should not be used until the burn is healed, or within 6 months to 2 years after a burn.

ii) Nondepolarizing muscle relaxants

(1) Burn patients tend to be resistant to nondepolarizing muscle relaxants, needing up to five times the normal dose.

e) **Pain management**

i) Opioids are still the mainstay for pain management.

ii) Opioid requirements are increased requiring very high doses.

iii) Other options should be considered—ketamine, dexmedetomidine, clonidine methadone, acetaminophen, NSAIDs, and benzodiazepines.[6]



After 48 hours, albumin-bound drugs and drugs metabolized by oxidative metabolism have a prolonged effect.

f) **Drug metabolism**

i) All drugs not administered IV have delayed absorption in the acute phase.

ii) Drugs metabolized by conjugation are not affected.

iii) Opioid requirements are increased.

iv) Inhalation agents may be poorly tolerated due to hypovolemia.

g) **Regional anesthesia**

i) Regional anesthesia may be contraindicated in patients with electric burns due to delayed neurological sequelae of the burn injury.[2]

ii) Local anesthesia injected into a graft or donor site by the surgeon site is helpful.

iii) Subcutaneous catheter infusions, peripheral nerve catheter, truncated blocks/catheters, or central neuraxial blocks should be considered for donor site harvesting and burn areas.[6]

h) **Blood loss**

i) It is difficult to estimate blood loss.

ii) Estimate of blood loss has been reported to be 2.6%-3.4% of the blood volume for every 1% TBSA excised.

iii) Coagulation should be closely monitored and aggressively treated.

7) **Postoperative concerns**

a) Postoperative care may best be done in a dedicated burn intensive care unit.

b) Pain control may be difficult and large doses of opioids may be required. Other pain options should be considered such as pregabalin, gabapentin, antipsychotics, antidepressants, NSAIDs, acetaminophen, and benzodiazepines.[6]

CHAPTER SUMMARY FOR BURNS

Burns	Burns are thermal injury to the skin and tissues Multiple organ systems are affected
Initial Assessment	Perform history and physical exam Assess risk of airway compromise and extent of injuries Hypovolemia is likely
Fluid Management	Based on urine output May use Parkland or other formulae; consider colloids early
Anesthetic Management	Choice of induction agent depends on risk of hypovolemia Monitoring may be difficult due to injury Patients are resistant to nondepolarizing neuromuscular blocking agents and may require higher doses Avoid succinylcholine after the first 24 h
Postoperative Management	Pain control may be difficult; increased doses of opioids and other options for analgesia should be considered Regional blocks have been shown to be helpful

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69 Arthritis

Jillian S. Vitter • Andrea J. Traynor

Arthritis is a general term referring to inflammation of joints. Rheumatoid arthritis (RA) and ankylosing spondylitis are systemic autoimmune diseases that affect primarily the joints and also multiple organ systems. Osteoarthritis (OA) is a degenerative process due to stress and trauma on joints over time and is not autoimmune. The perioperative approach to the patient with arthritis will be discussed in this chapter.

1) **Rheumatoid arthritis (RA)**
   a) **Pathophysiology and characteristics**[1]
      i) RA is a systemic autoimmune disease that is characterized by both inflammation and destruction of synovial membranes with the resultant articular destruction.
      ii) Any joint can be affected, but RA tends to affect the small- to medium-sized joints, especially in the hands and feet.
      iii) The thoracic and lumbar spine is usually spared.
      iv) Arthritis is usually symmetric.
      v) Occurs more often in women.
      vi) In addition to joint involvement, multiple organ systems may be affected, including the cardiovascular, pulmonary, and hematological systems.
   b) **Associated organ system involvement**. RA is a systemic disease affecting many organ systems. Extra-articular disease tends to correlate with more severe active disease.[2,3]
      i) Musculoskeletal (joint involvement)[4,5]
         (1) RA can involve the cervical spine, the temporomandibular joints (TMJ), and the cricoarytenoid joints.
            (a) Cervical spine involvement has been reported to be as high as 86% in RA patients, and the severity of peripheral joint involvement correlates to the degree of cervical involvement.
            (b) Patients may have severely diminished joint range of motion secondary to progressive fibrosis and ankylosis.
            (c) Instability of the atlantoaxial and subaxial joints may lead to subluxation.
               (i) This subluxation can cause spinal cord compression with flexion or extension of the cervical spine.
               (ii) Subluxation with spinal cord compression and sudden death may not be preceded by neurologic symptoms.



RA patients are at risk for cervical spine subluxation and spinal cord injury with neck manipulation.

The absence of preoperative neurologic symptoms does not rule out the presence of subluxation.

      ii) Cardiovascular
         (1) Cardiovascular involvement is common in RA patients and is the leading cause of death.
         (2) Cardiac granulomas can occur, which can cause conduction defects. In addition, nodules can form on valves, which may lead to valvular destruction.
         (3) Patients can also have pericardial disease with effusions.
         (4) Vasculitis is also associated with RA.

iii) Respiratory
(1) The chronic inflammatory state can cause restrictive and obstructive respiratory lung disease.
(a) Fibrosis, small airway disease, and pulmonary hypertension are all possible.
iv) Hematologic
(1) RA patients are often anemic.
(2) Immunosuppression is common due to immunosuppressive drugs and steroids used for treatment of the disease.
(3) Lymphoma and lymphoproliferative diseases are associated with RA.
v) Renal
(1) RA patients often have decreased renal function, usually due to medications.
vi) Hepatic
(1) Elevated liver enzymes are often observed.

c) **Treatment**[6]
i) Early recognition, diagnosis, and treatment initiation are linked with higher rates of disease remission.
ii) Disease-modifying antirheumatic drug (DMARD) therapy is first line and often begins with methotrexate. A short course of glucocorticoids may be used when beginning treatment but should be tapered.
iii) Nonsteroidal anti-inflammatory drugs (NSAIDs) are often used to treat pain and inflammation.
iv) Lifestyle changes (smoking cessation, weight loss, diet, and exercise) and physical and occupational therapy have also been shown to be effective when combined with DMARD therapy.

d) **Anesthetic considerations**[1,5]
i) Atlantoaxial instability is a serious concern in patients with RA and places them at risk for spinal cord injury, possibly resulting in quadriplegia, with neck manipulation.
ii) In any patient with RA, especially those with symptoms of pain or radiculopathy on neck movement, consider cervical spine flexion and extension x-rays to evaluate for atlantoaxial subluxation.
iii) Cervical spine instability may also interfere with vertebral artery blood flow, so the patient should be queried about syncope or dizziness with neck movement.
(1) If there is any question about cervical spine stability in a patient with RA, awake fiberoptic intubation should be performed.



Atlantoaxial instability is a serious concern in patients with RA and places them at risk for spinal cord injury, possibly resulting in quadriplegia, with neck manipulation.



If there is any question about cervical spine stability in a patient with RA, awake fiberoptic intubation should be performed.

2) **Ankylosing spondylitis**
a) **Pathophysiology and characteristics**[1,7]
i) Ankylosing spondylitis is an autoimmune condition similar to RA that affects the spine.
ii) Chronic inflammation of the vertebral articular surfaces leads to fusion of the spine, typically beginning in the lumbosacral region and ascending.
(1) The characteristic finding is a "bamboo spine" on x-ray.

b) **Anesthetic considerations**[7]
i) These patients are at risk for difficult airway due to severely limited cervical spine range of motion.
(1) Even trivial amounts of spine movement may cause fractures.
(2) A thorough preoperative history and physical should be done, paying particular attention to neck range of motion.

(3) Consider awake fiberoptic intubation.

ii) In addition, patients with ankylosing spondylitis appear to be at increased risk of developing an epidural hematoma following neuraxial anesthesia.

(1) Consider postoperative neurochecks after neuraxial anesthesia.

iii) Cardiac involvement is possible and may include aortic regurgitation, conduction abnormalities, and cardiomegaly.



Patients with ankylosing spondylitis are at increased risk of difficult airway with general anesthesia and epidural hematoma after neuraxial anesthesia.

3) **Osteoarthritis**

a) **Pathophysiology and characteristics**[1]

i) Degenerative changes in the joints caused by previous injury, repetitive use, and age.

ii) Characterized by pain, stiffness, and limited movement.

iii) Knees and hands are most commonly affected.

iv) Degenerative arthritis of the spine may be present and lead to intervertebral disc herniation and nerve root compression.

v) OA is not an autoimmune disease.

vi) Systemic corticosteroids are not part of routine treatment.



The fact that RA is a multisystem disease should be considered when formulating the anesthetic plan.

b) **Anesthetic considerations**

i) Obesity is a common comorbidity. Increased weight causes strain on joints, which causes difficulty with exercise and leads to weight gain.

ii) Cervical spine involvement is rare.



Osteoarthritis is the most common joint disease.

4) **Anesthetic management considerations for patients with arthritis**

a) **Preoperative considerations**[1,5,7]

i) A thorough history and physical should be performed, bearing in mind RA and ankylosing spondylitis affect multiple organ systems.

ii) Patients should be asked in the preoperative evaluation if they have pain radiating into the occiput, paresthesias, or sensory loss with head and neck movement.

(1) Flexion and extension cervical spine x-rays can help to determine the presence and severity of the subluxation in the cervical spine.

iii) Patients with RA and ankylosing spondylitis may have pulmonary fibrosis.

iv) Medications should be reviewed.

(1) Patients with autoimmune arthritis may be treated with chronic steroid therapy.

(2) Consider the possibility of medication-induced kidney .dysfunction

(3) Many patients have chronic pain requiring multiple medications, including opioids.



RA patients with a history of hoarseness, pain on swallowing, and stridor may have involvement of the cricoarytenoid joint, which places them at risk for difficult airway and postextubation laryngeal obstruction.

v) Laboratory and other testing

(1) Functional status is often difficult to determine due to limited mobility. Therefore, consider preoperative stress testing in appropriate patients.

(2) ECG should be obtained to evaluate for concomitant coronary artery disease, pericarditis, and conduction abnormalities.

(3) Chemistries may be helpful to determine the extent of any kidney dysfunction and electrolyte abnormalities.



Fiberoptic intubation should be considered in RA and ankylosing spondylitis patients.

(4) Blood counts should be obtained as anemia is common.

b) **Intraoperative considerations**[1,5,7]

i) Airway management

(1) RA and ankylosing spondylitis patients should be considered high risk for difficult airway.

(a) As a general rule, these patients should be assumed to have an unstable neck unless proven otherwise.

(b) RA patients undergoing cervical spine surgery may experience less postoperative airway obstruction with fiberoptic intubation when compared with standard airway management.

(2) The temporomandibular joint is often affected in RA and can cause severe mouth opening limitations.

(3) The cricoarytenoid joint is also very commonly involved in RA.

(a) Hoarseness, weak voice, stridor, and laryngeal obstruction can result.

(4) Patients with ankylosing spondylitis may have extremely limited cervical range of motion.

ii) IV access and invasive monitors

(1) May be difficult to place if the patient has significant bone deformities or obesity.

(2) Invasive monitors should be placed based on the procedure and the patient's coexisting organ dysfunction.

READ MORE
Steroids, Chapter 33

iii) Positioning

(1) Careful positioning and padding of all extremities are important to prevent nerve and soft tissue injuries.

(2) Some extremities may have limited movement. Do not force any position, but instead carefully position the extremity with padding.



Neuraxial block placement may be extremely difficult in patients with ankylosing spondylitis.

iv) Blood loss

(1) May be significant, especially in patients with RA or on chronic NSAIDs who have not had adequate time to discontinue medications.

(2) Blood availability should be strongly considered for major surgeries, including orthopedic cases.

v) Perioperative stress-dose steroids

(1) May be indicated if the patient has been treated with long-term steroids

c) **Postoperative considerations**[1,5,7]

i) Many patients have chronic pain, which may make postoperative pain management difficult.

ii) Peripheral nerve blocks and neuraxial analgesia should be considered. However, a preblock neurologic exam should be performed and documented.

CHAPTER SUMMARY FOR ARTHRITIS

Consideration	Rheumatoid Arthritis	Ankylosing Spondylitis	Osteoarthritis
Pathophysiology	Autoimmune	Autoimmune	Degenerative
Commonly Affected Joints	Neck; TMJ; cricoarytenoid; hands and feet	Neck, spine, and sacroiliac joint; TMJ	Knees and hands
Other Manifestations	LV dysfunction, pericarditis; pulmonary fibrosis; anemia	Chest wall rigidity; bowel dysfunction	Obesity; advanced age

Consideration	Rheumatoid Arthritis	Ankylosing Spondylitis	Osteoarthritis
Preoperative Considerations	Evaluate for coexisting disease and systemic manifestations; attempt to determine functional status; consider blood product availability; reconcile medications and evaluate for medication-induced disease		
Intraoperative Considerations	High risk for difficult airway and neurologic damage due to unstable cervical spine	High risk for difficult airway; neck fractures possible with manipulation; neuraxial anesthesia may be difficult	Neck and airway typically normal
	Positioning and IV access may be difficult		
Postoperative Considerations	Pain management may be difficult due to opioid tolerance, respiratory problems, and chronic pain		

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70 Systemic Lupus Erythematosus§

M. G. Lemley

Systemic lupus erythematosus (SLE) is a multisystem chronic inflammatory disease characterized by the presence of antinuclear antibodies. Any organ system can be affected, the clinical presentation ranges from mild to severe, and the disease course varies between relapse and remission.

1) **Overview**
   a) **Epidemiology**[1]
      i) The overall incidence in the United States is 1 in 2000.
      ii) African Americans and Hispanics have four times the incidence of Caucasians.
      iii) Women are affected seven times as often as men, and SLE is the most frequently encountered autoimmune disease in pregnancy.
      iv) The stress of surgery may exacerbate SLE.
   b) **Pathophysiology**
      i) The pathogenesis of SLE remains unclear, but, once triggered, disease progression involves B cells and B-lymphocyte stimulator (BLyS), which are key humoral mediators of autoantibody activity.[2]
      ii) The deposition of autoantibodies results in inflammation and end-organ damage.
   c) **Drug-induced SLE**
      i) Drugs associated with the development of SLE include hydralazine, isoniazid, procainamide, and α-methyldopa.
         (1) Drug-induced SLE is usually more mild and characterized by rash, fever, arthralgias, anemia, and leukopenia.



Drug-induced SLE is associated with hydralazine, isoniazid, procainamide, and α-methyldopa.

Patients with SLE are at increased risk of CAD with a four to eight times higher incidence than the general population.

2) **Organ involvement**
   a) **Cardiac**: Over half of all patients with SLE have some cardiac involvement. SLE can involve the pericardium, endocardium, myocardium, coronary arteries, and conduction system.[3]
      i) Pericardial disease is one of the most common cardiac manifestations, but hemodynamic compromise is rare.
      ii) Valvular abnormalities
         (1) Mitral and aortic valves are the most frequently affected.
         (2) Libman-Sacks endocarditis is a nonbacterial vegetation on the mitral valve.
         (3) Embolic complications are more common than hemodynamically significant lesions.
      iii) Myocardial dysfunction can occur secondary to acute myocarditis or related pathologies such as hypertension, renal disease, valvular lesions, and coronary artery disease (CAD).
      iv) CAD: Patients with SLE are at increased risk of CAD with a four to eight times higher incidence than the general population.
         (1) Young women have a 50-fold increased risk of developing CAD compared to age-matched controls.[3]

v) Dysrhythmias: Sinus tachycardia is common. Rarely, atrioventricular and bundle branch blocks can occur.

b) **Neurologic**: SLE can affect both the central and the peripheral nervous systems.
   i) Cognitive dysfunction, mood disorders, headaches, seizures, transverse myelitis, and cerebrovascular accidents have all been reported.
   ii) Sensory and autonomic neuropathy can also develop.[4]

c) **Renal**: Virtually all patients with SLE have some renal involvement, and 50% will develop renal disease.
   i) The most common cause is immune-complex deposition resulting in complement activation and inflammation.
   ii) Patients may have proteinuria, hematuria, and pyuria.
   iii) Patients with SLE can progress to end-stage renal disease.[5]

d) **Pulmonary**
   i) Pleuritis and autoimmune pneumonitis result in cough, dyspnea, and arterial hypoxemia.
   ii) Restrictive lung disease can develop due to the chronic inflammatory process.
   iii) Pulmonary hypertension and right heart strain may be present.

e) **Hematologic**
   i) Thrombocytopenia, leukopenia, and anemia are commonly seen.
   ii) There is a strong association with SLE and antiphospholipid antibody syndrome, which is associated with arterial and venous thrombus formation.
   iii) Patients may also be on chronic immunosuppressive medications.

3) **Treatment**

a) The treatment involves decreasing inflammation and suppressing the immune system to prevent further end-organ damage.

b) Patients are frequently on nonsteroidal anti-inflammatory drugs as well as more potent anti-inflammatory agents such as corticosteroids, hydroxychloroquine, methotrexate, azathioprine, cyclophosphamide, and rituximab.

c) A novel class of targeted biologic treatments for SLE, such as belimumab, a recombinant BLyS antibody, recently demonstrated safety and efficacy in clinical trials.[2]

d) Patients with a history of antiphospholipid antibody syndrome may be on anticoagulant medications.

4) **Anesthetic concerns:** A thorough understanding of the diverse and complex nature of the disease is important for the anesthesiologist caring for a patient with SLE.

a) **Preoperative**: A complete preoperative evaluation with particular focus on the cardiopulmonary as well as neurologic and renal systems is warranted. Disease severity and organ systems involved should be assessed.
   i) Assess the patient's exercise tolerance, with particular emphasis on any episodes of dyspnea or angina.
   ii) Check an ECG for conduction abnormalities and signs of ischemia.
      (1) A stress cardiac study may be indicated.
      (2) Echocardiography should be considered if there are findings on the cardiac exam suggestive of valvular disease or myocardial dysfunction.
   iii) Pulmonary function test and a chest x-ray are indicated in patients with dyspnea (rest or with mild exercise) to rule out pulmonary disease.
   iv) A careful neurologic exam should be performed to determine the extent of central or peripheral nervous system involvement.
   v) Laboratory studies
      (1) Complete blood count
      (2) Coagulation studies
      (3) Basic metabolic panel to determine the extent of renal disease and electrolyte status

vi) Patients who have been receiving steroids for the past 6 months may require perioperative stress-dose steroids.



Most SLE patients are immunosuppressed, and strict sterile technique should always be used for procedures.

b) **Intraoperative**
   i) Monitoring
      (1) Standard monitors should be used.
      (2) Other monitors, such as invasive BP monitoring, should be used as warranted by the patient's systemic disease and the surgical procedure.
      (3) Five-lead ECG monitoring should be considered based on the patient's risk for cardiac ischemia.
   ii) The choice of induction agent should be determined by the extent of the patient's systemic disease and the hemodynamic goals of the procedure.
      (1) Nephrotoxic medications should be avoided.
      (2) If the patient has renal impairment, drug clearance may be decreased.
   iii) Strict sterile technique should be used for any procedure, as SLE patients are immunosuppressed.
   iv) Patients with thrombocytopenia are at risk for hemorrhage.
c) **Postoperative**
   i) Monitor for signs of infection as patients may be immunosuppressed.
   ii) Continue to monitor for signs of cardiac ischemia.
   iii) Patients may be at increased risk of developing deep venous thrombosis and pulmonary embolism.

CHAPTER SUMMARY FOR SLE

Definition	SLE is a multisystem inflammatory disease affecting predominantly the cardiac, neurologic, hematologic, renal, and pulmonary systems	
Potential SLE Manifestations	Cardiac	Conduction abnormalities, pericarditis, valvular disease, premature CAD
	Neurologic	Cognitive dysfunction, CVAs, vasculitis
	Pulmonary	Restrictive lung disease, possible pulmonary HTN
	Renal	Glomerulonephritis can result in renal failure
	Hematologic	Thrombocytopenia, anemia, immunosuppression
	Endocrine	Adrenal suppression possible, consider stress-dose steroids
Preoperative Concerns	Preoperative evaluation, with particular focus on the cardiopulmonary, neurological, and renal systems Careful neurological exam to determine the extent of central or peripheral nervous system effects Blood chemistry to evaluate the degree of renal disease and check electrolyte balance prior to surgery	
Intraoperative Concerns	Consider invasive monitoring based on the presence of systemic disease and surgical procedure Nephrotoxic medications should be avoided Sterile technique for invasive procedures due to immunosuppression	
Postoperative Concerns	Monitor for signs of cardiac ischemia or infection May be at increased risk of developing DVTs and PEs	

CAD, coronary artery disease; CVA, cerebrovascular accident; HTN, hypertension; DVT, deep venous thrombosis; PE, pulmonary embolism.

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71 Myasthenia Gravis and Myasthenic Syndrome

Fatoumata Kromah

Recent advances in pharmacology and anesthetic care have created a better understanding and safer clinical environment for the management of myasthenia gravis (MG) in the perioperative setting.

1) **Overview**[1]
   a) MG is the most common neuromuscular junction (NMJ) disease.
   b) Affects approximately 10-20 people per 100 000 in the United States, mostly women under age 40 and men over age 50.
   c) The Myasthenia Gravis Foundation of America (MGFA) Clinical Classification has replaced older schemes (**Table 71-1**).
   d) Commonly administered anesthesia-related medications may result in exacerbation symptoms (**Table 71-2**).
2) **Pathophysiology**[6]
   a) **MG is an autoimmune disease (Fig. 71-1).**
      i) A breakdown in tolerance toward self-antigens through a T-cell–dependent process, mediated at least in part by the thymus that generates acetylcholine receptor (AChR) antibodies (occurs in 80%-90% of patients with MG).
      ii) The resulting complement activation destroys the postsynaptic membrane surface.
      iii) Decreased functional AChR limits muscle cell depolarization by decreasing the probability that a nerve impulse will be followed by an action potential.
      iv) Repetitive stimulation of the NMJ decreases the quantity of ACh released (normal phenomenon), decreasing even further the action potentials initiated in the muscle. This is the pathophysiological basis of fatigue in patients with MG.
      v) Seven percent of patients diagnosed with MG do not have antibodies against AChR, but they have antibodies against muscle-specific kinase (MuSK), a receptor for agrin (lipoprotein-related protein 4, a protein secreted by the axon and essential for the formation of the NMJ).
      vi) The rest of the patients are truly seronegative.
3) **Clinical presentation and diagnosis**[7,8]
   a) Main symptom of MG is painless skeletal muscle weakness.
   b) Muscle weakness is either localized or generalized and variable in nature.
   c) Associated with fatigue (increased weakness after exercise) but not with a sensation of tiredness.
   d) 50% or more patients with MG present with ocular symptoms (orbicularis oculi muscle weakness, diplopia, or ptosis) and 15% with oropharyngeal symptoms (dysarthria or dysphagia):



MG is frequently associated with other autoimmune diseases such as thyroiditis, rheumatoid arthritis, pernicious anemia, and systemic lupus erythematosus.

TABLE 71-1 MGFA Clinical Classification

MGFA Class I	Ocular MG
MGFA Class II[a]	Mild generalized MG
MGFA Class III[a]	Moderate generalized MG
MGFA Class IV[a]	Severe generalized MG
MGFA Class V	MG cases requiring intubation

[a]Classes II, III, and IV have subgroups "a" for limb/axial or "b" for oropharyngeal/respiratory involvement.

i) Dysarthria indicates laryngeal muscle weakness with possible ensuing extrathoracic obstructive flow pattern.
ii) Dysphagia is a common exacerbating factor for MG, and its presence increases the risk of aspiration.

e) Cardiac disease is typically not present, although thymomas may result in myocarditis.
f) **Exacerbating factors**
i) Infections, medications, trauma, hyperthermia, hypokalemia, pregnancy, hyperthyroidism, and emotional distress.
g) **Diagnosis**
i) In patients with obvious ocular involvement, the edrophonium test is a useful bedside test.
(1) 2 mg of edrophonium given every 1 minute up to 10 mg should result in decreased muscle weakness.
(2) Side effects are bradycardia and bronchospasm due to the muscarinic effects of ACh.
ii) In all patients, diagnostic confirmation should include the following:
(1) Serologic test for specific antibodies.
(2) Electrophysiological testing with repetitive nerve stimulation studies. Single-fiber electromyography is the most sensitive test and useful in seronegative patients.
iii) Other useful tests
(1) Chest CT or MRI to identify the presence of a thymoma

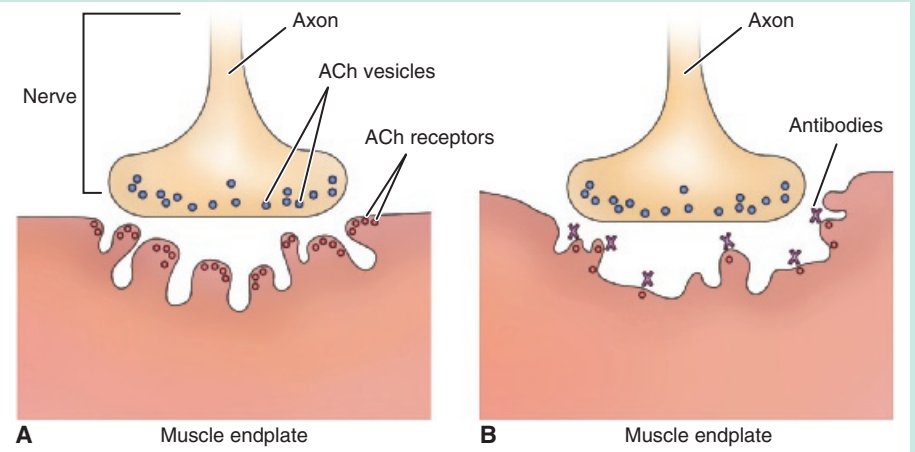
4) **Treatment**[8]
a) **Acetylcholinesterase inhibitors (AChEIs)** are used to treat the symptoms of MG.
i) Allows for ACh accumulation, which improves neuromuscular transmission.
ii) Neostigmine and pyridostigmine are most commonly used.
(1) Pyridostigmine is preferred.
(2) Neostigmine is less effective.

TABLE 71-2 Interaction of Anesthetic Agents With MG[2-5]

Agent	Action	Comments Regarding Patients With MG
Volatile anesthetics	Suppression of T1 and a 40%-70% decrease in T4/T1 ratio at 2 MAC	
Succinylcholine	Increase in ED95 from 0.31 to 0.82 mg/kg, ie, 2.6 times	Resistant Prolonged duration of action
Atracurium and cisatracurium	ED95 of atracurium is decreased from 0.24 to 0.07 mg/kg	Sensitive Advantageous due to their unique mechanisms of elimination

T1 and T4 represent amplitudes of the first and the fourth twitches in a train of four stimulations.

FIGURE 71-1 Myasthenia gravis



A. NMJ with normal postsynaptic membrane surface AChR. **B.** NMJ in MG showing autoimmune destruction of AChR. Reprinted with permission from Pugh S. Management of patients with Neurologic infections, Autoimmune disorders, and Neuropathies. In: Hinkle JL, Cheever KH. *Brunner & Suddarth's Textbook of Medical-Surgical Nursing*. 14th ed. Philadelphia, PA: Wolters Kluwer; 2018:2080.

iii) Approximate equivalent doses are neostigmine 15 mg po = neostigmine 1.5 mg IM = neostigmine 0.5 mg IV = pyridostigmine 60 mg po = pyridostigmine 2 mg IM = pyridostigmine 0.7 mg IV.
iv) Most common side effects are muscarinic

b) **Immunomodulators**
i) Suppress the production of AChR antibodies
ii) Most MG patients require immunomodulators in addition to AChEI to control the disease.
iii) Chronic treatment includes corticosteroids (prednisone) and other immunosuppressants such as azathioprine, cyclosporine, cyclophosphamide, methotrexate, tacrolimus, rituximab, and mycophenolate mofetil.
iv) Plasmapheresis
(1) Directly removes AChR antibodies from circulation.
(2) Reserved for myasthenic crisis (MC), preoperative or as an adjuvant.
(3) Removes plasma AChE, which may alter the metabolism of various drugs.
(4) Side effects include electrolyte imbalances, coagulopathies, and thrombotic events.
v) Intravenous immune globulin suppresses harmful inflammation and is proven effective in MG.

c) **Surgical treatment**
i) Thymectomy is generally effective for patients with and without thymoma.

Central anticholinergic syndrome is produced by the inhibition of the central nervous system muscarinic receptors (eg, atropine), manifested by confusion and treated with central acting AChEI (physostigmine).

5) **Differential diagnosis**
a) Differential diagnosis of chronic weakness includes myasthenic syndrome, motor neuron disease, and various myopathies and neuropathies.

b) **Cholinergic crisis**
   i) Caused by excessive AChEI medication
   ii) Manifested as weakness, which may or may not be accompanied by cholinergic symptoms (bradycardia, miosis, hypersalivation).
   iii) The edrophonium test is not reliable at differentiating between the two conditions and is not recommended in this setting.
   iv) Treatment is symptomatic with antimuscarinic agents (glycopyrrolate and atropine).

6) **Preoperative evaluation**[9,10]
   a) **History and physical examination should be performed with careful attention paid to the following:**
      i) Bulbar and respiratory muscle dysfunction, which increase the risk of aspiration and respiratory failure.
      ii) Vascular or airway obstruction caused by an enlarged thymus.
      iii) Chronic pulmonary disease, which increases the risk of postoperative ventilation.
         (1) Consider pulmonary function studies, and plan for intensive care admission in these patients. PFTs will be below normal in a patient with MG.
      iv) Associated autoimmune disorders
         (1) 12-lead ECG should be obtained to rule out cardiac conduction abnormalities
         (2) Possible cervical spine abnormalities may be present in patients with coexisting rheumatoid arthritis
   b) **Consider preoperative neurologic optimization**, possibly including preoperative plasmapheresis, if the patient is not at the baseline or in severe cases of MG.
   c) **Medication reconciliation**
      i) Doses, adherence to regimen, and side effects should be ascertained.
      ii) AChEI drugs should not be interrupted, especially in symptomatic patients.
      iii) Corticosteroid use is common.
         (1) Assess the need for perioperative supplementation.
         (2) In patients treated with high-dose steroids and nondepolarizing neuromuscular blockers (NMBs), quadriplegic myopathy may occur.[10]
      iv) Azathioprine-induced hepatotoxicity or bone marrow suppression.
      v) Cyclosporine-associated nephrotoxicity.
   d) MG patients should receive minimal sedation as hypoventilation could precipitate respiratory failure or aspiration. However, care should be taken to avoid stress and hyperventilation which could also worsen the clinical picture.
   e) Strongly consider aspiration prophylaxis and precautions with rapid sequence induction and intubation.

READ MORE
Perioperative Corticosteroids, Chapter 33.



Remifentanil, propofol, desflurane, sevoflurane, and cisatracurium are modern anesthetic agents that can be used with confidence in patients with MG.

7) **Intraoperative management**
   a) **Anesthetic agents in MG**
      i) Volatile anesthetics can profoundly decrease neuromuscular transmission in MG.[11] In fact, the combination of weakness from disease and muscle relaxation from volatile anesthesia may obviate the need for muscle relaxation during surgery.
      ii) Depolarizing NMBs (**Table 71-2**)
         (1) There is no contraindication to succinylcholine in MG in terms of risks of hyperkalemia or hypercatabolic syndromes.
         (2) The effect of succinylcholine may be unpredictable. Patients may exhibit resistance to succinylcholine.
         (3) The dose succinylcholine administered should be increased to approximately 2 mg/kg.[12]

(4) Onset of action may be delayed and neuromuscular blockade prolonged.
(5) Patients are more likely to exhibit signs of phase II block.

iii) Nondepolarizing NMBs
(1) Faster onset and prolonged duration of action in MG patients.
(2) Sensitivity can be maintained in patients thought to be in remission and in those with isolated ocular myasthenia.
(3) All the NMB agents have been used with safe results if appropriate precautions in dosing and monitoring are taken.

iv) NMB reversal
(1) Neostigmine may not effectively reverse the action of NMBs, and overdose may lead to a cholinergic crisis.
(2) Sugammadex successfully reverses steroidal NMBs in patients with MG without any interference with ACh pathways.
(3) Standard dosing of sugammadex avoids the undesirable effects noted with neostigmine.

v) Narcotics
(1) Associated with higher risk of respiratory depression, even after epidural administration, and should be carefully titrated.
(2) Remifentanil has been used successfully in association with both propofol and volatile anesthetics.

vi) Local anesthetics
(1) Depress both presynaptic and postsynaptic neuromuscular transmission.
(2) Amide local anesthetics are preferred (see below).

vii) Interactions with AChE inhibitor therapy
(1) Succinylcholine, mivacurium, and ester local anesthetics have impaired metabolism (ie, prolonged duration of action).
(2) Remifentanil and cisatracurium are unaffected.

All MG patients should be monitored intraoperatively with a nerve stimulator.

b) **Monitoring with peripheral nerve stimulator**

i) When eye muscles are affected, the usual pattern of the facial nerve being more resistant than ulnar nerve to the actions of NMBs could be reversed.
(1) Consider double neuromuscular monitoring at both sites in those patients.

Severe bradycardia is possible with neuraxial anesthesia and remifentanil administration in a MG patient treated with AChEI.[13]

c) **Anesthetic plan**

i) Whenever feasible, local, regional, or neuraxial anesthesia should be the first choice.
(1) Spinal anesthesia may be preferable to epidural due to the lower doses of local anesthetics required. However, high levels of sensory blockade are more likely to precipitate respiratory distress in MG patients with a baseline compromised respiratory status.
(2) Regional anesthesia does not eliminate the risk of MG exacerbation.
(3) In the presence of significant respiratory muscle involvement, general anesthesia may be necessary for aspiration prevention and respiratory support.

ii) If general anesthesia is indicated, avoidance of NMBs is recommended.
(1) Total intravenous anesthesia and the use of less soluble volatile anesthetics are the preferred techniques.
(2) In the cases where the use of NMBs is mandatory, dosage should be titrated, and effects checked with a peripheral nerve stimulator.

The possibility of postoperative mechanical ventilation and intensive care admission should be discussed with MG patients.

d) **Induction and intubation methods in order to avoid NMBs** (only to be used if feasible with regard to cardiovascular stability):
   i) Increase the dose of IV induction agent.
   ii) Use volatile agents to deepen anesthesia prior to attempting intubation.
   iii) Use remifentanil in induction doses.
e) For maintenance, a balanced anesthetic is the most common choice. Immobility can be obtained either with remifentanil or maintaining a deeper plane of anesthesia.
f) **Anesthetic emergence**
   i) Processed EEG monitoring might be useful to titrate agents toward the completion of surgery.
   ii) Immediate postoperative extubation can be accomplished in most cases.
   iii) Standard extubation criteria should be fulfilled.
      (1) Sustained head lift for 5 seconds is the gold standard to assess for neuromuscular strength.
      (2) Optimize patient's general condition (normothermia, normovolemia, electrolyte imbalance).
   iv) In the most severe cases, admission to intensive care and delayed extubation until the patient is awake and as close to baseline as possible is the preferred approach.
g) **Avoid medications that can exacerbate MG.**[14]
   i) A comprehensive and updated list of medications that exacerbate MG can be found at the MGFA website at (http://www.myasthenia.org/).



A comprehensive and updated list of medications that exacerbate MG can be found at the MGFA website at (http://www.myasthenia.org/)

8) **Postoperative considerations**
   a) Patients require a monitored setting that is able to provide continuous pulse oximetry, supplemental O_2, more frequent nursing interventions, and physician assessment.
   b) The handoffs should be comprehensive, with emphasis on resumption or titration of intravenous or parenteral AChEI as tolerated and avoidance of detrimental medication.
   c) Non-opioid-based analgesia can decrease risk for respiratory depression.
   d) Due to increased work of breathing and postoperative stress, MG patients may experience myasthenic exacerbation for several days after surgery.
9) **Special types of MG**
   a) **Neonatal MG**
      i) Caused by transfer of AChR antibodies from mother to infant
      ii) Characterized by generalized weakness, which resolves spontaneously within 6 weeks of birth
   b) **Iatrogenic MG**
      i) Caused classically by D-penicillamine, alpha-interferon, and bone marrow transplantation[14]
   c) **Congenital MG**[2]
      i) Genetic predisposition and deficiency in AChR, choline acetyltransferase deficiency, and other NMJ proteins.
      ii) Develop lifelong generalized weakness beginning in infancy or childhood.
      iii) Unlike autoimmune MG, patients with congenital MG typically do not respond to immunomodulator therapy.
10) **MC corresponds to MGFA class V**
   a) Defined by respiratory muscle weakness and impending respiratory failure.
   b) Most commonly precipitated by infections.
   c) Outcomes have improved due to advances in artificial ventilation, but the overall duration of the crisis is around 2 weeks.
   d) Management includes:
      i) Optimization and treatment of the precipitating factors.
      ii) Supportive care usually includes artificial ventilation and ICU care.
      iii) Treatment of neuromuscular dysfunction, usually with plasmapheresis.

e) When intubated, AChEI may be discontinued due to excess respiratory secretions, mucous plugging, and increased risk of arrhythmias.

11) **MG during pregnancy**[4,5]
   a) Fluctuating course due to altered hormonal levels ante- and postpartum. Close monitoring and aggressive treatment are essential.
   b) **AChEI considerations in pregnancy**
      i) Oral pyridostigmine is the first-line treatment.
      ii) Intravenous pyridostigmine is associated with increased uterine contractility secondary to uterine cholinergic receptor stimulation and should not be recommended during pregnancy.
      iii) There is minimal placental and breast milk transfer.
   c) Risk and benefits of diagnostic radiographic imaging, immunomodulating therapy, and surgical management should take maternal and fetal well-being into consideration.
   d) Tocolytic and preeclamptic medications (magnesium sulfate, terbutaline, and ritodrine) alone and in combination with NMBDs can exacerbate muscle weakness.
   e) Stage II of labor could be impaired due to muscle fatigue. Assisted delivery might be necessary.
   f) For vaginal or cesarean delivery, epidural or combined spinal-epidural is preferred if tolerated from a respiratory standpoint.

Lambert-Eaton Myasthenic Syndrome (LEMS)[15]

1) **Overview**
   a) Presynaptic acquired autoimmune disease characterized by an antibody to the voltage-gated calcium channels at the motor nerve terminal.
      i) Results in decreased ACh release from the vesicles
   b) **Signs/symptoms**
      i) Nonfluctuating weakness, more frequently proximal muscle weakness in the lower limbs.
      ii) Rare bulbar or ocular symptoms
      iii) Tendon reflexes are maintained.
      iv) Associated with autonomic failure and sensory neuropathy.
      v) Weakness improves with exercise.
   c) **Associated with small-cell lung cancer, other tumors, and autoimmune diseases**
   d) **Treatment**[16]
      i) Amifampridine or 3,4-diaminopyridine (3,4-DAP) is a potassium channel blocker at the motor nerve terminal and prolongs the action potential and enhances ACh release. It was approved by the FDA in 2018 to treat LEMS.
      ii) Amifampridine can cause hypersensitivity reactions such as anaphylaxis and is contraindicated in patients with seizure disorder.
      iii) Acute or chronic immunomodulation is achieved using the same approach as in MG.
      iv) Anticholinesterase drugs used in MG are not effective in LEMS.



In LEMS muscle weakness improves with exercise, but weakness worsens with exercise in MG.



LEMS patients are sensitive to succinylcholine and extremely sensitive to nondepolarizing agents unlike patients with MG, who are resistant to these drugs.

2) **Anesthetic management**
   a) Most of the considerations from MG discussion apply to patients with LEMS with the notable differences that LEMS patients are sensitive to succinylcholine and extremely sensitive to nondepolarizing agents. Avoidance of NMB is preferred.
   b) If NMB are used, doses should be reduced, and the patient carefully monitored.
   c) Reversal of NMB may be insufficient with AChEI, and extreme care should be taken to ensure muscular strength compatible with successful extubation.

CHAPTER SUMMARY FOR MYASTHENIA GRAVIS AND MYASTHENIC SYNDROME

	Myasthenia Gravis	Lambert-Eaton Myasthenic syndrome
Pathophysiology	Autoimmune disease where antibodies are formed against the ACh receptor	Autoimmune disease affecting voltage-gated calcium channels at motor nerve terminal; results in decreased ACh release
Preoperative Evaluation	Optimization of weakness; plasmapheresis; continue AChEI; assessment of risks of aspiration and postoperative respiratory failure	Amifampridine is the first-line treatment; associated with small-cell carcinoma of the lung
Intraoperative Management	Minimal sedation; if possible, perform regional or neuraxial techniques; avoidance of NMBs; unpredictable response to succinylcholine; sensitivity to nondepolarizing NMBs; "stress dose" of steroids; bradycardia risk; interference with the metabolism of drugs at esteratic sites	Sensitive to succinylcholine *and* sensitive to nondepolarizing NMB; other considerations similar to MG
Postoperative Considerations	Careful titration of narcotics, close monitoring of respiratory status	Careful titration of narcotics, close monitoring of respiratory status

AChEI, acetylcholinesterase inhibitor; ACh, acetylcholine; NMB, neuromuscular blocker; MG, myasthenia gravis.

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Part I: Psychiatric Disease and Substance Abuse

Psychiatric Diseases and Electroconvulsive Therapy

Dominique H. Schiffer

Patients with psychiatric disease are affected by a heterogeneous group of disorders influencing the central nervous system (CNS). Though the underlying pathophysiology may differ considerably in psychiatric illness, there are important anesthetic implications regarding the selection of drugs, techniques, monitoring, and resuscitation. Such patients often require careful preoperative assessment and interdisciplinary care coordination between psychiatrists, neurologists, and the perioperative physician.

1) **Depression**
   a) A complex and heterogeneous disorder that comprises disorders of mood, cognitive function, and neurovegetative functions (ie, energy, sleep, appetite, and sexual function)[1]
   b) **Treatment may use a multimodal approach that may include behavioral therapy, psychotherapy, pharmacotherapy, and education. In those with treatment-resistant depression, electroconvulsive therapy (ECT) may be helpful.**[2]
      i) Behavioral therapy
         (1) Psychotherapy is a nonpharmacologic method for treating depression.
         (2) It includes supportive counseling, cognitive therapy, problem-solving therapy, and insight-oriented therapy.
      ii) Pharmacological
         (1) Nearly half of moderate to severe episodes of depression will respond to medication.
         (2) When a depressed patient presents for surgery, he or she may be taking one or more medications with different mechanisms of action depending on their drug class (**Table 72-1**).
         (3) Antidepressants act by altering noradrenergic and/or serotonergic, dopaminergic, and glutamatergic neurotransmission. They increase the amount of neurotransmitter present in the synapse.[3]
         (4) It is imperative that the anesthesiologist be familiar with the various classes of antidepressant medication.
            (a) Selective serotonin reuptake inhibitors (SSRIs)
               (i) SSRIs are the most broadly prescribed class of antidepressants for mild to moderate depression.
               (ii) SSRIs and some of their metabolites inhibit certain cytochrome P-450 enzymes, with fluoxetine (Prozac) being the most potent inhibitor.
               (iii) Some β-adrenergic antagonists and several cardiac antidysrhythmic drugs (type 1C, eg, propafenone) are metabolized by the same enzyme system that SSRIs inhibit, thereby potentiating their effects.

TABLE 72-1 Common Classes of Psychiatric Drugs, Their Mechanisms of Action, and Associated Anesthetic Interactions

Drug	Mechanism of Action	Anesthetic Interactions
SSRI	Inhibits 5-HT reuptake increasing synaptic levels of 5-HT	Drugs metabolized by cytochrome P-450, eg, benzodiazepines, may have increased effect
Tricyclic antidepressants	Inhibit NE and 5-HT reuptake increasing their levels in the synapse	Unpredictable response to sympathomimetics
MAO inhibitor	• Inhibits MAO A and B • Increases presynaptic NE, DA, and 5-HT	Indirect sympathomimetics are contraindicated (ephedrine, cocaine)
Lithium	• Not well defined, effects on dopaminergic/noradrenergic transmission • May decrease availability of IP3 and ~DAG second messengers	Neuromuscular blockade may be prolonged
Antipsychotics	• Typical: block D2 receptor • Atypical: 5-HT 2A antagonist	CNS depressants may have enhanced effects

(iv) Other drugs metabolized by the cytochrome P-450 enzymes that could potentially interact with SSRIs are diazepam, midazolam, tricyclics, omeprazole, warfarin, phenytoin, theophylline, haloperidol, carbamazepine, tolbutamide, and clozapine.[4]

(v) A rare complication of SSRI treatment is SIADH with hyponatremia.[4]

(b) Tricyclic antidepressants (TCAs)

(i) Mechanism of action

1. Block reuptake of epinephrine and serotonin in nerve cells.[3]
2. Alter both noradrenergic and serotonergic systems.
3. Anticholinergic properties may cause orthostatic hypotension.
4. Cardiac conduction delay can result in dysrhythmias and heart block.
5. Response to sympathomimetics is unpredictable in patients taking TCAs.
   a. In patients recently started on TCAs, an exaggerated pressor response can be expected with both direct- and indirect-acting sympathomimetics, though the response may be more exaggerated with an indirect-acting sympathomimetic, such as ephedrine.[7]
   b. For those who are chronically treated, either sympathomimetic is acceptable, although one should start with a smaller dose.



In patients recently started on TCAs, an exaggerated pressor response can be expected with both direct- and indirect-acting sympathomimetics.

(c) Monoamine oxidase inhibitors (MAOIs)

(i) Mechanism of action

1. Inhibit the activity of monoamine oxidase, thereby preventing the breakdown of monoamine neurotransmitters.
2. The use of this class of drugs has significantly declined recently because of a wide spectrum of side effects, drug-drug interactions, and toxicities.
3. In patients taking MAOIs, the administration of propofol, barbiturates, and benzodiazepines can result in hypotension and potentiate their CNS and respiratory depressant effects.[3]
4. A hypertensive crisis can be induced by giving indirect sympathomimetics (ephedrine), methyldopa, and levodopa, in patients taking MAOIs.[3]

5. Patients taking MAO inhibitors can be safely anesthetized when keeping in mind the aforementioned potential side effects and drug interactions mentioned in.[4]



A **hypertensive crisis** can be induced by giving indirect sympathomimetics (ephedrine), methyldopa, and levodopa, in patients taking MAOIs.[3]

(d) Ketamine infusion therapy
   (i) Currently used off-label for treatment-resistant depression. Several small placebo-controlled, double-blinded trials and randomized trials have been completed and show rapid and robust effects, but the effects might be transient.[5,6]

2) **Bipolar disorder (manic-depressive illness)**
   a) **Characterized by cyclical disturbances in mood, cognition, and behavior, with episodes of mania and hypomania.** Some may present with rapid cycling including a change in mood over a period of hours. They may also exhibit depression and hypomania (appearing like anxiety) at the same time (a mixed state).
   b) **Treatment**
      i) Lithium is one of the most common drugs used for the treatment of bipolar disorder.[7]
         (1) Mechanism of action
            (a) May be related to its effects on dopaminergic and noradrenergic transmission or on complex transduction pathways
            (b) Lithium acts presynaptically to inhibit neuromuscular transmission.
            (c) Prolongation of neuromuscular blockade is possible with both depolarizing and nondepolarizing neuromuscular blockers. These drugs should be given in reduced doses. Monitoring the depth of neuromuscular blockade is essential.[3]



Lithium may prolong neuromuscular blockade with both depolarizing and nondepolarizing neuromuscular blockers. Reduce the neuromuscular blocker dose and monitor the depth of blockade closely.

         (2) Plasma concentration levels must be measured to ensure safe and effective use. The therapeutic range is 1.0-1.2 mEq/L.
         (3) Lithium can affect renal function; thus, renal function tests should be done every 6 months.
         (4) ECG changes may be observed with lithium treatment.
            (a) Flattening of the T wave without any clinical consequences is common.
            (b) Toxicity may result in QRS complex widening.
            (c) Sinoatrial node dysfunction has been described.
         (5) Hypothyroidism develops in 5% of patients.
         (6) Patients with therapeutic levels of lithium may experience delirium after ECT; therefore, psychiatrists usually will hold lithium the day prior to ECT.

3) **Schizophrenia**
   a) **A group of illnesses that are characterized by a thought disorder is demonstrated by delusions, hallucinations, behavioral disturbances, and paranoia.**
   b) **Antipsychotic medications are chemically diverse compounds.**
      i) Typical antipsychotics (chlorpromazine, haloperidol) block the dopamine D2 receptor.
      ii) Atypical antipsychotics (clozapine, olanzapine) act as 5-HT 2A antagonists.
      iii) Antipsychotic medication may enhance the effects of CNS depressants. The combination of antipsychotics and ECT is well tolerated.[3]

4) **Electroconvulsive therapy (Table 72-2)**
   a) **ECT provokes a generalized epileptic seizure**.

TABLE 72-2 Anesthetic Management for Patients Undergoing Electroconvulsive Therapy (ECT)

Indications	Treatment-resistant depression, schizophrenia, or psychotic depression
Preoperative evaluation	• Thorough history and physical • Evaluation of prior ECT records • Informed consent
Anesthetic plan	• Efficacy is determined by production of adequate seizure • Consider patient's comorbidities
Intraoperative Management	
Equipment	Standard monitors and equipment for general anesthesia
Induction agents	Methohexital (gold standard), propofol, etomidate
Muscle relaxants	Succinylcholine is preferred
Hemodynamic management	Significant swings in blood pressure and heart rate may be seen; anticholinergic, antihypertensive agents are frequently required
Postoperative management	Treat possible headache, nausea, myalgias, or confusion

b) **It is safe and effective and at times can be life-saving.**
c) **The average ECT course is about eight to nine treatments, usually administered three times a week. ECT also may be performed anywhere from once a week to once a month in order to prevent relapses.**
d) **ECT can be safely done in both in-patient and out-patient settings.**[2]
e) **Indications**
   i) Treatment-resistant depression
   ii) Schizoaffective disorder
   iii) Psychotic depression
   iv) Catatonia
   v) Bipolar depression including rapid cycling bipolar disorder or mixed state
   vi) Acute mania
   vii) The need for a rapid response due to psychosis or risk of suicide[2]
f) **Contraindications**
   i) There are not any absolute contraindications to ECT.
   ii) Conditions that increase a patient's risk of complications include:
      (1) Unstable cardiac disease
      (2) Cerebral vascular disease
      (3) Recent cerebral hemorrhage or stroke
      (4) Increased intracranial pressure
   iii) ECT is safe for those who:
      (1) Have cardiac pacemakers
      (2) Have implantable cardiac defibrillators
      (3) Are pregnant
   iv) The anesthetic must be tailored appropriately for these conditions as would be for any surgical procedure.[8]
g) **Mechanism**
   i) The mechanism is unknown, but effects may be secondary to alterations in neurophysiologic, neuroendocrinological, and neurochemical systems.[3] ECT itself is anticonvulsant, and its therapeutic action may be based on this brain stabilizing effect.
h) **Description of ECT procedure**
   i) The psychiatrist places an EEG monitor and either unilateral or bilateral electrodes to deliver the ECT stimulus.

ii) An additional BP cuff is placed on an available extremity, used to monitor the duration of the motor seizure.
iii) After preoxygenation with 100% oxygen, anesthesia is induced and the patient is ventilated via a facemask. A slight head-up position is useful in obese patients.
iv) After inflating the second BP cuff above the systolic BP, a muscle relaxant is administered.
v) The anesthesiologist places a bite block between the patient's teeth, followed by sweep with the fingers around the bite block to make sure the tongue is fully out of the way, and the electrical stimulus is applied to induce a seizure.
vi) The seizure is monitored by both EEG and the tonic-clonic activity in the extremity with the second blood pressure cuff.
vii) Generally, anesthesia is required for only about 3-5 minutes.
viii) When the seizure is over, and spontaneous respirations return, the patient is taken to a recovery area and monitored for at minimum, 30 minutes. Standard discharge criteria are used for release from the PACU.

i) **Physiologic responses to ECT**
i) At the moment the electrical current is applied, a parasympathetic surge occurs (15-20 seconds of bradycardia).
ii) Thereafter, a sympathetic surge ensues, with an increase in HR and BP.
(1) The rate pressure product may increase two- to fourfold, increasing myocardial oxygen demand. The anesthesiologist must understand this unique situation and be ready to treat the cardiovascular responses when necessary.
(2) Cerebral blood flow increases during the seizure to keep up with the increased oxygen utilization, the maximal blood flow velocity in the middle cerebral artery more than doubles.
(3) After the seizure terminates, a second parasympathetic response may occur but not as robustly as the first one. Ventricular escape beats may be seen and can be treated with an antiarrhythmic or atropine.

5) **Anesthetic considerations with ECT**
a) **Preoperative evaluation**
i) The quality of the preoperative evaluation should be the same as for any surgical procedure.[8]
(1) A thorough review of systems and focused physical exam, with an emphasis on cardiac, respiratory, and neurological systems, should be done in order to identify possible risk factors.
(2) Additional tests, laboratory exams, and imaging studies may be requested based on the patient's history and physical examination.
(3) Since most patients require multiple ECT treatments, it is very helpful to review previous anesthetic records. Once this is done, asking the patient and/or caregivers about complications or problems with the previous anesthetic can help guide anesthetic management.
(4) The anesthetic must be tailored appropriately for these conditions as would be for any surgical procedure.[8]
ii) Written informed consent should be obtained for ECT.[8]
(1) Patients who cannot give consent because of severe psychosis or catatonia may have consent be given by a family member with a durable medical power of attorney.[6]
(2) A court order may be obtained if there is no health care proxy.[8]

b) **Intraoperative considerations**
i) ECT requires a complete general anesthetic, providing loss of consciousness, analgesia, amnesia, and muscle relaxation.

ii) The challenge is to provide the anesthetic without suppressing the seizure.
iii) Concurrently, one must also manage the airway and the physiologic consequences of the electrically induced seizure.
iv) Monitoring and equipment
  (1) ECG, BP monitor, pulse oximeter, capnography
  (2) Invasive BP monitoring is only necessary in unique cases such as in a patient with a cerebral aneurysm.
  (3) Facemask appropriate for controlled ventilation
  (4) Standard circle system or bag-valve-mask system
  (5) Resuscitative equipment
  (6) Laryngoscope
  (7) Endotracheal tube
  (8) Laryngeal mask airway (LMA) for management of an airway emergency
  (9) Extra BP cuff to isolate circulation to an extremity
  (10) Bite block
  (11) BIS monitoring has been used in ECT with mixed results.[8]
v) Induction
  (1) The efficacy of ECT is based on seizure duration, with the goal of 25-50 seconds to produce an antidepressant effect.
  (2) Many induction agents have anticonvulsant properties.
    (a) Methohexital is considered the "gold standard" induction agent for ECT.
      (i) The American Psychiatric Association recommends a dose of 0.75-1.0 mg/kg.
    (b) Propofol at minimally hypnotic doses (0.75 mg/kg) is associated with similar seizure duration as methohexital. Larger doses (up to 1.5 mg/kg) can still lead to clinically acceptable seizure durations.
    (c) Etomidate in doses of 0.15-0.3 mg/kg has little impact on seizure threshold.
      (i) Associated with longer seizure duration than methohexital and propofol
      (ii) Associated with fewer hemodynamic changes than other induction agents
      (iii) Drawbacks include myoclonus, emetic symptoms, and increased confusional states post-ECT.
    (d) Ketamine's potential use as an induction agent for ECT remains controversial, and some recommend further study.
      (i) A recent publication suggests that it may have excellent antidepressant properties.[5]
vi) Muscle relaxants
  (1) Succinylcholine is the muscle relaxant of choice for ECT, due to quick onset and fast recovery that result.
    (a) The recommended dose is 0.5-1.0 mg/kg.
    (b) Contraindications to the use of succinylcholine include:
      (i) Pseudocholinesterase deficiency
      (ii) Malignant hyperthermia
      (iii) Neuroleptic malignant syndrome
      (iv) Organophosphate poisoning
      (v) Conditions that render patient sensitive to the potassium-releasing effects of succinylcholine
  (2) Nondepolarizing muscle relaxants can be used when succinylcholine is contraindicated.
    (a) In a recent crossover comparison study of rocuronium vs succinylcholine:



ECT requires a complete general anesthetic, providing loss of consciousness, analgesia, amnesia, and muscle relaxation.

(i) A 0.3-mg/kg dose of rocuronium was compared to succinylcholine 1 mg/kg for muscle relaxation at the induction of anesthesia
(ii) Reversal agent (10 μg/kg of atropine and 20 μg/kg neostigmine) was given immediately after completion of the ECT procedure when rocuronium was used.
(iii) Time to first spontaneous breath was 9.46 minutes with rocuronium and 8.07 minutes with succinylcholine.[10]
(iv) One would expect similar results using sugammadex as a reversal agent.

vii) Respiratory management
(1) Control of the airway during ECT is paramount.
(2) Hypocarbia induced by hyperventilation can be used to enhance seizure duration.
(3) After induction, if mask ventilation is difficult, airway devices including LMA can be used to achieve adequate ventilation.
(4) Intubation might be indicated in certain circumstances such as late pregnancy or a full stomach.
(5) Oxygen consumption and CO_2 production are increased by seizure activity. Ventilation must be increased appropriately.
(a) Hypoxia and hypercarbia can aggravate tachycardia and hypertension.

viii) Managing the hemodynamic effects of ECT
(1) Anticholinergic agents
(a) Recommended for patients with bradycardia or heart block or in those where a prolonged bradycardia may occur (ie, patients on β-adrenergic antagonists).
(i) Routine use of an anticholinergic agent results in increased tachycardia after ECT.
(b) β-Adrenergic antagonists
(i) Both esmolol and labetalol have been extensively studied in the management of ECT-induced hemodynamic responses, including attenuating the rise in blood pressure and heart rate.
(c) Calcium channel antagonists
(i) Intravenous nifedipine (0.1 mg/kg) and nicardipine (40 μg/kg) have been reported to reduce mean arterial pressure and heart rate after ECT.
(d) Other agents
(i) Clonidine, nitroprusside, nitroglycerin, and trimethaphan have all been used successfully to control the hemodynamic response in ECT.

c) **Postoperative concerns**
i) The most common side effects of ECT are confusion, agitation, amnesia, myalgias, nausea, and headache.[11]
(1) The confusional state can last up to an hour post-ECT.
(2) Headaches and myalgias can be successfully treated with both NSAIDs and narcotic analgesics. Intranasal sumatriptan is also effective.[2]
ii) ECT can cause transient memory deficits.
iii) The death rate from ECT is ~4 in 100 000 treatments, similar to that of general anesthesia.



ECT side effects such as headache and myalgias are treated with NSAIDS and narcotic analgesics.

CHAPTER SUMMARY FOR PSYCHIATRIC DISEASES

Psychiatric Condition and Pharmacologic Treatment	Mechanism of Action	Anesthetic Interaction and Considerations
Depression		
SSRI	SSRI: P-450 inhibitor	SSRI: potential potentiation of cardiac antidysrhythmic drugs (propafenone)
TCA	TCA (increases activity of synaptic norepinephrine)	TCA: potential exaggerated effects with direct-acting catecholamines (epinephrine)
MAOIs	MAOI (effects norepinephrine presynaptic stores)	MAO: indirect agents contraindicated (ephedrine) which could cause hypertensive crisis Recommend awareness of potential complications and careful hemodynamic monitoring in all patients taking antidepressant medications
Bipolar Disorder	Lithium	May prolong neuromuscular blockade of both nondepolarizing and depolarizing neuromuscular blockers; reduce the dose and monitor depth of blockade
Schizophrenia	Antipsychotics are a diverse group of compounds which block dopamine D2 receptors (eg, Haloperidol, typical antipsychotic), or 5-HT 2A antagonists (eg, clozapine, atypical)	Potential for impaired temperature regulation and potentiation of hypotensive and sedative effects of general anesthesia. Higher incidence of paralytic ileus
Electroconvulsive Therapy (ECT)	Indicated for treatment-resistant depression, schizophrenia, or psychotic depression	Significant swings in blood pressure and heart rate may be seen; anticholinergic and antihypertensive agents are frequently required. Treat possible postprocedure headache, nausea, myalgias, or confusion.

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73 Alcohol and Substance Abuse†

Adrian Hendrickse

An ever-increasing number of patients, using and/or addicted to recreational drugs, present for routine and emergency surgery. Intoxication may be acute, chronic, or one superimposed on the other. Multiple drugs may be present at any one time and withdrawal from intoxicants may prove problematic. Multiple organ systems can be affected.

1) **Ethyl alcohol (EtOH)**—With the possible exception of nicotine, EtOH is the most common substance of abuse. The acute effects of EtOH lead to impairment in judgment, balance, and motor control, often predisposing the user to traumatic injury. Chronic effects are listed in **Table 73-1**.
   a) **Effects on organ systems (see Table 73-1)**
      i) Central nervous system (CNS)
         (1) Depresses the CNS by augmenting inhibitory GABAergic synaptic transmission and by inhibiting excitatory glutamate-mediated NMDA transmission.
         (2) Results in generalized blunting and loss of higher motor, sensory, and cognitive function and a depressive effect on inhibitory pathways (disinhibition).
         (3) Chronic EtOH use is associated with peripheral nerve and neuropsychiatric disorders, which may be due to neurotoxic effects or to nutritional deficiencies (eg, Wernicke encephalopathy and Korsakoff psychosis).
            (a) Neuropathies have many features, including weakness of intrinsic muscles of the foot as well as proximal limb weakness, pain and numbness in the lower extremities (often in a stocking foot distribution), and decreased or absent deep tendon reflexes.
            (b) Autonomic dysfunction, manifesting as postural hypotension and delayed gastric emptying are also risks.
            (c) The EtOH-withdrawing patient may experience delirium and seizures, which can be fatal.
      ii) Cardiovascular (CV) system
         (1) Acute
            (a) Moderate acute ingestion of EtOH produces no significant changes in blood pressure (BP) or myocardial contractility.
            (b) Heart rate increases and flushing results from cutaneous vasodilatation.
            (c) At toxic levels of acute EtOH ingestion, a decrease in central vasomotor activity causes respiratory and cardiac depression.
            (d) Acute intoxication may have negative inotropic effects.
         (2) Chronic
            (a) Hypertension
            (b) Cardiac dysfunction is the leading cause of death.
            (c) Tachycardia and dysrhythmias (eg, acute atrial fibrillation, atrial flutter, ventricular ectopy, and tachycardia) may indicate alcohol-related cardiac dysfunction, and a 12-lead electrocardiogram (ECG) should be evaluated.
            (d) May cause conduction delays.
            (e) Severe use over an extended period may result in congestive cardiomyopathy with associated pulmonary hypertension and right-heart failure.

TABLE 73-1 Chronic Effects of Alcohol Abuse

Organ System	Physiologic and Clinical Effects
Neurologic	Neuropsychiatric disorders, peripheral nerve palsies, Korsakoff psychosis, seizures, Wernicke encephalopathy, delirium tremens
CV	Autonomic dysfunction, hypertension, dysrhythmias, congestive cardiomyopathy
Pulmonary	Pulmonary hypertension, right-heart failure, decrease in lung capacities, pneumonia
Gastrointestinal	Delayed gastric emptying, susceptibility to aspiration, esophagitis, gastritis, pancreatitis,
Hepatic	Impaired hepatic synthetic function, coagulation disturbances, unpredictable drug metabolism, hypoglycemia
Renal	Numerous electrolyte abnormalities
Immunologic	Decreased bone marrow function, susceptibility to infection

iii) Respiratory system
  (1) Acute
    (a) May cause depression of the ventilatory response to CO_2 and increases in dead-space ventilation
    (b) There is a generalized decrease in vital, functional residual, and inspiratory capacity.
    (c) Aspiration of gastric contents is a risk as laryngeal reflexes may be blunted.
    (d) EtOH users are more susceptible to pulmonary infections.

iv) Gastrointestinal and hepatobiliary systems
  (1) Alterations in glucose homeostasis are common.
    (a) Hypoglycemia should be considered if the patient has an altered mental status not explained by intoxication.
    (b) The patient subsisting mostly on EtOH calories may experience an adverse reaction to glucose infusions, including Wernicke encephalopathy and acute phosphate depletion with rhabdomyolysis and neurologic dysfunction.
  (2) EtOH use may cause esophagitis, gastritis, and pancreatitis.
  (3) Delayed gastric emptying and the risk of aspiration have been mentioned.
  (4) Chronic severe consumption of EtOH leads to irreversible cirrhosis and alcohol-induced hepatitis.
    (a) Hepatic synthetic function is impaired, as manifested by hypoalbuminemia and decreased production of coagulation factors II, V, VII, X, and XIII. This leads to a prolonged prothrombin time.
    (b) Distribution, metabolism, and excretion of drugs can be altered, so effects can be unpredictable.
    (c) Esophageal varices develop as portal pressure increases, and the value of placement of a nasogastric tube in such patients should be carefully considered due to the risk of severe variceal bleeding.

v) Fluid and electrolyte abnormalities
  (1) Hyponatremia, hypokalemia, hypophosphatemia, hypocalcemia, and hypomagnesemia are common.
  (2) EtOH inhibits ADH acutely and can result in dehydration.
  (3) Chronic use results in an edematous state due to secondary hyperaldosteronism. Ketoacidosis has been noted.

vi) Hematopoiesis

(1) Bone marrow function is depressed and may be reflected by pancytopenia. Macrocytic anemia is common.
(2) Patients are prone to infections, especially pneumonia.

vii) Nutritional deficiencies
(1) Thiamine deficiency
(a) Wernicke encephalopathy (confusion, nystagmus, ophthalmoplegia, ataxia) is usually reversible with treatment. Korsakoff psychosis (memory loss and associated confabulation) is often not.
(b) Polyneuropathy.
(c) Cardiac failure characterized by high cardiac output, low systemic vascular resistance, and loss of vasomotor tone.
(2) Folic acid deficiency
(a) Causes bone-marrow depression

b) **Effects on anesthetic agents**

i) Volatile anesthetics
(1) Acute intoxication
(a) Minimal alveolar concentration (MAC) of inhalational agents is reduced by 40% or more, depending on the blood alcohol level.
(2) Chronic use
(a) MAC for inhalational agents is increased.

ii) Intravenous anesthetics
(1) Acute intoxication
(a) Increased sensitivity to the effects of barbiturates, benzodiazepines, and opioids.
(2) Chronic use
(a) May require larger-than-normal anesthetic doses due to enzyme induction and cross-tolerance.
(b) Intravascular volume status and autonomic dysfunction may complicate management.
(i) Decisions as to drug dosing and fluid management must be individualized and based on response.

READ MORE
Diseases of the liver and biliary tract, Chapter 56.

iii) Muscle relaxants
(1) Cirrhotic patients are edematous and have a greater *Vd* for water-soluble drugs such as muscle relaxants.
(2) A larger initial dose of nondepolarizing relaxants can be needed, though drugs that rely on hepatic metabolism for their clearance may have a prolonged duration of action.
(3) Repeated dosing should be based on the assessment of neuromuscular blockade.
(4) Impaired hepatic synthetic function may result in decreased levels of plasma cholinesterase, prolonging the effects of succinylcholine.
(5) Cisatracurium (largely metabolised independently of liver function) is a good choice of muscle relaxant for patients with severe liver disease.



Patients who abuse alcohol may be at risk for hypothermia due to peripheral vasodilation and altered thermoregulation.

iv) Sympathomimetic medications
(1) Patients may be less sensitive to endogenous or parenteral catecholamines, and direct-acting medications are suggested.



Alcohol withdrawal can be fatal and should be treated aggressively.

c) **Withdrawal states**

i) Acute withdrawal syndrome usually develops within the first 24 hours but can often be delayed for several days.

ii) Early withdrawal symptoms include fever, tachycardia, anxiety, tremor, agitation, and hallucinations.
iii) Delirium tremens and seizures are risks and are potentially fatal.
iv) It should be noted that while medications such as clonidine decrease the symptoms of withdrawal, they are not protective for seizures.
(1) Benzodiazepines should be administered to EtOH-dependent inpatients.



The perioperative period is not the best time to limit the prescription of opioids in an attempt to wean the dependent patient.

2) **Cannabinoids (cannabis, marijuana, dope, grass, hash, pot, weed)**
a) General information
i) Cannabinoids are the most commonly used illicit drugs in the United States.
ii) Legislative changes have made cannabinoids a legal drug of abuse in some states.
iii) Δ-9-Tetrahydrocannabinol (THC) is the chemical primarily responsible for the desired psychoactive effects.
iv) The THC content of marijuana products has increased dramatically over the last half-century with advances in cultivation techniques.
v) Cannabinoid products are commonly smoked, inhaled, or ingested.
vi) New generation synthetic cannabinoids have similar effects to natural marijuana products. However, they are often unpredictable in terms of severity and duration.
b) Effects on organ systems (**Table 73-2**)
i) Central nervous system
(1) Rapid onset of a feeling of enhanced sensory awareness (especially spatial and temporal) and euphoria, followed by relaxation and sedation.
(2) Anxiety, increased appetite, and problems with coordination and memory are common.
(3) Psychosis and hyperemesis syndrome can be severe.
ii) Cardiovascular
(1) Tachycardia and peripheral vasodilation leading to an increase in cardiac output.
(2) Increased risk of myocardial infarction especially in the first hour after a dose.[1]
(3) Conjunctival injection—"red eyes"
(4) Synthetic cannabinoids have stronger effects compared to natural marijuana products.
iii) Pulmonary
(1) Bronchodilation and decreased airway resistance in both inhaled and ingested routes.
(2) Smoking carries an increased risk of upper airway reactivity.
(3) The higher temperature of smoked cannabis can cause airway damage and edema.

TABLE 73-2 Effects of Chronic Cannabinoid Abuse

Organ System	Physiologic and Clinical Effects
Neurologic	• Anxiety, depression, paranoia, psychosis, and schizophrenia • Memory, cognition, and sleep disturbance • Autonomic instability—orthostatic hypotension
CV	Atheromatous vascular disease, myocardial ischemia, hypertension
Pulmonary	Cough, bronchitis, emphysema, chronic pulmonary disease
Gastrointestinal	Hyperemesis
Endocrine	Gonadal suppression—gynecomastia in males, anovulation and galactorrhea in females

(4) Breatholding after cannabis inhalation can cause a pneumothorax or pneumomediastinum.

iv) Endocrine

(1) Suppression of gonadal function with gynecomastia in males and anovulation and galactorrhea in females.

(2) Hypoglycemia

c) **Anesthetic considerations**

i) Preoperative considerations

(1) Take a good history specific to the type of cannabinoid use, the regularity, and the method of ingestion.

(2) Delay surgery for at least an hour after acute intoxication to lower the risk of a perioperative myocardial infarction.[2]

(3) Use a multimodal pain plan including regional anesthesia.

ii) Intraoperative considerations

(1) Regular users may require higher doses of propofol for induction, airway instrumentation, and procedures performed under sedation.[3]



Regular cannabis users may require higher doses of propofol for induction, airway instrumentation, and procedures performed under sedation.[3]

iii) Postoperative considerations

(1) Acute intoxication can add to the sedative effect of anesthesia.

(2) More "rescue" medications in the immediate postoperative period.

(3) Consider cannabis withdrawal if the patient is excessively difficult to manage after surgery.[3]

d) Dependency and withdrawal states

i) Heavy, regular users will experience withdrawal in <24 hours. Mild cases take a few days to resolve, severe cases up to several weeks.

ii) Irritability, anxiety, depressed mood, headaches, anorexia, insomnia, tremors, and chills are common presenting features.

iii) Management of severe cases is supportive and symptom based. Synthetic THC and benzodiazepines along with other sedatives are increasingly being used.

e) Synthetic cannabinoids (Incense, Spice, K2)

i) Usually packaged as salts or oils. Volatile and lipid-soluble, synthetic cannabinoids are designed to be inhaled, "vaped," or ingested orally.

ii) Multiple active ingredients with agonistic and antagonistic action at cannabinoid receptors, which explain their often, mixed clinical picture.

iii) Severe intoxication is characterized by the sympathomimetic toxidrome: agitation, anxiety, psychosis, hyperthermia, tachypnea, hypertension, tachycardia, and chest pain.[4]

iv) Management is supportive and symptom based, although effects may be life threatening (rhabdomyolysis, kidney failure, myocardial ischemia, seizures, hyperthermia) and require intensive care treatment.



Naloxone can precipitate withdrawal, severe hypertension, and pulmonary edema in habituated patients.

3) **Opioid abuse**

a) This problem ranges from the patient who abuses prescription opioids to the patient who obtains injectable street drugs such as heroin.

b) Patients with acute and legitimate requirements for opioids may become tolerant to the effects of these medications, requiring increasing doses, but do not tend to develop addictive behaviors.

c) **Effects on organ systems (Table 73-3)**

i) Central nervous system

(1) Excessive doses may result in oversedation, apnea, and death from respiratory depression.

TABLE 73-3 Chronic Effects of Opioid Abuse

Organ System	Effect
CNS	Coma, respiratory arrest, hypoventilation
CV	Valvular disease, pulmonary edema
Pulmonary	Aspiration, atelectasis, hypoventilation, infections
Infectious	Sepsis, human immunodeficiency virus, tetanus, opportunistic infections
Dermatologic	Cellulitis, abscess formation
Gastrointestinal	Hepatitis
Endocrine	Adrenal suppression

(2) Pinpoint pupils in the setting of coma suggest opioid effect.
(3) The opioid antagonist, naloxone, delivered in divided doses, will reverse an opioid-induced coma.

ii) Cardiovascular
(1) Pulmonary embolism, pulmonary hypertension, cardiogenic pulmonary edema, endocarditis, and valvular disease have been described.

iii) Pulmonary
(1) Aspiration, pneumonia, atelectasis, respiratory depression, and noncardiogenic pulmonary edema are risks.

iv) Hematologic
(1) Predilection toward infectious diseases
(a) Mainly seen in users of injectable opioids
(i) Contaminated needles and nonsterile injection practices render these patients prone to diseases including hepatitis B and C, human immunodeficiency virus, bacterial endocarditis, pneumonia, cellulitis, abscess formation, sepsis, tetanus, fungal infections, etc.



Postoperative pain control is often challenging for patients with chronic opioid use, and regional anesthesia as an adjunct should be considered whenever possible.

d) **Effects on anesthesia**
i) Acute intoxication
(1) Opioids reduce volatile anesthetic requirements.
ii) Chronic use
(1) Patients often require more volatile anesthetic or higher opioid dosing than anticipated.
(2) Providing adequate postoperative analgesia may be challenging.
(a) Consider the merits of peripheral nerve blocks for peripheral procedures.
(3) Obtaining intravenous access may be difficult.

4) **Opioid withdrawal**
a) **Symptoms**
i) Restlessness, sweating, nausea, rhinorrhea, abdominal cramping, and lacrimation. Overt withdrawal results in piloerection, emesis, diarrhea, muscle spasms, fever, chills, tachycardia, and hypertension.
b) **Onset and duration of withdrawal varies with the drug used.**
i) For example, meperidine withdrawal symptoms peak in 8-12 hours and last for 4-5 days. Heroin withdrawal symptoms usually peak within 36-72 hours and may last for 7-14 days.
c) **Treatment**
i) Includes slow-onset, long-duration medications (eg, methadone, half-life 15-25 hours) that tend not to provide the euphoric feeling drug abusers crave.[5]

(1) Methadone has been associated with torsade de pointes in patients with prolonged QT syndrome, so an ECG is necessary prior to its administration.
(2) Sympathetic symptoms may be treated with β-adrenergic antagonists or α_2-agonists such as clonidine.
(3) Withdrawal is not usually life threatening.

5) **Cocaine** is a powerful, addictive stimulant that can be administered in its powdered form across mucosal membranes, dissolved and injected parenterally, or smoked as the highly addictive lipid-soluble formulation known as "crack."
   a) **Mechanism of action**
      i) Inhibition of presynaptic neuronal reuptake of norepinephrine (NE), epinephrine, dopamine, and serotonin leading to prolonged stimulation of the sympathoadrenal axis
      ii) NMDA receptor agonist
   b) **Physiologic effects**
      i) Mainly due to increases in NE
      ii) Increases in serum NE levels increase systolic, diastolic and mean arterial pressure, heart rate, and body temperature



Hyperthermia is a serious complication of cocaine intoxication and can cause death.

   c) **Metabolism**
      i) Hydrolyzed by plasma and liver esterases
      ii) Metabolites excreted by the kidney and can be detected in the urine for up to 60 hours
      iii) Patients with qualitative or quantitative cholinesterase deficiencies may be prone to cocaine toxicity at lower serum levels.
   d) **Effects on organ systems (Table 73-4)**
      i) Central nervous system
         (1) The patient may be euphoric or experience boundless energy but in excessive doses may become agitated, combative, or hallucinate.

TABLE 73-4 Effects of Cocaine Abuse

Organ System	Effect
CNS	• Euphoria, increased energy • Agitation • Seizures • CVA
CV	• ↑HR, ↑BP • Chest pain • Myocardial ischemia/infarction • Dilated cardiomyopathy • Aortic dissection • Ventricular arrhythmias • Sudden death
Pulmonary	• Pneumothorax • Pulmonary edema • Pulmonary hemorrhage and infarction
Hematologic	Disseminated intravascular coagulation
Kidney	• Acute renal injury • Rhabdomyolysis
Other	• Perforated nasal septum • Severe hyperthermia

(2) Cocaine is hypermetabolic and blocks the thermoregulatory process of vasodilation and sweating.

ii) CV system

(1) Vasoconstriction and vasospasm compromising tissue perfusion and oxygenation.

(2) Severe hypertension and ischemia-induced arrhythmias are common.

(3) Patients may develop angina, myocardial ischemia, or infarction. Cardiomyopathies, dissections, and strokes are rarer presentations.

(a) Treatment of cocaine-induced chest pain includes the use of selective β_1-adrenergic blockade, nitrates, calcium channel blockers, benzodiazepines, and α-adrenergic blockers.

(b) Avoid selective β_2 agents because it may lead to unopposed α_1-mediated coronary and peripheral vasoconstriction.[4]

(c) Cocaine blocks sodium channels in cardiac conducting tissue and is proarrhythmic.



Selective β_2-adrenergic blockade should be avoided in the cocaine-intoxicated patient as it can lead to unopposed α_1-mediated coronary and peripheral vasoconstriction.

e) **Anesthetizing the cocaine-intoxicated patient**

i) Preoperative considerations

(1) Prolongation of the QT interval predisposes the patient to torsades de pointes.

(a) An ECG must be obtained preoperatively.

(2) Elective surgery is safe in a cocaine-ingesting patient who is not acutely intoxicated and does not have significant alterations in vital signs or myocardial ischemia.[6]



Fatal ventricular arrhythmias and sudden death have been associated with cocaine use.

ii) Intraoperative considerations

(1) The patient should be well anesthetized prior to airway instrumentation to avoid severe tachycardia and hypertension.

(2) Ketamine and pancuronium potentiate the CV toxicity of cocaine and should be avoided.

(3) The MAC of inhalational anesthetic agents is increased when acutely intoxicated.

(4) Should hypotension require pressor therapy, direct-acting agents such as phenylephrine may be better as these patients may be catecholamine depleted.

(5) Lidocaine may potentiate its proarrhythmic effects.

(6) Hyperpyrexia may require active cooling measures.

(7) Benzodiazepine administration is important to prevent seizures.



Cocaine abuse can cause catecholamine depletion and render patients unresponsive to indirect-acting sympathomimetics such as ephedrine.

iii) Postoperative considerations

(1) Cocaine withdrawal may be problematic.

(a) Symptoms include agitation, anxiety, depression, fatigue, disturbed sleep, tremors, and myalgias.

6) **Phenylethylamines: Amphetamine and its derivatives (Methamphetamine, Crystal Meth, Ecstasy, MDMA)**

a) Historically used to suppress appetite in obese patients and is now often used for patients with attention deficit disorder/hyperactivity and narcolepsy.

b) As a drug of abuse, amphetamines and the derivatives such as methamphetamine and ecstasy (3,4-methylenedioxymethamphetamine—MDMA) are used to promote the feeling of euphoria, alertness, and enhanced energy.

c) Because of its relative ease of preparation and cost, "Crystal Meth" has become a significant drug of abuse in rural communities.

i) Effects are similar to cocaine.

ii) Usage results in increased availability of epinephrine, NE, dopamine, and serotonin at the synapse.
iii) Onset of effects occurs rapidly (seconds after smoking or injection, minutes after oral ingestion) and commonly lasts for 24 hours.

d) **Effects on organ systems**
i) Signs and symptoms of intoxication include mydriasis, anorexia, diaphoresis, hypertension, tachycardia, dysrhythmias, hyperthermia, metabolic acidosis, agitation, and psychosis.
ii) Electrolyte abnormalities including hyponatremia and hyperkalemia and hypoglycemia have been observed.
iii) Patients can also have myocardial infarctions, seizures, strokes, rhabdomyolysis, and renal failure.
iv) Rarely, hyperthermia develops, which may precipitate CV collapse and multiorgan failure.
v) The withdrawing patient will experience fatigue, irritability, insomnia, anxiety, and psychotic reactions.

e) **Anesthetic considerations**
i) Recommendations for anesthetizing a meth-abusing patient are similar to those for cocaine.
ii) Avoid anesthetizing the acutely intoxicated patient for elective procedures.
iii) Chronic users may be catecholamine depleted and will only respond to direct-acting sympathomimetics.



Dental decay from bruxism and poor dental hygiene is common in abusers of methamphetamine ("meth mouth") and may increase the incidence of dental trauma.

General anesthesia is preferable in PCP-abusing patients because of their frequent dissociative state and unpredictable violent behavior.

7) **Phencyclidine (PCP, angel dust)**
a) Structurally related to ketamine and is an NMDA antagonist.
b) Like ketamine, it is a CV stimulant with significant analgesic qualities.
c) It is usually smoked but can be snorted, ingested, or injected. Metabolised in the liver (90% first pass) and excreted in the urine.
d) It produces a dissociative state or "out-of-body" experience. It crosses the blood-brain barrier to produce amnesia, delirium, disordered thought processes, paranoia, hostility, and delusions.
i) Hallucinations and psychosis may resemble schizophrenia.
ii) Patients are often violent while under its influence and insensitivity to pain can make them formidable physical challenges.
iii) Patients often bounce between stimulation and sedation. Nystagmus, ataxia, and difficulty speaking may occur.
iv) Autonomic stimulation and cerebral vasodilation are also effects.
v) No significant ventilatory depression is observed, and airway reflexes are usually maintained. Irregular breathing patterns with episodic apnea and tachypnea occur.
vi) Copious oral secretions with airway obstruction may be noted.
e) The precise mechanism of action is unknown, but it is thought to depress cortical and thalamic function while stimulating the hippocampus, neocortex, and limbic system.
i) May block afferent impulses associated with the affective component of pain perception and suppress spinal cord activity.
ii) Can inhibit pseudocholinesterase.
f) Clearance is increased by acidification of the urine and nasogastric suction.

8) **Lysergic acid diethylamide (LSD), novel psychoactive substances (NPS), and other hallucinogens**
a) Alter sensory perception and mood remarkably, often in color.
b) Often have sympathomimetic effects: mydriasis, tachycardia, hypertension, and hyperthermia.
c) Linked to "serotonin syndrome": altered mental state, neuromuscular abnormalities, and autonomic hyperactivity.

d) Can cause profound delusional states and the behavioral consequences.
e) Otherwise do not have significant impact on the conduct of anesthesia.
f) Recovery should be in a quiet environment with supportive care.

CHAPTER SUMMARY FOR ALCOHOL AND SUBSTANCE ABUSE

General Considerations	Acute and chronic substance abuse significantly affects physiology and response to perioperative medications
Alcohol Abuse	Acute—Decreases MAC, respiratory, and cardiac depressant Chronic—Increases MAC; patients may have chronic neuropathy, liver failure, cardiomyopathy; withdrawal is life threatening
Cannabinoid Abuse	Unpredictable reaction to anesthesia depending on method of abuse. Chronic users often suffer from withdrawal syndrome during their hospital stay
Cocaine Abuse	Hyperthermia may be life threatening; observed arrhythmias include long QT and ventricular tachycardia with risk for sudden death; hypertension may be severe; may not respond to indirect-acting sympathomimetic agents
Methamphetamine Abuse	Similar physiologic effects as cocaine. Psychiatric symptoms are common in patients presenting to the emergency services. High risk for dental injury with airway manipulation
Opioid Use/Abuse	Chronic use results in tolerance with patients requiring high doses for adequate pain control; use of injected opioids increases risk of blood-borne infection and endocarditis

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74 Overview of Pediatric Anesthesiology

Christopher L. Ciarallo

Pediatric patients can unsettle even the most seasoned anesthesiologist. Providers must not only be comfortable with wide variation in anatomy and physiology (from the tiny premature infant to the adult with congenital diseases) but also have an understanding of psychological state and developmental issues throughout childhood. The care of children requires appropriately sized equipment and personnel experienced in the preoperative assessment and postoperative recovery of children.

1) **Anatomy and physiology**
   a) **Airway**
      i) Pediatric patients have a proportionately larger occiput and shorter neck than adults. As a result, a shoulder roll rather than a head pillow may improve airway patency during mask ventilation and endotracheal intubation.
      ii) Infants <6 months old are obligate nasal breathers.
      iii) The face mask used for ventilation should rest on the nasal bridge and the mentolabial sulcus (cleft of the chin). A mask one size larger than anticipated should be available in the event an oropharyngeal airway is necessary.
      iv) Manual jaw thrust combined with continuous positive airway pressure (CPAP) of 5-10 cmH_2O is the most efficacious technique to improve pediatric upper airway patency.[1]
      v) The pediatric epiglottis is long and floppy, and the pediatric larynx is more cephalad (at the level of the C3-C4 vertebral body rather than C5-C6 as in adults).
         (1) Straight laryngoscope blades (eg, Miller, Wisconsin, Wis-Hipple) may be more successful and should be used to directly lift the epiglottis in neonates, infants, and children <2 years of age.
         (2) Gentle external laryngeal manipulation over the thyroid cartilage may dramatically improve the view during laryngoscopy and intubation.
      vi) The pediatric cricoid cartilage is ellipsoid rather than round. The pediatric subglottic area is cylindrical with the narrowest dynamic portion at the glottis and the narrowest fixed point at the cricoid cartilage.[2]
      vii) Uncuffed endotracheal tubes (ETTs) may not provide effective occlusion to achieve adequate tidal volumes during positive-pressure ventilation and may lead to repeated laryngoscopy and intubation for tube exchanges.
         (1) Poorly fitted tubes result in unreliable ventilation and capnography, anesthetic gas pollution, and increased fresh gas waste.



A straight laryngoscope blade (Miller, Wisconsin, Wis-Hipple) is recommended for endotracheal intubation in children <2 years old.

READ MORE
Management of pediatric difficult airway, Chapter 96; Practical pediatrics anesthesia, Chapter 75



Insertion of LMA backward with partially inflated cuff and then rotating 180 degrees to the anatomic position provides the highest success rate.

viii) Pediatric cuffed ETTs
(1) Can be safely used for children as young as full-term neonates.
(2) Have smaller internal diameters and increase the work of breathing during spontaneous ventilation.
(3) The cuff should be positioned entirely below the glottis, and the pressure checked frequently when using nitrous oxide.
(4) Microcuff ETTs have a very low profile, short and more distally placed cuff. However, they lack a Murphy eye and may be more prone to distal obstruction.[3]



The most critical factor in selecting the appropriate ETT size is the ability to maintain an air leak at <20-25 cmH_2O pressure to minimize the risk of mucosal ischemia and subsequent subglottic stenosis.

ix) Appropriate ETT depth
(1) May be approximated by the various formulae

(eg, 4 + [age (in years)/4] for uncuffed; 3.5 + [age (in years)/4] for cuffed)

or by aligning the ETT double black line markers at the vocal cords
(2) The most reliable method of determining correct tube depth is to deliberately mainstem the ETT and withdraw it until bilateral breath sounds are heard. The ETT should be secured at a position 1-2 cm (depending on age) proximal to this point above the carina.[4]
(3) Pediatric tracheal and bronchial diameters correlate best with patient age rather than weight or height, while tracheal and bronchial length correlate slightly better with overall patient height.[5]

READ MORE
Practical pediatric anesthesia, Chapter 75

x) Single-lung ventilation
(1) Children under the age of 6 years old require a single-lumen mainstem intubation or a 5-Fr bronchial blocker.
(2) The smallest double-lumen ETT (26 Fr) has an outer diameter of 9.3 mm and is likely not appropriate for children under 8 years old.

xi) Laryngeal mask airways (LMAs)
(1) Sized primarily based on patient weight and can be utilized in infants as small as 3-5 kg
(2) In children 6 months to 6 years old, inserting the LMA backward with a partially inflated cuff and then rotating 180 degrees to an anatomic position provides the highest success rate and the lowest incidence of airway complications.[6]



In children 6 months to 6 years old, inserting the LMA backward with a partially inflated cuff and then rotating 180 degrees to an anatomic position provides the highest success rate and the lowest incidence of airway complications.[6]



The most reliable method of determining correct ETT depth is to deliberately mainstem the ETT and withdraw until bilateral breath sounds are heard.

b) Respiratory
i) Children have pliable ribs and increased chest wall compliance along with smaller airways, a reduced number of alveoli, and reduced parenchymal lung compliance.
(1) The resulting higher closing volumes and significantly reduced functional residual capacity are exaggerated with respiratory muscle relaxation under general anesthesia.

READ MORE
Practical pediatric anesthesia, Chapter 75

ii) Children have twice the oxygen consumption rate of adults (6-8 cc/kg/min compared to 3-4 cc/kg/min), providing limited reserve during periods of apnea.
(1) Accordingly, true rapid sequence induction is rarely utilized in pediatric anesthesia (eg, pyloric stenosis, small bowel obstruction).
(2) "Controlled/modified rapid sequence induction and intubation" utilizing gentle facemask ventilation (maximum peak inspiratory pressures of 10-12 cmH_2O) may reduce the incidence of significant oxygen desaturation without

increased risk of pulmonary aspiration even in children with a suspected full stomach. Reverse Trendelenburg positioning, nasogastric suctioning in patients with intestinal obstruction, and complete skeletal muscle paralysis are likely more important variables than gentle ventilation.[7]

iii) Neonates, particularly premature neonates, have an underdeveloped central respiratory drive and may react to hypercarbia and hypoxemia with apnea.
   (1) Opioids should be used judiciously in this population, and questionable neonates should be admitted to the intensive care unit for apnea monitoring.



All intravenous tubing and injectates must be meticulously deaired, as the incidence of intracardiac shunt and relative intravascular air volumes are higher in the pediatric population.

c) **Cardiovascular**
   i) The pediatric autonomic nervous system is parasympathetic dominant, and children frequently respond to noxious stimuli with bradycardia.
   ii) Infants and neonates have a relatively noncompliant left ventricle that cannot increase contractility to increase cardiac output.
   iii) Hypocalcemia is poorly tolerated and should be evaluated and corrected in hypotensive pediatric patients.
   iv) Blood pressure is typically preserved in mild to moderately hypovolemic pediatric patients, and systemic hypotension is a late and ominous sign (ie, 35%-40% blood volume loss).



Reliable locations for "blind" access are the saphenous vein just anterosuperior to the medial malleolus, the median cubital vein in the antecubital fossa, and the dorsal hand vein between the fourth and the fifth carpal bones.

d) **Vascular access**
   i) Intravenous access can be challenging in pediatric patients, and "blind" techniques may be necessary.
   ii) Reliable locations for "blind" access are the saphenous vein just anterosuperior to the medial malleolus, the median cubital vein in the antecubital fossa, and the dorsal hand vein between the fourth and the fifth carpal bones.
   iii) Intraosseous access is more precarious but may be obtained with an intraosseous or Tuohy needle in the proximal tibia, distal tibia, or distal femur.
   iv) Central venous access can also be difficult in pediatric patients, but the use of ultrasound has been shown to markedly increase success rates while reducing complications.[8]
   v) The Valsalva maneuver is the most effective technique to increase the cross-sectional area of the internal jugular or the femoral veins in children,[9] while inguinal compression will also effectively augment the femoral vein.[10]
   vi) Guidewire-assisted radial artery cannulation may be more efficient and successful than a direct cannulation technique in pediatric patients.[11]



Bradycardia is poorly tolerated in pediatric patients. Have a low threshold to treat with atropine, glycopyrrolate, or epinephrine early.

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Practical pediatric anesthesia, Chapter 75

e) **Hepatorenal**
   i) Neonates have relatively increased total body water and reduced plasma protein concentration.
   ii) Alpha 1-acid glycoprotein binds to amide local anesthetics and moderates local anesthetic systemic toxicity, but serum levels do not reach adult values until 10-12 months of life.
   iii) Hepatic P-450 enzymes mature over months to years, and glucuronidation does not mature until 6 months of life.
   iv) Importantly, amide local anesthetics, morphine, barbiturates, and diazepam are cleared more slowly in infants than older children.[12]

v) Morphine should be used with caution in unmonitored infants <6 months old.



Morphine should be used with caution in unmonitored infants <6 months old.

vi) Glomerular filtration and renal concentrating ability do not mature until 5-6 months of life, and excessive intravascular sodium cannot be effectively excreted. Postoperative hyponatremia is common; therefore, intraoperative fluid resuscitation should be made with an isotonic balanced salt solution.

vii) As hepatic glycogen stores are minimal during the first few months of life, prolonged fasting in neonates mandates blood glucose testing and/or supplemental IV dextrose at a rate of 6-8 mg/kg/min.



Maintenance fluids are slightly hypotonic at a rate per hour as calculated by the "4-2-1 rule" (4 mL/kg for the first 10 kg, 2 mL/kg for the subsequent 10 kg, and 1 mL/kg for each kg thereafter).

viii) In general, all children <3 months old and those with significant liver disease, sepsis, or prolonged parenteral nutrition should have intraoperative glucose supplementation.

f) **Hematologic**

i) Physiologic anemia occurs between the second and the 3rd months of life, with a hemoglobin nadir around 11-11.5 g/dL.

ii) Estimated blood volumes are around 100 mL/kg for premature neonates, 90 mL/kg for term neonates, 80 mL/kg for infants, and 70-75 mL/kg for small children.

iii) Red blood cell transfusions in neonates and immunocompromised children should be cross-matched, leukoreduced, and irradiated. Other children require only cross-matched and leukoreduced red blood cells.

iv) Stored blood may have elevated plasma potassium levels, but small-volume transfusions very rarely cause hyperkalemia.

(1) In the event of anticipated massive transfusion, fresh (<7 days from collection) or washed red blood cells may be requested from the blood bank.

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g) **Gastrointestinal**

i) Compared with adults, pediatric fasting gastric contents are of larger volume (on a mL/kg basis) and of lower pH.[13]

ii) The overall incidence of pediatric perioperative pulmonary aspiration is ~1:2000-1:3000 anesthetics, with 80% occurring during induction of anesthesia. Surprisingly, morbidity associated with pediatric pulmonary aspiration is very low, and patients not requiring supplemental O_2 within 2 hours of an aspiration event may be safely discharged home without sequelae[14] **(Table 74-1).**

TABLE 74-1 Pediatric NPO Guidelines

	American Society of Anesthesiologists Fasting Guidelines	Children's Hospital of Philadelphia Fasting Guidelines[15]
Clear liquids	2 h	2 h
Breast milk	4 h	3 h
Infant formula	6 h	4 h if < 6 mo old 6 h if ≥ 6 mo old
Nonhuman milk	6 h	6 h
Light snack	6 h	6 h
Large meal, fats, meats	8 h	8 h

h) **Dermatologic**
   i) Pediatric patients dissipate heat readily during anesthesia as a result of their relative large body surface area, thin skin, and insufficient fat stores.
   ii) Forced air warmers, heating lamps, and warmed IV fluids are important in pediatric temperature maintenance. Operating room temperatures may be raised from 75°F to 80°F.



Pediatric patients dissipate heat readily during anesthesia as a result of their relative large body surface area, thin skin, and insufficient fat stores.



Hypothermia may lead to delayed awakening, apnea, coagulopathy, insufficient reversal of neuromuscular blockade, and delayed drug metabolism.

2) **Preoperative evaluation**
   a) **Separation anxiety**
      i) During normal development, children become distressed when removed from their caregivers at around 8-9 months of life.
      ii) Oral midazolam at a dose of 0.3-0.5 mg/kg (maximum 20 mg) is an effective sedative and anxiolytic when given 10-30 minutes before induction.
      iii) Parental presence at induction (PPI)
         (1) Surprisingly, as compared with midazolam, parental presence during induction of anesthesia does not appear to alleviate the objective or the subjective anxiety in either the parent or the child.[16]
         (2) PPI appears to increase parental satisfaction with the anesthetic, provided they are forewarned of the peculiar ocular and body movements observed during stage II anesthesia (**Table 74-2**).
   b) **Preoperative labs**[12]
      i) Routine preoperative laboratory testing of pediatric patients is not indicated. However, certain disease states warrant further evaluation.
      ii) Former premature infants <56 weeks postconceptual age (PCA) should have a preoperative hematocrit, as a value <30 may be associated with apnea after anesthesia.
      iii) Pediatric patients with sickle cell disease should have a preoperative hemoglobin electrophoresis and hematocrit, as percentage of hemoglobin S >40% or Hct < 30 are risk factors for sickle crisis.
      iv) Hemophiliac patients should have preoperative factor VIII or IX level drawn to assist in factor replacement therapy.
      v) Patients with diabetes mellitus should have preoperative glucose checked.
      vi) Patients with diabetes insipidus should have preoperative electrolytes documented.
   c) **Congenital heart disease**
      i) Preoperative consultation with a pediatric cardiologist or a pediatric cardiac anesthesiologist is strongly recommended to

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Congenital heart disease, Chapter 85

TABLE 74-2 Pediatric Premedication

Premedication	Suggested Dose (mg/kg)
Midazolam (oral)	0.25-0.5
Midazolam (intranasal)	0.2-0.3
Midazolam (intravenous)	0.05-0.1
Ketamine (oral)	3-6
Ketamine (intramuscular)	2-3
Ketamine (intravenous)	0.5
Clonidine (oral)	0.002-0.004
Methohexital (rectal)	25

address the cardiac anatomy, function, and altered physiology in patients with congenital heart disease.

ii) Recent (ie, within the last 3-6 months) cardiac catheterization and echocardiography reports should be obtained.

d) **Asthma**

i) The patient's home medication schedule, known triggers, and overall control should be assessed.

ii) Prior emergency room visits, endotracheal intubation or intensive care admissions, and systemic corticosteroid use are markers of more severe disease.

iii) Parents should be questioned about familiarity with nebulizer masks or metered-dose inhalers.

iv) Home medications should be taken preoperatively, even in asymptomatic patients.

v) Airway management may consist of an LMA or ETT.

(1) Laryngeal masks may be less likely to induce bronchospasm but will be ineffective in delivering high positive airway pressures, if required.

(2) Adequate anesthesia is critical during intubation, and consideration should be given to deep extubation.

vi) In children with reactive airways, sevoflurane is a more effective bronchodilator than desflurane at 1 minimal alveolar concentration (MAC).[18]

vii) Inhaled beta-agonists and IV corticosteroids should be readily available.



Laryngeal masks may reduce the incidence of respiratory complications as compared to endotracheal intubation and may be safely utilized 2 or more weeks following a URI.[17]

e) **Upper respiratory infection (URI)**

i) Recent (within 2-4 weeks) or concurrent URI in children increases the perianesthetic risk of oxygen desaturation, breath-holding, laryngospasm, bronchospasm, and coughing.

ii) Independent risk factors for respiratory complications include endotracheal intubation in a child <5 years old, history of prematurity, history of reactive airway disease, paternal smoking, airway surgery, presence of copious secretions, and presence of nasal congestion[19] (**Fig. 74-1**).

f) **Ex-premature infant**

i) The care of an ex-premature infant must include documentation of the patient's gestational and postconceptual age (PCA [current chronologic age corrected for prematurity]) and consideration of associated comorbid diseases (eg, patent ductus arteriosus, intraventricular hemorrhage, necrotizing enterocolitis, chronic lung disease).

ii) A history of prolonged intensive care, endotracheal intubation, or O_2 therapy should alert the anesthesiologist to an increased risk of potential airway (eg, stenosis/malacia) or respiratory complications.

iii) As mentioned above, a preoperative hematocrit should be evaluated.

iv) During the neonatal period, the patient's oxygen saturation should be maintained between 88% and 93% to minimize the O_2-mediated risk of retinopathy.[20]

v) Postanesthetic apnea is a concern in newborn and former premature infants.

(1) Most postanesthetic apnea occurs within 12 hours of discontinuation of anesthesia, and ex-preterm infants <44 weeks PCA display the highest risk.[21]

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Anesthesia for the premature infant, Chapter 89

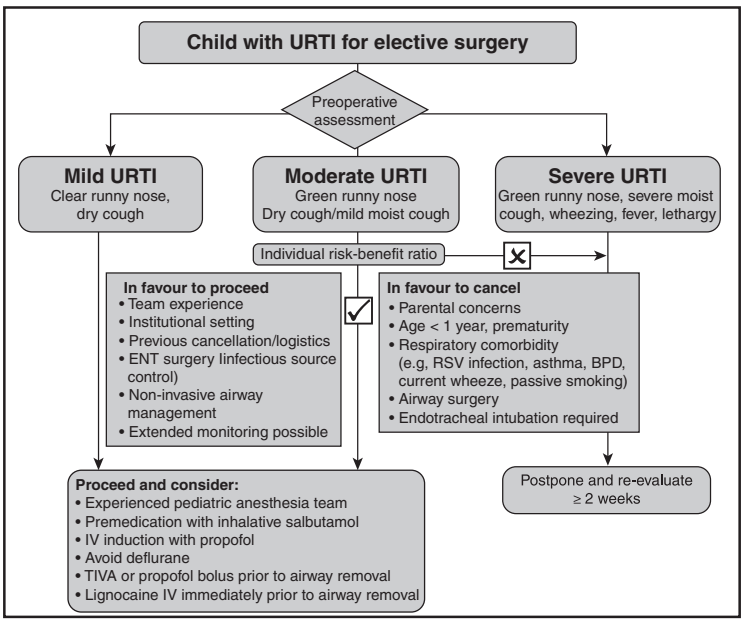


Postanesthetic apnea is a concern in newborn and former premature infants.



Ex-premature infants <60 weeks PCA are at risk for postoperative apnea and bradycardia.

FIGURE 74-1 Decision Tree for a Child With URI.



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(2) Very conservative studies recommend continuous apnea monitoring (pneumocardiography) for all ex-premature infants <60 weeks PCA, for a duration of 36 hours if <45 weeks PCA or 24 hours if >45 weeks PCA.[22]

(3) The risk decreases to <1% after 56 weeks PCA for ex-preterm infants and 44 weeks PCA for term infants, and many institutions have instituted these thresholds for 24-hour postoperative admission and monitoring.

3) **Induction, maintenance, and emergence**

a) **Inhalational induction**

i) For most pediatric patients at low risk for pulmonary aspiration, an inhalational mask induction may be better tolerated than an awake IV placement and induction.

ii) However, children with multiple risk factors for perioperative respiratory events (URI ≤ 2 weeks, wheezing ≤ 12 months, exercise-induced wheezing, nocturnal dry cough, eczema, passive smoking, family history of asthma or eczema) are significantly more likely to have minor and serious perioperative respiratory adverse events when induced with sevoflurane as compared with intravenous propofol.[23]

iii) Nitrous oxide may marginally reduce the inhaled induction time with sevoflurane, but it minimizes the preoxygenation reserve of the patient in the event the airway becomes compromised.

iv) Pediatric patients may better tolerate the mask adjacent to their face during induction, with the mask closely approximated only after they are asleep.

(1) Agitated patients are best managed with the clinician's hand securely fitting the mask to the face, while the other hand is stabilized against the patient's occiput.

(2) Gentle bag-mask assistance of the patient in sync with their respiratory pattern will help the progress through stage II anesthesia.

v) For anticipated awake IV access, EMLA (lidocaine 2.5%/prilocaine 2.5%) cream (APP Pharmaceuticals, Schaumburg, IL, USA) may be beneficial. It should be preemptively applied at multiple sites, covered with an occlusive dressing, and allowed 60 minutes to work. Alternatively, LMX (lidocaine 4%) cream may be applied in a similar fashion and allowed 30 minutes to work (**Table 74-3**).



Succinylcholine is not indicated for routine use in pediatrics, because of the risk of undiagnosed myopathy and hyperkalemic cardiac arrest.

b) **Skeletal muscle relaxation**

i) Succinylcholine is contraindicated for routine use in pediatric patients because of the risk of rhabdomyolysis, hyperkalemia, and cardiac arrest in patients with undiagnosed skeletal muscle myopathy.

ii) The emergency pediatric intubating dose of succinylcholine is 2 mg/kg IV or 4 mg/kg IM.

iii) The refractory laryngospasm treatment dose for succinylcholine is 0.1 mg/kg IV.[25]

iv) As neonates and infants are particularly prone to bradycardia, succinylcholine should be coadministered with atropine 0.01-0.02 mg/kg IV or IM.

v) Pediatric patients are routinely intubated without skeletal muscle relaxation.

(1) Deepening an inhaled anesthetic with intravenous propofol (1-3 mg/kg) OR remifentanil (3-5 µg/kg for neonates/infants, 1-2 µg/kg for older children) AND atropine (0.01-0.02 mg/kg) or glycopyrrolate (0.004-0.01 mg/kg) are appropriate choices to assist with laryngoscopy.

vi) Topical lidocaine (2-4 mg/kg) to the vocal cords and subglottis may blunt the coughing and laryngospasm reflexes during intubation.

vii) As in adults, nondepolarizing muscle relaxants are dosed according to weight and should be monitored and reversed according to train-of-four monitoring.

(1) Of note, vecuronium is a long-acting muscle relaxant in neonates and infants, with only 10% recovery of neuromuscular function at 60 minutes.[12]

viii) Neuromuscular reversal of all nondepolarizing relaxants is strongly encouraged, as children are at an increased risk of postoperative complications.

(1) Sugammadex is not currently approved by the FDA for use in pediatric patients, but a meta-analysis of off-label pediatric use studies suggests similar reversal characteristics and side effect profiles as seen with adult patients using the 2 and 4 mg/kg doses.[26]

TABLE 74-3 % Vapor to Achieve One Minimum Alveolar Concentration (MAC) as a Function of Age[24]

Age	Approximate Sevoflurane (%)	Approximate Isoflurane (%)	Approximate Desflurane (%)
Preterm	<2.5	1.3	<9.3
Neonate	3.2	1.6	9.3
6 mo old	2.5	1.8	9.9
12 mo old	2.6	1.7	8.7
2 y old	2.5	1.6	8.5
6 y old	2.5	1.5	8.2

c) **Deep extubation**
   i) Unless contraindicated by aspiration risk, altered mental status, or difficult mask/intubation, deep tracheal extubation may be appropriate for pediatric patients.
   ii) The clinician should be comfortable with managing a pediatric airway, and appropriate personnel and equipment should be immediately available to monitor and assist with complications during emergence.
   iii) No difference in supplemental O_2 requirements or airway-related complications was apparent between awake and deep tracheal extubation in elective cases.[27]
   iv) Laryngospasm has an incidence of 0.1%-1.7%. Failure to effectively recognize and treat laryngospasm may result in hypoxemia, bradycardia, or negative pressure pulmonary edema.



Laryngospasm should be aggressively treated with sustained positive airway pressure, followed by deepening of anesthesia with IV agents, muscle relaxation, and possible tracheal intubation.[28]

d) **Stridor**
   i) Postextubation stridor may be a result of edema (ie, sustained coughing or an inappropriately sized ETT), inflammation (ie, URI), or glottic secretions/foreign body.
   ii) Initial management should include supplemental O_2 (consider positive pressure) and dexamethasone (0.5 mg/kg IV).
   iii) Nebulized racemic epinephrine (0.5 mL of 2.25% solution in 2.5 mL normal saline) may help reduce tissue edema but mandates a 4-hour posttreatment observation period because of the risk of rebound edema and stridor.
   iv) Refractory stridor may necessitate tracheal reintubation with a smaller ETT and possible otolaryngology evaluation.

e) **Emergence agitation**
   i) Across multiple studies, emergence agitation in pediatric patients receiving volatile anesthesia is ~30%.
   ii) Desflurane, sevoflurane, and isoflurane appear to be of higher risk than halothane or propofol.[29,30]
   iii) Propofol (1 mg/kg IV) at the end of surgery may reduce the incidence of emergence agitation.[31] Dexmedetomidine (0.25-0.5 µg/kg IV over 10 minutes) is also very effective with a low incidence of cardiopulmonary complications.
   iv) Hypoxemia, hypercarbia, and urinary retention should be excluded as contributing factors to emergence agitation.
   v) Addressing this issue preoperatively with the family may help preempt escalation of parental anxiety in this situation.



Propofol 1 mg/kg IV given at the end of surgery may reduce the incidence of emergence agitation.

f) **Postoperative nausea and vomiting**
   i) Postoperative vomiting is rare in neonates and infants. The risk increases from 3 years of age until adolescence.
   ii) Strabismus surgery, middle ear surgery, and adenotonsillectomy are associated with a high risk of postoperative nausea and vomiting.
   iii) The combination of dexamethasone (0.15-0.5 mg/kg IV) and 5-hydroxytryptamine receptor antagonist (eg, ondansetron 0.1-0.15 mg/kg IV) provides a 50%-60% relative risk reduction.[32] Metoclopramide is not a reliable antiemetic in children and is not recommended for the management of postoperative nausea and vomiting.[33]



Acetaminophen may be given rectally 35-40 mg/kg as a one-time loading dose.

4) **Pain management**

a) **Non narcotic analgesics**
   i) Acetaminophen may be given IV or orally (15 mg/kg every 6 hours) or rectally (35-40 mg/kg one-time loading dose THEN 15 mg/kg/dose) and is a mild to moderately effective analgesic.

ii) Maximum dosages of acetaminophen are 60 mg/kg/24 hours in infants under 3 months old and 90 mg/kg/24 hours for older children.

iii) Acetaminophen suppositories are uniformly distributed medications and may be manually divided, but clinicians are notoriously inaccurate in their estimation.

iv) Other effective adjunct analgesics include
   (1) Ibuprofen (10 mg/kg po or IV every 6 hours–max 40 mg/kg or 2400 mg IV)
   (2) Ketorolac (0.5 mg/kg IV, maximum 30 mg)

v) Aspirin and aspirin-containing products should not be routinely given to children under the age of 19 years old because of the risk of developing Reye syndrome.



To avoid overdosing, families must be alerted to combination medications that contain acetaminophen (eg, Tylenol No. 3, Tylenol No. 4, Lortab, Vicodin, Percocet, and other over-the-counter products).

b) **Narcotics**

i) Most synthetic and semisynthetic opioids have been successfully used in pediatric patients.

ii) Methadone and morphine should be used cautiously in unmonitored neonates and infants due to their impaired clearance.

iii) Continuous opioid infusions in intubated neonates should be preservative-free formulations.

iv) Commonly administered parenteral opioids include
   (1) Morphine (0.05-0.1 mg/kg IV)
   (2) Fentanyl (0.0005-0.001 mg/kg IV = 0.5-1 μg/kg IV)
   (3) Hydromorphone (0.01-0.015 mg/kg IV)

v) Commonly administered oral opioids include
   (1) Oxycodone elixir (0.05-0.15 mg/kg)
   (2) Hydrocodone (with acetaminophen) elixir (0.1-0.15 mg/kg of hydrocodone)
   (3) Codeine and tramadol are contraindicated to treat pain in any patient under 12 years of age and contraindicated after adenotonsillectomy in patients under 18 years of age.



Clinician should decide appropriate interval for redosing medication based on clinical situation and patient condition.

c) **Regional anesthesia**

i) Regional anesthesia has been employed with increasing frequency in pediatric patients, particularly since the introduction of ultrasound-guided techniques.

ii) The documented regional anesthesia complication rate in pediatric patients is 0.09% evaluated over 24 000 anesthetics.[34]

iii) Local anesthetic toxicity must be considered in all pediatric patients. The weight-based acceptable doses of local anesthetic must be calculated.
   (1) The pediatric intralipid dosing regimen for local anesthetic toxicity has not been established. Weight-based adult dosing regimens are currently recommended. (1.5 mL/kg of 20% lipid over 2-3 minutes followed by 0.25 mL/kg/min infusion for 30-60 minutes. The 1.5 mL/kg bolus may be repeated twice, and the maximal recommended infusion is 0.5 mL/kg/min. Total recommended maximum dose 12 mL/kg.)

iv) Neonates and infants have lower serum levels of alpha-1-acid glycoprotein than adults and thus have higher unbound amide local anesthetic levels for equivalent doses. In addition, the time to peak serum levels after injection is prolonged in infants and small children.



READ MORE
Caudal epidural anesthesia, Chapter 95; Atlas of peripheral nerve block procedures, Section XV



The maximum subcutaneous dose of bupivacaine in infants <3 months old is 1.5-2 mg/kg. After 3 months, the maximum is 2.5 mg/kg.

v) As in adults, continuous epidural infusions may be performed at the caudal, lumbar, and thoracic levels.
   (1) Radiographic or ultrasound confirmation of the final catheter location is recommended.
   (2) The distance from skin to epidural space in the lumbar spine of children 6 months to 10 years old is ~1 mm/kg body weight.[35]
   (3) Epidural test dose should be performed with 0.5 µg/kg epinephrine.
   (4) Bolus dose should be approximated at 0.04-0.05 mL/kg/dermatome level.



The distance from skin to epidural space in the lumbar spine of children 6 months to 10 years old is ~1 mm/kg body weight.[35]

vi) Peripheral nerve blocks are safe and efficacious in pediatric patients.
   (1) The dose for individual blocks varies greatly by location and technique (eg, loss of resistance, ultrasound guided, stimulator guided).
   (2) Most peripheral nerves can be successfully anesthetized with 0.2-0.3 cc/kg or less of local anesthetic.
   (3) In general, pediatric nerve blocks are shorter in duration than adult blocks.

CHAPTER SUMMARY FOR PEDIATRIC ANESTHESIA OVERVIEW

Overview	Pediatric anesthesia is a specialized practice of anesthesiology that requires specific skills and knowledge of anatomy, pathophysiology, and procedures unique to children and infants.
Airway Anatomy	Pediatric patients can have long and floppy epiglottis and the larynx is more anterior and cephalad. Using a straight blade and having patient in a sniffing position can help with intubation.
Preoperative Issues	Separation anxiety begins after 8-9 mo of life. Consider oral midazolam 0.3-0.5 mg/kg (max 20 mg) for sedation and anxiolysis. Presence of a parent at induction does not appear to reduce anxiety compared to midazolam but may increase parental satisfaction provided they are given appropriate preparation.
Intraoperative Issues	Most pediatric patients have a low risk of pulmonary aspiration. Inhalational mask induction is generally better tolerated than awake IV placement and IV induction. Succinylcholine is contraindicated for routine use in pediatrics.
Postoperative Issues	Postextubation stridor may be due to edema, inflammation, or glottic secretions/foreign body and may be treated with steroids, nebulized racemic epinephrine, and supplemental oxygen. Laryngospasm should be aggressively treated with sustained positive airway pressure, deepening anesthesia with IV agents, and muscle relaxation. Endotracheal intubation may be necessary.

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75 Practical Pediatric Anesthesia

Michael Lennig • Ellen Choi • Calvin Kuan

The practice of pediatric anesthesia is marked by a variety of differences from the practice of adult anesthesia. Physiology, comorbidities, and socio-emotional needs are uniquely different in the pediatric patient. Their smaller stature and less mature organ systems make for a broad spectrum of normal vital signs as well as special considerations for appropriate equipment sizing and drug dosing. This chapter will attempt to provide a concise collection of information to address the most basic differences in the pediatric population. In addition, various clinical pearls are provided to enhance the quality and safety of care for the pediatric patient.

Normal Pediatric Vital Signs (Table 75-1)

Pediatric Airway

1) **Routine airway management**
   a) **Endotracheal intubation** (**Table 75-2**)
      i) Smallest cuffed ETT available is 3.0.
      ii) Smallest UNcuffed ETT available is 2.0.
      iii) Formula for calculating approximate UNcuffed ETT size:

(Age in years/4) + 4

      iv) Formula for calculating approximate CUFFED ETT (=0.5 size smaller than uncuffed for similar age) size:

TABLE 75-1 Pediatric Vital Signs and Approximate Weight

Age	Weight (kg)	Heart Rate	Systolic Blood Pressure	Diastolic Blood Pressure	Respiratory Rate
Premature infant	<3.0	120-170	55-75	35-45	45-60
Term neonate	3.5	100-150	65-86	40-45	40-60
6 mo	7	90-120	70-90	50-65	30-50
1 y	10	80-120	80-100	55-65	30-50
2 y	12	70-110	90-100	55-70	25-40
3 y	14	70-110	90-110	55-70	25-35
4 y	16	65-110	90-110	60-75	25-35
5 y	18	65-100	90-110	60-75	20-30
6 y	20	65-100	90-110	60-75	20-30
8 y	25	60-90	100-120	60-75	20-30
10 y	30	60-90	100-120	60-75	20-25
12 y	40	60-90	100-120	60-75	<20
14 y	50	55-85	110-135	60-75	<20
Adult	70	55-85	110-135	60-75	<20

(Age in years/4) + 3.5

Quick formula to estimate weight: weight (kg) = (age in years × 2) + 9

v) Quick rule of thumb for appropriate size of ETT suction catheter and the depth at which to tape the ETT for premature infants and neonates: "Weight 1, 2, 3… Suction catheter 4, 5, 6… Distance 7, 8, 9" (**Table 75-3**).

vi) Formula for approximate ETT depth from lip/gums to mid-trachea for oral intubation:

Size of ETT × 3 = cm marking on ETT at lips/gums

Rule of thumb for lowest acceptable mean arterial pressure (MAP) in the neonatal period = approximate gestational age, for example, a 2-week-old full-term (40 week) infant should have MAP around 42 mm Hg (=40 + 2).

vii) The most reliable method for determining appropriate position for an oral or nasal ETT is the mainstem method[1]:

(1) After tracheal intubation has been confirmed and while continuing to hand ventilate, advance the ETT while listening with stethoscope over the left chest. The depth at which the breath sounds disappear indicates the transition from carina to right mainstem bronchus (in patients with normal situs solitus). Take note of the depth of the ETT at this point.

(2) Then continue to listen over the left chest as you pull back the ETT for return of breath sounds indicating the transition of the ETT from mainstem bronchus to trachea. This depth should be within 0.5 cm of the previous depth.

(3) Position the ETT above the carina 1-3 cm depending on patient age and surgical situation. We recommend 0.5-1 cm above carina for neonates/infants; 1-2 cm for toddlers; and 2-3 cm for older children.

(a) For abdominal or thoracic surgeries, the position of the ETT may change with manipulation of abdominal contents, diaphragm, or heart/lungs.

Formula for approximate UNcuffed ETT size: (age in years/4) + 4.

The most reliable method for determining correct ETT depth is the mainstem intubation method.

Quick method for initial approximation of ETT depth: size of ETT × 3 = distance from lips/gums to midtrachea.

viii) Checking for a leak around ETT:

(1) For most healthy children with normal lung compliance, the leak around the ETT should be no greater than 20-25 cm H_2O to avoid tracheal mucosa ischemia.

(2) A leak <10-15 cm H_2O with an UNcuffed ETT may necessitate a change to a larger size or a cuffed ETT to adequately ventilate.

(3) To check for a leak:

(a) With a stethoscope, auscultate the upper airway at the mouth or neck.

TABLE 75-2 Laryngoscope Blades and Sizes

Age	Miller	Wis-Hipple	MacIntosh
Preterm	0[a]		
Full term	0 or 1[a]	1	
1 y	1	1 or 1.5[a]	1
2 y	1 or 2	1.5[a]	1 or 2
3–5 y	2	1.5[a]	2[a]
6–10 y	2[a]		2 or 3
>10 y	2[a]		3[a]

[a]Recommended for first attempt.

TABLE 75-3 Suction Catheter and Depth of ETT Placement for Premature Infants

Weight (kg)	Size of Suction Catheter for ETT (Fr)	Distance from Gums to Mid-Trachea (cm)
1	4 (or 5)	7
2	5 (or 6)	8
3	6	9

(b) Turn the APL valve on the anesthesia machine to the maximal accepted peak pressure (30-40 cm H_2O).
(c) Allow the airway pressure to rise to desired peak pressure.
(d) Watching the manometer, note the pressure at which air is first heard "leaking" around ETT in the airway.

ix) Special anesthetic considerations:
(1) The ETT size and target leak for an individual case should take into consideration the patient's pulmonary compliance and the likelihood of change of compliance with surgery or disease process. For example, a child with worsening ARDS may require increased peak pressures to ventilate in the near future.
(2) Take care to ensure that gas flow is not too high relative to patient size to avoid overinflating lungs.
(3) Take care not to close the APL valve completely.
(4) If patient is unstable, squeeze the anesthetic bag to accelerate lung inflation and allow for quicker leak check.



For most children, the leak around the ETT should be no >20-25 cm H_2O. However, the clinician should take into consideration the patient's lung compliance and likelihood of change in compliance with surgery or disease process.

2) **Alternative airway equipment (Table 75-4)**
a) **Airway exchange catheters (AEC)** (Cook Medical, Inc., Bloomington, IN) (**Table 75-5**):
i) Used to facilitate exchange or replacement of ETT in an intubated patient.
ii) Used to stiffen a guidewire that has been passed through a fiberoptic bronchoscope for intubation through an LMA.
iii) Sizes 8-19 French have a Luer lock adaptor for jet ventilation connection or 15-mm ETT connector (Rapi-Fit Adaptor) to connect to a breathing circuit or an ambu bag.
iv) Connector may also be used to attach to $ETCO_2$ monitor for verification of endotracheal vs esophageal position.

READ MORE
Management of the pediatric difficult airway, Chapter 96.

b) **Single-lung ventilation (Table 75-6):**
i) Single-lung ventilation in the pediatric population can be challenging because of the difficulty in finding the appropriate tube or device for lung isolation in accordance with the patient's size without obstructing the upper airway.
ii) In many situations, endobronchial intubation may be easier and less time consuming than attempting to place and maintain a bronchial blocker when the patient is too small for a double-lumen tube.
iii) Fiberoptic bronchoscopy is useful to place and verify the position of bronchial blockers and double-lumen tubes, especially when access to auscultation is limited during surgery.

Vascular Access

1) **Central venous catheter insertion**[3]
a) See **Table 75-7** for formulae to determine length of catheter to insert based on patient height for central venous catheter placement via RIJ (right internal jugular) or RSC (right subclavian) vein.
b) **Special considerations for central venous catheter placement:**
i) Visualization with an ultrasound prior to cannulation is recommended to assess size, verify patency of the vein, and determine position of vein relative to artery.

TABLE 75-4 Difficult Airway Equipment: Laryngeal Mask Airway, Endotracheal Tube (ETT), Video Laryngoscopes, and Fiberoptic Bronchoscopes[2]

Patient Weight (kg)	Classic or Unique LMA Size[a]	Maximum Size ETT[b] That Will Fit Through LMA (Recommended by the Manufacturer)	Maximum Size ETT[b] That Will Fit Through LMA (Our Recommendation)	Maximum Fiberoptic Bronchoscope Size (mm)
<5.0	1.0	3.5 uncuffed	3.0 uncuffed	2.7
5.0-10.0	1.5	4.0 uncuffed	4.0 uncuffed[c]	3.0
10.0-20.0	2.0	4.5 uncuffed	4.5 uncuffed 4.5 cuffed[d]	3.5
20.0-30.0	2.5	5.0 uncuffed	5.0 uncuffed 5.0 cuffed[d]	4.0
30.0-50.0	3.0	6.0 cuffed	6.0 cuffed	5.0
50.0-70.0	4.0	6.0 cuffed	6.0 cuffed	5.0
70.0-100.0	5.0	7.0 cuffed	7.0 cuffed	5.0
Patient Weight (kg)	**Air-Q LMA Size[e]**	**Maximum Size ETT That Will Fit Through LMA (Recommended by the Manufacturer)**	**Maximum size ETT That Will Fit Through LMA (Our Recommendation)**	**Maximum Fiberoptic Bronchoscope Size (mm)**
4-7	1.0	4.5	4.5 cuffed	3.5
7-17	1.5	5.0	5.0 cuffed	4.0
17-30	2.0	5.5	5.5 cuffed	4.0
30-50	2.5	6.5	6.0 cuffed	5.0
50-70	3.5	7.5	7.0 cuffed	5.0
70-100	4.5	8.5	8.0 cuffed	5.0

Glidescope AVL (Advanced Video Laryngoscopy) Preterm/Small Child System	
Weight Range (kg)	**Laryngoscope Blade Size**
<1.5	GVL 0
1.5-3.6	GVL 1
1.8-10	GVL 2
10-28	GVL 2.5

C-MAC for Neonatology and Pediatrics/Video Laryngoscopes	
Blade Type/Style	**Blade Size**
Miller	0
Miller	1
MacIntosh	0
MacIntosh	2
"D"	NA

[a]LMA Classic and LMA Unique (LMA North America, Inc., San Diego, CA).
[b]Mallinckrodt Intermediate Hi-Lo ETT (Covidien-Nellcor and Puritan Bennett, Boulder, CO).
[c]Generous amounts of lubrication required to pass easily.
[d]The cuffed 4.5 and 5.0 ETT will fit into the shaft of the LMA, but the balloon will not fit through the LMA connector. Refer to Chapter 96: "Management of the Pediatric Difficult Airway" for more detailed discussion of alternative solutions.
[e]air-Q Disposable LMA (Cookgas, Saint Louis, MO).
Data from LMA Unique product brochure at www.lmaco.com (LMA North America, Inc., San Diego, CA).

TABLE 75-5 Airway Exchange Catheters

Catheter Size (Fr)	Catheter Length (cm)	Catheter ID (mm)	Smallest ETT Size (ID mm)
8	45	1.6	3.0
11	83	2.3	4.0
14	83	3.0	5.0
19	83	3.4	7.0

Permission for use granted by Cook Medical, Bloomington, Indiana.

ii) Position of the catheter should be confirmed with one or more of the following:
   (1) Ultrasound visualization of the catheter in the vein or right heart
   (2) Transducing a venous waveform
   (3) Confirmation with blood gas measurements
iii) Confirmation of position of tip of catheter can be made by chest radiograph or transesophageal echocardiographic visualization of tip in SVC or RA.

2) **Arterial access**
   a) **Arterial catheter size**
      i) Radial, ulnar, posterior tibial, or dorsalis pedis arteries
         (1) Patients <10 kg → 24 or 22 gauge
         (2) Patients 10-40 kg → 22 gauge
         (3) Patients >40 kg → 22 or 20 gauge
      ii) Femoral or axillary arteries
         (1) Patients <10 kg → 22 or 20 gauge (3-4 Fr)
         (2) Patients 10-40 kg → 18 or 16 gauge (4-5 Fr)
         (3) Patients >40 kg → 18, 16 or 14 gauge (5-6 Fr)
      iii) Umbilical artery
         (1) Patients <10 kg → 3.5-5 Fr



Use ultrasound to verify position, size, and patency of vessels prior to cannulation.

TABLE 75-6 Options for Single-Lung Ventilation[3]

Age (y)	ETT (ID in mm)	Bronchial Blocker (Fr)	Univent (ID in mm)	Double-Lumen Tube (Fr)
0.5-1	3.5-4.0	2-3	Not available	Not available
1-2	4.0-4.5	3	Not available	Not available
2-4	4.5-5.0	5	Not available	Not available
4-6	5.0-5.5	5	Not available	Not available
6-8	5.5-6	5	3.5	Not available
8-10	6.0 cuffed	5	3.5	26
10-12	6.5 cuffed	5	4.5	26-28
12-14	6.5-7.0 cuffed	7	4.5	32
14-16	7.0 cuffed	7	6.0	35-41[a]
16-18	8.0-8.5 cuffed	7-9	7.0	35-41[a]

[a]For adult-sized patients, double-lumen tube sizing should be based on tracheal diameter. Adapted with permission from Golianu B, Hammer GB. Pediatric thoracic anesthesia. *Curr Opin Anaesthesiol.* 2005;18:5-11.

TABLE 75-7 Central Venous Catheter Insertion Depth and Sizing[4]

Height (cm)		Formula
<100		**(Height [cm]/10) – 1 = distance (cm) to SVC/RA junction**
>100		**(Height [cm]/10) – 2 = distance (cm) to SVC/RA junction**
Weight (kg)	**Length (cm) of catheter insertion from skin to SVC/RA junction**	**Size (Fr) of central venous catheter for right IJ cannulation**
2.0-2.9	4	4
3.0-4.9	5	4
5.0-6.9	6	4
7.0-9.9	7	4
10-12.9	8	5 or 5.5
13.0-19.9	9	5 or 5.5
20.0-29.9	10	5 or 5.5
30.0-39.9	11	7
40.0-49.9	12	7
50.0-59.9	13	9
60.0-69.9	14	9
70.0-79.9	15	9
>80.0	16	9

Adapted with permission from Andropoulos D, Bent ST, Skjonsby B, et al. The optimal length of insertion of central venous catheters for pediatric patients. *Anesth Analg.* 2001;93:883-886.

Hematologic Considerations in the Pediatric Patient (Tables 75-8 and 75-9)

1) The amount of blood loss in a pediatric surgery will vary by surgical complexity and anatomy, urgency of the procedure, and patient comorbidity.
2) A vital consideration is the overall blood volume of the patient undergoing the procedure and the rapidity with which the patient will achieve a critical amount of blood loss.
3) Pediatric red cell transfusion should be calculated according to the following formula[5]:

Volume of PRBC to transfuse (mL) = 1.6 × weight (kg) × desired rise in Hct (%)

4) Maximum allowable blood loss (MABL):

MABL= [EBV x (starting Hct - minimal acceptable Hcg)] / starting Hct.

EBV = estimated blood volume (~70-100mL / kg depending on age)



A vital consideration is the overall blood volume of the patient undergoing the procedure and the rapidity with which the patient will achieve a critical amount of blood loss.

TABLE 75-8 Transfusion Estimates[6]

	Volume to Transfuse	Expected Change
PRBC	10-15 mL/kg	Increases Hgb by 2-3 or Hct by 6%-9%
Fresh frozen plasma	10-15 mL/kg	Increases factor level by 15%-20%
Platelets	10 mL/kg	Increases platelets by 50 000-100 000/μL
Cryoprecipitate	1-2 units/10 kg	Increases fibrinogen by 60-100 mg/dL

TABLE 75-9 Estimated Total Blood Volumes by Age[6,7]

Age	Approximate weight (50th percentile) (kg)	Blood Volume Estimate (mL)
Birth	3.5	300
2 mo	5	400
12 mo	10	800
3 y	14	1120
6 y	20	1400
12 y	40	2800
20 y	65	4550

TABLE 75-10 Commonly Used Drugs: Pediatric Dosing

Drug	Suggested Dose	Maximum Single Dose
Induction		
Propofol	2-3 mg/kg IV	
Ketamine	1-3 mg/kg IV 3-10 mg/kg IM	
Etomidate	0.2-0.3 mg/kg IV	
Muscle Relaxant		
Cisatracurium	0.1-0.2 mg/kg IV	
Rocuronium	0.6-1.2 mg/kg IV	
Vecuronium	0.1 mg/kg IV	
Succinylcholine	1-2 mg/kg IV 4-5 mg/kg IM For laryngospasm: 0.1 mg/kg IV	
Muscle Relaxant Reversal Agents[8]		
Sugammadex[9]	For rocuronium and vecuronium: 2-4 mg/kg IV For rocuronium only: 16 mg/kg IV for rapid reversal of RSI dose	
Neostigmine	0.05-0.07 mg/kg IV	
Glycopyrrolate	0.01-0.015 mg/kg IV	
Edrophonium	0.3-1 mg/kg IV	
Benzodiazepines		
Midazolam	0.1 mg/kg IV 0.5-1 mg/kg PO	20 mg PO
Opioids		
Fentanyl	1 µg/kg IV	
Morphine	0.1 mg/kg IV	
Hydromorphone	10-20 µg/kg IV	
Naloxone (opioid reversal)	0.25-0.5 µg/kg titrating to effect	2 mg; can give up to 100 µg/kg

(*Continued*)

TABLE 75-10 Commonly Used Drugs: Pediatric Dosing (Continued)

Drug	Suggested Dose	Maximum Single Dose
Other Analgesics		
Ketorolac	0.5 mg/kg IV	60 mg
Acetaminophen	30-40 mg/kg PR single loading dose, then 15 mg/kg PO/PR IV formulation (Ofirmev): Newborns and children <2 yo: 15 mg/kg IV q8h or 12.5 mg/kg IV q6h Children >2 yo: 15 mg/kg IV q6h or 12.5 mg/kg IV q4h	1000 mg; watch total daily dose
Ibuprofen	10 mg/kg PO q6h 10 mg/kg IV q4-6h	800 mg 40 mg/kg or 2400 mg, whichever is less
Antiemetics		
Dexamethasone	0.1-0.2 mg/kg IV	20 mg
Ondansetron	0.1 mg/kg IV	4 mg
Metoclopramide	0.15 mg/kg IV/PO	
Antibiotics		
Cefazolin	30 mg/kg IV q3h during surgery	2000 mg
Steroids		
Dexamethasone	*Airway edema*: 0.5-2 mg/kg/day IV, IM, or PO, divided into q6h beginning 24 h before extubation and continuing for 4-6 doses afterwards *Physiologic replacement*: 0.03-0.15 mg/kg/d divided into q6-12h dosing	
Hydrocortisone	*Acute adrenal insufficiency*: Older children: 1-2 mg/kg bolus and then 150-250 mg/d in q6-8h divided doses Younger children and infants: 1-2 mg bolus and then 25-150 mg/d divided into q6-8h doses	

Antifibrinolytic (usage with CPB)	Age and Weight Ranges	Dosing (Loading; Infusion; CPB Prime)
Tranexamic acid	a) 0-2 mo b) 2-12 mo c) >12 mo	a) 120 mg/kg; 16 mg/kg/hr; 0.15 mg/mL b) 65 mg/kg; 16 mg/kg/h; 0.15 mg/mL c) 30 mg/kg; 16 mg/kg/h; 0.15 mg/mL
Aminocaproic acid	a) 0-1 mo b) 1 mo to 50 kg c) >50 kg	a) 40 mg/kg; 30 mg/kg/h; 100 mg/L b) 75 mg/kg; 75 mg/kg/h; 250 mg/L c) 5 g; 10 mg/kg/h; 5 g total

Procoagulants	Dosing
FEIBA (anti-inhibitor coagulant complex)	Perioperative: 50-100 units/kg q6-12h; Intraoperative: conservative starting dose is 10 units/kg per dose incrementally to minimize risk of hypercoagulation.
Riastap (human fibrinogen concentrate)	Fibrinogen level unknown: 70 mg/kg Fibrinogen level known: [(target fibrinogen) − (measured fibrinogen)/1.7] × (patient weight in kilograms) = total dose

TABLE 75-11 Vasoactive Drugs for Infusion[a]

Drug	Dose
Dopamine	1-20 µg/kg/min
Dobutamine	1-20 µg/kg/min
Epinephrine	0.05-1 µg/kg/min
Isoproterenol	0.05-1 µg/kg/min
Norepinephrine	0.05-1 µg/kg/min
Milrinone	0.25-1 µg/kg/min
Phenylephrine	0.1-1 µg/kg/min
Vasopressin	0.0001-0.0005 units/kg/min
Clevidipine[10]	1-7 µg/kg/min

[a]All drugs should be titrated to effect.

Pharmacologic Considerations in the Pediatric Patient (Tables 75-10 and 75-11)

Most medication dosing in the pediatric population is done on a per-weight basis. The safe selection and preparation of medications for the pediatric patient continually involves vigilance with regard to dosage, concentration, and side effect profile making it unique from the practice of adult anesthesia. Using clinical pearls for the estimation of drug infusion volumes can help minimize waste by not using a larger vial when a smaller vial would be sufficient.

How to determine the volume of propofol to draw up for a case to minimize waste:

1) Convert patient weight in kg to mL of propofol (eg, for a 12-kg child, draw up 12 mL).
2) That amount will last 1 hour at a dose of 166 µg/kg/min.
3) Estimate the duration of the case, multiply the hours times the volume per hour for the patient, and you can determine the amount needed.
4) Knowing the amount will help decide whether to open a large or small vial and may help to minimize waste.

This formula can also be used for other drugs as long as the concentration is prepared in multiples of 10. Using the same example: for remifentanil if prepared as 10 µg/mL, 12 mL will last 1 hour at a dose of 0.166 µg/kg/min. For a drug prepared as 1 mg/mL, 12 mL will last 1 hour at a dose of 16.6 µg/kg/min.



Children are not just little adults? The safe selection and preparation of medications for the pediatric patient continually involves vigilance with regard to dosage, concentration, and side effect profile making it unique from the practice of adult anesthesia.



To help minimize waste, use this formula: for propofol (conc 10 mg/mL), taking the patient's weight in kg and draw up that volume of propofol in mL to be infused, the syringe will last 1 hour at a dose of 166 µg/kg/min. Example: for a 15 kg pt, a syringe of 15 cc of propofol will last for 1 hour at an infusion rate of 166 µg/kg/min.

Ensuring Safety in the Pediatric Patient

1) Avoid intravascular entrainment of air
   a) Preoperatively examine for and tap out all visible bubbles within the line with special care noted at each entry and connection point of the tubing. Bubbles are most effectively flushed when distal tubing (furthest from the fluid bag) is held at a level above proximal tubing (ie, the air bubble will float "up" the tubing).
   b) Allow fluid to flow to meet the tip of the female Luer tip of your manifold before attaching a syringe.
   c) Draw back on a syringe pulling from your fluid bag to dislodge and withdraw any air remaining hidden in your manifold.

d) Medications should be injected with a "plunger-highest" technique in order to assure that all air will rise away from the entry point to the tubing.

2) Constant preparation for medication injection for unforeseen developments
   a) A syringe of normal saline flush should be constantly connected in-line upstream from all other injection points so that a rescue drug can be administered with the least possible delay.
   b) A syringe of propofol attached to the IV tubing after emergence/extubation and throughout transport until the patient is fully awake in order to provide immediate treatment for laryngospasm or any other complications.



Debubble all lines and syringes before use. The most effective way is to flush the tubing holding the distal tubing at a level HIGHER than the proximal tubing so that air will float up.

Draw back on the syringe before injecting EVERY TIME you attach a new syringe to a port/stopcock, and inject with plunger higher than the injection port site to avoid injection of air.

Have a syringe of normal saline flush connect in line and upstream from all other injection points and medications so that a rescue drug can be administered with the least delay possible.

A syringe of propofol (or other rapid-acting agent) should be attached to the IV tubing after emergence/extubation and throughout transport until the patient is fully awake in order to provide immediate treatment for laryngospasm or other complications.

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76 Management of Pediatric Arrest and Resuscitation

Rania K. Abbasi • Samuel A. Mireles

There are significant differences in etiology and management of cardiac arrest between children and adults. This chapter will highlight key features of pediatric cardiac arrest management, as well as post-arrest management and resuscitation in special situations.

1) **Pediatric perioperative cardiac arrest**
   a) **Etiology**
      i) According to the Pediatric Perioperative Cardiac Arrest Registry, 49% of perioperative deaths during 1998–2004 were anesthesia related.[1]
      ii) Cardiovascular causes were the most common (41% of all perioperative arrests).
         (1) Hypovolemia from blood loss and transfusion-related hyperkalemia were the most common identifiable cardiovascular causes (12% and 5%, respectively).
      iii) Respiratory causes were the second most common cause (27% of arrests), with laryngospasm being the most common respiratory cause (6%).
      iv) Medication-related arrests were the third most common cause at 18%.
   b) **Presentation**
      i) Bradycardia, dysrhythmia, and shock can all occur secondary to respiratory failure.
      ii) The development of hypotension in a child with previously compensated shock is an ominous sign of impending arrest. The practitioner should be alert to signs of compensated shock and should not wait for hypotension to begin treatment.[2]
   c) **Prevention**
      i) Pediatric arrest may possibly be prevented by maintaining HR and oxygenation, particularly in patients with minimal cardiopulmonary reserve and higher parasympathetic tone, such as neonates and premature infants.
2) **Circulation (Table 76-1)**
   a) **Chest compressions**
      i) In an intubated child, a sudden drop in $ETCO_2$ may indicate decreased cardiac output. Quickly rule in or rule out airway issues, and simultaneously feel for a pulse.
      ii) Chest compressions are a critical part of resuscitation as they provide the only means of end-organ perfusion in the setting of inadequate cardiac output.
      iii) Careful attention should be paid to the rate (100-120/min) and depth of compressions, allowing time for adequate chest recoil. The upper rate of compressions (120) was recently added to prevent inadequate recoil with excessive compression rates.[4] It is important to minimize interruptions to ensure optimal perfusion.



49% of pediatric perioperative deaths in the US during 1998-2004 were anesthesia related.

READ MORE
Neonatal resuscitation, Chapter 113



The 2015 PALS guidelines recommend beginning with chest compressions ("C-A-B") rather than the previous emphasis on rescue breaths first ("A-B-C").[3]

TABLE 76-1 Summary of CPR Maneuvers Based on Age[2–4]

Maneuver	Adult (Adolescent or Older)	Child (1 y to Adolescence)[a]	Infant (<1 y)
Airway	Head tilt/chin lift (jaw thrust if trauma)		
Breathing: initial	Two breaths at 1 s/breath		
Breathing: subsequent (without advanced airway)	10-12 breaths/min	12-20 breaths/min	
Breathing: subsequent (with advanced airway)	8-10 breaths/min		
Foreign body	Abdominal thrusts		Back blows and chest thrusts
Circulation: pulse check	Carotid		Brachial or femoral
Compression landmarks	Lower half of sternum/between nipples		Lower half of sternum/ just below nipples
Compression method	Heel of one hand, other hand on top	Heel of one hand, or as for adults	2 thumbs of encircled hands (2 providers) or 2-3 fingers
Compression depth[b]	2 in	>2 in but no more 6 in	1.5 in
Compression rate	100-120/min		
Compression/ventilation ratio	30:2	30:2 (single rescuer) 15:2 (2 rescuers)	
Defibrillation AED	Adult pads	Use pediatric system for 1-8 y, if available	

[a]Note that pediatric hospitals and intensive care units may choose to continue the use of PALS for adolescents as well as younger children.
[b]If an arterial line is in place, use presence and shape of arterial waveform to judge effectiveness of compressions.

iv) If a functional arterial line is in place, use presence and shape of arterial waveform to judge effectiveness of compressions.

b) **Defibrillation—for pulseless ventricular tachycardia (VT) and ventricular fibrillation (VF)**
   i) Use infant paddles for children <10 kg.
   ii) Use adult paddles for children ≥10 kg.
   iii) A dose of 2 J/kg is recommended for the first shock (monophasic or biphasic): ASYNCHRONOUS.[5]
   iv) A dose of 4 J/kg is recommended for subsequent shocks (monophasic or biphasic): ASYNCHRONOUS.[5]

c) **Cardioversion—for atrial tachycardias, SVT, or symptomatic VT with pulse present**
   i) A dose of 0.5-1 J/kg is recommended (monophasic or biphasic): SYNCHRONOUS with QRS.
   ii) Energy for subsequent shocks is NOT escalated.



To determine whether compressions are resulting in adequate cardiac output, assess for (a) presence of $ETCO_2$; (b) BP tracing on arterial line; and (c) palpable femoral, brachial, or carotid pulses.

d) **IV Fluids**
   i) According to the 2015 PALS guidelines, children in shock (presumably relatively hypovolemic) should receive a bolus of 20 mL/kg IV of isotonic fluid that does not contain dextrose.[3]
   ii) Excess IV fluid administration may lead to pulmonary edema and require mechanical ventilation.

3) **Airway**
   a) **Bag-valve-mask (BVM)**
      i) An appropriate initial maneuver before tracheal intubation is BVM ventilation with jaw thrust. The use of an oral airway may also be necessary.
      ii) Gastric inflation due to excessive ventilation pressures can impede lung expansion and cardiac filling, resulting in decreased cardiac output. It also carries significant risk of pulmonary aspiration.
      iii) Adequate ventilation should be judged by bilateral chest excursion, as well as auscultation.
   b) **Endotracheal intubation**
      i) The most reliable verification of successful endotracheal intubation is visualization of the endotracheal tube passing through the cords and bilateral auscultation of breath sounds.
      ii) End-tidal CO_2 ($ETCO_2$) confirmation of endotracheal intubation.
         (1) $ETCO_2$ will only be present in the setting of effective pulmonary blood flow.
         (2) $ETCO_2$ may not be detected even with appropriate endotracheal intubation if there is no cardiac output (eg, asystole or ineffective chest compressions).
      iii) Effective BVM ventilation without endotracheal intubation is preferable to multiple failed attempts at intubation by inexperienced personnel. Chest compressions should not be halted or delayed to perform endotracheal intubation.
4) **Breathing (Table 76-1)**
   a) Excessive ventilation is common during resuscitation after cardiac arrest.
   b) Hyperventilation (even with normal peak inspiratory pressures) can lead to decreased venous return, decreased cardiac output, and decreased cerebral perfusion secondary to decreased $PaCO_2$.
   c) Once an artificial airway is placed, ventilation should be given at 8-10 breaths/min without any pause during chest compressions.
   d) While adult guidelines suggest an option for compression-only CPR by lay-persons, rescue breaths are still recommended in pediatric patients requiring resuscitation.[6,7]
5) **Medications for pediatric resuscitation and arrhythmias (Table 76-2)**
   a) The following medications can be administered via the ETT: Lidocaine, Atropine, Naloxone, Epinephrine. Mnemonic = LANE.
   b) The total volume administered through the ETT with each drug dose should not exceed 5 mL in the child and 10 mL in the adult. Drug concentrations should be adjusted with these volume limits in mind.[8]
   c) There is no evidence to support routine premedication with atropine (nor a minimum dose) prior to intubation to prevent bradycardia.[3,9]
   d) High-dose epinephrine (100 µg/kg) has been shown to result in worse outcomes in pediatric arrest and should be avoided except via the endotracheal route.[10]
6) **Postresuscitation management**
   a) Hypothermia has not been proven to improve outcomes; however, hyperthermia should be avoided in comatose children after resuscitation.[11]
   b) Systolic blood pressure should be maintained ≥5th percentile for age with fluids and inotropes.[8]



$ETCO_2$ will only be present in the setting of effective pulmonary blood flow. $ETCO_2$ may not be detected even with appropriate endotracheal intubation if there is no cardiac output (eg, asystole or ineffective chest compressions).



Effective BVM ventilation without endotracheal intubation is preferable to multiple failed attempts at intubation by inexperienced personnel. Chest compressions should not be halted or delayed to perform endotracheal intubation.



Excessive ventilation increases intrathoracic pressure and leads to decreased venous return, decreased cardiac output, decreased cerebral perfusion, and barotrauma.



High-dose epinephrine (100 µg/kg) has been shown to result in worse outcomes in pediatric arrest and should be avoided except via the endotracheal route.

TABLE 76-2 Medications for Pediatric Resuscitation[2,9,10]

Medication	Dose	Comments
Adenosine	0.1 mg/kg (max. 6 mg), repeat dose 0.2 mg/kg (max. 12 mg)	Monitor ECG; Rapid IV/IO bolus.
Amiodarone	5 mg/kg IV/IO, repeat up to 15 mg/kg (max. 300 mg)	Monitor ECG and blood pressure; caution when used with other QT-prolonging drugs.
Atropine	0.02 mg/kg IV/IO, 0.03 mg/kg ET	May need higher dose with organophosphate poisoning. A minimum dose of 0.1 mg is often reported but is unsupported in literature and may be toxic for smaller patients.[7]
Calcium chloride	10-20 mg/kg IV/IO	Via large bore, preferably central line or IO.
Calcium gluconate	30-60 mg/kg IV/IO	Central line preferred, peripheral IV OK.
Epinephrine	0.01 mg/kg IV/IO, 0.1 mg/kg ET; max. dose 1 mg IV/IO and 10 mg ET	May repeat q3-5min.
Glucose	0.5-1 g/kg IV/IO	D10 preferred over D50 in infants due to association between D50 and intraventricular hemorrhage.
Lidocaine	1 mg/kg IV/IO, followed by 20-50 µg/kg/min infusion IV/IO; 2-3 mg/kg ET	Consider decreased dose with hepatic disease, hypoproteinemia, or severely decreased cardiac output. Toxic metabolites may accumulate with renal insufficiency.
Magnesium sulfate	25-50 mg/kg IV/IO (max. dose 2 g)	
Naloxone	<5 y or ≤20 kg: 0.1 mg/kg IV/IO/ET	Lower doses recommended to reverse respiratory depression in nonarrest situation (1-15 µg/kg).
	≥5 y or ≥20 kg: 2 mg IV/IO/ET	
Sodium bicarbonate	1 mEq/kg IV/IO slowly	After adequate ventilation established.

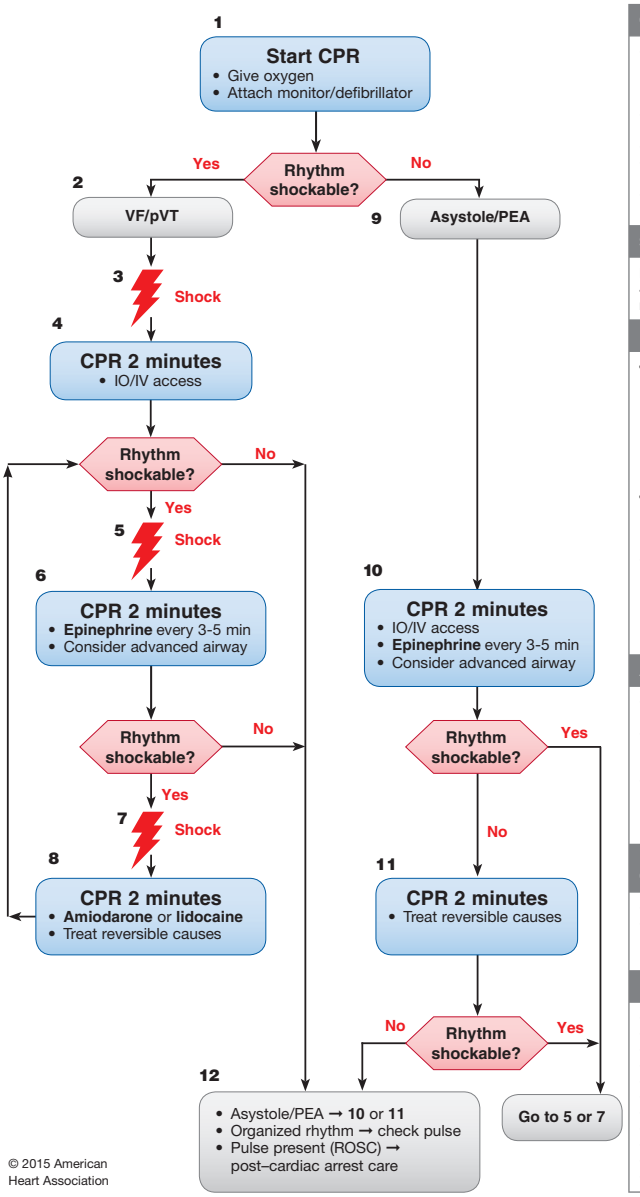
IV, intravenous; IO, intraosseous; ET, endotracheal.

7) **Resuscitation in special scenarios**
   a) **Local anesthetic toxicity**[12,13]
      i) Initial focus should be on airway management and resuscitation algorithms. Resuscitation efforts may be prolonged.
      ii) Seizures should be treated with benzodiazepines.
      iii) Lipid emulsion 20% is the key therapy: bolus 1.5 mL/kg IV over 1 minute followed by 0.25 mL/kg/min infusion until circulatory stability is attained.
      iv) Avoid vasopressin, calcium channel blockers, beta blockers, local anesthetics, and large doses of propofol.



Medications that can be given via ETT: use the mnemonic "LANE" = Lidocaine, Atropine, Naloxone, Epinephrine.

Pediatric Cardiac Arrest Algorithm—2015 Update



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CPR Quality

- Push hard (≥ of anteroposterior diameter of chest) and fast (100-120/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Rotate compressor every 2 minutes, or sooner if fatigued.
- If no advanced airway, 15:2 compression-ventilation ratio.

Shock Energy for Defibrillation

First shock 2 J/kg, second shock 4 J/kg, subsequent shocks ≥4 J/kg, maximum 10 J/kg or adult dose

Drug Therapy

- **Epinephrine IO/IV dose:** 0.01 mg/kg (0.1 mL/kg of 1:10,000 concentration). Repeat every 3-5 minutes. If no IO/IV access, may give endotracheal dose: 0.1 mg/kg (0.1 mL/kg of 1:1,000 concentration).
- **Amiodarone IO/IV dose:** 5 mg/kg bolus during cardiac arrest. May repeat up to 2 times for refractory VF/pulseless VT.
- **Lidocaine IO/IV dose:** Initial: 1 mg/kg loading dose. Maintenance: 20-50 mcg/kg per minute infusion (repeat bolus dose if infusion initiated >15 minutes after initial bolus therapy).

Advanced Airway

- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions

Return of Spontaneous Circulation (ROSC)

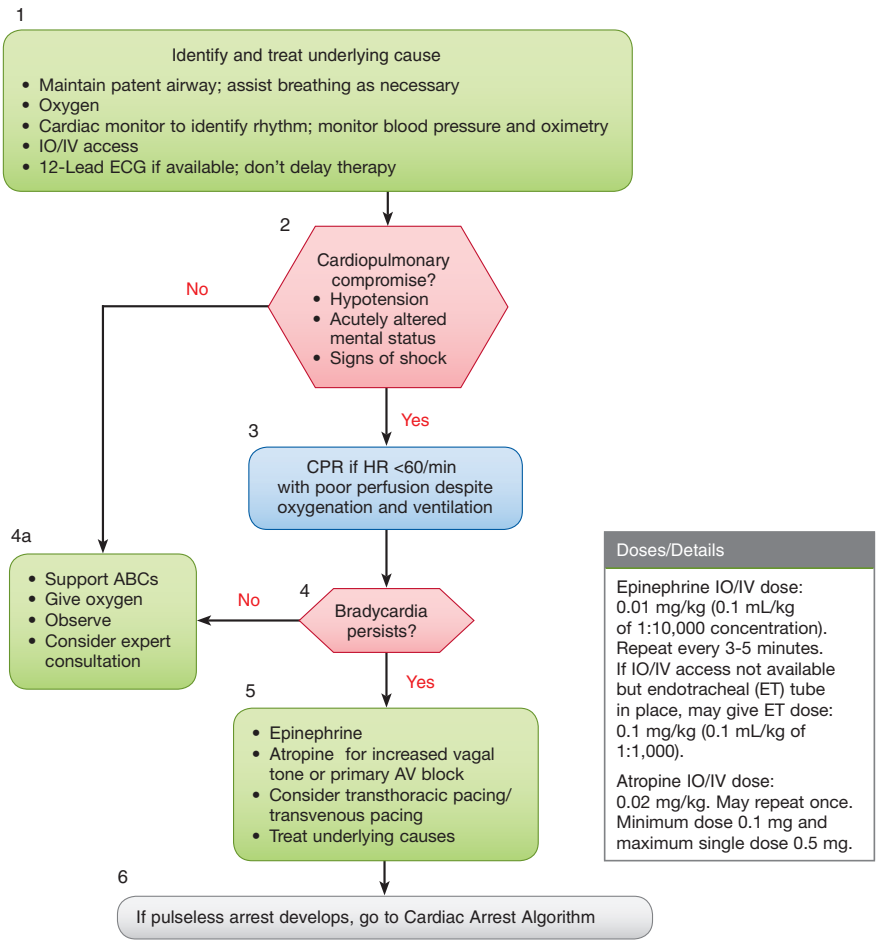
- Pulse and blood pressure
- Spontaneous arterial pressure waves with intra-arterial monitoring

Reversible Causes

- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypoglycemia
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

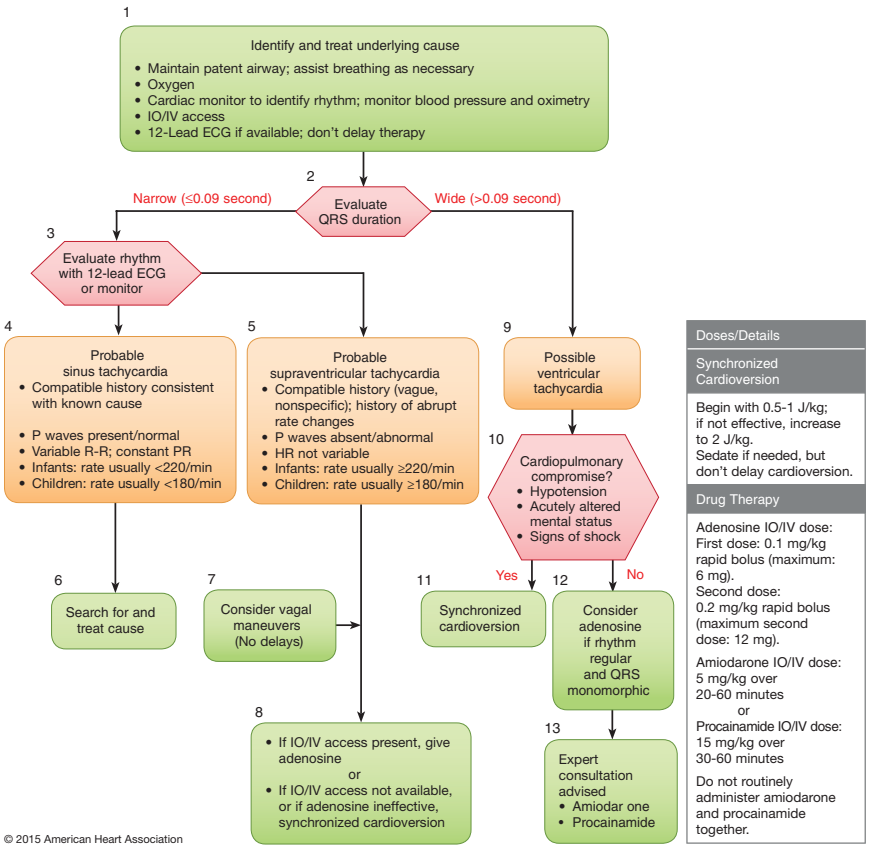
Following algorithms from Barash PG, Cullen BF, Stoelting RK, et al., eds. *Clinical Anesthesia*. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2017. Adapted from American Heart Association. Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care: International Consensus on Science. *Circulation*. 2000;102(8):I241-I243.

Pediatric Bradycardia With a Pulse and Poor Perfusion Algorithm



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Pediatric Tachycardia With a Pulse and Poor Perfusion Algorithm



CHAPTER SUMMARY FOR MANAGEMENT OF PEDIATRIC ARREST AND RESUSCITATION

Cardiovascular causes are the most common cause of perioperative pediatric arrests (41%), with respiratory causes, particularly laryngospasm, being second (27%), and medication reactions being third (18%).

"C-A-B" is the preferred sequence when initiating pediatric resuscitation according to the 2015 AHA guidelines.

Defibrillation (asynchronous) is for pulseless ventricular tachycardia (VT) and ventricular fibrillation (VF). Cardioversion (synchronous) is for atrial tachycardias, SVT, or symptomatic VT with pulse present.

After resuscitation, systolic blood pressure should be maintained ≥5th percentile for age with fluids and inotropes.

Local anesthetic toxicity: lipid emulsion 20% is the key therapy. Bolus 1.5 mL/kg IV over 1 minute followed by 0.25 mL/kg/min infusion until circulatory stability is attained.

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77 Ethical Considerations in Pediatric Anesthesiology

Alyssa M. Burgart

Anesthesiologists routinely face ethical issues when caring for pediatric patients. Anesthesiologists caring for children must be facile in assessing childhood development and its influence on care decisions. In addition to recognizing ethical standards related to pediatric practice, anesthesiologists must become familiar with relevant local laws that may significantly impact shared decision-making with parents and children. While this chapter focused on pediatrics, many of the principles here can be used to guide ethical dilemmas in adults as well.

1) **Anesthesiologists as moral agents**
   a) Anesthesiologists may experience a lack of control over ethical matters related to their practice, particularly when patients present on the day of surgery with unresolved moral implications of treatment.
   b) Such scenarios may be uncomfortable, and anesthesiologists may feel morally conflicted as to their role in intervening. This may be a source of moral distress.
   c) The anesthesiologist is in a unique position of power to halt or delay a procedure when needed to resolve moral conflict and protect the patient.
   d) **Moral distress: Distress caused by a perceived inability to act in alignment with one's moral values.**[1,2]
      i) Sources of moral distress include real or perceived powerlessness, inadequate resources, inadequate staff, and insufficient administrative/organizational support, and specific clinical contexts.
      ii) Unresolved moral distress contributes to burnout and therefore has implications for practice longevity and one's sense of self and purpose.
      iii) Moral courage: Feeling fear to act on one's moral values, but doing so anyway.
2) **Skills of the pediatric anesthesiologist**
   a) **Anesthesiologists who work with children must maintain a wide range of skills, including the ability to:**
      i) Assess developmental stages, milestones, and make adjustments for developmental conditions.
      ii) Engage children in developmentally appropriate dialog.
      iii) Simultaneously assess patient and parental anxiety.
      iv) Assess parental influence on decision-making, between multiple parents, or between parent-patient dyads.
      v) Engage parents' concerns honestly, without causing undue patient or parental anxiety.
      vi) Impart a sense of trust in both parents and patients.
      vii) Obtain and disclose sensitive information from children, adolescents, and parents.
   b) **Anesthesiologists are especially adept at using these skills to quickly develop a relationship with the family preoperatively.**
   c) **Respectful communication**[3,4]
      i) Patients and parents deserve to be referred to with language that aligns with their identity, including name pronunciation and gender pronouns.

ii) If you are unsure how to say a patient or parent's name or are unsure how the person wishes to be addressed, it is best to ask, rather than assume.

iii) To normalize introductions, you can provide your name and gender pronouns "Hello, my name is Dr. Burgart and I use she/her pronouns. How should I address you?" You can ask every patient and parent, rather than just the ones that you suspect of having a trans or nonbinary identity.



If you are unsure how to say a patient or parent's name or are unsure how the person wishes to be addressed, it is best to ask, rather than assume.

3) **Minors' human rights include the following**
   a) Being treated with respect
   b) They have developing autonomy and the right to ask questions, express their views and values.
   c) The right to high-quality medical care
   d) The right to be protected from harm, such as abuse, neglect, and violence
   e) Adolescents have a right to privacy.
   f) Minors' legal rights to consent to health care vary by state and by situation.
4) **Pediatric developmental continuum (Table 77-1)**
   a) Rule of 7s is one simple way to broadly assess pediatric development.
   b) The ages provided are estimates and physician judgment may determine different decision-making capacity for a particularly mature or immature patient.
   c) This rule is not appropriate to assess children with significant intellectual delays.
5) **Important concepts in pediatric ethics**
   a) **Best Interest Standard**: When a pediatric patient does not have the developmental capacity to participate in the decision-making process, parents and physicians evaluate the patient's "best interest" to determine how to proceed.
      i) What is "best" is a value laden judgment and reasonable people may arrive at differing conclusions.
      ii) The decision maker selects the "best" option for the given situation.
      iii) The best interest of a child is a high standard, much higher than the standard adults may apply to their own choices.
   b) **Parental zone of discretion**: The zone of decisions that parents are allowed to make without interference.[5]
      i) These decisions may not meet a best interest standard but do not fall below the threshold of harm.
      ii) When a decision is determined to be outside the zone of parental discretion (ZPD), and parents refuse treatment or attempt to cause harm, legal avenues may be pursued to limit parental authority.
   c) **Harm principle**: The principle that individual actions should only be limited if they harm another person[6]

TABLE 77-1 The Rule of 7s for Child Development

Age	Ability to Participate in Decisions
<7 y	• Assume child does NOT HAVE CAPACITY to understand risks, benefits, alternatives • Rely on best interest standard
7-13 y	• Child has developing sense of autonomy and increasing level of decision-making capacity • Rely on combination of the best interest standard from the parents and informed permission from patient
14-17 y	• Child assumed to HAVE CAPACITY unless there is evidence to the contrary • Rely on patient's informed assent/permission or consent if legally appropriate

i) This is a threshold that may be used to justify state interference in parental decisions.
ii) "Harm" is also a subjective, value laden principle.

d) **Child abuse/neglect**: Child abuse is parental or caretaker action, or failure to act, that results in harm to a child, including death, serious physical or emotional harm, sexual abuse, or exploitation, or which places the child in an imminent risk of serious harm.
i) Child abuse laws balance the right of children to be safe from harm against
(1) The right of families to privacy
(2) The rights of parents to raise and discipline their children without interference

6) **Truth telling**

a) **Avoid lying to children.** Children are learning to appreciate honesty and truthfulness. Lying to children risks damaging your therapeutic alliance and creating lasting distrust of health care providers.

b) **Even though children under age 7 may not have the capacity to meaningfully participate in health care decisions, they are conscious of their body and have opinions about what happens to them.**
i) Developmentally appropriate language should be used to explain what is happening in a clinical encounter and why.
(1) This can reduce fear and improve likelihood that children will be willing participants in their care.

c) **When parents have neglected to notify the child that they are having surgery/anesthesia, it is best to reset the dialog.** Parents who avoid educating the child often do so from a place of protection, believing that awareness and anticipation of the surgical plan will be harmful. This is generally a reflection of parental anxiety. Additionally, parents may feel entirely uneducated regarding what information should be shared.
i) While pharmacologic sedation can mask preoperative anxiety, be cognizant that even young children will still understand that *something* happened to their body, and sense the loss of time after emergence.

d) **Avoid offers/promises to a child that cannot be delivered.** Be cognizant that not all families will handle the postoperative period or parent in the same way. Promises of screen time, certain foods, etc., may be seen as overstepping and unwelcome interference in the parent-child relationship.

e) **Take caution when discussing what children will or will not remember from the anesthetic experience.**
i) Midazolam is not amnestic for all patients; patients may recall their anesthetic induction.
ii) Even if a child does not remember an entire encounter, times of high emotions or trauma may leave lasting marks on a child's psyche.

7) **Consent and assent[7] (Tables 77-1 and 77-2)**

a) **Consent:**
i) Parents have the moral and legal authority to make decisions for their children, based on the assumption that parents generally act in the best interest of their children.[6] Unless there is evidence to the contrary:



To read more consult: The International Convention Rights of the Child http://www.ohchr.org/en/professionalinterest/pages/crc.aspx

This document outlines comprehensive rights of children. Unfortunately, it has not been ratified in the United States.



Avoid lying to children. This damages your therapeutic alliance with them and creates a lasting distrust of health care providers.



Parents may avoid telling their children about surgery out of their own anxiety and/or ignorance. Anesthesiologists are in a position to reset the dialog and facilitate communication.



Always assume the child is listening to you, no matter how distracted they appear. Do not say or do anything in front of a child that you do not want them to hear or see.

TABLE 77-2 Consent and Assent

Informed Consent	Parental Permission	Informed Assent
Using the value of self-determination, patients who understand the risks, benefits, and alternatives to a treatment may allow it. Parental consent on behalf of a child is consent by proxy.	Has the same elements as informed consent, but acknowledges that parents are acting on behalf of their child. Informed consent and informed permission are often used interchangeably.	Even when children are not ready to make complex decisions alone, they can participate to the extent their development allows, and give their own permission for treatment. Assent empowers children to the extent of their capacity.

(1) Parents are assumed to care about their child.
(2) Know the unique needs of the child better than other people.
(3) Are uniquely invested in the child's well-being
(4) Parents must manage the consequences of medical decisions for the child.

ii) Parents must meet four requirements to be surrogate decision makers:[8]
(1) Be competent to make reasoned judgments
(2) Possess adequate knowledge and information
(3) Be emotionally stable
(4) Be committed to the incompetent patient's interests

iii) Unless there are special circumstances, parents are tasked with provision of written consent for their children.

iv) Generally, only one parent is needed to consent to treatment. Law or hospital policy may require both parents to consent to procedures or treatments (**Fig. 77-1**).

b) **Assent**

i) Assent is a minor's active agreement to participate in medical care.

ii) Children, especially adolescents, are capable of participating in the decision-making process.

iii) Children should be solicited for their opinions and questions and should be taken seriously.

iv) The adolescent voice is often the primary influence on parental decision-making. Parents frequently support or defer to teens in such decisions.

v) Children should be assessed for their level of understanding of a procedure and assent obtained whenever possible. It is the responsibility of the person obtaining assent to develop the necessary skills to discuss the topic in developmentally appropriate terms (**Fig. 77-2**).[5]

FIGURE 77-1 Informed Consent

What is needed to provide informed consent?

1. Adequate provision of information including:
   - the nature of the ailment or condition,
   - the nature of the proposed diagnostic steps or treament and the probability of their success;
   - the existence and nature of the risks involved
   - the existence, potential benefits, and risks of recommended alternative tretments (including the choice of no treatment)
2. Assessment of the patient's understanding of the above information
3. Assessment, if only tacit, of the capacity of the patient or surrogate to make the necessary decisions.
4. Assurance, insofar as it is possible, that the patient has the freedom to choose

FIGURE 77-2 Requirements for Assent

What is needed to provide assent?

1. Helping the patient achieve a developmentally appropriate awareness of the nature of their condition.
2. Telling the patient what he or she can expect with tests and treatment.
3. Making a clinical assessment of the patient's understanding of the situation and the factors influencing how they are responding (including whether there is inappropriate pressure to accept testing or therapy).
4. Soliciting an expression of the patient's willingness to accept the proposed care.

c) **Minors as decision makers**

i) Parental consent is the default requirement in most states for children under the age of majority (usually 18).[9]

ii) In specific situations and jurisdictions, select minor patients may have the legal right to provide informed consent.

iii) Mature minors

(1) The mature minor doctrine states that adolescents who have capacity to make informed decisions have an ethical right to do so. Most states use age 14 as a benchmark (based on the Rule of Sevens), but the closer a patient's age to the age of majority, the more likely the right will be conferred.

(2) A minority of states have official "mature minor" exceptions to parental consent, by statute or judicial ruling. States vary on how this is determined, such as by age, graduation from high school, or evaluation by a clinician.

(3) Thirty-four states have no clearly defined mature minor exception. Many states grant temporary or permanent consent authority to adolescents in specific situations, such as reproductive and mental health care.

(4) Emancipated minors: Have global right to make health care decisions. Certain states grant emancipation when a minor is married, enters military service, and becomes a parent. Otherwise, it is determined by judicial ruling.

(5) Clinicians should consult with a local expert (ie, risk management) when unsure about a minor's rights.

(6) Minors are able to receive emergency care without parental consent.

(a) A few states authorize minors to give their own consent for emergency care.

(b) Parents should be notified as soon as possible after emergency care is provided.



Adolescent rights vary widely by jurisdiction and specific situation, and it is prudent to engage experts in risk management and/or social work if you are unsure of your obligations to the patient and parents/legal guardians.

8) **Emergency care**

a) Emergency anesthetic care may be needed for minors to reduce the risk of death, disability, or long-term complications.

b) Treatment should not be delayed due to lack of parent or other surrogate decision maker to provide consent.

c) If time permits and a parent is available by phone, consent may be obtained in this manner. If not, parents should be notified as soon as they are available.

d) If developmentally appropriate and time permits, assent should be sought from the patient.

9) **Unique parents and guardians**
   a) Children may present with biological, adoptive, or foster parents, temporary or permanent guardians.
   b) Some children may be in foster care as wards of the state. Depending on the arrangement with the court, biological parents may retain the right to consent to medical treatments. Therefore, the guardian who presents with the patient may not have the legal right to consent to treatment. Review court documents to ensure consent is obtained from the appropriate party.
   c) Court orders for care may be required and should be reviewed prior to initiating any treatment.
   d) **Elective cases:** If a parent or legal guardian does not present with a child, the responsible party must be contacted for consent before the procedure may move forward. If parents/guardians are unreachable and there is no court order for care, then the procedure should be rescheduled.
   e) **Minors as parents:** In 30 states, underage parents have the rights to consent to medical care for their children.[10]
10) **Impaired parents**
   a) Parents may make impaired judgments, especially if under the influence of drugs, alcohol, or medical condition.
   b) When no parents can serve as a decision maker for the child, an alternative decision maker must be determined. If none is available, one may be selected by the courts/child protective services. This is not generally a fast process and is unlikely to be completed when such matters become apparent in the preoperative area.
   c) If a parent is unable to act as an appropriate surrogate, the anesthesiologist and surgeon must carefully consider whether to proceed based on urgency. In such cases, it is wise to consult your organization's risk management/legal service or ethics consultant regarding responsibilities for patient welfare and appropriate documentation.
   d) It may not be possible to discharge the patient home with the parent (eg, acutely impaired parents should not drive.)
   e) Consider social work consultation to determine how to best support the child and family.
   f) Parents' behavior may be disruptive or even dangerous.
   g) It may be necessary to direct the parent to the emergency room for evaluation.
   h) Protect yourself and the patient from harm.
11) **Refusal of treatment by minor patient**
   a) Patient refusal should be taken seriously and respected. Anesthesiologists should not engage in coercion.
   b) If one doesn't intend to weigh a minor's views seriously, then do not ask.
   c) In situations where a patient will receive treatment despite their objection, the patient should be notified.
   d) Children with the ability to participate in a meaningful way with decisions might make a valid refusal. Children as young as 9 may meet this standard, children 12 or older are likely to be able to validly refuse.
   e) **Elective cases**: Based on the patient's age and maturity, consider if the procedure should be cancelled/delayed until a resolution has been reached with the patient and parents.
   f) **Life-threatening emergencies**: It may be that the child's dissent should be overruled to preserve his/her life. If the harm is serious and the minor lacks decision-making capacity and/or their reasons for refusal are significantly shortsighted, then overriding the refusal and proceeding with a best interest–based approach may be justified.

The physician should provide detailed documentation regarding the discussion with the parent and patient.

12) **Pediatric limitations on life-sustaining therapy (Fig. 77-3)**
   a) Life-sustaining therapies (LST) include do not resuscitate, do not intubate orders, and other orders that limit treatment.
   b) It is a common misconception that LST orders must be automatically rescinded to go to the operating room.
   c) Determination regarding whether to maintain LST orders in the operating room should be made among the patient, parents, surgeon, and anesthesiologist.
   d) These orders should not be changed unilaterally by clinicians.
   e) The American Society for Anesthesiology,[11] American College of Surgeons,[12] and American Academy of Pediatrics[13] all support supporting patient self-determination regarding LST orders during surgery.
   f) **Alternative goals may include the following:**
      i) Full resuscitation
      ii) Limited resuscitation based on specific procedures
      iii) Limited resuscitation based on the patient's goals and values
   g) **If an agreement cannot be reached between the patient and care team:**
      i) If the anesthesiologist finds the plan counter to his/her moral values, he/she may consider withdrawing from the case and finding an alternative provider.
      ii) If the plan is counter to generally accepted standards of care, the law, or hospital policy, the anesthesiologist should openly express this concern to the patient and surgeon, and escalate the discussion to the appropriate institutional representative.
      iii) If there is not time to manifest the previous alternatives, the case may proceed with an attempt to adhere to the patient's stated directives, goals, and values.

13) **Medically ineffective treatment**
   a) Parents may request or demand treatments that are ineffective or inappropriate.
   b) Clinicians are not obligated to comply with unreasonable or unjustified parental demands.



It is a common misconception that life-sustaining therapy orders must be automatically rescinded to go to the operating room.

FIGURE 77-3 Considerations on Life-Sustaining Therapy

Issues to consider when discussing life-sustaining therapy

Clinical assessment:
- Expected benefits to the patient
- Expected risks of procedure/anesthetic (risk of iatrogenic effects)
- Likelihood of resuscitation (in operating room vs unwitnessed arrest) and range of possible outcomes
- How would anesthesiologst respond due to atrogenc events, including interventions and their risks
- Post-op care location, range of possibilities

Timing:
- If orders are to be rescinded of goal-directed for the perioperative period, how is that period defined

Documentation:
- Thorough documentation of the conversation and decisions
- Appropriate/timely adjustment of orders in the EMR

c) Whenever possible, disagreements should be resolved using respectful communication and shared decision-making.
d) For cases where communication becomes protracted, hospital policies on medically ineffective treatment may help guide and respond to such demands.
e) If parents are obstructing provision of regular care, it may be necessary to engage the courts or child protective services.

14) **Jehovah's Witnesses and refusal of blood transfusions**

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a) Jehovah's Witness parents believe that blood is sacred and, as a result, may refuse to consent to blood transfusions for their children.[14]
b) Parents of Jehovah's Witnesses are unlikely to refuse any other medical treatment.
c) **Caring for the children of Jehovah's Witness parents requires thoughtful and open communication about blood transfusion.**
   i) This is a religious belief, not a logical or scientific argument.
   ii) Not all members of the faith agree regarding which blood products or "blood fractions" are permissible and may use their discretion as a Christian. Do not assume you know the specific values of the patient or individual parent.
   iii) Some parents may be willing to consent to transfusion.
   iv) The Jehovah's Witness community offers a hospital liaison service to its members. Such liaisons can facilitate communication between the family and medical team.
d) Adult informed refusal of blood transfusion has been consistently upheld by the courts as an expression of self-determination.
e) The courts have consistently (although not universally) intervened in parental refusal of blood transfusions for minors. As *parens patriae*, the state has an obligation to protect those who are not capable of making informed decisions.
f) **When parents refuse to consent to blood transfusions for their child, there are several means to address this:**
   i) Court order for transfusion: A local court may be petitioned for a court order for blood transfusion. This secures a legal means to transfuse the child. Unfortunately, due to court variability, there can be unintended consequences, such as the court removing the child from parental custody or opening an investigation into the custody rights of other children in their care.
      (1) When the likelihood of transfusion is moderate or high, the court order may be obtained preoperatively.
      (2) When the risk is low, the court may be petitioned during the surgery if the need to transfuse appears to be approaching. If the need becomes dire before the court order is in effect, the patient should not be harmed by lack of transfusion, and the anesthesiologist is ethically and legally obligated to proceed with emergency care, including transfusion.
   ii) Parental acknowledgement: In an effort to avoid court ordered treatment, hospitals are increasingly turning to alternatives to transfusion consent. Such alternative consent documents allow parents to clearly voice that they are not able to consent to transfusion but also to acknowledge that physicians have a legal and moral obligation to prevent their child's death due to lack of transfusion. This removes the burden of the decision from the parents, allowing anesthesiologists to transfuse when it is necessary.
g) **Adolescent Jehovah's Witnesses**
   i) Depending on the jurisdiction, adolescents may have the right to refuse treatment.
   ii) Teens may choose to petition the court for the right to refuse transfusion. Courts have been inconsistent in these cases.

iii) If the patient is close to age 18 and surgery can be safely delayed, consider doing so until the patient may legally provide an informed refusal.

h) Unanticipated, life-threatening bleeding has occurred in Jehovah's Witness patients, placing the surgeon and anesthesiologist in an uncomfortable situation. Beware of the assumption that "this case won't bleed."

i) Preoperative optimization: Methods to reduce the likelihood of transfusion should be taken. Various ways to reduce the risk should be discussed with the parents.

j) For elective procedures, workup may include referral to hematology for management of underlying anemia. All efforts should be made to minimize the need for transfusion and increase potential to comply with parents' preferences.

k) **Children of Jehovah's Witnesses should receive the same level of care provided to any other patient.**
   i) Anemia may be well tolerated. Prior to induction of anesthesia, it is preferable to transparently communicate with the family which circumstances might trigger transfusion.

l) **Emergency transfusions**
   i) In an emergency setting, where the patient will die or be injured from lack of transfusion, do not delay transfusion for a court order.
   ii) Adequately document decision-making rationale.

15) **Suspicion of child abuse**[15]
   a) **Abuse falls into four domains: neglect, physical abuse, sexual abuse, and emotional abuse.**
      i) As many as 1 in 4 children has been maltreated.
      ii) Abuse is often not confined to one domain.
      iii) An estimated 1700 children die annually due to abuse and neglect.[16]
   b) As mandatory reporters, physicians are required to report even a suspicion of child abuse.
   c) Physicians should not assume that abuse is a one-time event.
   d) **In the operating room, staff may observe physical exam findings that raise suspicion of child abuse.**[17]
      i) Bruises or burns in shape of objects (ie, fingers, hands, irons)
      ii) Developmentally inappropriate injuries
      iii) Injuries not consistent with reported history
   e) **Any concerning physical examination findings should be documented thoroughly.**
      i) Many EMRs now support insertion of photographs and can be very valuable in documenting injuries.
   f) **Munchausen by proxy:** An unusual form of child abuse where a parent causes or fictionalizes a child's illness.
      i) May be identified by perioperative care staff as patients may be seen for unusual procedures or with high frequency.
   g) Additional resources: Hospitals may have an abuse specialist on staff or on contract.

16) **Confidentiality**
   a) Essential component of patient-physician and parent-physician relationships is necessary so that both parties can communicate without censorship.
   b) Teens may delay or decline important medical care out of concern of their privacy being violated.
   c) Teens may have a moral right to confidentiality but may not have a legal right to it.
   d) Teens should be interviewed in private to enhance trust and prevent accidental breach of confidentiality during the interview.

e) Unauthorized and unnecessary disclosure of sensitive information should be avoided.
f) Before teen discloses anything, anesthesiologists should notify teens that they are mandated reporters and how this role impacts confidentiality.
g) Minors who are either deemed mature by legal statute, or emancipated by the court, are essentially adults and entitled to the same level of confidentiality and privacy that adults enjoy.
h) **Risk of minor harm**: For scenarios where disclosure is likely to cause minimal harm, teens can be encouraged to disclose to parents and supported in that conversation.
   i) If the teen decides not to disclose, they should be respected.
i) **Risk of serious harm**: If the risk to the teen is severe, it may be ethically necessary or legally required to disclose information to parents.
   i) These times may include parental notification laws, mandated reporting laws, credible threat to self or another person.
j) When teens act as their own health care decision maker, HIPAA regulations apply.



Teens should be interviewed in private to enhance trust and prevent accidental breach of confidentiality during the interview.



If you cannot guarantee privacy (eg, due to mandatory reporting responsibilities), do not offer it! This can significantly damage your therapeutic relationship with the teen patient.

17) **Pregnancy testing**
a) Pregnancy tests should only be ordered when a positive result will impact care.
b) Teens and parents should be notified that a pregnancy test has been ordered, but generally, it is ethically justified to only disclose the positive results to the minor.
c) Clinicians who order pregnancy testing should be trained regarding thoughtful disclosure of positive result and refer patient to appropriate resources.
   i) Clinicians must also be aware of any additional rights the teen gains when a pregnancy test is positive.
d) Determination as to whether to cancel a case should be made based on the specific risks and benefits of the case. For example, cancelling an elective surgery may be justified, but more urgent procedures will need a more nuanced approach.
e) Maintaining the patient's confidentiality in these cases is important, as disclosure to parents may have far reaching impact for the teen.
   i) It is best to talk with the teen before discussing with parents. Deception of parents in such cases may be ethically justified. It is legally required in states where the pregnant teen has consent authority.



Work with the minor to determine how to safely navigate the result of a pregnancy test.

18) **Minor pregnancy**
a) Laws surrounding pregnant adolescents differ from state to state, with pregnant teens having full consent authority in some states, and having no additional rights in others.
b) Pregnant patients who meet certain criteria have consent authority for reproductive health care decisions. This may not necessarily include the right to consent to general medical procedures (ie, a teen may have the right to consent to prenatal care, but not have the right to consent to a cholecystectomy during her pregnancy).
c) While the legal landscape is murky and social disagreement on this issue persists, both the American Academy of Pediatrics and the American Medical Association support adolescent confidentiality when considering reproductive health, including abortion.
d) Even in states with clear mature minor statutes, parental permission or judicial waiver may be required for minors to obtain an abortion.

19) **Labor epidurals in teens**
   a) Twelve states do not allow minors to self-consent for prenatal/pregnancy care. Delivery itself is essentially treated as emergency care. In seven of these states, if the teen is legally married, she gains consent authority.[18]
   b) In those states, parental consent is required for all treatment. If no parent is present, the patient is an unrepresented minor with no decision-making authority. Parents who are present may use any reason to withhold consent to a labor epidural (ie, punishment for pregnancy).

Even though minors may not have the right to make health care decisions for themselves, they may have the legal right to make health care decisions for their own children.

20) **Workplace safety and workplace violence**
   a) Every employee has the right to a safe work environment.
   b) The Federal Occupational Health and Safety Act of 1970 requires employees be protected from hazards expected to cause harm or death. States may have regulations whose protections exceed those set out by the federal government.
   c) **According to National Institute for Occupational Safety and Health (NIOSH), violence is an occupational hazard in hospitals. Examples of violence include the following:**
      i) Threats: Expressions of intent to cause harm, including verbal threats, threatening body language, and written threats
      ii) Physical assaults: Attacks ranging from slapping and beating to rape, homicide, and the use of weapons such as firearms, bombs, or knives
      iii) Muggings: Aggravated assaults, usually conducted by surprise and with intent to rob[19]
   d) **If a patient, parent, or staff member demonstrates violent behavior:**
      i) Call security for support. Security guards have special training in de-escalation and will help secure the physical space. It may even be necessary to call local police.
      ii) Calling for help from security or police is not a sign of weakness, but utilization of an important service to maintain a safe work environment.

If a patient, parent, or staff member demonstrates violent behavior:

Call security for support. Security guards have special training in de-escalation and will help secure the physical space. It may even be necessary to call local police.

21) **Ethics consultation**
   a) All hospitals accredited by the Joint Commission have some mechanism to address ethical questions that arise in practice.
   b) Ethics consultants often work closely with risk management, and there is important overlap between the two disciplines.
   c) **Reasons one might request an ethics consultation**
      i) Lack of clarity around consent
      ii) Concern for abuse/neglect
      iii) Moral distress
      iv) Disagreements among physician, parent, and patient
      v) Unclear ethical responsibilities

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78 Part A: Diseases in Pediatric Patients

Congenital Diaphragmatic Hernia

Sally Vender

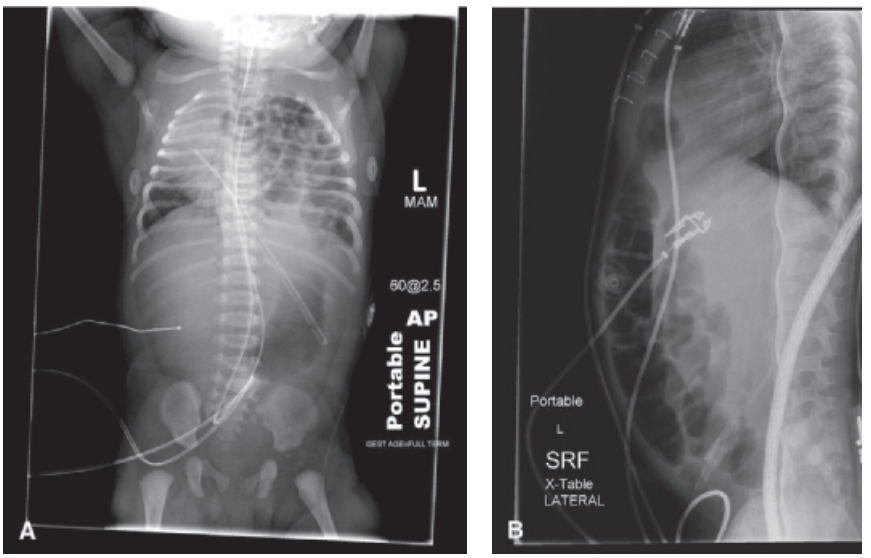
The diaphragm contains a number of foramen in which large structures traverse the abdomen to the chest. Defects in diaphragmatic fusion during embryogenesis can result in herniation of the viscera into the chest cavity causing marked developmental consequences to the cardiopulmonary system. Congenital diaphragmatic hernia (CDH) occurs in ~1:4000 live births and the mortality rate is as high as 50%.[1] Prenatal diagnosis is ideal in order to plan for delivery and management of these challenging patients at a tertiary care center.

1) **Pathophysiology**
   a) The diaphragm develops during weeks seven through ten of gestation and failure to fuse results in intrusion of the abdominal contents into the chest cavity. Compression of developing lung tissue results in pulmonary hypoplasia.
   b) The most common type of CDH is the Bochdalek type (80%), which occurs in the posterior lateral diaphragm, and is most commonly a left-sided defect.
   c) Rarely, a Morgagni type hernia (9%) occurs and is a result of failure of the anterior diaphragm to fuse.[2]
   d) The presence of abdominal viscera in the thorax leads to pulmonary hypoplasia and decreased lung compliance.
   e) Impaired ventilation and gas exchange due to reduced surface area causes respiratory acidosis and hypoxemia.
   f) Abnormal pulmonary vasculature occurs due to lung hypoplasia and results in persistent pulmonary hypertension.[2]
   g) Morbidity and mortality are associated with the degree of pulmonary hypoplasia and persistent pulmonary hypertension.
2) **Signs and symptoms/clinical findings**
   a) Neonates with Bochdalek hernias present at birth with respiratory distress and acute hypoxemia.
   b) **Physical exam ination** findings include a scaphoid abdomen, barrel-shaped chest, and absent breath sounds on the ipsilateral side.
   c) **Associated findings**
      i) Cardiac anomalies occur in 25% of CDH patients including atrial septal defects, ventricular septal defects, and persistent fetal circulation (eg, patent foramen ovale and patent ductus arteriosus) due to acidemia and hypoxemia. Preoperative echocardiographic evaluation strongly recommended.
      ii) Chromosomal abnormalities are found in 5%-30% of cases with trisomy 13 and 18 being the most common.[3]
      iii) Intestinal malrotation and gastroesophageal reflux are common.
   d) Chest x-ray confirms diagnosis by demonstrating herniated viscera in the thorax with the cardiac silhouette deviated to the contralateral side (**Fig. 78-1**).



Because CDH is frequently associated with cardiac anomalies, preoperative echocardiogram is strongly recommended.

FIGURE 78-1 Radiographs of Neonates With Left-Sided Congenital Diaphragmatic Hernias



A. Left-sided Bochdalek CDH in a neonate—AP. **B.** Left-sided Bochdalek CDH in a neonate—lateral view. Photo courtesy of Dr. Allyson Richards, University of New Mexico School of Medicine.

3) **Surgical treatment**
   a) CDH repair is not a surgical emergency. Timing of repair does not seem to influence survival, so ensuring that the patient is hemodynamically stable is important.[4]
   b) Surgical repair can be performed at the bedside while the patient is on high-frequency oscillatory ventilation (HFOV) or while on extracorporeal membrane oxygenation (ECMO).
   c) CDH can be repaired thoracoscopically (reserved for cases performed in the operating room) or via an open approach. Thoracoscopic surgery is associated with greater derangements in $PaCO_2$, PaO_2, and pH, which worsen pulmonary hypertension.[4] This method of repair is usually reserved for less severe cases of CDH.
   d) Patch or primary closure through an open approach is common. A subcostal incision is made on the ipsilateral side, the abdominal contents returned to the abdomen, and the defect in the diaphragm is closed.
4) **Anesthetic management**
   a) **Preoperative considerations**
      i) Ideally, neonates with CDH will be intubated immediately after birth.
      ii) Mask ventilation should be avoided as air may be insufflated into the herniated stomach and bowel, further compressing lung parenchyma and worsening the already compromised ventilatory status.
      iii) Cephalad displacement of the carina in infants with CDH may result in right main stem intubation and subsequent development of pneumothoraces, which are associated with increased mortality.[5]
      iv) Orogastric or nasogastric tube should be in place and on either continuous or intermittent suction to help reduce bowel distension.



In CDH, mask ventilation can cause gastric distention and respiratory and hemodynamic compromise. Secure the airway with an ETT as soon as possible.

TABLE 78-1 Ventilatory Strategy for Management of Congenital Diaphragmatic Hernia

Peak inspiratory pressure	<25 cm H_2O
PEEP	3-5 cm H_2O
Respiratory rate	40-60 breaths/min
FiO_2	<1, especially in premature infants (oxygen causes retinopathy of prematurity) Higher FiO_2 may be necessary in patients with pulmonary hypertension
Preductal saturation	Between 80% and 95%
$PaCO_2$	<65 mm Hg
pH	>7.2
Lactate	<5 mmol/L

v) Ventilatory strategy should include (**Table 78-1**) the following:
   (1) Maintenance of peak inspiratory pressure <25 cm H_2O
   (2) PEEP 3-5 cm H_2O
   (3) Respiratory rate between 40 and 60 breaths per minute
   (4) FiO_2 less <1
   (5) Preductal oxygen saturation 80%-95%
   (6) Adequate ventilation is defined by $PaCO_2$ < 65 mm Hg.[6]

vi) Signs of adequate end-organ perfusion include a pH >7.2, lactate <5 mmol/L, and urine output >1 mL/kg/h.[6]

vii) Signs of persistent pulmonary hypertension such as worsening ventilatory requirements, hypoxemia, and diminished end-organ perfusion may indicate that the patient is not optimized and repair may need to be delayed.

b) **Preoperative studies and tests**

i) Echocardiogram should be completed prior to surgery to evaluate cardiac structure, cardiac function, and signs of pulmonary hypertension.

ii) Cranial ultrasound should be performed to rule out intraventricular hemorrhage in premature infants or patients who are anticoagulated on ECMO.

iii) Renal ultrasound is useful for evaluating structural kidney disease.

iv) Labs
   (1) Type and cross-match should be done and blood immediately available during the surgery. Significant blood loss is unusual unless the patient is anticoagulated on ECMO.
   (2) Baseline hemoglobin, hematocrit, and platelet count may be helpful if there is significant bleeding or coagulopathy during the surgery.
   (3) Electrolytes and blood gases are useful clinical indicators of adequate respiratory status.

c) **Monitors and lines**

i) Ideally, the neonate will be intubated with umbilical vein and umbilical artery catheters.

ii) Standard ASA monitors are indicated along with extra pulse oximeters. Consider monitoring pre and postductal saturations in infants with congenital heart disease.

iii) Arterial line placement is recommended if an umbilical artery catheter is not in place. Blood gases and beat-to-beat blood pressure monitoring help guide anesthetic management.

iv) Consider placing a central venous line if an umbilical vein catheter is not in place. This is the safest way to deliver high concentration dextrose solution, inotropic infusions, and calcium.

v) Additional peripheral IVs should be placed if central venous access is not available.

d) **Intraoperative management**

i) Induction

(1) Most patients will be intubated and sedated in the NICU with lines already in place.

(2) More stable patients who are not intubated require preoxygenation with an FiO_2 of 100% unless cardiac issues preclude its use.

(3) The airway should be secured immediately after induction with minimal mask ventilation, which could lead to gastric distention and increased intrathoracic organ compression.

(4) Avoid the use of nitrous oxide. Bowel distention will occur and cause worsening pulmonary compression.

(5) Place an orogastric or nasogastric tube after intubation for gastric decompression.

(6) Consider using muscle relaxant in order to facilitate endotracheal intubation and decreased peak pressures if mask ventilation must be performed.

(7) If the patient is on ECMO or HFOV and the surgical procedure is being performed at the bedside, continue the current sedation regimen and add additional anesthetic agents and muscle relaxant.

ii) Maintenance

(1) A high FiO_2 may be necessary to minimize pulmonary vascular resistance unless there are cardiac contraindications.

(2) Muscle relaxant use will help facilitate mechanical ventilation, reduction of the abdominal viscera, and closure of the diaphragmatic defect.

(3) Inhaled anesthetics may be used unless the patient is hemodynamically unstable or being ventilated using an ICU ventilator.

(4) Adequate analgesia is necessary for preventing an increase in sympathetic tone and increased pulmonary vascular resistance.

(5) Patients on ECMO will require more opioids as their volume of distribution within the circuit is increased.

iii) Anesthetic considerations

(1) Cardiovascular

(a) Reduction of hernia contents back into the abdomen can cause hypotension and increased peak inspiratory pressures as the diaphragm is displaced cephalad and the inferior vena cava is compressed.

(b) Hemodynamic instability may require initiation of inotropic support.



Sudden decrease in cardiac output, lung compliance, or oxygenation could signal contralateral pneumothorax.

(2) Pulmonary/ventilation

(a) In patients with severe lung disease or prematurity, the use of an ICU ventilator should be considered for transport and during the surgery.

(b) Avoid hypoxemia and acidosis, which may worsen pulmonary vascular resistance or cause myocardial compromise.

(c) Maintain gentle ventilation with permissive hypercapnia to avoid barotrauma.

(d) Sudden decrease in cardiac output, lung compliance, or oxygenation could signal contralateral pneumothorax.

(e) High-Frequency Oscillatory Ventilation (HFOV) is used when conventional ventilation modes fail to adequately oxygenate or ventilate the patient.

iv) Indication for use of HFOV includes PIP >28 cm H_2O on conventional ventilation, preductal saturation <85%, or postductal saturation <70%.[6]

v) HFOV delivers small tidal volumes that are less than the anatomical dead space yet promotes lung protective gas exchange at an extremely high rate (300-3000/min). The faster rate allows the airway pressure to oscillate around a constant and lower mean.[6]

vi) Positioning and transport of a patient on HFOV is complicated by the limited length of the circuit to the ETT and the requirement of keeping the circuit in a straight line.
   (1) Extracorporeal membrane oxygenation (ECMO) is used in patients with pulmonary hypertension as a temporizing measure until the pulmonary vascular resistance decreases.
      (a) Requires anticoagulation, limiting the ability to place new lines and increasing the risk of surgical and intracranial bleeding
      (b) Requires perfusionists or specially trained ECMO nurses to operate and troubleshoot the circuit around the clock
      (c) Transport to the operating room is tedious, dangerous, and complicated so surgical procedures are often done at the bedside.
   (2) Inhaled nitric oxide (iNO) is a selective pulmonary vasodilator that can improve pulmonary vascular resistance and decrease right to left shunt in patients with severe CDH and pulmonary hypertension.
      (a) Postoperative management/disposition
         i) Patients are usually left intubated after repair.
         ii) Despite repair of the diaphragmatic defect, the lungs are still hypoplastic and pulmonary hypertension risk is still present.
         iii) Sedative and analgesic infusions should be continued to diminish catecholamine release from postsurgical pain and worsening pulmonary vascular resistance.

CHAPTER SUMMARY FOR CONGENITAL DIAPHRAGMATIC HERNIA

General Considerations	CDH is a failure of diaphragmatic fusion that results in herniation of abdominal contents into the thorax. >80% of CDH occur through the left posterior lateral foramen of Bochdalek.
Preoperative Considerations	Neonates present at birth in respiratory distress with acute hypoxemia, scaphoid abdomen, barrel-shaped chest, and absent breath sounds on the ipsilateral side. Hypoplastic lungs have decreased compliance and increased pulmonary vascular resistance. Cardiac anomalies are relatively common (25%) so preoperative echocardiogram is recommended. Intestinal malrotation and gastroesophageal reflux are common. CDH is not a surgical emergency. Management of confounding pulmonary hypertension and hemodynamic instability, if present, should be addressed prior to repair.
Intraoperative Considerations	The airway should be secured immediately with minimal mask ventilation. Hypotension and compromised ventilation can occur when the intrathoracic viscera distend. Placement of a gastric tube after intubation can help decompress the distended viscera and improve ventilation. Consider using a high FiO_2 to help decrease the pulmonary vascular resistance. Use of an ICU ventilator instead of an anesthesia machine should be considered in patients with severe lung disease or prematurity and in whom disconnection would compromise ventilatory status. If hemodynamic instability, poor lung compliance, and hypoxemia occur suddenly, consider contralateral pneumothorax. Use of gentle ventilation techniques with permissive hypercapnia (<65 mm Hg) reduces barotrauma when peak inspiratory pressure is kept low (<25 cm H_2O).

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79 Tracheoesophageal Fistula

Amy Babb • Julianne M. Mendoza • Calvin Kuan

Tracheoesophageal fistula (TEF) occurs in 1:3000 births. The survival rate is 90% in TEF patients who do not have other congenital anomalies. However, up to 50% of TEF patients have other associated congenital anomalies, and 35% have associated cardiac defects.

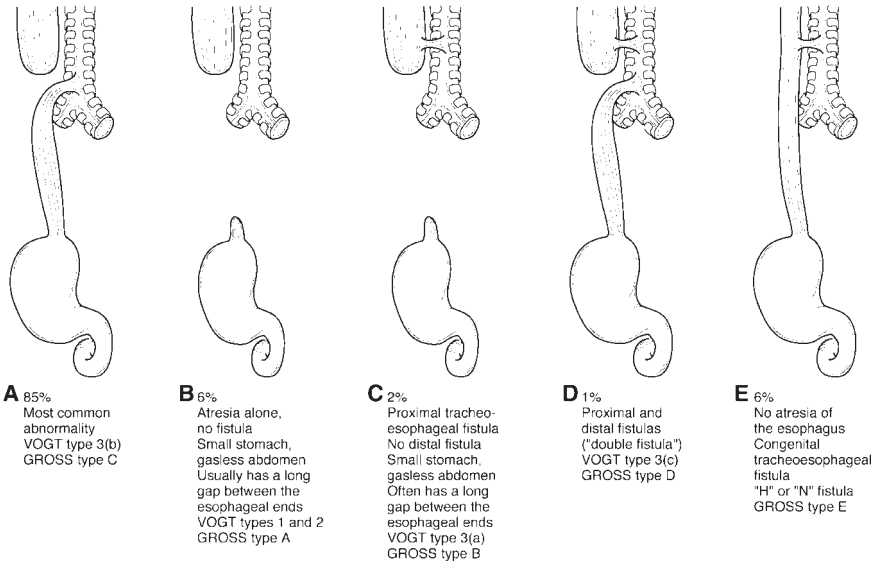
1) **Pathophysiology/anatomy**
   a) The median pharyngeal groove develops into the respiratory and digestive tubes during gestation. Failure of mesenchyme separation between the tubes results in tracheoesophageal fistula (TEF).
   b) There are five types of congenital TEF as described by Gross[1,2] (**Fig. 79-1**).
      i) Gross type A (6% of cases): isolated EA without fistula.
      ii) Gross type B (2% of cases): EA with proximal TEF.
      iii) Gross type C (85% of cases): esophageal atresia (EA) with distal TEF is the most common type.
      iv) Gross type D (rare, 1%): EA with proximal and distal fistulae is the least common type.
      v) Gross type E (6% of cases): TEF without EA. Also known as H-type.
   c) The usual location of the fistula is one to two tracheal rings above the carina; however, it is not always visible.[3]

The most common combination of congenital anomalies seen with TEF is termed the vertebral, anal, cardiac, tracheo-esophageal fistula, radial aplasia or renal defects, and limb association (VACTERL).

2) **Signs/symptoms/clinical findings**
   a) Prenatal diagnosis via ultrasound is classically associated with absent gastric bubble and polyhydramnios, but this may not always be present.
   b) Concern for EA/TEF after birth occurs with regurgitation during the first feeding and corresponding coughing, choking, and cyanosis.
   c) Inability to pass an orogastric catheter past 9-10 cm at the lip is diagnostic of Gross types A, B, C and D.
   d) Radiograph showing coiling of the catheter in the upper mediastinum then confirms the diagnosis of EA/TEF.
   e) Aspiration and subsequent pneumonia are common, especially if diagnosis is delayed.
3) **Surgical treatment**
   a) Most infants have surgical repair in the neonatal period. This consists of division of the fistula and primary esophageal anastomosis ("short-gap atresia").
   b) Patients with a wide separation between the proximal and the distal esophagus, known as long-gap TEF, may require cervical esophagostomy with delayed esophageal repair. Complete repair then occurs after sufficient esophageal growth or interposition with jejunum or colon.
   c) Surgical approach may be through a right thoracotomy or thoracoscopically in the left lateral position.
   d) Rigid bronchoscopy is often done prior to repair to identify the size and location of the fistula and can also help with correct endotracheal tube placement.

Diagnosis of TEF (except H-type) can be confirmed by placing a catheter in the esophagus and visualizing it in a blind upper esophageal pouch on chest radiograph.

FIGURE 79-1 Types of Esophageal Atresia and Tracheoesophageal Fistula.



A. Esophageal atresia and distal TEF. **B.** Esophageal atresia alone (no fistula). **C.** Esophageal atresia with proximal distal fistula. **D.** Esophageal atresia with double fistula. **E.** "H" fistula—percentage reflects approximate incidence. Reproduced with permission from Beasley SW. Esophageal atresia and tracheoesophageal fistula. In: Oldham KT, Colombani PM, Foglia RP, Skinner MA, eds. *Principles and Practice of Pediatric Surgery*. Vol 2. Philadelphia, PA: Lippincott Williams & Wilkins, 2005:1042.

e) Some children have staged surgeries with initial gastrostomy tube placement for stomach venting, and to allow growth before definitive esophageal repair.

4) **Anesthetic management**

a) **Preoperative considerations**

i) The presence of other congenital anomalies should be sought before surgical repair is undertaken—especially VACTERL association.

(1) Cardiac

(a) Echocardiographic evaluation should be completed prior to surgery to evaluate for cardiac anomalies.

(b) Cardiac anomalies that require patency of the ductus arteriosus by prostaglandin infusion, such as hypoplastic aortic arch, have been associated with increased mortality of 57% vs 10% in patients with non–ductal-dependent cardiac lesions.[4]

(c) Confirmation of aortic arch location (ie, presence of a right or left arch) is important for surgical planning.

(2) Renal ultrasound should be obtained preoperatively to rule out structural kidney disease.

(3) Pulmonary

(a) Patients may have respiratory compromise from aspiration and/or prematurity, making intraoperative oxygenation and ventilation challenging.



Up to 50% of patients with TEF have other congenital anomalies, and 35% have congenital heart disease. Preoperative echocardiogram is highly recommended.

ii) Understanding the specific subtype of TEF as well as the location of the fistula is necessary for appropriate airway management.
  (1) Gross types A and B are not at risk for gastric inflation.
  (2) Gross type D has more than one fistula.
iii) An orogastric or a nasogastric tube should be inserted into the stomach or the blind esophageal pouch to remove secretions and prevent aspiration.
iv) Labs
v) Low birth weight <1500 g has been associated with increased morbidity and mortality though surgical repair is generally not delayed.
  (1) Type and cross should be completed, and blood should be immediately available during the procedure.
  (2) Electrolytes should be checked and dehydration corrected with volume resuscitation.

b) **Monitors/lines**
i) Standard ASA monitors.
ii) Additional pulse oximeters.
  (1) In case of failure of single oximeter due to poor perfusion.
  (2) Consider placing preductal and postductal monitors for patients with congenital heart disease.
iii) An arterial line is usually indicated for blood gas and hemodynamic monitoring especially for patients with cardiac anomalies and those whose lesions are ductal-dependent.
iv) IV access sufficient to give rapid volume resuscitation and blood products is required (usually at least one 22-gauge peripheral IV).
v) Central venous line is usually indicated to provide high-concentration dextrose solutions, calcium replacement, or other inotropic infusions.

c) **Intraoperative management**
i) Induction
  (1) Copious secretions tend to be present in TEF patients, and frequent suctioning is required. A preinduction dose of glycopyrrolate 10 μg/kg IV may assist in decreasing secretions for intubation.
  (2) Historically, neonatologists have performed awake intubations to avoid mask ventilation.
  (3) Inhalational induction is often done to help maintain spontaneous ventilation and avoid excessive gastric distention.[5]
  (4) Bronchoscopy may be performed by the surgeon after induction, with the patient breathing spontaneously. This confirms fistula location and excludes multiple fistulae.
  (5) Mask ventilation
    (a) Positive pressure ventilation prior to intubation should be avoided to prevent air going down the fistula instead of the trachea.
    (b) If positive pressure mask ventilation is required, keep peak inflation pressures as low as possible. No more than10-15 cm H_2O is recommended. This can be challenging in patients with compromised respiratory status.
  (6) Intubation
    (a) Ideally, the tip of the endotracheal tube (ETT) should be positioned below the fistula but above the carina, so breaths can be directed into both lungs and not through the fistula. This will not be possible if the fistula is at the carina or in a mainstem bronchus.[6]
    (b) After intubation, the ETT should be advanced initially into the right mainstem bronchus.

(c) While listening with a stethoscope over the left chest, the ETT is pulled back until air is heard in the stomach and then advanced slightly until bilateral breath sounds are heard with no air heard in the stomach.

(d) Alternatively, the ETT can be pulled back until breath sounds are audible in the left chest. If bilateral breath sounds are equal, and no air is heard in the stomach, the ETT can be assumed to be positioned between the carina and fistula.



With positive pressure ventilation (mask or ETT), keep PIP as low as possible (<10-15 cm H_2O) to avoid gastric inflation.

ii) Maintenance

(1) Total IV anesthetic may be indicated because leaking of anesthetic vapor is common during surgical repair of fistula.

(a) An analgesic and anesthetic/hypnotic should be used such as fentanyl or ketamine with midazolam or propofol.

(b) Paralysis should be employed to assist in ventilation and exposure during surgical repair.



The ideal position of ETT is below the fistula and above the carina.

iii) Special anesthetic considerations

(1) Ventilation

(a) The infant should be ventilated with 100% inspired oxygen during the surgical procedure.

(i) An exception to this would be patients with single ventricle physiology who require lower inspired O_2.

(b) Single left-lung ventilation may be desired for optimal exposure during surgical repair but may not be possible secondary to the neonate's small size and inability to adequately oxygenate with single lung ventilation.

(c) The right lung is often compressed with retractors to obtain exposure, and a decrease in SpO_2 indicates that the retracted lung needs to be re-expanded.

(d) If the fistula is located at the carina or the mainstem bronchus, adequate ventilation may be problematic or impossible.

(i) Adjust settings to achieve desired minute ventilation with low peak inspiratory pressures, low tidal volumes, and increased respiratory rates, hand ventilate if necessary.

(ii) Occlusion of the fistula or venting of stomach may be needed to ventilate.

(2) Gastrostomy

(a) The surgeon may place gastrostomy before surgery to allow intermittent venting and ameliorate gastric distension.

(b) Emergent gastrostomy may be rarely necessary.

(3) Occlusion of fistula

(a) Under bronchoscopic guidance, a balloon-tipped catheter can be passed down the trachea to occlude the fistula.

(b) If gastrostomy is in place, a balloon-tipped catheter can be passed retrograde from the stomach into the esophagus to occlude the fistula.



Gastrostomy or occlusion of fistula with balloon-tipped catheter may be necessary if gastric inflation interferes with adequate ventilation.

iv) Emergence/analgesia

(1) Decision about extubation should be individualized based on comorbidities, anesthetic used, and surgical/airway concerns, but most patients will remain intubated in the immediate postoperative period.

(2) Children who have had aspiration events or pneumonia will likely require mechanical ventilatory support after a thoracotomy.

(3) Thoracic epidural catheter threaded up from the caudal space can provide excellent analgesia postoperatively.
(4) Patients with vertebral anomalies should not have an epidural catheter placed without careful consideration.

5) **Postoperative management**
   a) Care should be taken to avoid disrupting the esophageal anastomosis by extending the neck or suctioning the esophagus. Postoperative ventilation and sedation are often necessary to ensure stabilization of the anastomosis.
   b) **Postoperative complications**
      i) Anastomotic leaks may develop but infrequently require surgery.
      ii) A right chest tube is placed to detect leaks and treat pneumothorax in the immediate postoperative period.
      iii) Gastroesophageal reflux may develop with increased risk of aspiration.
      iv) Esophageal stricture is common and often requires multiple dilations.

6) **Anesthetic considerations in children who have a history of repaired TEF**
   a) Careful placement of an ETT after TEF is critical.
      i) Inadvertent placement of the ETT into a residual pit or stump at the fistula ligation site may occur.
         (1) No lung or stomach sounds will be heard on auscultation.
         (2) No end-tidal CO_2 will be observed.
         (3) Treatment involves withdrawing the ETT back slightly until breath sounds are heard.
   b) Patients may have chronic upper GI dysmotility, strictures, and reflux predisposing them to aspiration long term.

CHAPTER SUMMARY FOR TEF

General Considerations	The most common type of TEF is type C defined as distal TEF with proximal atretic esophageal pouch
	The most common combination of congenital anomalies seen with TEF is termed VACTERL association or syndrome
Preoperative Considerations	Up to 50% of patients with TEF have other associated congenital anomalies, and 35% have congenital cardiac defects. Preoperative echocardiogram is recommended
	Understanding the specific subtype of TEF as well as the location of the fistula is necessary for appropriate airway management
Intraoperative Considerations	Positive pressure (via mask or ETT) should employ as low peak pressures as possible to avoid gastric insufflation Position tip of ETT below the fistula and above carina for optimal ventilation Gastrostomy or occlusion of fistula with balloon may be necessary if gastric inflation interferes with adequate ventilation
Postoperative Considerations	Postoperative intubation is usually necessary secondary to patient age/size, comorbidities, and stabilization of the esophageal anastomosis. A right chest tube is often left in place to detect leaks and treat pneumothorax in the immediate postoperative period

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80 Myelomeningocele

Debnath Chatterjee

Myelomeningocele (MMC) is the most common open neural tube defect, characterized by a cleft in the vertebral column with a corresponding epithelial defect, resulting in exposed spinal cord and meninges. The most common location is the lower lumbosacral region. Almost all patients develop a Chiari II malformation, which includes a small posterior fossa and hindbrain herniation. Approximately 70%-90% of the patients develop hydrocephalus and are shunt dependent for life. MMC defects are associated with several other lifelong comorbidities including motor and sensory deficits in lower extremities, neurogenic bladder and bowel function, and cognitive impairment. Postnatal management consists of early surgical repair, within the first 72 hours of life to decrease the risk of infection. The major anesthetic considerations are positioning during intubation to avoid direct pressure on the MMC defect and prone positioning during surgical repair.

1) **Overview**
   a) **Spina bifida (SB)** refers to a group of congenital anomalies of the spine in which the midline structures fail to fuse. There are two distinct types: SB occulta and SB cystica.
   b) **SB occulta** (**Fig. 80-1**)
      i) Mildest form.
      ii) Underlying neural tube defect (NTD) is masked by intact overlying skin.
      iii) External signs are often absent or subtle.
      iv) Most cases go undiagnosed until late childhood or even adulthood.
   c) **SB cystica** (**Fig. 80-1**)
      i) Characterized by an epithelial skin defect in addition to the NTD and includes myelomeningocele (herniation of spinal cord and meninges) and meningocele (herniation is limited to meningeal layers)
   d) **Myelomeningocele (MMC)** (**Fig. 80-2**)
      i) Most common open NTD.
      ii) Most commonly located in the lower lumbosacral region.[1]
      iii) The prevalence of MMC is highly variable and depends on ethnic, geographic, and nutritional factors.
      iv) In the United States, the prevalence ranges from 0.3 to 0.4 per 1000 live births.[2]
   e) The etiology of NTDs is multifactorial and includes both genetic and environmental factors[1] (**Table 80-1**).
   f) Folic acid supplementation during pregnancy decreases the risk of the first occurrence and recurrence of isolated NTDs.[2]

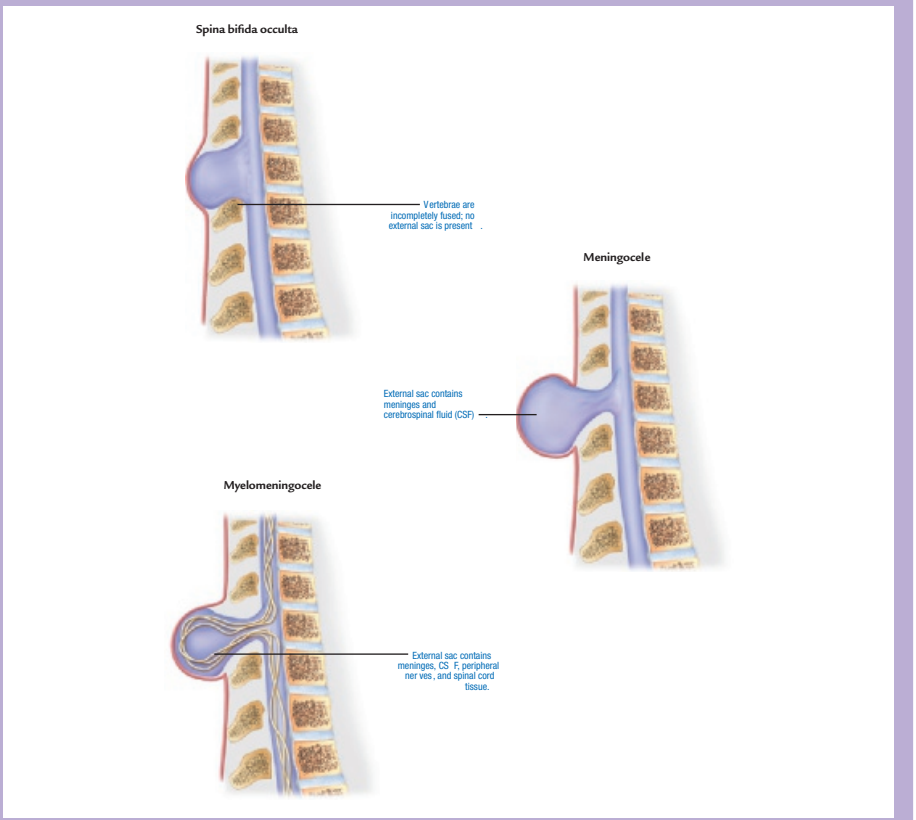


Myelomeningocele is the most common open neural tube defect and is most commonly located in the lower lumbosacral region.

Folic acid supplementation decreases the risk of neural tube defects. All women planning a pregnancy or capable of becoming pregnant should take 400 μg of folic acid supplementation daily.

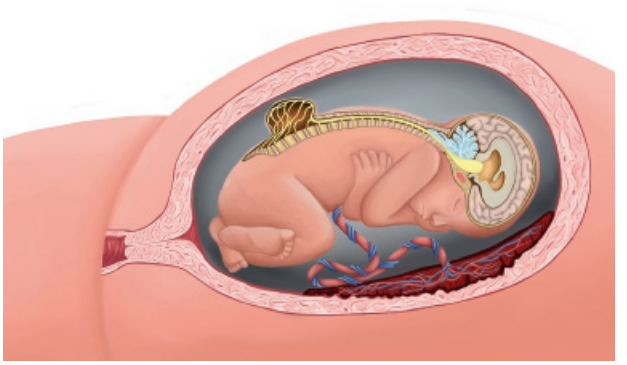
2) **Pathophysiology/embryology**
   a) MMC is caused by a failure of primary neurulation, where the lateral edges of the neural plate become elevated to form the neural folds that subsequently fuse to form the neural tube.[1]
      i) This process normally occurs between the 3rd and 4th week of gestation, a time when most women are not aware that they are pregnant.

FIGURE 80-1 Types of Spina Bifida.



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FIGURE 80-2 Myelomeningocele Defect.



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TABLE 80-1 Etiology of Neural Tube Defects

Etiology of Neural Tube Defects	
Genetic factors	• High recurrence risk (2%-3%) • Risk nearly triples after each affected pregnancy • Increased risk in first-degree relatives (2%-3%)
Environmental factors	• Inadequate folate intake • Use of certain antiepileptic drugs (eg, valproate, carbamazepine) • Maternal fever in first trimester • Pregestational diabetes • Prepregnancy obesity • Lower socioeconomic status • Pesticide exposure

b) There is growing evidence to support a two-hit hypothesis, with failure of primary neurulation being the first hit, followed by a combination of spinal cord inflammation from exposure to amniotic fluid and mechanical trauma to the exposed spinal cord.[2]



Myelomeningocele is caused by a failure of primary neurulation that occurs between the 3rd and 4th week of gestation, followed by mechanical trauma to the exposed spinal cord.

3) **Signs/symptoms/clinical findings**
   a) Screening for NTDs is recommended for all pregnant women using ultrasonography in the second trimester between 18 and 22 weeks of gestation.[2]
   b) Elevated maternal serum alpha-fetoprotein levels to 2.5 multiples of the median (MoM) in the second trimester, between 15 and 18 weeks of gestation, have also been used as a screening tool.[2]
   c) Following prenatal diagnosis, the pregnant woman should be referred to a multidisciplinary team of specialists for counseling and delivery planning. There is no clear consensus on the preferred method of delivery, and the decision should be made individually.[2]
   d) The degree of neurologic deficits depends upon the level of the lesion and is usually severe, resulting in complete paralysis and absence of sensation in the lower extremities.[1] The bowel and bladder are affected as well, resulting in urinary and fecal incontinence.
   e) Almost all patients with MMC develop a Chiari II malformation, characterized by a small posterior fossa, brainstem abnormalities, and downward displacement of the cerebellar tonsils and medulla.[1] Chiari II malformation is also responsible for the cognitive and brainstem dysfunction, swallowing difficulties, and apneic episodes.
   f) 70%-90% of patients with MMC develop obstructive hydrocephalus that requires cerebrospinal fluid diversion by shunting or endoscopic procedures.[1]
   g) Long-term prognosis is dependent on level of MMC lesion, severity of Chiari II malformation, and presence or absence of hydrocephalus.[3]



Ultrasonography in the second trimester between 18 and 22 weeks gestation is recommended for all pregnant women as screening for anomalies, including neural tube defects.

Long-term prognosis is dependent on the level of the defect, severity of Chiari II malformation, and presence or absence of hydrocephalus.

4) **Postnatal care**
   a) After delivery, the newborn should be stabilized and positioned in the prone or lateral position to avoid pressure on the MMC defect.
   b) A sterile, saline-moistened gauze dressing should be used to cover the defect, and prophylactic antibiotics should be administered.[3]

c) These patients are at an increased risk of developing latex sensitivity secondary to repeated exposures, and so latex precautions should be instituted from birth.[3]



Latex precautions should be used in all patients with a MMC defect.

d) A detailed neurological evaluation must be performed to determine the extent of muscle weakness and paralysis, response to sensation, and deep tendon reflexes.[3]

e) Preoperative investigations should include a complete blood count (as baseline), basic metabolic profile (to rule out electrolyte disturbances and dehydration from large defect), type and cross match, cranial ultrasound (to determine ventricular size), renal ultrasound (to rule out genitourinary abnormalities), and echocardiography (to rule out cardiac defects).

READ MORE
Allergy and Anaphylaxis, Chapter 49.

f) Surgical repair can be performed safely up to 72 hours after birth.[3] However, morbidity increases if repair is delayed beyond 72 hours. Immediate CSF diversion before MMC closure is required in a small proportion of patients with severe hydrocephalus.



Early surgical repair within 72 hours after birth is recommended to decrease the risk of infection.

5) **Anesthetic considerations**

a) Positioning during induction and intubation must avoid direct pressure on the MMC defect by using towels or donut rings to support the uninvolved portion of the patient's back.

i) Alternatively, the patient may be induced and intubated in the left lateral decubitus position.

b) **Surgical repair is performed in the prone position.**

i) Rolls must be placed under the chest and hips to allow the abdomen to hang freely.

ii) The patient's head is typically rotated to one side and supported by padding, to avoid direct pressure on the eyes and nose and to keep the ear flat.

c) Standard ASA monitors, including core temperature monitoring, should be used. With the patient being a neonate, maintaining normothermia and euglycemia is critical. Arterial and central lines are rarely necessary.

d) Blood loss can be considerable with large defects, and therefore adequate intravenous access must be obtained.

e) Either intravenous or volatile anesthetic agents may be used for maintenance of general anesthesia. Intraoperative neuromonitoring and neuraxial anesthetic blocks are typically not performed.

f) Postoperative ventilation in the neonatal intensive care unit is not uncommon as the neonates are typically kept in the prone position for 1-3 days following operative closure.



Positioning during induction and intubation must avoid direct pressure on the MMC defect.

CHAPTER SUMMARY FOR MYELOMENINGOCELE

General Considerations	• Myelomeningocele is the most common open neural tube defect, characterized by exposed spinal cord and meninges. • Caused by failure of primary neurulation that normally occurs between the 3rd and 4th week of gestation followed by mechanical trauma to the exposed spinal cord. • Almost all patients develop a Chiari II malformation, which includes a small posterior fossa and hindbrain herniation. • 70%-90% of the patients develop hydrocephalus and are shunt dependent for life. • MMC defects are associated with several other lifelong comorbidities including motor and sensory deficits in lower extremities, neurogenic bladder and bowel function, and cognitive impairment.

Preoperative Considerations	• Early surgical repair should be performed within 72 hours after birth to decrease the risk of infection. Latex precautions must be used. Preoperative investigations should include a complete blood cell count, basic metabolic profile, cranial ultrasound, renal ultrasound, and type and cross match.
Intraoperative Considerations	• Standard ASA monitors should be used. Positioning during induction and intubation must avoid direct pressure on the defect. Adequate venous access must be obtained. Maintaining normothermia and euglycemia is critical and the patient must be well padded in the prone position.
Postoperative Considerations	• Postoperative ventilation in the neonatal intensive care unit is not uncommon.

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81 Patent Ductus Arteriosus

Heidi M. Boules • Calvin Kuan

The patent ductus arteriosus (PDA) is a remnant from fetal circulation extending from the main pulmonary artery to the descending aorta usually immediately distal to the origin of the left subclavian artery. Functional closure usually occurs within the first day of life. Anatomic closure, where the PDA loses the ability to reopen, may not occur for several weeks. Up to 60% of preterm infants weighing <1500 g may have a PDA; the incidence is even higher in infants weighing <1000 g (**Fig. 81-1**).

1) **Pathophysiology**
   a) PDA results in left-to-right shunting from the descending aorta to pulmonary artery once pulmonary vascular resistance (PVR) has decreased below systemic vascular resistance (SVR).
   b) The left-to-right shunt results in pulmonary overcirculation and volume overload of the left heart.
   c) If PVR remains high, blood may shunt right to left, resulting in cyanosis.
2) **Signs/symptoms/clinical findings**
   a) **Cardiovascular**
      i) Harsh systolic ejection murmur that may be continuous at left upper sternal border (LUSB)
      ii) Bounding pulses, hyperdynamic precordium, and palmar pulses may also be present.
      iii) Tachycardia may occur in response to increased volume load.
      iv) In young children, particularly premature infants, the shunt effectively steals blood flow from the aorta, resulting in relative hypotension (especially low diastolic pressures) and hypoperfusion of coronary arteries and abdominal viscera, which may require inotropic support.
      v) Older children may develop pulmonary vascular changes and CHF.
   b) **Respiratory**
      i) Isolated PDA occurs usually with left-to-right shunt, and therefore SpO_2 should be normal.
      ii) With complex congenital heart disease, shunt may be right to left or bidirectional, so the patient may be cyanotic.
      iii) Pulmonary overcirculation may cause tachypnea and require increased ventilatory support and O_2 requirements with an inability to wean from ventilator.
   c) **Others**
      i) Intraventricular hemorrhage
      ii) Necrotizing enterocolitis due to pulmonary steal
      iii) Renal failure due to pulmonary steal
      iv) Chronic lung disease
   d) **Older children/adults may be asymptomatic.**



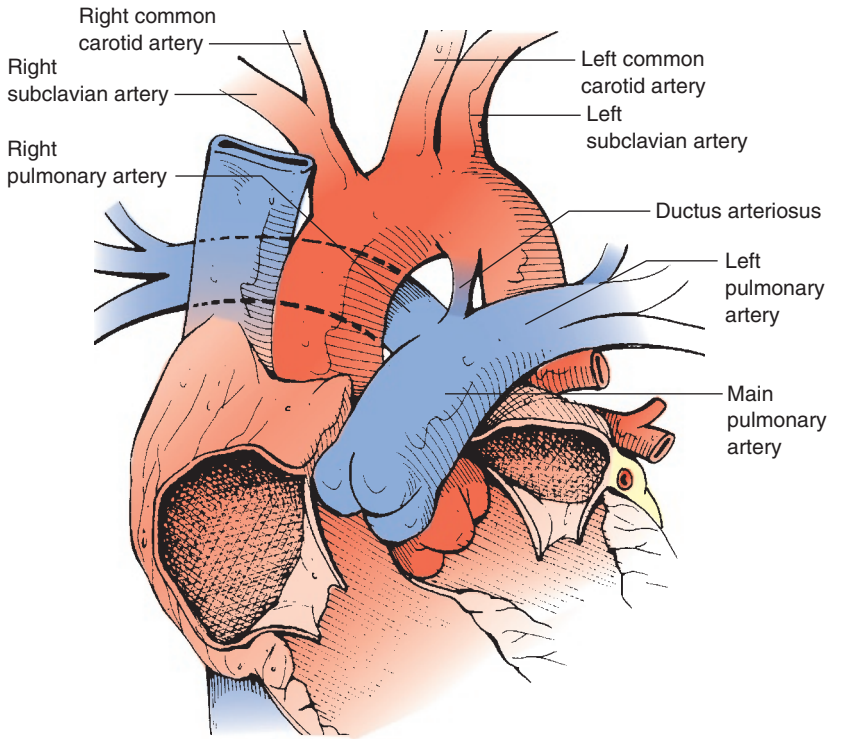
Functional closure of DA usually occurs within the first day of life. Anatomic closure may occur several weeks later.

A PDA shunting left to right will cause pulmonary overcirculation. A PDA shunting right to left will cause cyanosis.

Cardiovascular problems may include low diastolic blood pressure and systemic hypoperfusion requiring inotropic support.

Pulmonary problems may include pulmonary overcirculation and inability to wean off the ventilator.

FIGURE 81-1 Patent Ductus Arteriosus. The ductus arteriosus connects the junction of the main and left pulmonary arteries to the descending aorta adjacent to the origin of the left subclavian artery



Reproduced with permission from Rouine-Rapp K, Miller-Hance WC. Transesophageal echocardiography for congenital heart disease in the adult. In: Perrino AC, Reeves ST, eds. *A Practical Approach to Transesophageal Echocardiography*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008:381.

e) EKG may be normal or show left ventricular hypertrophy in small to moderate PDA. Bilateral ventricular hypertrophy may be seen with a large PDA.
f) Chest radiograph (CXR) may show cardiomegaly and increased pulmonary vascular markings with large left-to-right shunts.
g) Echocardiographic findings include dilated left-sided chambers and diastolic flow reversal through the descending aorta.

3) **Treatment**
   a) **Medical**
      i) Management may include diuretics and fluid restriction.
      ii) Indomethacin/cyclooxygenase inhibitors may be administered to stimulate closure.[2]
         (1) Risk of renal and platelet dysfunction and intestinal perforation
         (2) Relatively contraindicated in infants with a history of single kidney, renal dysfunction, intracranial hemorrhage, or necrotizing enterocolitis

iii) Acetaminophen is gaining favor as a second-line agent to promote ductal closure, showing similar efficacy with improved side effect profile compared to cyclooxygenase inhibitors.[3]

b) **Surgical**
   i) Ligation of the PDA, usually through thoracotomy (left or right depending on direction of aorta), either at bedside in neonatal intensive care unit (NICU) or in OR

c) **Percutaneous intervention**
   i) Interventional cardiac catheter insertion of various occlusion devices in children 6 kg and greater, with select centers trialing devices in children <1 kg

d) **Choice of treatment depends on patient comorbid conditions and is center-specific.**

e) **Surgical closure of PDA has the lowest reintervention rate;** however, it is associated with increased risk of retinopathy of prematurity, pneumothorax, and increased length of stay. There is no greater mortality associated with surgical intervention over percutaneous intervention.[4]

4) **Anesthetic management**

a) **Preoperative evaluation**
   i) Assess overall cardiovascular status and degree of pulmonary overcirculation and shunting.
   ii) Assess pulmonary status and arterial blood gas results if available.
   iii) Cross-matched blood should be available at bedside.

b) **Monitors/lines**
   i) Standard monitors with two pulse oximeters—one on right hand (preductal) and second one in lower extremity (postductal)
      (1) Loss of waveform in lower extremity monitor will help identify inadvertent clamping of aorta.
      (2) If the pulmonary artery is ligated accidentally, SpO_2 in both extremities and end-tidal CO_2 will decrease.
   ii) Invasive BP monitoring is desirable but may not be necessary depending on hemodynamic status of the individual patient.
   iii) If an arterial line is not present, noninvasive BP should be measured in the right arm to best estimate cerebral perfusion.



Use two pulse oximeters: one on preductal right hand and second on lower extremity to catch inadvertent clamping of aorta

If arterial line not available, NIBP should be measured in the right arm to best estimate cerebral perfusion.

c) **Intraoperative management**
   i) Induction
      (1) Patients are usually intubated in the NICU and do not require premedication.
      (2) If patient is hemodynamically unstable and/or on inotropic support, consider waiting until surgeon is ready to make incision before administering anesthetic agents.
   ii) Maintenance
      (1) Analgesia
         (a) Fentanyl 1-2 mg/kg IV q5-10 min as needed
         (b) Ketamine 1-2 mg/kg IV q5-15 min as needed
      (2) Muscle relaxation
         (a) Pancuronium 0.1-0.2 mg/kg IV
         (b) Vecuronium 0.1-0.2 mg/kg IV
         (c) Rocuronium 0.5-1 mg/kg IV
   iii) Special anesthetic considerations
      (1) Continue dextrose containing IV fluids or parenteral nutrition at basal rate.
      (2) Airway/ventilation
         (a) Know the location of the ETT as surgical retraction during surgery may cause endobronchial intubation with desaturation or bradycardia.

(b) If an uncuffed ETT is in place, one may need to reintubate with cuffed ETT to facilitate adequate ventilation after administration of paralysis and during surgical manipulation.

(c) Ventilation strategy of mild hypoventilation and minimizing FiO_2 may avoid lowering PVR and reduce pulmonary overcirculation and systemic hypotension.



Surgical retraction of lungs during procedure may cause endobronchial intubation and desaturation or bradycardia.



Ventilation strategy of mild hypoventilation and minimizing FiO_2 may avoid lowering PVR and reduce pulmonary overcirculation and systemic hypotension resulting in coronary ischemia and other end-organ ischemia.

(3) With successful PDA ligation, arterial diastolic and mean pressure should increase and the PDA murmur disappear.

d) **Postoperative management**

i) Patients are usually left intubated to recover in the NICU.

ii) Extubation readiness should be left to the discretion of the neonatologist as other medical issues may need to be resolved.

iii) For postoperative analgesia, an intercostal nerve block may be performed by surgeon.

CHAPTER SUMMARY FOR PDA LIGATION

General Considerations	Patients are commonly premature infants in the NICU.
Preoperative Considerations	Cardiovascular status—blood pressure and peripheral perfusion and pulmonary overcirculation. Pulmonary status—degree of cyanosis and ventilatory settings. Also assess renal function and acid-base status.
Intraoperative Considerations	Have two pulse oximeters—one on right hand and second on lower extremity. Lung retraction may cause endobronchial intubation and desaturation or bradycardia. Ventilation strategy and FiO_2 may affect degree of left-to-right shunting.
Postoperative Considerations	Patients usually left intubated and sedated in NICU.

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82 Omphalocele and Gastroschisis

Romy Yun • Calvin Kuan

Omphalocele and gastroschisis are congenital abnormalities of the anterior abdominal wall. These abdominal wall defects (AWD) are a significant source of morbidity and mortality in the neonatal population despite advances in neonatology and surgical care. They can be considered surgical emergencies due to the risk for bowel ischemia, bowel obstruction, and sepsis.

1) **Pathophysiology/embryology**
   a) **Omphalocele**
      i) The herniation of abdominal contents through a defect at the base of the umbilicus; extruded contents are covered by a membranous sac
      ii) Speculated to result from incomplete return of bowel into the abdominal cavity after physiologic umbilical herniation during embryonic development



Omphalocele is covered by a membranous sac. Gastroschisis is not covered.

   b) **Gastroschisis**
      i) Extruded contents are uncovered.
      ii) Likely results from a vascular accident involving the omphalomesenteric artery during gestation or failed development of one of the lateral folds of the embryo.
2) **Signs/symptoms/clinical findings** (**Table 82-1**)
   a) **Omphalocele**
      i) Midline abdominal wall defect.
      ii) Depending upon the size of defect, extruded viscera may include small or large bowel, stomach, spleen, or liver.
      iii) The herniated viscera are covered with a membranous sac composed of fused layers of the amnion and peritoneum, and the bowel is usually morphologically and functionally normal.
      iv) Omphalocele is often associated with other anomalies.
         (1) Chromosomal anomalies
         (2) Congenital heart disease
         (3) Pulmonary hypoplasia
         (4) Genitourinary anomalies (ie, exstrophy of the bladder)
         (5) Neural tube defects
         (6) Beckwith-Wiedemann syndrome
         (7) Pentalogy of Cantrell



Patients with omphalocele often have other serious congenital anomalies.

   b) **Gastroschisis**
      i) An isolated full-thickness defect lateral (usually on the right) to the normally inserted umbilicus.
      ii) The herniated viscera and intestines are not covered and are exposed to amniotic fluid and then air at time of delivery leading to inflammation, edema, and functionally abnormal bowel.
      iii) Animal models suggest that amniotic fluid is directly toxic to exposed bowel and may lead to dysmotility and a thickened fibrous peel (damaged bowel that is stiff and inflamed).[1]

TABLE 82-1 Abdominal Wall Defects

	Incidence (Live Births)	Gender	Associated Anomalies	Prematurity Incidence
Omphalocele	1:3 000-10 000	M > F	High association with other anomalies (50%-76%); Cardiovascular 15%-25%	33%
Gastroschisis	1:30 000	M = F	Rare	58%

Reproduced with permission from Holzman RS, Mancuso TJ, Polaner DM. *A Practical Approach to Pediatric Anesthesia*. Philadelphia, PA: Lippincott Williams & Wilkins; 2008:295-298.

iv) Although there are less associated anomalies with gastroschisis, nearly 25% of cases have associated gastrointestinal problems including atresia, volvulus, stenosis, and compromised bowel function.[2]
v) Intrauterine growth retardation is noted in a significant number of gastroschisis cases.[3]

3) **Surgical treatment**
   a) Primary or complete surgical reduction is carried out if the abdomen is large enough to accommodate return of viscera without excessive intra-abdominal pressure that may compromise organ perfusion and decrease ventilatory reserve.
   b) Staged reduction involves covering viscera with prosthetic Silon pouch and gradually reducing the size of the pouch in stages allowing for the abdominal cavity to accommodate to the increased mass over 1-2 weeks.[4]
4) **Anesthetic management**
   a) **Preoperative considerations**
      i) Management of these lesions from birth until surgical repair is directed by the following goals:
         (1) Minimizing heat and fluid loss from exposed surfaces
         (2) Normalizing intravascular fluid status
         (3) Maintaining perfusion to the herniated viscera
         (4) Prevention of sepsis and hypothermia
      ii) Cover mucosal surfaces with sterile, saline-soaked dressing; plastic wrap further decreases evaporative losses.
      iii) Nasogastric tube placement helps to prevent intestinal distention.
      iv) Continually assess intravascular fluid status.
      v) Empiric antibiotics are administered given high risk of infection from peritonitis, ischemia, and parenchymal disease.
      vi) Identification of associated anomalies (eg, cardiac, chromosomal, Beckwith-Wiedemann).[5]
   b) **Monitors/lines**
      i) Standard ASA monitors
      ii) Additional pulse oximeters recommended for upper and lower extremities as lower extremity perfusion may be compromised after closure.
      iii) Arterial line recommended for hemodynamic monitoring and blood gases to gauge effects of abdominal pressure on ventilation.
      iv) Central venous line is recommended for additional access and/or postoperative parenteral nutrition.
   c) **Intraoperative management**
      i) Induction
         (1) An orogastric tube should be placed prior to induction to minimize aspiration and decrease gastric distention.

(2) The method of induction should be determined based on the clinical condition of patient including factors such as airway, degree of obstruction, and hemodynamic stability.
(3) Choose an endotracheal tube (ETT) size that will allow for ventilation with higher peak inspiratory pressures that may be required after closure.
(4) Consider using a cuffed ETT.



Choose an ETT that will allow for ventilation with higher peak inspiratory pressures that may be required after abdominal closure. Consider using a cuffed ETT.

ii) Maintenance
(1) Avoid nitrous oxide.
(2) Anesthetic with vapor vs TIVA vs balanced techniques depend on clinical situation and availability of access.
(3) Muscle relaxation is highly advised and may be requested by surgeon to facilitate closure.

iii) Special anesthetic considerations
(1) Higher peak inspiratory pressures may be required to maintain minute ventilation after closure.
(2) Use of PEEP may help minimize atelectasis after closure.
(3) Communicate with surgeons regarding neuromuscular blockade in order to evaluate intra-abdominal pressures.[5]
(4) Assessment of intra-abdominal pressure; if <20 mm Hg, primary closure is likely possible.
(5) Consider using peak inspiratory pressure or transduction of intragastric or bladder pressures as diagnostic adjuncts.[5]

iv) Emergence
(1) Except for the most benign small-volume herniations, most patients will require postoperative ventilatory support and NICU care.
(2) Many may require continued paralysis to minimize intra-abdominal pressure.

d) **Postoperative management**
i) Perioperative risks include respiratory failure, ARDS, abdominal compartment syndrome, renal failure, infection, coagulopathy, and hypothermia.[3,5]
ii) Necrotizing enterocolitis has been reported to complicate the postoperative course of up to 20% of neonates with gastroschisis.[3]
iii) Patients with prolonged ileus may require parenteral nutrition.
iv) Pain control may be achieved with epidural analgesia via caudal, lumbar, or thoracic approaches with radiographic confirmation.[5]
v) Outcomes in gastroschisis have improved over past few decades with contemporary overall survival rate as high as 90%-95%.[6,7]

CHAPTER SUMMARY FOR OMPHALOCELE AND GASTROSCHISIS

General Considerations	Omphalocele is the herniation of abdominal contents through a defect at the base of the umbilicus; extruded contents are covered by a membranous sac Gastroschisis is a defect off midline (usually to the right); extruded contents are uncovered These are considered a neonatal surgical emergency due to the concern for major fluid deficits, bowel obstruction, ischemia, and sepsis
Preoperative Considerations	Omphaloceles have a high association with other anomalies Although there are less associated anomalies with gastroschisis, nearly 25% of cases have associated gastrointestinal problems including atresia, volvulus, stenosis, and compromised bowel function Management of these lesions from birth until surgical repair is directed at minimizing heat and fluid loss from exposed surfaces, normalizing intravascular fluid status, maintaining perfusion to the herniated viscera, and preventing development of sepsis and hypothermia Naso-/orogastric tube placement to prevent intestinal distention
Intraoperative Considerations	Choose an ETT size that will allow for ventilation with higher peak inspiratory pressures that may be required after closure Consider using a cuffed ETT Muscle relaxation highly advised and may be requested by surgeon to facilitate closure Higher peak inspiratory pressures may be required to maintain minute ventilation after closure Use of PEEP may help minimize atelectasis after closure
Postoperative Considerations	Most patients will require postoperative ventilatory support and NICU care

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83 Intussusception

Nicholette Kasman

Intussusception is the second most common cause of bowel obstruction in neonates after pyloric stenosis. There is a higher incidence in males at a ratio of 2:1. The bowel telescopes upon itself leading to ischemia and edema and occasionally perforation and sepsis. Many of the management principles discussed here also apply to any child with a bowel obstruction.

1) **Pathophysiology**
   a) Intussusception is the second most common cause of bowel obstruction in young children after pyloric stenosis.[1]
   b) Altered peristalsis causes telescoping of the bowel. This can result in bowel edema, ischemia, necrosis, and peritonitis.[4]
   c) Approximately 30% of patients experience an antecedent viral illness before the onset of intussusception.[1]
   d) It occurs in males more frequently, at a ratio of 2:1.[2]
   e) The majority of cases are classified as idiopathic and ileocolic.[2,3]
   f) Some of these idiopathic cases may be caused by hyperplastic lymphoid tissue, which acts as a **"lead point"** around which the bowel turns.[2,3]
   g) Known pathologic lead points include Meckel diverticulum, polyps, bowel wall tumors, and hemangiomas such as those in Henoch-Schönlein purpura.



Many of the management principles for intussusception also apply to any child with a bowel obstruction.

2) **Signs/symptoms/clinical findings**
   a) The classic presentation of intussusception includes
      i) Colicky abdominal pain
      ii) "Red currant jelly" stools
      iii) Vomiting
   b) Neonates with acquired lesions rarely present with all symptoms. Symptoms mimic any form of bowel obstruction or viral gastroenteritis and include abdominal pain, vomiting, and diarrhea.
   c) Dehydration, vomiting, and possible bowel perforation may lead to aspiration pneumonia, dehydration, hypovolemia, and metabolic abnormalities.



Classic presentation of intussusception is (a) colicky abdominal pain, (b) red currant jelly stools, and (c) vomiting.

3) **Treatment**
   a) Diagnosis is made with ultrasound, which has a sensitivity and specificity of 98%.
   b) The classic ultrasound finding is a hypoechoic ring of bowel with central echogenicity known as the "target sign" or "doughnut sign." Seen in transverse view, it is called the "pseudokidney sign".[2]
   c) Air enemas are the treatment of choice. They are successful in as many as 90% of cases. Recurrence occurs at a rate between 8% and 15%.
   d) Perforation is the most concerning potential complication of reduction by enema, having an occurrence rate of 1%-4%.
   e) Perforation with air could lead to pneumoperitoneum, which can be treated by needle decompression of the abdomen.
   f) Operative treatment is indicated for patients who are hemodynamically unstable or have bowel necrosis, perforation, or peritonitis or when facilities to perform an enema reduction are not available.[4]

g) If possible, laparoscopic surgical techniques are preferred to open given the decreased length of stay and decreased postoperative complications.

4) **Anesthetic management**

a) **Preoperative consideration**

i) Evaluation of fluid status

(1) Hypovolemia secondary to vomiting may cause an initial contraction alkalosis followed by a metabolic acidosis.

(2) Distributive or relative hypovolemia may occur from sepsis due to infection or ischemic bowel.

(3) Baseline electrolytes should be obtained; however, there may not be time to wait to correct abnormalities prior to surgery.

ii) Anemia may be present secondary to intra-abdominal bleeding. Type and cross should be sent.

iii) Full stomach precautions should be taken. The patient may benefit from placement of a preoperative nasogastric or orogastric tube.[5]

b) **Monitors/lines**

i) Standard ASA monitors may be adequate in patients who have been medically optimized.

ii) A preoperatively placed IV may be adequate for access unless patient is hemodynamically unstable or septic.

iii) In hemodynamically unstable patients, an additional arterial line and possibly a central venous line for evaluation of fluid status may be necessary.

c) **Intraoperative management**

i) Induction

(1) The patient should be approached as a "full stomach."

(2) Place a preoperative IV and proceed with a rapid sequence induction.

(3) Placement of an NG/OG prior to induction can decrease risk of aspiration.

(4) Vigorous suctioning can induce bradycardia. Consider glycopyrrolate 10 mcg/kg as a premedication.

(5) Despite standard preoxygenation, infants desaturate rapidly. Judicious, low-pressure ventilation while maintaining cricoid pressure may be indicated.

(6) Infants have a high volume of distribution. Watch the clock and give the muscle relaxant time to work, otherwise gagging, coughing, laryngospasm, and bronchospasm are likely to occur.

(7) Patients are often dehydrated. Albumin and calcium may be useful for sudden drops in blood pressure.



Suction out the stomach prior to induction to minimize risk of aspiration. Then proceed with rapid sequence induction.

(8) Agents

(a) Induction

(i) Ketamine, etomidate, propofol, or other agents at anesthesiologist's discretion depending on patient stability

(b) Muscle relaxants

(i) Use succinylcholine or high-dose rocuronium (preferred) for rapid sequence induction.

(c) Narcotics

(i) Midazolam may be considered for preoperative sedation.

(ii) Fentanyl, morphine, or hydromorphone may be used for analgesia.

(d) Inhaled agents

(i) Mask induction is relatively contraindicated.

ii) Maintenance
  (1) Inhaled agents are standard, though alternatively an IV infusion of ketamine (0.5-2 mg/kg/h IV) may offer greater hemodynamic stability.
  (2) Although there is conflicting evidence regarding the occurrence and significance of bowel distension with the use of N_2O, many surgeons prefer that it be avoided.
  (3) Regional techniques:
    (a) Rectus sheath and/or transversus abdominis blocks can be placed under ultrasound. Consult with the surgeon about the location of the trocars.
    (b) An epidural or caudal epidural might be desirable for a laparotomy; however, the risk of neuraxial infection in the setting of a potentially septic patient needs to be considered.[6]

iii) Special anesthetic considerations
  (1) In severe cases, there may be significant fluid losses from open procedures and third spacing.
  (2) Gangrenous bowel may result in severe metabolic acidosis and require correction of electrolyte abnormalities.
  (3) Vigilance must be maintained for developing sepsis.
  (4) Blood transfusion may be indicated with bleeding and/or fluid shifts.

iv) Emergence
  (1) The decision of whether or not to extubate should be made based on the stability of the patient.

d) **Postoperative management/analgesia**

i) There is a risk of persistent postoperative ileus, venous thrombosis, infection, and abdominal compartment syndrome.

ii) Systemic analgesics combined with intraoperative regional anesthetic techniques are generally adequate for postoperative pain control.

iii) If there is significant edema or third spacing, the surgeon may not be able to close the abdomen. Postoperative mechanical ventilation in a pediatric intensive care unit may be necessary.

CHAPTER SUMMARY FOR INTUSSUSCEPTION

General Considerations	Intussusception occurs when a more proximal portion of bowel invaginates into more distal bowel. Initially, compression of blood vessels causes venous congestion and bowel edema; however, as the obstruction progresses, the arterial supply may become compromised, leading to ischemia, bowel necrosis, and gangrene.
Preoperative Considerations	The classic presentation of intussusception includes colicky abdominal pain, "red currant jelly" stools, and vomiting. Neonates rarely present with all of the classic symptoms.
Intraoperative Considerations	Full stomach precautions should be taken. Stomach should be suctioned out with a preinduction orogastric tube prior to rapid sequence induction. Fluid resuscitation is critical. Patients are likely to be dehydrated, and there may be significant evaporative and third-space losses.
Postoperative Considerations	Systemic analgesics combined with intraoperative regional anesthetic techniques are generally adequate for postoperative pain control. Patients are at risk for persistent postoperative ileus, venous thrombosis, infection, and abdominal compartment syndrome.

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84 Hypertrophic Pyloric Stenosis

Pikulkaew Dachsangvorn • Calvin Kuan

The classic presentation of pyloric stenosis is a 3-week-old male infant with acute nonbilious emesis after feeding who is still hungry afterward. Most patients present between 2 and 12 weeks of age. The incidence is ~1 in 500 live births with a male-to-female ratio of 4:1. Of interest, 30% of patients with pyloric stenosis are firstborn males.

1) **Pathophysiology**
   a) Gradual hypertrophy of muscularis layer of pylorus resulting in progressive gastric outlet obstruction.
   b) Persistent vomiting leads to loss of fluids and electrolytes (eg, hydrochloric acid from the stomach), resulting in classic hypovolemia with hypochloremic, hypokalemic metabolic alkalosis.
   c) Increased aldosterone induces a renal response to retain sodium and hydrogen by secreting potassium instead, which initially normalizes serum pH.
   d) Upon depletion of electrolytes (eg, total body potassium), the kidneys secrete hydrogen (paradoxic aciduria), further increasing metabolic alkalemia.
   e) Continued emesis can result in prerenal azotemia, hypovolemic shock, and metabolic acidosis.



The classic electrolyte abnormality in patients with pyloric stenosis is a hypochloremic, hypokalemic metabolic alkalosis.

2) **Signs/symptoms/clinical findings**
   a) Early
      i) Nonbilious emesis after feeding. Infant is awake and hungry.
      ii) Persistent episodic projectile vomiting develops in 70% of patients.
   b) Later: evidence of hypovolemia
      i) Tachycardia
      ii) Decreased urine output
      iii) Poor skin turgor
      iv) Decreased activity
   c) Much later: severe dehydration
      i) Weight loss
      ii) Lethargy
      iii) Shock
      iv) Sunken anterior fontanelle
3) Diagnosis
   a) Clinical
      i) History of emesis as described above.
      ii) Palpation of "olive-shaped mass" in right upper quadrant near the midline in the abdomen.
      iii) Severe dehydration or electrolyte abnormalities may be absent due to early diagnosis.
   b) Radiographic
      i) The imaging modality of choice is abdominal ultrasound, which shows pyloric muscle wall thickness >3 mm, pyloric canal length 14-20 mm, pylorus diameter >12 mm, and vascularity of the pylorus mucosa and muscles[1] (**Figs. 84-1** and **84-2**).
      ii) Upper GI barium swallow is performed when the ultrasound is nondiagnostic.

FIGURE 84-1 Sonographic Measurement of Pylorus Muscle Wall Thickness and Pylorus Canal Length

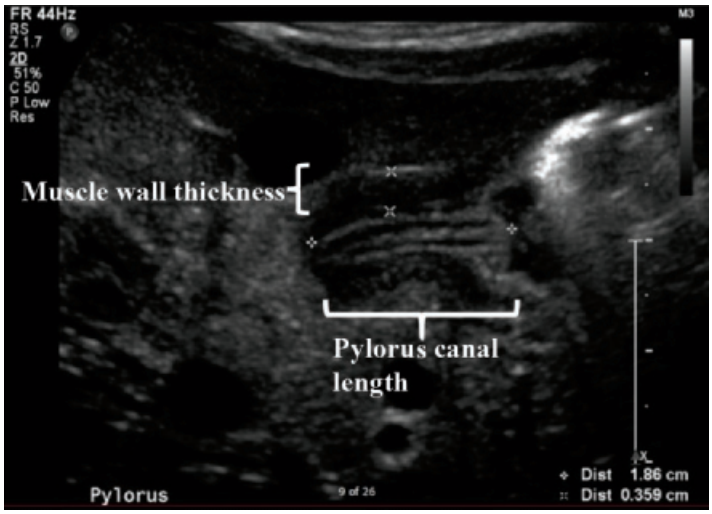
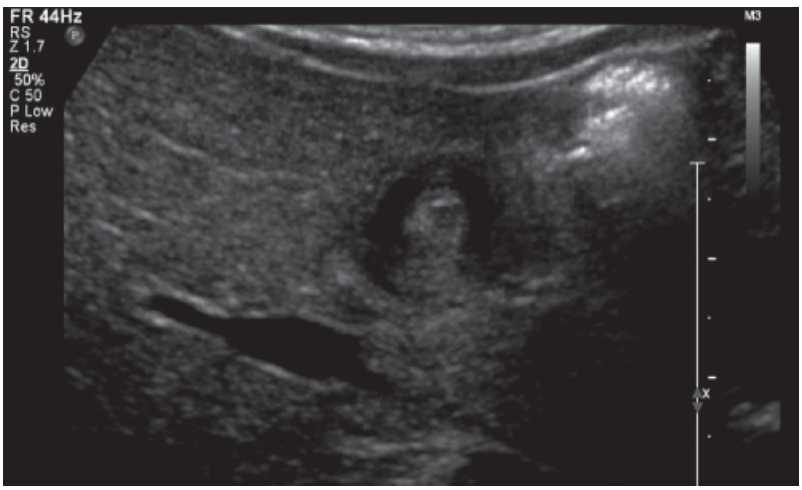


FIGURE 84-2 Target Sign



Pylorus in transverse plane in pyloric stenosis.

4) **Surgical treatment**
   a) **Surgical pyloromyotomy** (open vs laparoscopic) with typical surgical time 0.5-1 hour without significant blood loss or fluid shifts.
   b) **Laparoscopic pyloromyotomy** (LAP) may lead to decreased length of stay and shorter time to full feeds. However, LAP has a slightly increased risk of incomplete pyloromyotomy. The potentially increased risk of mucosal injury and duodenal perforation remains debatable[2,3].
   c) **This is not a surgical emergency**—failure to restore intravascular volume and metabolic stabilization can lead to significant risk of mortality.
   d) Nonoperative management[4]:
      i) Atropine sulfate is the only nonoperative treatment; 80% success rate.
      ii) Requires weeks of hospitalization and parenteral nutrition.
      iii) It should only be considered for extremely high-risk neonates or in parts of the world where it is unsafe to perform neonatal surgery.
      iv) Atropine sulfate can be used as a rescue therapy for incomplete pyloromyotomy.



Surgical repair of pyloric stenosis is not an emergency. Correction of volume and metabolic disturbances should be first priority.

5) **Anesthetic management**
   a) **Preoperative considerations**
      i) Clinical evaluation of hydration status
         (1) Urine output >0.5 mL/kg/h.
         (2) Normal capillary refill time, moist mucous membranes.
         (3) Normal wakefulness and activity level.
         (4) Normal saline may be more effective than lactated Ringer solution at restoring chloride and bicarbonate level.[2]
         (5) After achieving adequate urine output, maintenance fluid must contain potassium to avoid worsening alkalosis.
      ii) Laboratory evaluation
         (1) Goal of plasma chloride >100 mEq/L.
         (2) Goal of plasma bicarbonate <29 mEq/L.
         (3) Treatment of hypokalemia should be initiated only after alkalemia is resolved and urine output verified. Urine electrolytes are no longer routinely ordered for patients with pyloric stenosis.
   b) **Monitors/lines**
      i) Standard ASA monitors. Invasive monitoring is usually not necessary.
      ii) Single functioning peripheral IV should be in situ preoperatively.
   c) **Intraoperative management**
      i) Induction
         (1) Prior to induction, perform awake orogastric suctioning in supine and left and right lateral positions with large-bore orogastric tube (OG) to minimize the risk of aspiration.
         (2) Preoxygenate with 100% oxygen.
         (3) Awake intubation in infants can carry the risk of soft tissue injury, breath-holding, laryngospasm, and aspiration.[2] Only consider in cases of anticipated difficult airway.
         (4) Induction agents:
            (a) Vomiting and aspiration can result from inadequate depth of anesthesia and insufficient paralysis during intubation.[2]
            (b) Short-acting IV anesthetics are recommended for induction (propofol or ketamine).
            (c) Anticholinergic agent can be used to minimize vagal response during OG tube insertion and intubation. However, this is not evidence-based.

(5) Modified rapid sequence induction (RSI) or controlled RSI.
(a) Consider performing gentle mask ventilation with pressure <12 cm H_2O to minimize oxygen desaturation.
(b) Cricoid pressure is controversial as inappropriate cricoid pressure can interfere with mask ventilation and intubation in neonates.[2]
(c) Rocuronium and succinylcholine are both appropriate for paralysis.
(6) Replace OG tube following intubation.



Prior to induction, perform awake orogastric suctioning of stomach contents in supine and lateral positions.

ii) Maintenance
(1) Inhalational agent or TIVA with propofol or balanced technique. Nitrous oxide is not recommended.
(2) Local anesthetic infiltration is recommended. Avoid opioids, given the risk of post-op apnea due to patient age and abnormal CO_2 response caused by metabolic alkalosis.

iii) Special anesthetic considerations
(1) Aspiration risk is high due to nature of disease despite appropriate fasting time.
(2) Timing and dosage of muscle relaxants must be considered as the surgical time is short.
(3) These neonates are at increased risk of postoperative apnea due to abnormal CO_2 response from preoperative metabolic alkalosis. Postoperative apnea monitoring and avoidance of opioid is recommended.

iv) Emergence
(1) Awake extubation with full reversal of nondepolarizing blockade is recommended.

v) Regional anesthesia
(1) Spinal, thoracic epidural, and caudal anesthesia have been used in place of general anesthesia in highly experienced centers.
(2) Further studies are needed to assess the risk of local anesthetic toxicity and inadequate anesthesia requiring sedation or conversion to general anesthesia.
(3) Currently, regional anesthesia should only be practiced by those with significant experience in neonates and with careful attention to local anesthetic dosing.

d) **Postoperative management**

i) Analgesia
(1) Acetaminophen may be administered rectally or intravenously during perioperative period.
(2) Ketorolac is not recommended due to bleeding risk, particularly in infants <21 days of age.[2]
(3) The use of opioid as a rescue should be rare as the combination of local anesthetic and acetaminophen are generally adequate.



Patients should be monitored for apnea for at least 12 hours following surgery due to abnormal CO_2 response caused by the preoperative metabolic alkalosis.[5]

ii) Disposition
(1) Patients should be monitored for apnea in the hospital for at least 12 hours following surgery due to abnormal CO_2 response caused by the preoperative metabolic alkalosis.[5]

iii) Mortality
(1) Mortality is approaching zero in most specialty medical centers. However, high mortality rate remains in parts of the world with limited resources.

CHAPTER SUMMARY FOR HYPERTROPHIC PYLORIC STENOSIS

General Considerations	Hypertrophy of muscularis layer of pylorus resulting in high-grade gastric outlet obstruction. Classic findings are of hypovolemia with hypochloremic, hypokalemic metabolic alkalosis.
Preoperative Considerations	Surgical repair is not an emergency. Failure to ensure adequate volume resuscitation and electrolyte correction prior to surgery can significantly increase mortality risk.
Intraoperative Considerations	Suction out stomach with orogastric tube in supine and lateral positions prior to induction. Preoxygenate with 100% FiO_2. Controlled rapid sequence induction with succinylcholine or rocuronium recommended. Avoidance of perioperative opioid is highly recommended.
Postoperative Considerations	Patients should be monitored for apnea for at least 12 hours following surgery due to risk of apnea from altered CO_2 response from metabolic alkalosis.

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85 Congenital Heart Disease

Sheila Rajashekara • Calvin Kuan

Pediatric congenital heart diseases (CHD) encompass a wide spectrum of anatomic lesions. Some are relatively benign and are definitively repaired with minimal sequelae, while others can only be palliated and result in a lifetime of medical care.

1) **Introduction**
   a) To safely manage patients with congenital heart lesions, the anesthesiologist must:
      i) Thoroughly understand each individual patient's anatomy (visualizing the path of blood flow throughout the body including any abnormal pathways and shunts).
      ii) Understand how the lesion affects the patient's physiology.
      iii) Be able to predict the interactions of surgery and anesthesia—including pain, blood loss, fluid management, positive pressure ventilation, as well as the effects of oxygen and anesthetic agents.
   b) It is strongly recommended that children with CHD be treated at institutions with experienced multidisciplinary teams and that there is adequate communication between all the services involved—primary care physician, pediatric cardiologist, neonatologist, pediatric/cardiac intensivist, surgeon, anesthesiologist, and any relevant subspecialist.



Patients with CHD are at significantly increased risk of morbidity and mortality for all types of anesthetics.

2) **Risks of anesthesia**
   a) Children, particularly neonates and infants, with major cardiac anomalies have significantly increased risk of both cardiac arrest and mortality for noncardiac surgeries.[1–4]
      i) In one study, 54% of cardiac arrests in children with CHD occurred in the general operating rooms for noncardiac surgeries, as well as in the interventional catheterization lab[1–4].
      ii) Outcomes appear significantly better for the patient with CHD when cared for by teams with experience managing such patients[1–4].
   b) With rare exceptions, patients with the following lesions are at low risk and do not require special care.
      i) Repaired patent ductus arteriosus (PDA)
      ii) Repaired or unrepaired secundum atrial septal defect (ASD)
      iii) Repaired or unrepaired asymptomatic, small ventricular septal defects (VSD)
      iv) Asymptomatic pulmonary stenosis (PS)
   c) Patients with the following lesions are at highest risk and should be treated at a comprehensive pediatric cardiac center:
      i) Any patient with single ventricle physiology
         (1) Hypoplastic left heart syndrome (HLHS)
         (2) Tricuspid atresia
         (3) Double-outlet right ventricle (DORV)
         (4) Unbalanced atrioventricular canal (AVC)
         (5) Double-inlet left ventricle (DILV)
         (6) Severe Ebstein anomaly
      ii) Pulmonary hypertension (PH)
      iii) Ventricular dysfunction or heart failure, particularly cardiomyopathies



Knowledge of the specific lesion and history and communication with the patient's cardiologist are essential to implementing a safe anesthetic plan.

iv) Moderate to severe valvular obstruction or regurgitation (particularly aortic stenosis)
v) Systemic to pulmonary artery surgically placed shunt (BT shunt, central shunt, aortopulmonary window)
vi) Status post recent heart transplantation

d) Patients with other lesions/conditions fall in between the two categories and should be assessed individually as there is a wide range of symptoms with varying pathophysiology.

3) **Preoperative evaluation**

a) Know the diagnosis
   i) It is absolutely essential to know the correct cardiac anatomy as each lesion has different implications for anesthetic care.

b) Get accurate information
   i) It is not uncommon for patients and parents to have an incomplete understanding of a patient's physiology and complex medical history.
   ii) Accurate information should be obtained directly from the patient's cardiologist and from a thorough review of medical records and studies.

c) Past medical/surgical history
   i) Anatomy/physiology: Understanding the natural history of the individual lesions is essential. For example:
      (1) A child with a lesion that depends on a PDA to deliver blood may deteriorate when the duct closes a few days after birth.
      (2) A child with a large VSD may be minimally symptomatic at 1-year-old but develops Eisenmenger physiology at 20 years of age.
   ii) Surgical history: The anatomy and physiology may change significantly with each surgical procedure. For example:
      (1) A child with a history of a Blalock-Taussig shunt may have inaccurate blood pressure readings on the ipsilateral arm.
      (2) A child who has had a heart transplant may not respond to atropine given for bradycardia and may require epinephrine instead.
   iii) Past medical history/review of systems: Review the patient's past medical history and how illnesses of other organ systems may affect the anesthetic management. For example:
      (1) A child with severe aortic stenosis may not tolerate the tachycardia caused by albuterol given to treat bronchospasm.
      (2) A child with heterotaxy/asplenia should be treated as being relatively immunocompromised.

d) Physical examination
   i) Vital signs: Know the normal values at rest for the individual patient with the particular lesion. For example:
      (1) A patient on β-adrenergic blockade should be expected to have a slower-than-normal heart rate.
      (2) A teenager with an unrepaired coarctation of aorta may be expected to have baseline hypertension.
   ii) Oxygen saturation: It is essential to know what the oxygen saturation measurement should be for each patient whether cyanotic or acyanotic and to know whether the patient has an O_2 requirement at rest to maintain the saturation. For example:
      (1) A neonate with an unrepaired hypoplastic left heart syndrome (HLHS) may have baseline saturations of 75%-85%, which is optimal. Saturations of 95% may actually be too high and indicate pathologic pulmonary overcirculation.

(2) A child with pulmonary hypertension who requires an FiO_2 of 40% at baseline may require induction with an FiO_2 of at least 40% to avoid the risk of an increase in pulmonary vascular resistance due to hypoxia.

iii) Cardiovascular/fluid/volume status: It is important to evaluate the fluid status of children with heart disease as they may not tolerate relative hypovolemia especially under anesthesia or after fasting. For example:
(1) The patient's blood pressure may be in the normal range when awake, but administration of anesthetic agents may lower systemic vascular resistance (eg, inhaled agents, propofol) and result in hypotension.
(2) Children with systemic to pulmonary artery shunts are at an increased risk of clotting the shunt off when hypovolemic.

iv) Exercise tolerance: When one does not have documented recent echocardiography examinations or cardiac catheterizations, the subjective reporting of exercise ability is a useful assessment tool of cardiac function. Recent changes should raise the concern for deterioration in cardiac function. Examples of questions for parents or patients are:
(1) "Does the child do physical exercise at school? If so, what sort of activities or sports can she do?"
(2) "Can the child run, jump, and play? And can he keep up with other children his age?"
(3) "How long can she play before she gets tired, short of breath, or turn blue?"
(4) "Has the child's ability to do any of the above changed noticeably recently?"

v) Recent changes should raise the concern for deterioration in cardiac function and should prompt additional investigation prior to anesthesia and surgery.

e) Studies
i) Electrocardiogram
(1) All children with a history of cardiac disease should have a baseline EKG as close to anesthetic date as possible.
(2) Having a baseline to compare with intraoperative changes may avoid unnecessary interventions.

ii) Echocardiography examination
(1) Except for children who have had a complete repair of their lesions, are asymptomatic, and have regular follow-up with their cardiologist, a recent echocardiogram (within 3-6 months) by the child's pediatric cardiologist is recommended.
(2) Decision whether to proceed with anesthesia without a recent examination should be made on an individual basis depending on the changes in cardiac physiology expected since the last examination and the information that would be gained.



A recent echocardiogram (within 3-6 months) by the child's pediatric cardiologist is recommended.

iii) Cardiac catheterization findings
(1) Cardiac catheterization is not required for all patients.
(2) Severity of pulmonary hypertension (PH) affects perioperative outcomes, and a more recent cardiac catheterization may be required if the patient is known to have significant PH.
(3) If the patient has had a catheterization, useful information may be available:
(a) Measuring gradients across valves or shunts
(b) Measuring systolic and diastolic pressures that may indicate degree of ventricular function or failure
(c) Identifying stenoses not seen on echocardiogram
(d) Identifying occluded vessels that might be chosen for venous or arterial access

(e) Evaluating anatomy and patency of coronary arteries
(f) Measuring the Qp:Qs (ratio of pulmonary to systemic blood flow), which may give information on the hemodynamic effects of O_2
(i) Qp/Qs < 1: indicates right-to-left shunt and cyanosis.
(ii) Qp/Qs = 1: indicates no shunt, or balanced shunt. Patients are usually asymptomatic.
(iii) Qp/Qs > 1: indicates left-to-right shunt, with pulmonary overcirculation and potential congestive heart failure (CHF).

f) Labs
i) CBC (complete blood count)
(1) Know the goal hemoglobin/hematocrit (Hb/Hct) for the patient.
(a) Some children may require much higher than normal baseline Hct. For example, cyanotic children in general have a high Hct to increase O_2 content of blood.
(b) Our institutional practice is to keep the Hb ≥15 g/dL (Hct > 45-50%) for cyanotic children in the perioperative period to optimize O_2-carrying capacity.
(2) Baseline platelet count
(a) Patients on long-term aspirin therapy may have decreased platelet function despite normal platelet counts.
(b) Patients on left ventricular assist devices are often placed on antiplatelet medications, so thrombocytopenia and abnormal platelet function should be anticipated.
ii) Type and cross
(1) Patients who have had multiple surgeries and transfusions may develop antibodies to blood products. Additional time may be required for the blood bank to crossmatch blood.
iii) Chemistries
(1) Potassium
(a) Hyperkalemia is a much greater concern and risk of morbidity than hypokalemia in the pediatric population.
(b) An exception to the above recommendation is that patients on digoxin should have K^+ ≥3.5. Digoxin competes with K^+ in the myocardium; hypokalemia increases risk of digoxin toxicity.
(c) Consider using washed RBC or freshest RBC if large-volume transfusion is anticipated.
(2) Calcium
(a) Children, particularly neonates, are dependent on calcium for myocardial function and do not tolerate hypocalcemia.
(b) Patients with DiGeorge syndrome may have relative hypocalcemia and require additional supplementation.
(c) Administration of blood products or albumin may cause hypocalcemia.
iv) Coagulation studies
(1) Patients may be anticoagulated for prosthetic valves or a history of thromboses. Negotiation may be necessary between a surgeon who wants to do a craniotomy and a cardiologist who wants to prevent clots on the aortic or the mitral valve.
v) Medications
(1) Angiotensin-converting enzyme inhibitors (ACEI) and angiotensin receptor blockers (ARB) should be held for at least 12 hours or more prior to administration of anesthesia due to risk of significant hypotension after induction of anesthesia[5].

(2) Plan to hold diuretics on the morning of surgery to avoid further dehydration as patients will be NPO.
(3) Recommend continuing antiarrhythmics unless presenting for EP study; then, discuss with EP physician.
(4) Recommend continuing aspirin in shunt-dependent patients.

vi) NPO guidelines
(1) Patients should be scheduled as the first case of the day to minimize fasting time. Clear fluids should be encouraged until 2-3 hours before surgery. Consider admitting single ventricle patients, and patients with Williams syndrome or Alagille syndrome the night before for pre-op IV hydration.
(2) Patients receiving continuous nutrition via feeding tube or TPN may develop hypoglycemia, and symptoms may be masked under general anesthesia. Consider checking serum glucose level and providing a dextrose infusion.

4) **Special anesthetic considerations**
a) "A bubble is a bullet to the brain/heart": It is absolutely critical to ensure that all fluids entering the patient are cleared of air bubbles that may embolize to the brain or the coronary arteries.
b) "Oxygen is a drug": O_2 causes pulmonary vascular vasodilation.
i) For a patient with pulmonary hypertension, it may be beneficial in lowering pulmonary vascular resistance.
ii) For a child with single ventricle physiology whose cardiac output depends on a balance between systemic vascular resistance (SVR) and pulmonary vascular resistance (PVR), even a slight increase in FiO_2 may cause a decrease in PVR and result in systemic hypoperfusion and myocardial ischemia.



A bubble in the IV tubing is a bullet to the brain or the heart.

c) Postoperative care/disposition
i) Patients should be closely monitored postoperatively by PACU personnel experienced with taking care of patients with CHD.
ii) Patients who are young, unrepaired, and cyanotic or have single ventricle physiology might require monitoring overnight depending on the surgical procedure.
iii) Special care should be taken when administering narcotics due to effects on respiratory drive, oxygenation, and ventilation and their effects on cardiac physiology.



Oxygen is a drug. Know how it may affect your patient's physiology.

d) Vascular access
i) Vascular access (venous and arterial) may be extremely difficult in patients who have undergone several major surgeries or have been hospitalized for a long time.
ii) It is not uncommon for vessels to be stenotic or occluded and therefore unusable.
iii) Some patients may require very specific placement of lines. For example, the patient with a coarctation will require an arterial line in the right arm.



Consultation with a pediatric cardiologist or pediatric cardiac anesthesiologist is strongly recommended before initiating anesthesia.

5) **Selected lesions and anesthetic concerns:** The purpose of this section is to provide some basic understanding of select congenital cardiac lesions. Please refer to recommended references for additional information.

ANESTHETIC CONSIDERATIONS FOR SELECTED CONGENITAL LESIONS

Lesion: describes the anatomy of the defect(s).

Clinical findings: describes the signs, symptoms, and pathophysiology.

Repair: describes the usual surgical repair.

Prerepair concerns: describes some of the major anesthetic concerns for patients with an unrepaired lesion:
- What is the expected oxygen saturation on room air?
- Can 100% oxygen be used during induction?
- Is there a need for SBE prophylaxis?
- Other special considerations.

Postrepair concerns: describes some of the major anesthetic concerns for patients outside of the immediate period of surgical repair.
- Does the patient have normal anatomy and circulation?
- What is the expected oxygen saturation on room air?
- Can 100% oxygen be used during induction?
- Is there a need for subacute bacterial endocarditis (SBE) prophylaxis?
- Other special considerations.

ATRIAL SEPTAL DEFECT (FIG. 85-1)

Lesion: Defect in the atrial septum. There are four types that may occur individually or in combination:
- **Primum:** (5%-7%) associated with AV canal (AVC) defect.
- **Secundum:** most common (85%). Asymptomatic.
- **Sinus venosus:** (2%-5%) associated with partial anomalous pulmonary venous return (PAPVR).
- **Coronary sinus:** associated with persistent left SVC.

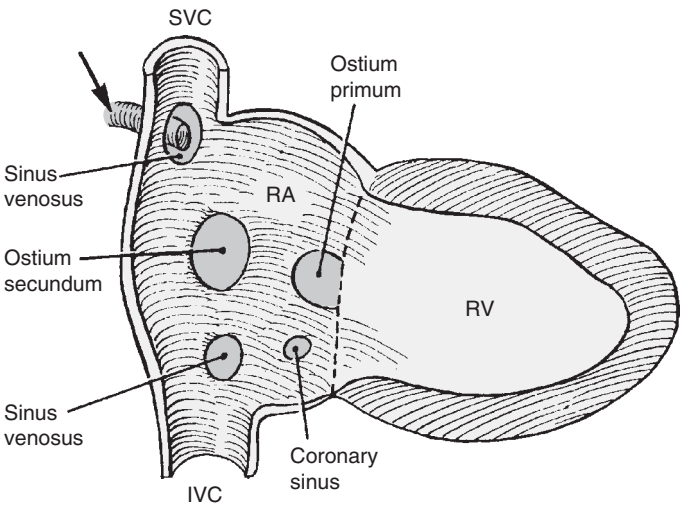
Clinical findings
- Usually asymptomatic in children.
- Usually left-to-right shunt; may have bidirectional shunt but does not result in cyanosis.
- Over time (ie, decades), right heart volume overload may lead to CHF and pulmonary hypertension (PH).

Repair
- Device closure in cath lab.
- Primary closure via sternotomy with cardiopulmonary bypass.

Prerepair concerns
- SpO_2 should be normal.
- No restrictions on O_2 use.
- SBE prophylaxis not required[6].
- Special anesthetic considerations
  - Care must be taken to avoid air bubbles in all lines and injections.
  - Otherwise, there are no significant hemodynamic concerns.

Postrepair concerns
- Anatomy is considered normal after repair.
- Expect normal SpO_2 on room air.
- No restrictions on O_2 use.
- SBE prophylaxis required only for the first 6 mo after repair[6].
- No other special anesthetic considerations.

FIGURE 85-1 Location of Atrial Septal Defects



View of the atrial septum showing locations of the different types of atrial septal defects. SVC, superior vena cava; IVC, inferior vena cava; anomalous pulmonary venous return associated with sinus venosus location, *arrow*; RA, right atrium; RV, right ventricle. Reused from Rouine-Rapp K, Miller-Hance WC. Transesophageal echocardiography for congenital heart disease in the adult. In: Perrino AC Jr, Reeves ST, eds. *A Practical Approach to Transesophageal Echocardiography*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008:372, with permission.

VENTRICULAR SEPTAL DEFECT (FIG. 85-2)

Lesion: Defect in the ventricular septum. There are multiple types that may occur individually or in combination:
- Perimembranous, or membranous, is the most common (70%)[7].
- Muscular (5%-20%) may be single or multiple (eg, "Swiss cheese" type).
- Inlet (5%-8%) is also known as AV canal type and is associated with primum ASDs.
- Supracristal is also known as infundibular, conoventricular, conal, subpulmonary, subarterial, or doubly committed VSD. These account for 5%-7% of all VSDs in the Western world and up to 30% of VSDs in the East.

Clinical findings
- Depends on the location of defect, size, and age of patient.
- Primary problem is pulmonary overcirculation due to a left-to-right shunt.
  - If left untreated, this may eventually lead to pulmonary vascular changes and Eisenmenger physiology, which is pulmonary hypertension causing a reversal of shunt and severe cyanosis.
  - A large, unrestrictive VSD may result in congestive heart failure earlier in life due to volume overload of the left ventricle.

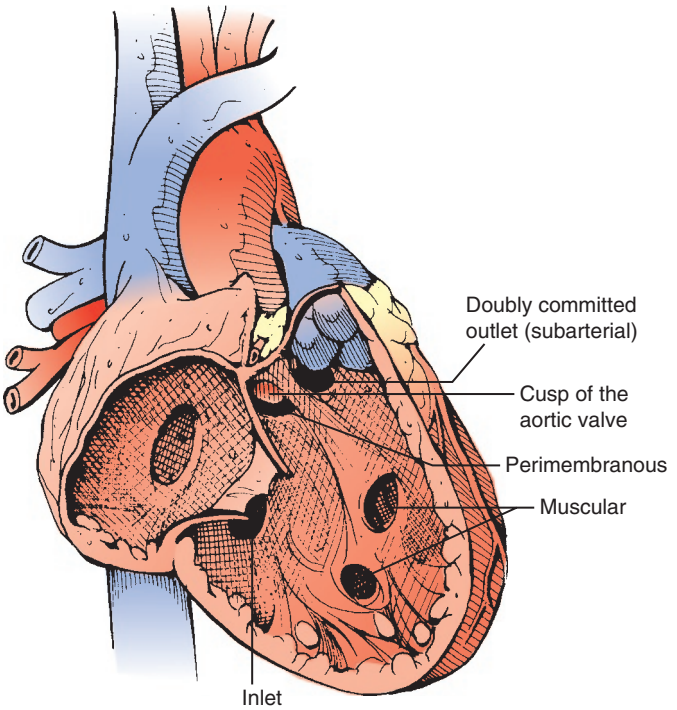
Repair
- Device closure in cath lab.
- Primary closure via sternotomy with cardiopulmonary bypass.

Prerepair concerns
- Expected SpO_2 depends on amount and direction of shunt.
  - SpO_2 should be normal with left-to-right shunts.
  - SpO_2 may be as low as 75%-80% with right-to-left shunts.

O_2 may be used for induction with the caveat that it may decrease pulmonary vascular resistance, increasing the left-to-right shunt and exacerbating pulmonary overcirculation.
SBE prophylaxis is not required unless patient is cyanotic.
Special anesthetic considerations
- Care must be taken to avoid air bubbles in all lines and injections.
- Anesthetic plan should take into consideration the degree of cyanosis and heart failure. If the patient is a teenager or an adult, there should be suspicion for pulmonary hypertensive changes.

Postrepair concerns
- Anatomy is considered normal after repair.
- Barring pulmonary hypertension, SpO_2 should be normal.
- No restrictions on O_2 use.
- SBE prophylaxis required only for the first 6 mo after repair[6].
- There are no other special anesthetic considerations.

FIGURE 85-2 Location of Ventricular Septal Defects



View of the interventricular septum from the right ventricle showing common locations of the different types of VSDs. Reused with permission from Rouine-Rapp K, Miller-Hance WC. Transesophageal echocardiography for congenital heart disease in the adult. In: Perrino AC Jr, Reeves ST, eds. *A Practical Approach to Transesophageal Echocardiography*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008:377.

COARCTATION OF AORTA (FIG. 85-3)

Lesion: Narrowing of the aorta usually at the descending aorta distal to left subclavian artery and at insertion site of ductus arteriosus.

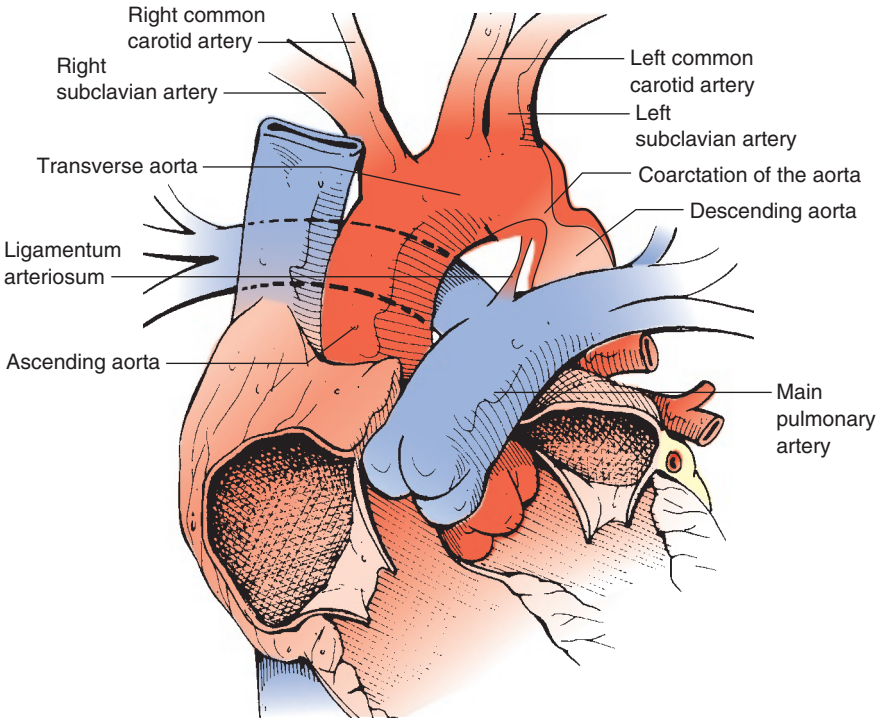
Clinical findings
- Associated with bicuspid aortic valve.
- Critical coarctation usually presents in the newborn period with poor cardiac function and shock.
- Outside of neonatal period, children present with upper extremity hypertension and decreased pulses distal to narrowing (eg, lower body).
- In long-standing coarctation, the patient may present with hypertension and left ventricular hypertrophy.

Repair
Primary closure via thoracotomy usually without cardiopulmonary bypass.
Balloon dilation in cath lab for patients with prior repair.

Prerepair concerns
- SpO_2 should be normal.
- No restrictions on O_2 use.
- SBE prophylaxis not required.
- Special anesthetic considerations:
  - For critical coarctation, alprostadil (PGE_1) may be needed to maintain patency of the ductus arteriosus to perfuse the body distal to the coarctation.
  - BP measured in the right arm more accurately reflects flow to the brain and coronary arteries.
  - Measuring BP both in the right arm and legs allows measurement of gradient across coarctation.

Postrepair concerns
- There may be residual coarctation. Hypertension may take days to weeks to resolve.
- SpO_2 should be normal.
- No restrictions on O_2 use.
- SBE prophylaxis is not required[6].

FIGURE 85-3 Coarctation of the Aorta



Most common location of aortic coarctation—juxtaductal distal to the left subclavian artery. Reused with permission from Rouine-Rapp K, Miller-Hance WC. Transesophageal echocardiography for congenital heart disease in the adult. In: Perrino AC Jr, Reeves ST, eds. *A Practical Approach to Transesophageal Echocardiography*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008:382.

TETRALOGY OF FALLOT (FIG. 85-4)

Lesions
- Anterior malalignment VSD.
- Right ventricular outflow tract obstruction (RVOTO).
- Overriding aorta.
- Right ventricular hypertrophy.

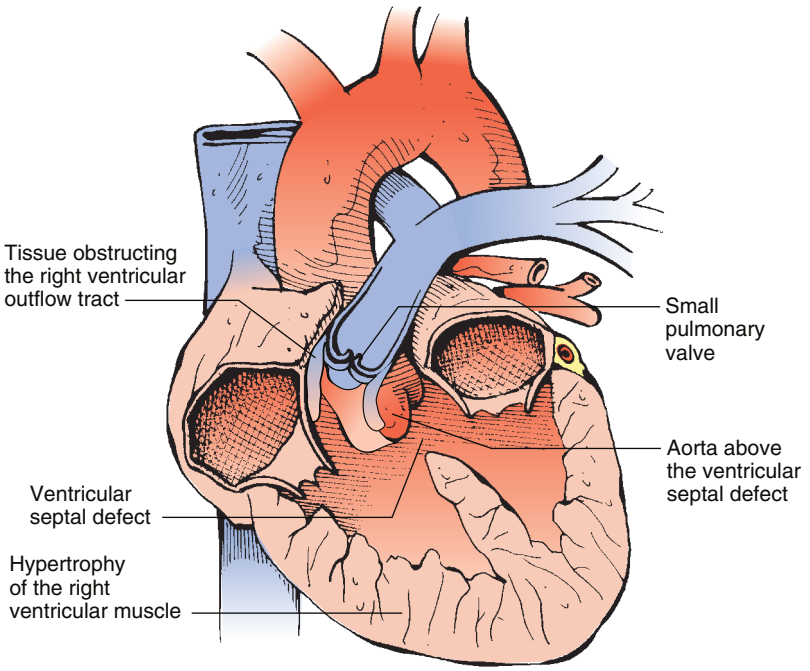
Clinical findings
- "Tet spells" aka hypercyanotic spells occur with RVOT infundibular spasm causing increased right-to-left shunt and cyanosis.
  - "pink Tet": mild RVOTO. Most of blood from the right ventricle goes into the pulmonary artery.
  - "blue Tet": more severe RVOTO. Blood flow to the lungs is obstructed and gets shunted through VSD out to the body without getting oxygenated.
- Medical treatment includes maneuvers to relax infundibular spasm and/or increase systemic vascular resistance.
  - 100% O_2.
  - Morphine or other opiates.
  - Phenylephrine.

Repair: Primary repair usually as neonate includes closure of VSD and enlargement of RVOT under cardiopulmonary bypass.

Prerepair concerns
- SpO_2 should be normal except during a hypercyanotic spell.
- No restrictions on O_2 use. O_2 is beneficial and patients should be preoxygenated with 100% O_2.
- SBE prophylaxis is recommended[6].
- Special anesthetic considerations:
  - Avoid catecholamine surges that might instigate hypercyanotic spell.
  - Premedication is recommended even in young infants.
  - Consider deep induction prior to intubation and deep extubation.
  - Have alpha-adrenergic agonist (eg, phenylephrine) available to treat hypercyanotic spell.

Postrepair concerns
- Anatomy and physiology:
  - Surgeon may leave a patent foramen ovale (PFO) as a "pop-off valve" if there is RV dysfunction.
  - There may be residual pulmonary insufficiency that may lead to RV volume overload, especially if the repair was with a transannular patch.
  - Pulmonary stenosis may develop over time.
- SpO_2 should be normal unless there is shunting through a PFO.
- No restrictions on O_2 use.
- SBE prophylaxis recommended for 6 months after repair and may be required afterward if prosthetic material was used.

FIGURE 85-4 Tetralogy of Fallot



Components of tetralogy of Fallot: right ventricular outflow tract obstruction, ventricular septal defect, right ventricular hypertrophy, and overriding aorta. Reused with permission from Rouine-Rapp K, Miller-Hance WC. Transesophageal echocardiography for congenital heart disease in the adult. In: Perrino AC Jr, Reeves ST, eds. *A Practical Approach to Transesophageal Echocardiography*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008:387.

Glossary of Congenital Heart Disease Terms

Aortopulmonary window (aka APW or aortopulmonary septal defect) May be a pathological congenital lesion or a surgical procedure performed to increase pulmonary blood flow resulting in side-to-side connection of the ascending aorta to the main pulmonary artery. Physiologically similar to central shunt.

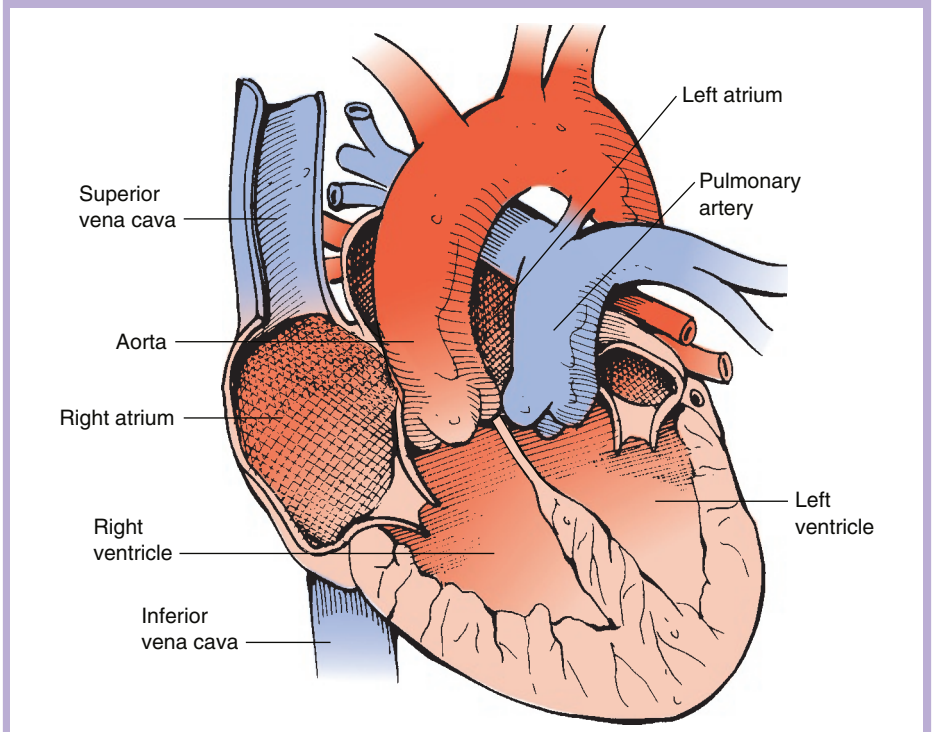
Arterial switch procedure (aka Jatene procedure) Surgical procedure to repair D-TGA (see entry below and **Fig. 85-5**) by switching malpositioned aorta and pulmonary artery. Requires moving the coronary arteries to a new location (**Fig. 85-6**).

Blalock-Taussig shunt (BTS) Surgical procedure that creates a shunt between the subclavian artery and the pulmonary artery to supply additional pulmonary blood flow in some congenital lesions.

- **Classic BTS** End-to-side anastomosis of native subclavian artery to ipsilateral pulmonary artery.
- **Modified BTS** Artificial shunt placed between the subclavian artery and the ipsilateral pulmonary artery.

Central shunt Artificial shunt placed side to side between the ascending aorta and the main pulmonary artery to increase pulmonary blood flow. Physiologically similar to aortopulmonary window.

FIGURE 85-5 D-Transposition of the Great Arteries



D-TGA: the aorta arises from the right ventricle carrying deoxygenated blood. Pulmonary artery arises from the left ventricle carrying oxygenated blood. Parallel circulation requires mixing of blood for patient to survive. Reused with permission from Rouine-Rapp K, Miller-Hance WC. Transesophageal echocardiography for congenital heart disease in the adult. In: Perrino AC Jr, Reeves ST, eds. *A Practical Approach to Transesophageal Echocardiography*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008:390.

Complete transposition of the great arteries (aka D-TGA aka dextro-TGA) Congenital lesion where the aorta arises from the right ventricle and pulmonary artery arises from the left ventricle resulting in two parallel circulations. Survival depends on adequate mixing of oxygenated and deoxygenated blood (**Fig. 85-5**).

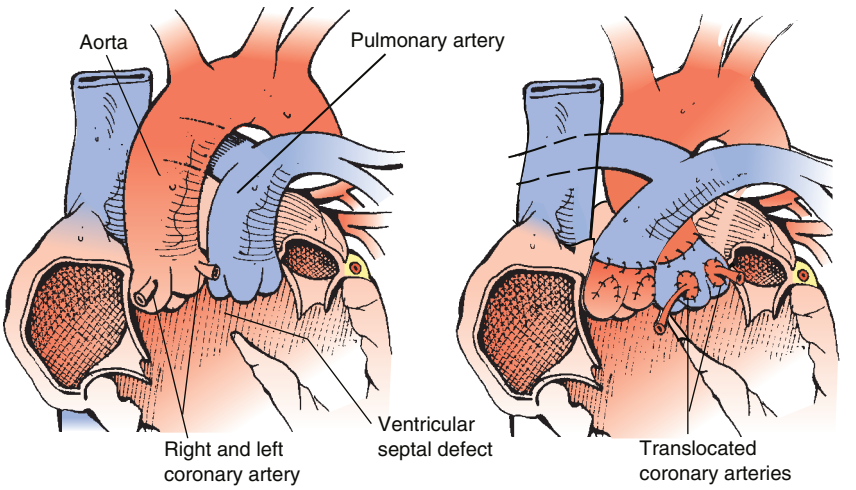
Damus-Kaye-Stansel (DKS) Surgical procedure used for various congenital lesions with systemic outflow tract hypoplasia or obstruction. Main pulmonary artery is transected and connected end to side to the ascending aorta to allow blood to go systemically. An alternative source of pulmonary blood flow is created (see entries for BT shunt, Sano, or RV to PA conduit).

Double-outlet right ventricle (DORV) Congenital lesion where both the aorta and the pulmonary artery arise from the right ventricle. A VSD is present for exit of blood from the left ventricle. Pathophysiology depends on position of VSD and other factors. May result in single or dual ventricle physiology.

Double switch procedure Surgical procedure to repair L-TGA (see entry below, and **Fig. 85-7**) with an atrial-level switch (see entries for Mustard or Senning) and arterial switch procedure. Usually, it requires preconditioning with pulmonary artery banding (PAB).

Ebstein anomaly "Atrialization" of the right ventricle. Abnormal and downward displacement of tricuspid valve with a spectrum of clinical manifestations from asymptomatic to single ventricle pathway. Often associated with dysrhythmias.

FIGURE 85-6 Arterial Switch Procedure



Arterial switch procedure: the aorta and pulmonary arteries are transected and switched to normal positions. Coronary artery buttons must be moved to new aortic position. ASD/VSD must be closed. Reused with permission from Rouine-Rapp K, Miller-Hance WC. Transesophageal echocardiography for congenital heart disease in the adult. In: Perrino AC Jr, Reeves ST, eds. *A Practical Approach to Transesophageal Echocardiography*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008:391.

Endocardial cushion defect (ECD aka AV canal, aka atrioventricular septal defect [AVSD]) Congenital lesion with a spectrum of manifestations including primum ASD, inlet VSD, and mitral and tricuspid valve abnormalities. Often associated with trisomy 21.

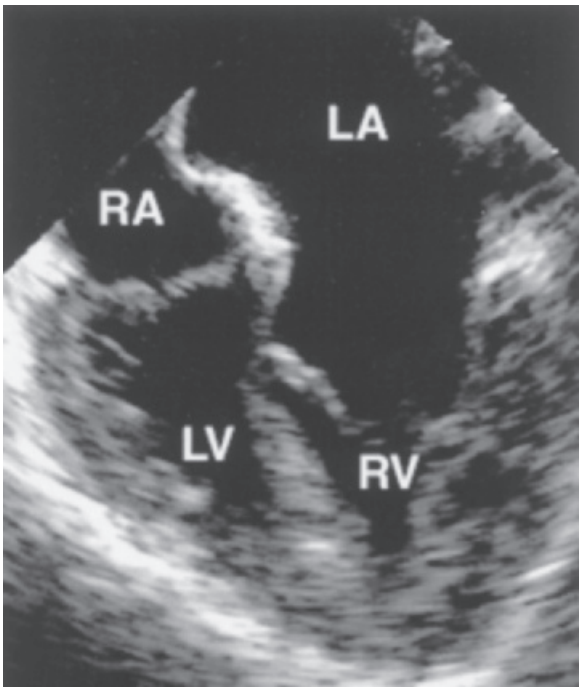
Fontan procedure Surgical procedure that is the final stage of palliation for single ventricle lesions usually done at 12-24 months of age. The goal of this procedure is to direct all venous return (now the IVC, in addition to the SVC return that was already redirected during the Glenn procedure) to the pulmonary circulation, bypassing the heart completely, depending on passive drainage into pulmonary arteries. After this surgery, the patient will ideally have 100% SpO_2.

- **Original Fontan** End-to-end connection of SVC to right pulmonary artery, separation of right and left pulmonary arteries at confluence, anastomosis of right atrial appendage to the proximal separated end of right pulmonary artery (RPA), closure of ASD, and ligation of the main pulmonary artery. It is not performed any longer.
- **Extracardiac Fontan** Use of Dacron tube conduit to connect the IVC to the RPA. It has fewer complications from dysrhythmias.
- **Lateral tunnel Fontan (aka intracardiac)** Use of the patient's native right atrial tissue to create a tunnel connecting the IVC to the PA, without any blood flow to the right ventricle. Associated with higher risk of dysrhythmias.

Glenn procedure (aka cavopulmonary shunt) In patients with single ventricle physiology, a surgical procedure that connects the SVC to the pulmonary artery allowing venous drainage from the upper body to bypass the heart. Usually done to volume unload the heart and to increase pulmonary blood flow.

- **Classic Glenn** In the original surgery, the right pulmonary artery was transected and anastomosed end to end to the SVC. The left pulmonary artery (now discontinuous from the right) continued to get flow from the heart via the main pulmonary artery.

FIGURE 85-7 L-TGA or Congenitally Correct Transposition (CC-TGA)



Transesophageal echo showing the heart with L-TGA. Note ventricular inversion with left ventricle on right and right ventricle on left. Reused with permission from Rouine-Rapp K, Miller-Hance WC. Transesophageal echocardiography for congenital heart disease in the adult. In: Perrino AC Jr, Reeves ST, eds. *A Practical Approach to Transesophageal Echocardiography*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008:392.

Bidirectional Glenn The current modification connects the SVC to the right PA, but allows venous blood to drain both to right and left pulmonary arteries, hence "bidirectional."

Bilateral bidirectional Glenn For patients with right and left SVCs, both SVCs must be connected to ipsilateral pulmonary arteries, hence "bilateral" shunts.

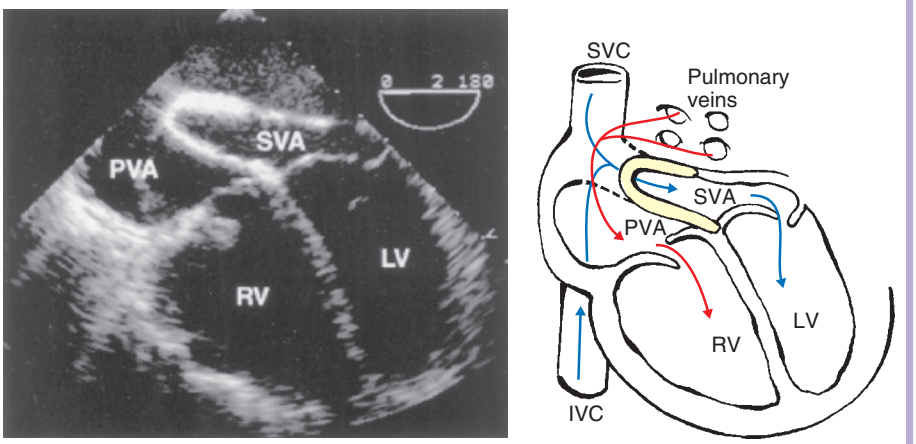
Hybrid procedure Most commonly done with HLHS patients as it avoids a cardiopulmonary bypass run in the neonatal period. Current approach involves stenting of the PDA to provide pulmonary blood flow and bilateral pulmonary artery banding to avoid pulmonary overcirculation.

Jatene procedure see arterial switch procedure.

Levo-Transposition of the great arteries (aka L-TGA or congenitally corrected TGA [CC-TGA]) Congenital lesion of ventricular inversion where the right-sided ventricle (that pumps to the pulmonary artery) is morphologically a left ventricle and the left-sided ventricle (that pumps to the aorta) is morphologically a right ventricle. Functionally normal anatomy, but often associated with dysrhythmias, other cardiac lesions, and potential failure of the systemic ventricle (**Fig. 85-7**).

Mustard procedure Atrial redirection procedure using pericardial or artificial material to create a baffle (ie, rerouting of blood flow) in the atria to palliate D-TGA. It is similar to the Senning procedure (**Fig. 85-8**).

FIGURE 85-8 Dextro-Transposition of the Great Arteries Following an Atrial Redirection Procedure



A: Two-dimensional transesophageal echocardiographic midesophageal four-chamber view that demonstrates the features of an atrial redirection procedure. In this procedure, venous return from the systemic and pulmonary circulations is rerouted through a baffle. Following placement of a baffle, desaturated blood from the superior and inferior venae cavae drains into the systemic atrium (SVA) and then through the mitral valve into the left ventricle (LV) and then into the pulmonary artery. Blood from the pulmonary veins drains into the pulmonary atrium (PVA) and then through the tricuspid valve into the right ventricle (RV) and then into the aorta. The RV remains the systemic ventricle. **B:** Illustration is included to clarify the route of blood flow following an atrial redirection procedure. SVC, superior vena cava; IVC, inferior vena cava. Reproduced with permission from Rouine-Rapp K, Miller-Hance WC. Transesophageal echocardiography for congenital heart disease in the adult. In: Perrino AC Jr, Reeves ST, eds. *A Practical Approach to Transesophageal Echocardiography*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008:391.

Norwood procedure First-stage surgical procedure in the pathway to palliate hypoplastic left heart syndrome. Repair includes creation of neo-aorta using native main pulmonary artery that is transected. Alternative source of pulmonary blood flow is created—for example, Sano RV to PA shunt. Subsequent stages include Glenn and Fontan procedure (**Fig. 85-9**).

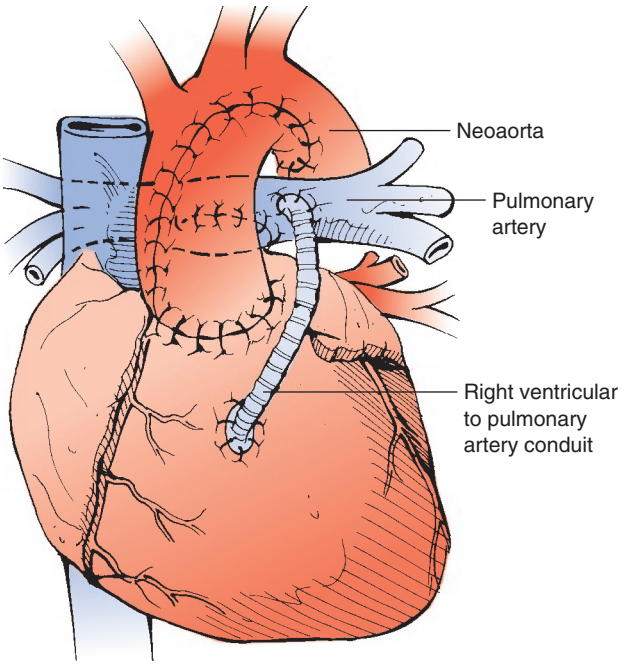
Partial anomalous pulmonary venous return (PAPVR) Congenital lesion where some of the pulmonary veins do not drain into the left atrium, but drain into the right atrium or some other part of the venous system. When none of the pulmonary veins drain normally into the left atrium, the lesion is termed total anomalous pulmonary venous return (TAPVR).

Pentalogy of Cantrell A congenital lesion with five distinct traits—anterior diaphragmatic defect, defects in the pericardium, abdominal wall defect (eg, omphalocele), lower sternum defect, and heart defects (including VSDs, ASD, and tetralogy of Fallot and rarely ectopia cordis where the heart is outside the thoracic cavity). This syndrome may be associated with other congenital issues including cleft lip/palate, limb defects, and neural tube defects.

Potts shunt Surgical procedure creating a shunt between the descending aorta and the left pulmonary artery.

Pulmonary atresia with intact ventricular septum (PA/IVS) Congenital lesion with an atretic pulmonary valve and no VSD for exit of blood from the right ventricle. An atrial shunt (PFO or ASD) is necessary for survival. Coronary sinusoids may develop from the elevated RV pressures, resulting in increased risk of right ventricular ischemia.

FIGURE 85-9 Norwood Procedure With Sano Modification



Norwood procedure consists of using native pulmonary artery to create neo-aorta. Sano modification is valved conduit from RV to PA to provide pulmonary blood flow. Reused with permission from Rouine-Rapp K, Miller-Hance WC. Transesophageal echocardiography for congenital heart disease in the adult. In: Perrino AC Jr, Reeves ST, eds. *A Practical Approach to Transesophageal Echocardiography*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008:395.

Pulmonary artery banding (PAB) Surgical procedure to tighten the pulmonary artery to decrease the amount of pulmonary blood flow and/or to strengthen the ventricle.

Rashkind procedure Procedure performed at bedside or in the cardiac cath lab to enlarge a patent foramen ovale or ASD in lesions that require increased mixing of systemic and pulmonary circulation, for example, D-TGA with an intact atrial septum.

Rastelli procedure Surgical procedure used to correct certain lesions with pulmonary artery abnormalities. The pulmonary artery is transected. The VSD is closed with a patch that simultaneously directs blood from the LV to the aorta. Finally, an RV to PA conduit is created to supply pulmonary blood flow.

Sano modification (**Fig. 85-9**): Surgical procedure creating a valved RV to PA conduit as a modification of the Norwood procedure.

Senning procedure Atrial redirection procedure using native atrial tissue to create a baffle in the atria to palliate D-TGA. Similar to Mustard procedure.

Tetralogy of Fallot See TOF entry in chapter above.

Tetralogy of Fallot with absent pulmonary valve (TOF/APV) Congenital lesion that is a small subset of TOF where the pulmonary valve is completely absent resulting in severe pulmonary regurgitation. A pulmonary artery aneurysm may develop that may be so large as to compress the trachea and bronchi, causing severe airway problems.

Tetralogy of Fallot with pulmonary atresia and MAPCAs (major aortopulmonary collateral arteries) (TOF/PA/MAPCAs) Congenital lesion previously described as truncus arteriosus type IV. An extreme form of TOF with complete pulmonary atresia. Pulmonary blood flow is from PDA or major aortopulmonary collateral arteries (MAPCAs). Surgical repair with unifocalization procedure.

Total anomalous pulmonary venous return (TAPVR) None of the pulmonary veins drain into the left atrium. The specific anatomy determines the physiology and the urgency of repair. Obstructed TAPVR is a surgical emergency.

Tricuspid atresia Congenital lesion with absent or atretic tricuspid valve and hypoplastic right ventricle. Repair of this single ventricle lesion eventually leads to Fontan procedure.

Truncus arteriosus Congenital lesion where only one great vessel leaves the heart and supplies blood flow to pulmonary arteries, aorta, and coronary arteries. A VSD is necessary for survival. Associated with interrupted aortic arch and DiGeorge syndrome.

Unifocalization procedure Complex surgical procedure for treatment of patients with TOF/PA/MAPCAs where aortopulmonary collaterals are separated from the aorta and connected to the pulmonary artery to supply deoxygenated blood to the lungs. May be done as single surgery or staged procedure depending on degree of pulmonary hypoplasia.

Waterston shunt Surgical procedure creating a shunt between the ascending aorta and the right pulmonary artery.

CHAPTER SUMMARY FOR CONGENITAL HEART DISEASE (CHD)

Overview	CHD comprises a wide spectrum of lesions with varying manifestations. Children with CHD are at significantly higher risk of morbidity and mortality with anesthesia. Communication with patient's caregivers, cardiologist, and surgeon is critical.
Preoperative	Understanding the anatomy and physiology is essential to providing safe anesthesia. Know what the baseline oxygen saturation should be at rest. Assess the patient's exercise tolerance as an indication of cardiac function. Evaluate recent studies and consider getting new ones if studies are outdated. Consultation with a pediatric cardiologist or a pediatric cardiac anesthesiologist is strongly recommended before initiating anesthesia.
Intraoperative	A bubble is a bullet to the brain or the heart. Take great care not to inject any air into intravenous lines. Oxygen is a drug. Know whether increased FiO_2 may adversely affect the patient.
Postoperative Issues	Patients should be closely monitored postoperatively by PACU personnel experienced with taking care of patients with CHD. Special care should be taken when administering narcotics due to effects of respiratory drive, oxygenation, and ventilation and their effects on cardiac physiology.

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86 Children With Special Neurodevelopmental Needs

Mary Lyn Stein

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by (1) impairment in social communication and interactions and (2) restricted, repetitive interests and behaviors, which are (3) present from early childhood and (4) cause significant impairment. ASD includes a wide range of clinical symptoms.

Cerebral palsy (CP) is a group of motor disorders resulting from injury to the central nervous system during early development and is a leading cause of disability in childhood. CP is clinically distinct from ASD, but children with CP may have comorbid ASD.

This chapter is meant to provide a brief overview of common issues associated with these conditions and their impact on anesthetic management.

Autism Spectrum Disorder

1) **Epidemiology**
   a) Diagnosed in 1 in 88 children in the United States, four times more prevalent in males than females[1]
   b) Prevalence is 1% in the general population.
   c) Cause is unknown, likely due to genetic and neurodevelopmental factors with multiple subtypes.



When caring for a person with autism, ***Plan ahead!***[3]

2) **Clinical features**[2]
   a) **Impairment in social communication:**
      i) Variable severity ranging from difficulty engaging in and maintaining social conversation to complete absence of spoken language
      ii) Problems with nonverbal communications including
         (1) Difficulty interpreting body language
         (2) Anxiety associated with making eye contact
            (a) This is likely due to impaired visual processing, and forcing eye contact can cause great anxiety.
   b) **Stereotyped behavior, including:**
      i) Strict adherence to routine
      ii) Repetitive movements
      iii) Intense, highly restricted interests
         (1) These interests may be engaged to decrease anxiety and elicit patient cooperation.
   c) **Sensory processing difficulties**
      i) Patients may have difficulty managing new sensory inputs particularly in the unfamiliar hospital environment.



People with autism have sensory sensitivities. This may include bright lights, loud noises, smells, textures, and even colors.

3) **Associated concerns**
   a) **Comorbid mental health problems**
      i) A large subset of children with ASD also have comorbid mental health diagnoses including the following:[4,5]
         (1) Anxiety disorders
         (2) ADHD

(3) Conduct disorder
(4) Mood disorders

ii) Medication interactions
(1) Antipsychotics (eg, risperidone, clozapine)[6]
(a) Frequently prescribed to patients with ASD as mood stabilizers
(b) Associated with hypotension and cardiac dysrhythmias under anesthesia
(2) Stimulant medications (eg, methylphenidate)[7]
(a) Frequently prescribed for ADHD
(b) Do not appear to impact the likelihood of hypotension on induction
(c) Do not appear to alter behavior or need for anxiolysis prior to induction

b) **Aggression and self-injury**
i) Prevalence of self-injurious behavior is 25%-50% in individuals with ASD.[4,5,8]

c) **Epilepsy**
i) Prevalence of 10%-39% of individuals with ASD[4,5]

d) **Caregiver concerns**
i) Caregivers may raise concerns based on their own research and understanding of their child's disease.
ii) No particular anesthetic regimen has been shown to be superior for children with ASD.[3–5]

4) **Anesthetic management**

a) **Preoperative considerations**
i) Early planning and conversation with caregivers to facilitate collaborative interactions
ii) Obtain as much information as possible during preadmission evaluation in order to minimize changes to plans on day of surgery[3]
iii) Quiet room, minimal waiting
iv) Technology
(1) Electronic tablets may facilitate communication or serve as distractors.
(2) Children may be very motivated by special interests, which may be used to elicit cooperation with clinical care and activities.
v) Sensory processing differences may lead to patients experiencing significant distress related to standard intake procedures.
(1) Physical sensation of standard monitors, particularly noninvasive blood pressure cuff
(2) Beeping from standard monitors
(3) Sensation of hospital gowns
(4) Bright lights in the operating room
(5) Ask about sensitivites ahead of time and minimize exposure to things about which an individual patient is sensitive
vi) Preparation
(1) Where available, involve child life specialists to prepare child and family and explain care in developmentally appropriate ways.
(2) Encourage families to use calm, factual stories or visual timelines to prepare patient for perioperative experience at home ahead of time.
vii) Day of Procedure – Practical Steps
(1) Use simple clear language and special interest to promote comfort in a new environment
(2) Allow the child to stay in his or her own clothes
(3) Minimize external stimulation and associated anxiety by dimming lights, avoiding noisy waiting areas and long wait times



Use simple clear language. Minimize external stimulation. Limit the number of providers interacting with the patient.[4,5]

(4) Turn down or off audible beeping on monitors
(5) Limit the number of providers interacting with the patient

b) **Premedication**

i) Patients should be continuously monitored once premedication has been administered.
ii) Resuscitation equipment including oxygen, suction, and bag and mask should be immediately available.
iii) Caregivers should be made aware of the potential for extended sedation following premedication.
iv) Agents—there are not sufficient data to recommend a particular premedication regimen.[4,5]
(1) Benzodiazepines
(a) Oral midazolam (0.25-1 mg/kg, usual max dose 20 mg)
(b) Intramuscular and intranasal administration may also be considered.
(c) May be used alone or in combination with other agents, including ketamine
(2) Ketamine (3-6+ mg/kg oral, 3-5 mg/kg IM)
(a) Should be administered with a benzodiazepine
(b) May cause increased salivation
(3) Dexmedetomidine (4 µg/kg oral, 2 µg/kg intramuscular, 1-3 µg/kg intranasal)
(a) Alpha-2 agonist
(b) May cause bradycardia
v) Practical considerations–choose familiar medication or combination of medications so as to:
(1) Prepare the patient and caregivers
(2) Maximize the chance of successful administration
(3) Facilitate rapid identification and treatment of medication side effects



Use of premedication is recommended to ease patient anxiety and facilitate cooperation.

c) **Intraoperative management**

i) Induction
(1) Discuss with caregivers the advantages and disadvantages of parent present induction for each child/situation.
(2) Discuss with all OR staff ahead of time and provide one *single voice* throughout induction.
(3) Maintain a calm, quiet operating room environment.
(4) Avoid use of physical restraints if at all possible.
ii) Maintenance
(1) There is no evidence that ASD impacts minimum alveolar concentration (MAC).[4,5]
(2) Ensure adequate analgesia, including multimodal therapy with acetaminophen, anti-inflammatory medications, and regional anesthesia.
(3) Prophylaxis of postoperative nausea and vomiting
(4) Appropriate hydration
iii) Emergence
(1) Plan the anesthetic to minimize risk of emergence agitation.
a) Alpha-2 agonists and propofol may be useful in this setting.
(2) Ensure all lines and drains are adequately secured prior to emergence and consider camouflaging/decorating if possible.



A calm, quiet environment with a **single**, gentle voice throughout induction may improve patient comfort and cooperation.

d) **Postoperative management/disposition**

i) Pain control
ii) Quiet, private room

iii) Caregivers present as early as possible.
iv) Minimize procedures and invasive lines.
(1) Consider early removal of IV as it can be a trigger for behavioral outbursts.
v) Early discharge where medically appropriate

Cerebral palsy

5) **Major subtypes include spastic diplegia (21%), hemiplegia (21%), and quadriplegia (27%).**[9,10]
a) Spastic quadriplegia is associated with feeding problems, aspiration, epilepsy, and intellectual disability.
b) Patients with spastic diplegia and hemiplegia are most likely to have normal intelligence.
6) **Patients with speech impairment may have normal intelligence. Difficulty in communication may be due to dysarthria, impaired hearing, or lack of cooperation.**
7) **Comorbid conditions may include GERD, epilepsy, chronic pulmonary disease, aspiration, and behavioral problems including ASD.**
a) Severe restrictive lung disease may be present in patients with scoliosis.
8) **Common surgeries include the following:**
a) Tendon transfer and release to improve mobilization
b) Spine surgery to limit progression of scoliosis
c) Neurosurgical procedures
d) Dental rehabilitation
e) Imaging such as MRI
f) GI procedures including feeding gastrostomies and endoscopy
9) **Children with CP require special consideration when undergoing anesthesia for surgery or other procedures.**
a) Latex allergy: The risk may be increased if the patient has had multiple medical and surgical interventions from an early age.
b) Positioning: Special care should be taken when positioning patients with contractures who have an increased risk of nerve damage and pressure sores.
c) Temperature regulation: Patients with CP may have borderline hypothermia and require warming and temperature conservation measures.
d) Neuromuscular blockade: Patients with CP have a slight increased sensitivity to succinylcholine, which is not usually clinically relevant.
i) They have increased resistance to vecuronium.[9,10]
e) MAC is decreased in patients with CP and may be further decreased in patients taking some antiepileptic medications.[9,10]
f) Regional and neuraxial anesthesia used in combination with GA is effective for surgery in children with CP and can reduce painful post-op muscle spasms as well as reduce postoperative narcotic consumption.



Children with CP have a slight increased sensitivity to succinylcholine, and they have ***resistance*** to vecuronium.



Consider regional anesthesia in combination with general anesthesia for children with CP

CHAPTER SUMMARY FOR AUTISM SPECTRUM DISORDER AND CP	
General Considerations	Patients with ASD may have difficulty managing new sensory inputs particularly in the unfamiliar hospital environment and may have behavioral outbursts associated with fear and anxiety.
Preoperative Considerations	***Plan ahead!*** Discuss clinical care plan with caregivers and staff ahead of time. Minimize stimulation, provide private rooms, and limit the number of providers interacting with the patient.
Intraoperative Considerations	Use premedication to ease patient anxiety and facilitate cooperation with needed care. Provide a calm, quiet environment with a single, gentle voice throughout induction. Take steps to minimize the risk of emergence agitation. Patients with CP have lower MAC and may be resistant to depolarizing neuromuscular blockade.
Postoperative Considerations	Provide a calm, quiet environment for recovery and have caregivers available as soon as possible to minimize anxiety of waking up in a foreign environment. Plan for early discharge when possible.

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87 Muscular Dystrophy

Evan Serfass

The muscular dystrophies are a group of inherited disorders of muscle that have in common progressive weakness of involved muscle groups. Although typically thought to primarily affect the skeletal muscle, cardiac and smooth muscle also can be severely affected. Common surgeries for patients with muscular dystrophy include release of contracture and correction of scoliosis. Patients with severe manifestations of a muscular dystrophy are at significantly greater risk of morbidity during and after anesthesia and sedation, with the most concerning perioperative problems resulting from weakness of respiratory muscles or abnormal cardiac dysfunction.

1) **Pathophysiology**
   a) This group of diseases is caused by defective or absent proteins in the cytoskeleton of muscle cells.
   b) Connections between the cytoskeleton and the extracellular matrix are weak, resulting in damage to muscle cell membranes and degeneration of muscle fibers.
   c) There is progressive development of skeletal weakness, contractures, and involvement of cardiac, respiratory, and smooth muscle.
2) **Types of muscular dystrophy**
   a) **Duchenne muscular dystrophy (DMD)** is the most severe type.
      i) Sex linked, affecting 1 in 3500 male births.
      ii) Usually presents by 5 years of age.
      iii) Although DMD remains a fatal disease, the advent of corticosteroid therapy has improved outcomes such that these patients currently survive to a median age of 35.[1]
   b) **Becker muscular dystrophy (BMD)**
      i) Presents similarly to DMD but later and with slower progression.
      ii) It affects 1 in 30 000 male births, and patients usually present in early adolescence.
      iii) Patients typically live into their mid-forties.[1]
   c) **Myotonic dystrophy (MyoD)** is the most prevalent muscular dystrophy in Caucasians.
      i) It affects 1 in 8000 and is usually transmitted in an autosomal dominant fashion.
      ii) The age of onset and disease severity are highly variable; MyoD type 1 can present in infancy, childhood, or adulthood, with greatest severity in the congenital form and least in the adult, while MyoD type 2 only has onset in adults and is less severe.[2–4]
   d) **Emery-Dreifuss muscular dystrophy**
      i) Characterized by mild skeletal muscle manifestations.
      ii) Cardiac conduction defects and arrhythmias are prominent and may cause syncope or sudden death.[2]
   e) Other muscular dystrophies include limb-girdle, facioscapulohumeral, and oculopharyngeal muscular dystrophy, with the names indicating the primary muscle groups affected.[1]
3) **Signs/symptoms/clinical findings**
   a) **Early**
      i) DMD and BMD:

(1) The earliest sign is often difficulty running, jumping, or ascending steps.
(2) Waddling gait, lumbar lordosis, and calf enlargement are also signatures.
(3) Gowers sign (the use of arms to help rise from a sitting position) is characteristic.[1,2]

ii) Myotonic dystrophy (MyoD)
(1) Initial symptoms vary, with weakness being a prominent presenting symptom the earlier the onset of the disorder.
(2) Patients with onset of MyoD type 1 in adulthood often present with other initial complaints such as arrhythmias, cataracts, or infertility.
(3) Hands, forearms, facial muscles, and ankle dorsiflexors (→ foot drop) are the commonly affected muscle groups.
(4) Muscle pain is common.
(5) Myotonia—best appreciated in the hand—is prominent in the early stages.[2–4]

b) **Late**
i) Respiratory
(1) Decreased vital capacity, maximal inspiratory pressure (MIP), and maximal expiratory pressure (MEP) are common.[5]
(2) Pharyngoesophageal weakness develops, resulting in aspiration.
(3) In DMD and BMD, progressive scoliosis results in restrictive lung disease.

ii) Cardiac dysfunction[5]
(1) MyoD: Atrial fibrillation and flutter are the most common arrhythmias, but ventricular arrhythmias can also occur. Left ventricular hypertrophy or dilatation is not uncommon, and cardiomyopathies can occur. Earlier age of initial symptoms correlates with earlier onset of cardiac dysfunction.[4]
(2) DMD: Primary dilated cardiomyopathy and conduction abnormalities develop in the teenage years. Significant mitral regurgitation may be present. Patients may not have cardiac symptoms, as their skeletal muscle weakness makes exercise impossible. Patients may be on ACE inhibitors or β-blockers.
(3) BMD: Similar to DMD but may have earlier onset of cardiac dysfunction as their ability to exercise puts strain on the heart.
(4) Emery-Dreifuss muscular dystrophy: Conduction abnormalities are common and are associated with a significant risk of sudden death; a permanent pacemaker or implantable defibrillator may be necessary. Weakness progresses slowly compared to other forms.

iii) Smooth muscle involvement is most common in MyoD, with irritable bowel syndrome–like symptoms, dysphagia, and possibly aspiration pneumonia.[3,5]



Succinylcholine is *absolutely* contraindicated in patients with DMD or BMD. Succinylcholine is *relatively* contraindicated in pediatric patients due to risk of undiagnosed myopathies.

c) **Associated findings**
i) Elevated creatine kinase (often 10-100 times normal in DMD patients).[2]
ii) Developmental delay is common in DMD and less common in BMD, and cognitive impairment may occur in MyoD (with greater severity and frequency the younger the age at presentation).[1,4]
iii) Side effects of steroid use in DMD and BMD, as chronic steroid therapy is common.[1]



Inhalational anesthetics may cause rhabdomyolysis and cardiac arrest in DMD and BMD patients. TIVA is recommended.

4) **Anesthetic management for patients with DMD or BMD**
a) Succinylcholine can lead to rhabdomyolysis and hyperkalemia, precipitating cardiac arrest. The black box warning on succinylcholine is due to deaths in undiagnosed DMD children who received this medication. DMD and BMD are absolute contraindications to the use of succinylcholine.[5]

b) Inhalational anesthetics may also precipitate rhabdomyolysis and cardiac arrest, even in the absence of succinylcholine, with greater frequency in younger patients. A total intravenous anesthesia (TIVA) is suggested as safest, although brief exposure to inhalational anesthetics in older children may be considered in situations where benefit appears to outweigh risk.[2,5,6]
c) Cardiac arrest may even occur postprocedure in the postanesthesia care unit (PACU) due to ongoing rhabdomyolysis and potassium release.[7]
d) Rhabdomyolysis is most common in patients <8 years old, as these patients have large numbers of fragile regenerating muscle fibers.[2]
e) There is no increased risk of malignant hyperthermia, although rhabdomyolysis and other complications can present a similar clinical picture.[7]
f) Careful use of nondepolarizing neuromuscular blockers is required, as both the onset and the recovery are prolonged. Consider the use of sugammadex for reversal.[5]
g) Possible difficult mask ventilation may occur due to macroglossia and limited mandibular and C-spine mobility. No increased risk of unanticipated difficult intubation has been reported.[5]
h) Patients are at risk for respiratory complications due to muscle weakness, such as obstruction, hypoventilation, atelectasis, respiratory failure, and difficulty weaning from mechanical ventilation. Consider extubation in the ICU. Preoperative pulmonology consultation is strongly advised.[2,5,8]
i) Patients are at risk for cardiac complications, including arrhythmias and heart failure due to myocardial depression or fluid overload. Preoperative cardiology consultation is strongly advised. Consider the use of invasive hemodynamic monitors. Treatment with inotropic agents may be necessary for severely affected patients.[5,9]
j) Chronic steroid use in DMD may lead to obesity, glucose intolerance, and osteoporosis and may necessitate stress-dose steroids.[10]
k) Advance directives should be clarified prior to surgery.[8,9]

5) **Anesthetic management for patients with myotonic dystrophy (MyoD) type 1:**[4]
a) Succinylcholine is not absolutely contraindicated; however, it may cause muscle contracture in these patients and is probably best avoided.
b) Drugs that cause pain with injection may precipitate myoclonus.
c) Inhalational anesthetics appear to be safe for induction and maintenance.
d) The patients may have increased sensitivity to IV hypnotics, sedatives, and opioids.
e) There is no increased risk of malignant hyperthermia or rhabdomyolysis in patients with MyoD.
f) These patients are at increased risk for regurgitation and aspiration, both during induction and postoperatively; modified rapid sequence induction with awake extubation is often the preferred approach.
g) Nondepolarizing neuromuscular blockers may be safely used; however, careful titration and monitoring are essential as low doses may be sufficient; reversal can be accomplished with neostigmine or sugammadex.
h) Respiratory complications are common postoperatively; patients may need postoperative respiratory support, extended stay in the PACU, or recovery in the PICU. Consider preoperative pulmonology consultation.
i) Cardiac complications are often arrhythmias; however, cardiac failure due to cardiomyopathy can also occur. Patients may present with pacemakers or internal defibrillators in place. Consider preoperative cardiology consultation.
j) Advance directives should be clarified prior to surgery.



Both onset of and recovery from nondepolarizing neuromuscular blockers are prolonged in patients with DMD.



Respiratory weakness is common: expect difficulties with spontaneous ventilation intraoperatively and postoperatively.

6) **Summary of DMD management guidelines** from the American College of Chest Physicians consensus statement.[5] Use clinical judgment in applying these to BMD, MyoD, or other muscular dystrophy patients.
   a) **Preprocedure**
      i) Anesthesia pre-op evaluation with a detailed review of systems, focusing on strength and motor capabilities.
         (1) Family medical and anesthetic history may point to undiagnosed muscular dystrophy (especially MyoD, which is often undiagnosed due to its varying severity); however, spontaneous mutations are responsible for at least 30% of cases of muscular dystrophy.[2]
      ii) Pulmonary consultation should be obtained in any muscular dystrophy patient with suspected respiratory compromise. Spirometry should include forced vital capacity (FVC), MIP, MEP, peak cough flow (PCF), and SpO_2 on room air.
         (1) If FVC < 50% predicted → increased risk of respiratory complications.
         (2) If FVC < 30% predicted → high risk of respiratory complications.
         (3) If SpO_2 < 95%, $PaCO_2$ or $EtCO_2$ should be measured prior to surgery.
         (4) Training in noninvasive positive pressure ventilation (NPPV) is suggested if FVC < 50% predicted.
         (5) Training in assisted cough if PCF < 270 L/min or MEP < 60 cm H_2O.
      iii) Cardiology consultation should be obtained for evaluation and medication optimization.
      iv) Nutritional assessment and optimization.
      v) Discussion of increased risk of anesthesia or sedation, resuscitation parameters, and advance directives (if appropriate) should occur prior to the procedure.



The myocardium is often affected in patients with DMD—life-threatening cardiomyopathies and dysrhythmias are common.

   b) **Intraoperative management**
      i) Setting and personnel are recommended to be of higher acuity or training than for the same procedure in a patient without muscular dystrophy.
      ii) Procedures normally performed outside the OR may need to be performed in the OR or PACU.
      iii) A respiratory therapist and an anesthesiologist with experience with muscular dystrophy patients may be required for intraprocedure and postprocedure care.
      iv) If FVC < 50% of predicted, use assisted or controlled ventilation for procedures normally done with spontaneous ventilation alone.
      v) Closely monitor cardiac and fluid status due to risk of congestive heart failure; consider using arterial lines, central lines, and transesophageal echocardiography.
   c) **Postoperative management**
      i) Strongly consider an ICU bed for postprocedure care, especially in DMD or older BMD patients.
      ii) Consider extubation to NPPV if FVC < 50% predicted or the patient is on NPPV preoperatively. If possible, use the patient's home NPPV device.
      iii) Extubation in the ICU avoids the risk of transporting a spontaneously ventilating patient with respiratory compromise.
      iv) Supplemental O_2 may mask problems with ventilation. Common causes of hypoxemia include hypoventilation, atelectasis, secretions, and obstruction.
      v) Consider manually or mechanically assisted coughing.
      vi) Optimize post-op pain control.
         (1) Continuous NPPV or delayed extubation may be necessary.
         (2) Strongly consider local, regional, or neuraxial techniques.

d) **Follow-up**
   i) Previously diagnosed patients: Appropriate postprocedure care is dictated by disease progression. Severely affected patients may require ICU recovery even for minor procedures using solely sedation.[5]
   ii) In undiagnosed patients who experience rhabdomyolysis or cardiac arrest due to hyperkalemia, ICU care will be necessary. Geneticists can establish the diagnosis and test family members; neurology, pulmonology, and cardiology follow-up are all appropriate.[7]

CHAPTER SUMMARY FOR MUSCULAR DYSTROPHY

General Considerations	The muscular dystrophies (MD) are inherited disorders that result in weakness of skeletal, cardiac, and smooth muscles. Succinylcholine and volatile anesthetics (alone or together) can precipitate rhabdomyolysis and cardiac arrest due to hyperkalemia in those with Duchenne or Becker MD. Diagnosis of MD is often not made until 3-5 y of age or later. Succinylcholine is therefore relatively contraindicated in all pediatric patients.
Preoperative Considerations	Preoperative consultation with pulmonologists and cardiologists should be sought due to weakness of respiratory muscles, cardiomyopathies, and arrhythmias. Surgery should ideally take place in a setting equipped to deal with respiratory and cardiac complications.
Intraoperative Considerations	Onset and recovery from nondepolarizing neuromuscular blockers may be significantly prolonged.
Postoperative Considerations	Recovery in the ICU may be necessary for patients with compromised respiratory or cardiac function.

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88 Cystic Fibrosis

Tammy Wang • Samuel A. Mireles

Cystic fibrosis (CF) is an autosomal recessive disease caused by a dysfunctional transmembrane chloride channel in epithelial cells of the airway, intestines, sweat, pancreatic and biliary ducts, and vas deferens. It is the most common lethal inherited disorder among Caucasians, with an incidence of 1:3500 births in this population, but can affect all racial groups.[1,2] Due to advances in medical management, median predicted survival is now >47 years for those born in 2016.[3]

1) **Clinical findings**
   a) **Pulmonary**
      i) Although disease manifestations vary widely, CF is progressive with pulmonary disease being the main cause of morbidity and mortality.
      ii) Patients have viscous mucous secretions in the airway and impaired ciliary clearance, which leads to mucus plugging, chronic infection, inflammation, and epithelial injury.
      iii) Chronic infections lead to bronchiectasis, increased airway resistance, and bullae, but there is also evidence of impaired airway function in infants prior to clinically recognized infections.[4]
      iv) Pulmonary function tests are consistent with obstructive disease with increased functional residual capacity but decreased forced expiratory volume, peak expiratory flow, and vital capacity.
      v) Tachypnea often leads to hypocapnia, but in severe disease, hypercapnia may prevail.



Chronic lung disease in patients with CF may lead to pulmonary hypertension and cor pulmonale.

   b) **Cardiac**
      i) Chronic hypoxemia can lead to pulmonary hypertension and right ventricular enlargement in CF patients, although the incidence is unclear.[5]
      ii) End-stage pulmonary disease may be associated with cor pulmonale, right ventricular failure, and fluid retention, which are associated with poor prognosis.[6]
   c) **Gastrointestinal**
      i) Malnutrition secondary to malabsorption is a common manifestation of the disease.
      ii) Low weight and body mass index are closely associated with poor lung function.[7]
      iii) Gastroesophageal reflux is common.



Malnutrition, malabsorption, and liver dysfunction may lead to coagulopathies.

   d) **Hematologic**
      i) CF patients have malabsorption of vitamin K and decreased synthesis of factors II, VII, IX, and X.
   e) **Hepatic**
      i) CF patients have decreased plasma cholinesterase.
      ii) Patients with end-stage disease and cor pulmonale may also have hepatomegaly and gastroesophageal varices.

f) **Pancreatic**
   i) Greater than 25% of patients aged 20 years have CF-related diabetes mellitus, increasing to 40%-50% at age 40. Greater than 75% of these patients are on chronic insulin therapy. CF-related diabetes may contribute to lung disease and worse outcomes.[2,8]

2) **Surgeries/procedures commonly performed in CF patients**
   a) Nasal polypectomy, endoscopic sinus surgeries
   b) Bronchoscopy, pulmonary lavage, management of pneumothoraces
   c) Investigation or correction of gastrointestinal disease such as meconium ileus in the neonate, gastroesophageal endoscopy, cholecystectomy, and sclerosis of varices due to portal hypertension in end-stage disease
   d) Insertion of venous access devices
   e) Organ transplantation, especially the lungs

3) **Anesthetic management**
   a) **Preoperative evaluation**
      i) Focus on the severity and optimization of pulmonary disease since this is the primary cause of morbidity and mortality in CF patients and the most common cause of perioperative complications.
      ii) History should assess the following:
         (1) Extent of multisystem involvement
         (2) Recent hospitalizations
         (3) Recent infections and antimicrobial sensitivities
         (4) Quality and quantity of secretions
         (5) Response to bronchodilators
         (6) Exercise tolerance
      iii) Physical exam should include:
         (1) Weight and body mass index
         (2) Baseline SpO_2
         (3) Cardiopulmonary assessment
      iv) Studies
         (1) Routine studies are not indicated.
         (2) Depending on the severity of disease and the extent of multisystem involvement, the following may be appropriate:
            (a) Arterial blood gas
            (b) Chest radiograph
            (c) Pulmonary function tests
            (d) Electrocardiogram
            (e) Echocardiogram
            (f) Glucose
            (g) Liver function tests
            (h) Coagulation studies
         (3) As with all preoperative assessment, invasive studies need only be obtained when the results are expected to alter management decisions.
      v) Pulmonary optimization
         (1) Pulmonary function should be optimized with chest physiotherapy, bronchodilators, and humidified nebulizer treatments.
         (2) Nebulized saline treatments may be administered up until induction.
         (3) Nebulized hypertonic saline (7% sodium chloride) has been shown to improve mucous clearance and lung function.[9]
         (4) Dornase alfa (DNase) has been shown to reduce air trapping, improve FEV_1, and decrease pulmonary exacerbation rate.[10]

b) **Monitoring/lines**
   i) Standard ASA monitors are usually all that is required.
   ii) Invasive lines are rarely necessary but may be indicated depending on the severity of disease and the surgical procedure.
   iii) In patients with severe lung disease, cor pulmonale, or intermediate- to high-risk procedures, invasive monitoring may be indicated.

c) **Intraoperative management**
   i) General anesthesia may be necessary due to patient age, coagulopathy, or surgical procedure.
   ii) Regional anesthesia offers the advantage of improved respiratory mechanics and is preferred over general anesthesia when not contraindicated.
      (1) Because of disease-related coagulopathy, consider coagulation studies prior to performing regional anesthesia, especially neuraxial procedures.
   iii) Induction
      (1) Anxiolysis with benzodiazepines is appropriate with anxious patients.
      (2) Inhalation induction may be prolonged due to increased functional residual capacity, small tidal volumes, and *V/Q* mismatch.[11]
      (3) IV induction and aspiration prophylaxis should be considered in patients with significant gastroesophageal reflux.
      (4) A cuffed endotracheal tube is preferred because higher airway pressures may be needed to ventilate poorly compliant lungs.
   iv) Maintenance
      (1) A deep anesthetic should be employed to prevent airway hyperreactivity.
      (2) Short-acting agents, local anesthetics, and nonopioid analgesics should all be considered to prevent postoperative respiratory depression.
      (3) Humidification of anesthetic gases is extremely important to reduce inspissated secretions and mucus plugging.
   v) Emergence
      (1) Ideally, the patient will be spontaneously breathing and extubated at the end of the procedure.
      (2) Complete reversal of neuromuscular blockers must be assured.
      (3) Deep extubation, when not contraindicated by difficult airway or gastroesophageal reflux, may decrease airway hyperreactivity in patients who are able to demonstrate adequate spontaneous respirations prior to emergence.
      (4) A head-up position will improve respiratory mechanics.
   vi) Complications
      (1) Most common are bronchospasm, laryngospasm, obstruction from inspissated secretions, hypoxemia, and atelectasis.
      (2) Less common complications include pneumothorax, pneumonia, postoperative respiratory failure, and opioid induced ileus.



Optimize pulmonary function preoperatively with bronchodilators, hypertonic saline treatment, and DNase.



Humidify all gases during the anesthetic, and consider suctioning ETT to prevent plugging of airways.

d) **Postoperative management/disposition**
   i) The goals are to provide adequate analgesia without respiratory depression, improve clearance of secretions, and minimize atelectasis.
   ii) Early ambulation, chest physiotherapy, nebulized saline treatments, and bronchodilators as needed should be utilized in postoperative care.
   iii) Monitored wards or ICU should be considered for severely affected patients.

CHAPTER SUMMARY FOR CYSTIC FIBROSIS

General Considerations	Cystic fibrosis is a multisystem disease with pulmonary, cardiac, gastrointestinal, endocrine, and hematologic manifestations.
Preoperative Considerations	Optimize pulmonary function preoperatively by the use of chest physiotherapy, bronchodilators, and humidified nebulizer treatments.
Intraoperative Considerations	Maintain deep anesthetic to prevent airway hyperreactivity and humidify anesthetic gases to improve mucous clearance.
Postoperative Considerations	Avoid postoperative respiratory depression by the use of regional techniques when feasible, short-acting anesthetics/opioids, and nonopioid analgesics.

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89 Part B: Anesthetic Concerns and Procedures in Pediatric Anesthesia

Anesthesia for the Premature Infant

Tammy N. Wang • Julie L. Williamson

Premature birth is defined as birth prior to 37 weeks' gestational age and is an important cause of morbidity and mortality in children. In the United States, since 2014, the rate of premature birth has risen to 9.85% of live births with the highest incidence in late preterm (34-36 weeks' postgestational age) births.[1] Extremely premature (24-28 weeks' gestational age) babies are the most likely to have major morbidities that result in frequent and challenging presentations for anesthetic care.[2] Even in the absence of underlying anomalies or with term and late preterm infants, careful anesthetic management is needed to optimize the infant's vulnerable physiologic state.

1) **Physiology and implications of prematurity**
   a) **Airway**
      i) Endotracheal tubes (ETTs)
         (1) Very small ETTs (2.0-3.0) are at increased risk for obstruction due to kinking and secretions.
         (2) Inspired gases should be warmed and humidified to prevent hypothermia and plugging of the ETT.
         (3) Frequent suctioning may be required.
      ii) Subglottic stenosis is common in previously intubated infants. Smaller-than-anticipated ETTs should be prepared.
      iii) Tracheomalacia or bronchomalacia may cause airway collapse on exhalation. This can be overcome with continuous positive airway pressure (CPAP) or positive end-expiratory pressure (PEEP).
      iv) While awake intubation by direct laryngoscopy is not common in anesthesiology, it is performed in the neonatal intensive care unit (NICU) and may be the most prudent method in this patient population if a difficult airway is suspected.



Awake intubations are sometimes performed by neonatologists and may be the most prudent method of securing the airway of a premature infant with a potentially difficult airway.

   b) **Central nervous system**[3]
      i) Periventricular leukomalacia and hemorrhagic infarction are common in premature infants of 27 weeks' gestational age and younger.
         (1) Grade I intraventricular hemorrhage (IVH) is bleeding into the fragile germinal matrix of the ventricles and is the most common site of bleeding in premature infants. This does not correlate with an adverse neurologic outcome.
         (2) Grade II IVH indicates bleeding into the ventricle.
         (3) Grade III IVH causes enlargement of the ventricle.
         (4) Grade IV IVH involves intraparenchymal bleeding. This may require placement of a ventricular peritoneal shunt and is associated with adverse neurologic outcomes.

ii) Cerebral autoregulation and complications
  (1) The limits of cerebral autoregulation in the premature infant are unknown. Therefore, a key anesthetic goal is avoidance of extreme swings in blood pressure.
    (a) Hypotension is a risk factor for cerebral ischemia.
    (b) Hypertension may increase risk of IVH.
    (c) It is common to use gestational age as a guideline for mean arterial blood pressure, but blood pressure rises significantly in the first few days of life, so all preterm infants should have a mean pressure >30 mm Hg by 72 hours of life.[4]
  (2) Coagulopathy should be aggressively treated prior to undergoing surgery.

iii) Retinopathy of prematurity (ROP) is pathology of retinal vasculature in which aberrant vascular development can cause retinal tears and detachment and may ultimately lead to blindness.
  (1) Risk factors for ROP include prematurity, low birth weight, and exposure to supplemental O_2. Up to 70% of neonates weighing <1000 g will be affected.
  (2) Supplemental O_2 should be avoided when possible in infants until 44 weeks' postgestational age.[5]
  (3) Supplemental O_2 should be given with a goal SpO_2 above 90% for the neonate.

iv) Parasympathetic tone predominates in the neonate. Removal of sympathetic tone with anesthetic agents can cause profound bradycardia and hypotension.
  (1) It is common to premedicate infants with vagolytic drugs prior to induction or manipulation of the peritoneum or eyes.
  (2) Atropine 0.01-0.02 mg/kg or glycopyrrolate 0.01-0.015 mg/kg may be used to prevent bradycardia on induction.[6]

c) **Respiratory system**

i) Central response to hypoxia and hypercapnia is dysregulated in the premature infant. Although ventilation may initially increase, the infant quickly may become apneic.

ii) Anesthetic agents will suppress the response to hypoxia or hypercapnia, placing the emerging or the postoperative infant at increased risk for apnea.
  (1) Apnea may be both centrally mediated and obstructive in premature infants and neonates.
    (a) Central apnea may result from hypoxia, hypercarbia, intracranial hemorrhage, hypothermia, airway stimulation, or without any apparent inciting event.
    (b) Obstructive apnea is associated with oropharyngeal dyscoordination and hypotonia and is commonly due to obstruction from the tongue.
  (2) Oral airways are often needed to effectively mask ventilate premature infants.
  (3) Postoperative apnea >15 seconds or associated with bradycardia or desaturation is common in the premature infant. Its occurrence is proportional to early gestational


Avoid extreme swings in blood pressure to minimize risk of intraventricular bleeding.


Coagulopathy should be aggressively treated to minimize risk of intracranial bleeding.


Supplemental O_2 should be avoided when possible in infants until 44 weeks' postgestational age.


Parasympathetic tone predominates in the neonate, so atropine or glycopyrrolate may be used to prevent bradycardia on induction.


Apnea is both centrally mediated and obstructive in premature infants and neonates.


Oral airways are often needed to mask ventilate premature infants.

age and is associated with anemia (Hct < 30%) and invasive surgical procedures.[7]

iii) Functional residual capacity is 30 mL/kg, similar to that of an adult. However, alveolar ventilation is two to three times (100-150 mL/kg/min) that of an adult, reflecting the greater O_2 consumption of the infant.

iv) Preoxygenate whenever feasible to mitigate the rapid desaturation of the infant during induction.



Preoxygenate the patient to mitigate the rapid desaturation during induction.

v) Surfactant is not endogenously produced until 34 weeks of gestation.

(1) Respiratory distress syndrome is a consequence of inadequate (premature infants) or delayed (infants of diabetic mothers) surfactant production. Lung volumes and compliance are decreased, and intrapulmonary shunt, *V/Q* mismatching, and risk of pneumothorax are increased in this disease state. A longer expiratory time may aid emptying in infants with lung disease.[7]



Goal SpO_2 for the neonate should be >90%.

(2) Continuous PEEP may aid oxygenation. Decreased lung compliance may require high peak inflating pressures that risk pneumothorax and interstitial emphysema.

(3) Pressure-controlled ventilation and monitoring of peak pressures and tidal volumes are needed to avoid lung damage.

(4) Decompensation of a ventilated infant should raise concern for pneumothorax, which can be evacuated with a 22-gauge IV catheter placed at the second intercostal space in the anterior axillary line.

(5) Continued supplemental O_2 requirement at 36 weeks' gestational age is referred to as chronic lung disease. These children are at increased risk of perioperative laryngospasm and bronchospasm into the early school-age years.

vi) Awake spinal anesthesia may be an appropriate alternative to general anesthesia in the preterm infant at risk of apneas for certain surgeries such as inguinal hernia repairs.[8]

(1) All ex-premature infants should be monitored for apneas in the late postoperative period as spinal anesthesia decreases the risk of early but not late apneas.[9]

d) **Cardiovascular system**

i) A poorly organized myocardium, poor diastolic function, and increased amount of connective tissue lead to decreased compliance in the neonatal heart.

ii) Premature infants have limited ability to augment stroke volume; therefore, cardiac output is strongly dependent on heart rate.

iii) The neonatal myocardium is preload dependent and requires adequate circulating volume to maintain an adequate cardiac output.

iv) The combination of high resting heart rate, decreased stroke volume reserve, and low blood volume predisposes the premature infant to cardiovascular collapse during induction of anesthesia or major surgical events.[3]

v) Vasodilation, myocardial dysfunction, and bradycardia from volatile agents and other anesthetics are poorly tolerated in premature infants.



Vasodilation, myocardial dysfunction, and bradycardia from volatile agents are poorly tolerated in premature infants.

vi) The transition to extrauterine physiology is often delayed in these infants. Failure of the ductus arteriosus and the foramen ovale to close is common.

(1) Strict attention should be paid to avoid air bubbles in IV tubing and injections to prevent "paradoxical" shunting of air emboli from the right to the left heart.
(2) Patent ductus arteriosus (PDA) can cause congestive heart failure and even pulmonary hypertension.
(a) Infants in the NICU with PDA are often treated with fluid restriction and diuretics, which may lead to a hypovolemic infant with electrolyte derangements presenting for surgery.
(b) Fluid resuscitation and sodium and potassium imbalance may need to be corrected prior to induction.



Preterm infants may be taking *fortified* breast milk that has an NPO time of 6 hours.

e) **Gastroenteric system**
i) Hyperbilirubinemia requiring phototherapy is ubiquitous in premature infants, and treatment with "bili lights" will increase insensible fluid losses.
ii) NPO times are generally the same as older children: 2 hours for clears, 4 hours for breast milk, and 6 hours for formula. Preterm infants may be taking fortified breast milk, which has an NPO time of 6 hours. IV fluid repletion with dextrose will often be needed to avoid hypoglycemia and hypovolemia.



Dextrose-containing fluids should be used for maintenance in the premature neonate as glucose requirements are relatively high. For hypoglycemia (<40 mg/dL), bolus 2-4 mL/kg of 10% dextrose over 1 minute IV.

f) **Fluid, electrolytes, and nutrition**
i) Glucose requirement is relatively high. Infusion of 5 mL/kg/h of 10% dextrose-containing solution will provide the 6-8 mg/kg/min glucose infusion rate that the premature infant needs.
ii) If a concentrated dextrose solution (eg, total parental nutrition with 15% dextrose) is changed to a lower concentration or rate, hypoglycemia can occur rapidly. Regular monitoring of serum glucose levels intraoperatively is recommended.
iii) Use dextrose-containing fluid as a maintenance solution, and then replace losses with non–dextrose-containing isotonic crystalloid, colloid, or blood product as needed.
iv) For hypoglycemia below 40 mg/dL, bolus 2-4 mL/kg of 10% dextrose over 1 minute to provide 200-400 mg/kg of glucose.[7]
v) The neonatal myocardium is relatively dependent on extracellular calcium release, so an ionized calcium level >1.0 mmol/L will aid cardiac output.
(1) Calcium chloride should be infused via a central line. Calcium gluconate may be slowly infused peripherally.[10]
vi) Sodium and potassium are not repleted in the first day of life. Daily requirements thereafter are similar to adults.[11]
vii) Newborn premature infants may have up to 90% of total body water as extracellular fluid volume.
(1) Spontaneous diuresis over the first few days of life may be augmented by diuretic therapy to treat the respiratory distress syndrome.
(2) These infants often present to the OR with intravascular volume depletion requiring fluid resuscitation prior to induction.



Administering calcium can augment cardiac output because the neonatal myocardium is more dependent on calcium release.

viii) Immature hepatic and renal function, altered volume of distribution, and decreased protein binding all lead to altered pharmacokinetics in premature infants. It is important to carefully titrate doses of medications to effect (eg, use of train-of-four monitoring to redose neuromuscular blockade).

g) **Hematologic system**
i) Estimated blood volume of the premature infant is 90-100 mL/kg.
ii) The normal hematocrit of a term newborn is 60%, but an early preterm neonate may have a Hct of 40%.

iii) Hct below 30% is poorly tolerated and is associated with apnea.
iv) Blood should be filtered, warmed, and administered slowly to avoid hyperkalemia.
v) Blood products for infants should be irradiated to prevent graft vs host reaction, a lethal complication of transfusion in neonates.[12]
vi) Newborn premature infants commonly have vitamin K deficiency and thrombocytopenia.

Blood products for infants should be irradiated to prevent graft vs host reaction.

h) **Temperature regulation is important in small infants.**
i) The room should be warmed to 27°C/80°F to avoid metabolic stress.
ii) Radiant warmers can be used.
iii) Inhaled gases should be heated and humidified. Transfused fluids and irrigation fluids should be warmed.

The estimated blood volume of the premature infant (90-100 mL/kg) is relatively higher than that of older children and adults.

2) **Preanesthetic evaluation**
a) History and physical should include assessment of cardiac anatomy if hypoxemia or other congenital malformations exist.
b) Volume status should be carefully assessed as preoperative fluid resuscitation may be needed; a sunken fontanelle, decreased skin turgor, and decreasing weight are signs of hypovolemia in the infant.

3) **Anesthetic management**
a) Anesthesia has been safely delivered to these small, fragile children for decades using both regional and general techniques.
b) **Induction**
i) Preoxygenate the infant with 100% FiO_2
ii) Consider aspirating gastric contents by orogastric or nasogastric tube prior to induction.
iii) Assure that peripheral IV is patent if IV induction is planned.
iv) Consider atropine or glycopyrrolate to decrease secretions and prevent bradycardia.
v) Opioids, midazolam, ketamine, and propofol are commonly used induction agents. Propofol may cause disproportionate hemodynamic decompensation in hypovolemic or sick infants.
vi) After loss of consciousness, use an oral airway and ventilate gently to avoid overinsufflation of the stomach, which could affect lung expansion and compress the heart affecting cardiac output. This is particularly important for laparoscopic cases.
vii) Paralysis may be obtained with 0.6-1 mg/kg of rocuronium (up to 1.2 mg/kg for rapid sequence intubation).



Gentle mask ventilation is recommended to avoid overinsufflation of the stomach, which could affect lung expansion and compress the heart affecting cardiac output.

c) **Maintenance**
i) MAC (minimum alveolar concentration) of isoflurane is reduced in the premature infant; even low levels of volatile agent may lead to hemodynamic decompensation.
ii) Ketamine in doses of 2-4 mg/kg IV every 20-30 minutes provides analgesia and anesthesia and maintains circulatory tone in most circumstances.
iii) Opioids are well tolerated with regard to hemodynamics, and titration of high-dose opioids (eg, 10-20 µg/kg of fentanyl) may be used for maintenance if postoperative mechanical ventilation is planned.
iv) Remifentanil infusion is also well tolerated and allows for earlier postoperative extubation. It is typically used in combination with other anesthetic agents (volatile or intravenous) to maintain anesthesia and the required dose may be higher in children compared to adults.[13,14]

d) **Postoperative management/disposition**
   i) Postanesthetic monitoring is recommended for all premature infants even in the absence of comorbidities and even if no surgical procedure was performed (eg, imaging studies).
   ii) Apnea is possible even without opioid use and has been reported even when solely regional anesthesia was performed.
   iii) Current recommendations are to defer surgery and anesthesia until after 60 weeks' postgestational age when possible.
   iv) Anemia (Hct < 30%) increases risk of postoperative apnea.
   v) Conservative recommendations are to admit infants <60 weeks' gestational age after regional or general anesthesia for postoperative monitoring for apnea of prematurity.[15]
   vi) At our institution, infants born at <37 weeks' gestational age who are <52 weeks' gestational age at the time of the anesthetic are admitted for monitored observation overnight.
   vii) Infants who were born earlier than 37 weeks and who are <60 weeks' gestational age at the time of anesthetic are admitted for monitoring if they have any remaining significant comorbidities.



Postanesthetic monitoring is recommended for all premature infants even in the absence of comorbidities and even if no surgical procedure was performed. Apnea is possible even without opioid use and has been reported even when solely regional anesthesia was performed.

4) **Anesthesia and neurotoxicity**
   a) Recently, there has been concern regarding the safety of anesthesia in infants because animal studies have shown exposure to general anesthesia results in pathohistological changes associated with behavioral changes.
   b) Two large current prospective trials (GAS and PANDA) in children have studied if early exposure to anesthesia may have long-term neurologic consequences. Initial results from the GAS trial demonstrate no effect from a single brief exposure to general anesthesia compared to awake regional anesthesia.[16]

CHAPTER SUMMARY FOR ANESTHESIA FOR PREMATURE INFANTS

Definitions	Prematurity: gestational age <37 weeks Small for gestational age (SGA) are those <10% tile for gestational age or >2 standard deviations below the mean
Airway	Humidify and warm gases to prevent occlusions Watch for kinking and obstruction in small ETTs
Neurologic	Monitor postoperatively for central apnea Avoid swings in BP, which can cause IVH Avoid supplemental O_2 causing ROP High parasympathetic tone—consider pretreatment with atropine or glycopyrrolate
Respiratory	Increased alveolar ventilation leads to rapid desaturation Surfactant deficiency leads to high peak inspiratory pressures and risk of pneumothorax
Cardiovascular	Cardiac output is heart rate dependent Avoid air bubbles due to presence of shunts
Fluids, Electrolytes, Nutrition	Deliver adequate glucose infusion rate Supplement calcium if hemodynamically unstable Assess for hypovolemia and resuscitate prior to induction
Hematologic	PRBCs should be warmed, filtered, and irradiated Hct < 30% is associated with apnea

Induction	Preoxygenate with 100% oxygen Pretreat with vagolytic agent
Maintenance	Volatile agents and propofol may cause hemodynamic instability Monitor neuromuscular blockade due to variable pharmacokinetics Maintain thermal environment >80°F
Postoperative	High risk for apnea Monitor infants <60 wk postgestational age overnight

ETT, endotracheal tube; IVH, intraventricular hemorrhage; ROP, retinopathy of prematurity; PRBCs, packed red blood cells.

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90 Adenotonsillectomy

Dondee Almazan • Calvin Kuan

Adenotonsillectomy, or tonsillectomy and adenoidectomy (T&A), was a nearly universal procedure for children in the early 1900s for a wide range of medical conditions. It is still one of the most commonly performed pediatric surgical procedures in the world. The indication for surgery is now limited to recurrent tonsillitis or obstructive sleep apnea (OSA). Currently, OSA accounts for over 75% of the clinical indication for T&A in children.[1]

1) **Pathophysiology**
   a) Nasal and pharyngeal upper airway obstruction can cause chronic hypoxemia and hypercarbia, which may lead to pulmonary vascular changes resulting in pulmonary hypertension, right ventricular hypertrophy, and even cor pulmonale—all of which significantly increase risks of anesthesia.
   b) Obstructive sleep apnea (OSA) is defined as sleep-disordered breathing caused by partial or complete airway obstruction. It is an abnormal respiratory pattern while asleep that leads to obstructive events.
      i) The gold standard for diagnosis of OSA is polysomnography (PSG), which is an electrographic recording of physiological parameters for identifying and quantifying sleep-disordered breathing.
         (1) Though there are no established guidelines for ordering PSG, it is highly recommended for the following patients with the following comorbidities: obesity, Down syndrome, craniofacial abnormalities, neuromuscular disorders, sickle cell disease, or mucopolysaccharidoses.
   c) Pediatric OSA classification: apnea-hypopnea index (AHI) is the number of hypopneas and apneas per hour as determined during PSG (**Table 90-1**).
   d) Recurrent and chronic tonsillitis is the most common indication for T&A in adults, and not OSA.[4]
      i) Chronic or recurrent infections are not associated with cardiopulmonary changes.
      ii) Chronic tonsillitis is defined as infection or inflammation of the tonsil for at least 3 months. Symptoms typically recur after antibiotic cessation.
      iii) Most are caused by viral infection and are typically benign and self-limiting.



Refer to the American Society of Anesthesiologists (ASA) and the American Academy of Pediatrics (AAP) guidelines for more information about the perioperative management of children with OSA.[2,3]



Assess patients for risk of OSA because of increased perioperative complications—airway obstruction and respiratory depression.

2) **Signs/symptoms/clinical findings**
   a) **History**
      i) Sleep problems
      ii) Loud snoring
      iii) Daytime somnolence
      iv) Observed pauses during sleep
      v) Irritability
      vi) Speech problems

TABLE 90-1 Pediatric OSA Classification

Severity	AHI
Mild	1-4.9
Moderate	5-9.9
Severe	>10

AHI, apnea-hypopnea index.

b) **Physical exam**
   i) Majority of children are overweight with BMI > 95 percentile for age/gender; however, some have normal weight and maybe even underweight.
   ii) Craniofacial abnormalities affecting the airway (eg, Treacher Collins).
   iii) Upper airway obstruction.
   iv) Coughing.
   v) Halitosis.
   vi) Acute or recurrent otitis media and/or adenoiditis or tonsillitis.

c) Less common or more serious findings include:
   i) Failure to thrive
   ii) Hypoxemia and hypercarbia
   iii) Cardiac abnormalities
      (1) Left ventricular dysfunction
      (2) Right ventricular hypertrophy
      (3) Pulmonary hypertension
      (4) Cor pulmonale
   iv) Peritonsillar abscess
   v) Recurrent lower respiratory tract infections

3) **Surgical treatment**
   a) **Tonsillectomy** is the surgical excision of the palatine tonsils.
   b) **Adenoidectomy** is usually performed in conjunction with tonsillectomy, especially in the presence of associated chronic adenoiditis, recurrent otitis media, and/or nasal obstruction.
   c) Many different surgical techniques are employed, including dissection, ultrasound ablation, and electrocautery.

4) **Anesthetic management**
   a) **Preoperative considerations**
      i) Determine the indication for surgery, and include review of medical records and previous anesthetics, medical history, physical exam, and sleep studies.
      ii) Focus on airway, cardiac, pulmonary, and hematologic systems.
         (1) It is most important to identify children with sleep-disordered breathing or OSA because they are at greater risk of developing perioperative complications.[1]
            (a) There is no official definition for diagnosis of OSA in children.
            (b) Many children may be referred for surgery without formal or extensive workup, but they are still at risk for OSA-associated complications.
            (c) The practitioner must have a high index of clinical suspicion and need for workup to determine the severity of disease.
         (2) Patients with obstructive symptoms may have underlying craniofacial abnormalities that are associated with difficult airways.
         (3) Recent upper respiratory infections may necessitate postponing surgery due to increased respiratory complications and increased risk for postoperative bleeding.[5] Clinicians should also recognize that children (especially school age) may have multiple respiratory infections throughout the year, and it may be

difficult to expect a period of 4-6 weeks without respiratory symptoms.

(4) Any history of easy bleeding or bruising must be investigated to rule out underlying coagulopathy.



Premedication should not automatically be given to patients with OSA without adequate monitoring preoperatively.

iii) Additional labs/studies prior to surgery for the otherwise healthy child are not necessary and should be ordered on a case-by-case basis (eg, history of easy bleeding).

iv) Premedication should be individualized, rather than routine.

(1) Children with sleep-disordered breathing or OSA have increased sensitivity to sedatives and opiates[6]—a dose of midazolam 0.5 mg/kg po may result in hypoxia or even apnea.

(2) Consider avoiding premedication due to high risk of respiratory depression for these patients. Alternatives to premedication include parental presence at induction, use of child life specialist, and visual-audio technology distractions.

(3) If premedication is given, children should be closely monitored for obstruction, hypoventilation, and hypoxemia, including pulse oximetry and ECG monitoring.

b) **Intraoperative considerations**

i) Monitors/lines/position

(1) Standard ASA monitors with a single IV line are sufficient for routine T&A cases.

(2) Patients with cardiac or pulmonary involvement may require additional invasive monitors.

(3) The patient is typically turned 90 degrees away from the anesthesiologist.

ii) Induction



Children with OSA have greater incidence of respiratory problems on induction compared to children with tonsillitis.

(1) Inhalational induction is typically initiated with sevoflurane and O_2.

(a) N_2O may be used if needed.

(b) Preoxygenation with 100% O_2 will allow greater oxygen reserve in case of airway problems.

(2) Patients with OSA have a greater incidence of respiratory problems (eg, laryngospasm, breath holding, and desaturation) on induction compared to children with tonsillitis.[7]

(3) IV induction may be safer for children with significant cardiac or pulmonary comorbidities.

(4) For older children, IV induction is also preferred for faster and more controlled induction.

iii) Airway may be secured either with ETT or LMA depending on preference of the anesthesiologist and surgeon. However, ETT is preferred due to limited studies validating the use of LMA. There are also reports of failed LMA intraoperatively leading to eventual endotracheal intubation, especially for obese patients.[8]

(1) Reinforced or armored LMA

(a) Classic or nonreinforced LMAs are likely to kink or dislodge.

(b) Spiral reinforced LMA Flexible can be positioned safely under the retractor without kinking or dislodging.

(c) The smallest size Flexible currently available is size 2 for children of 10-20 kg.

(d) LMA use is associated with decreased incidence of postoperative stridor and laryngospasm but does not protect airway in case of aspiration.[9]

(e) There is no evidence that LMA decreases morbidity over an ETT.

(2) Cuffed oral RAE ETT

(a) Cuffed ETT are preferred to minimize number of intubations and to minimize leak of O_2 or anesthetic gases.

(b) Oral RAE tubes are preferred due to the preformed angle for better surgical exposure and decreased risk of kinking.
(c) Armored cuffed ETT are also less likely to kink.
(d) Advantages of the ETT over the LMA include:
(i) Provision of a secured airway
(ii) Ability to safely paralyze and ventilate a patient if necessary
(iii) Isolation of the trachea with cuffed ETT from blood/fluids
(iv) Ability to minimize leak of O_2 into surgical field, decreasing risk of airway fire

iv) Maintenance
(1) Volatile vs IV anesthesia
(a) Volatile anesthetics
(i) Sevoflurane is associated with a smoother induction and a slightly longer emergence compared to desflurane.
(ii) Desflurane has a faster offset compared to sevoflurane when used for maintenance but can cause tachycardia and severe laryngospasm during emergence and extubation.
(iii) Both have similar incidence of emergence delirium and time to patient discharge.
(b) IV anesthetics may be used to supplement volatile anesthetic or as a total intravenous anesthesia (TIVA).
(i) Due to the typical short duration of surgery, TIVA with short-acting agents may be preferred.
1. Propofol has antiemetic properties and smoother emergence. Propofol may be used as a supplement to a balanced anesthetic to decrease the required amount of volatile agent or other intravenous anesthetics.
2. Remifentanil's potent and ultrashort-acting analgesic properties facilitates general anesthesia without the long-term effects of a long-acting opioid. However, remifentanil has been associated with hyperalgesia and, due to its rapid metabolism, can lead to extreme postoperative pain upon discontinuation.
(ii) Dexmedetomidine may reduce the incidence of early agitation and can be used with a balanced inhalational anesthetic. Initial loading dose of 1 μg/kg is followed by a maintenance infusion of 0.4 μg/kg/h.[10]
(iii) Opioids should be carefully titrated in during the case as needed in anticipation of exaggerated post-op opioid sensitivity.



Spontaneous ventilation during case allows for titration of narcotics/analgesics to respiratory rate to achieve adequate analgesia prior to emergence.

(2) Controlled vs spontaneous ventilation
(a) Controlled ventilation
(i) May be necessary for patients who require muscle relaxation for induction/intubation or treatment of airway obstruction. If necessary, use of short-acting muscle relaxant is preferred followed by full reversal.
(ii) Use of succinylcholine is discouraged due to its black box warning for concerns of hyperkalemia and rhabdomyolysis causing cardiac arrests in children with undiagnosed muscular dystrophies.
(iii) Advantages
1. The ability to use paralytic agent to guarantee immobility during surgery.

2. The availability of sugammadex may permit avoidance of succinylcholine, but still allow for rapid reversal of neuromuscular blockade.

(iv) Disadvantages
1. Need to administer reversal agents with associated risks of nausea/vomiting and residual neuromuscular blockade
2. Unable to use patient's spontaneous respiratory rate to gauge titration of opiates for postoperative analgesia

(b) Spontaneous ventilation
(i) Advantages
1. Respiratory rate and $ETCO_2$ level can be used as a guide for titration of opioid or other analgesics during the case to achieve a smoother emergence.
2. Theoretically faster emergence as there is no need to wait for return of spontaneous respirations.

(ii) Disadvantages
1. May require deeper level of anesthesia to prevent movement interfering with surgery
2. May not be an option if paralysis was necessary on induction

c) **Special anesthetic considerations**

i) Anesthesia for T&A has a high incidence of upper airway obstruction and airway reactivity (ie, laryngospasm and bronchospasm), especially for patients with OSA.

ii) The airway is shared between the anesthesiologist and the surgeon, so be aware of high risk of accidental extubation due to surgical manipulation.

iii) A mouth gag is usually placed by the surgeon and must be removed at the end of the case.



A mouth gag is typically placed by the surgeon and must be removed at the end of the case.

iv) Be vigilant for tube kinking and lips/tongue being pinched especially during retractor insertion. Consider asking the surgeon to release airway retractor every 15 minutes to lessen the risk of airway ischemia and swelling.

READ MORE
ENT and laser surgery, Chapter 122.

v) Airway fire
(1) Risk depends on surgical technique.
(2) Use the lowest tolerated FiO_2.
(3) Use of cuffed ETT with minimal leak around cuff decreases risk.
(4) N_2O is noncombustible but will accelerate burning of combustible material.
(5) Discuss with surgeon and nursing staff regarding a treatment plan before an airway fire occurs. Awareness is paramount to prevention. Surgical fire is a never event and should be included as part of the presurgical time out discussion.



Due to risk of airway fire, keep FiO_2 as low as tolerated by the patient.

vi) Acute postobstructive pulmonary edema
(1) Acute upper airway obstruction in spontaneously breathing patient may result in negative pressure pulmonary edema.
(2) Relief of chronic upper airway obstruction may cause noncardiogenic pulmonary edema.
(3) Treatment of both includes endotracheal intubation with higher than normal PEEP and supportive care.
(4) Since the problem is not primarily fluid overload, diuresis is contraindicated and may be detrimental.

d) **Emergence/analgesia**
   i) Extubation recommendations to decrease PONV.
      (1) Historically recommended to extubate patients awake, but many practitioners choose to extubate patients deep.
      (2) Full reversal of neuromuscular blockade is recommended.
      (3) Deep extubation
         (a) Advantages
            (i) Theoretically less risk of postoperative bleeding from coughing or bucking on emergence.
            (ii) Children whose indication for surgery is infection rather than OSA have a lower risk of airway problems with deep extubation.
         (b) Disadvantages
            (i) Increased risk of airway obstruction and laryngospasm
         (c) Strategies to decrease the risk of airway obstruction
            (i) Propofol maintenance over vapor anesthetic.
            (ii) Select older/larger patients with less severe obstructive histories for deep extubation.
      (4) Awake extubation
         (a) Advantages
            (i) Ability to protect the airway from blood/secretions prior to extubation
         (b) Disadvantages
            (i) Coughing and straining on emergence may cause bleeding from surgical sites.



Turn patient on side prior to extubation to allow secretions/blood to pool out of the mouth and decrease risk of laryngospasm.

   ii) Positioning patient for emergence
      (1) Tonsil or recovery position: patient in lateral position to allow fluid to pool away from and out of the mouth.
      (2) Patient may be kept in this position for transport to recovery.
   iii) Analgesic management
      (1) Adenotonsillectomy is very stimulating during the surgery and can be very painful postoperatively.
      (2) The goal of complete analgesia must be weighed against risk and side effects of drugs used (airway obstruction and hypoventilation).
      (3) Dexamethasone
         (a) A single dose 0.5-1 mg/kg IV (maximum dose: 20 mg) given intraoperatively is effective for procedures using electrocautery.
         (b) In addition to its analgesic properties, dexamethasone has been shown to decrease the risk of PONV.
      (4) Acetaminophen
         (a) Effective adjunct analgesic may be given during surgery and continued postoperatively.
         (b) Due to its better pharmacodynamic profile, IV acetaminophen is preferred over po and rectal routes (dose is 15 mg/kg IV).



Children with OSA are extra sensitive to narcotics—give incremental doses titrating to respiratory rate and analgesic effect.

      (5) Opioids
         (a) Due to concerns for PONV and respiratory depression, opioid sparing with multimodal analgesia is highly recommended.
         (b) Weigh risk-benefits of giving long-acting vs short-acting agents in children.
         (c) Codeine should not be given to children with OSA who are not monitored due to risk of active metabolites causing respiratory depression.[11]
         (d) Children with OSA.

(i) Smaller than expected opioid dose may produce exaggerated respiratory depression.[12]
(ii) Titrate doses to effect while watching analgesic response and respiratory rate to avoid overdosing.

(6) Nonsteroidal anti-inflammatory drugs (NSAIDs) should only be used after consulting with the surgeon.
(a) Effective postoperative opioid-sparing adjunct.
(b) Reported increase in postoperative bleeding, but systematic reviews have not supported this finding.
(c) Aspirin increases bleeding risk, but other NSAIDs do not.

(7) Ketamine
(a) May decrease need for opiates and have preemptive analgesic properties

iv) Recommendations to decrease PONV
(1) The incidence of PONV may be as high as 75% after T&A. Aggressive PONV prophylaxis is highly recommended.[13]
(2) Suction the stomach and oropharynx prior to extubation.
(3) Consider the use of propofol for maintenance.
(4) Prophylactic serotonin antagonist (eg, ondansetron) given intraoperatively is more effective than metoclopramide.[14]
(5) Dexamethasone is also effective in preventing PONV as well as treating pain.[15]
(6) Minimize use of opioids and avoid neuromuscular reversal agents if possible.

5) **Postoperative considerations**

a) **Disposition**



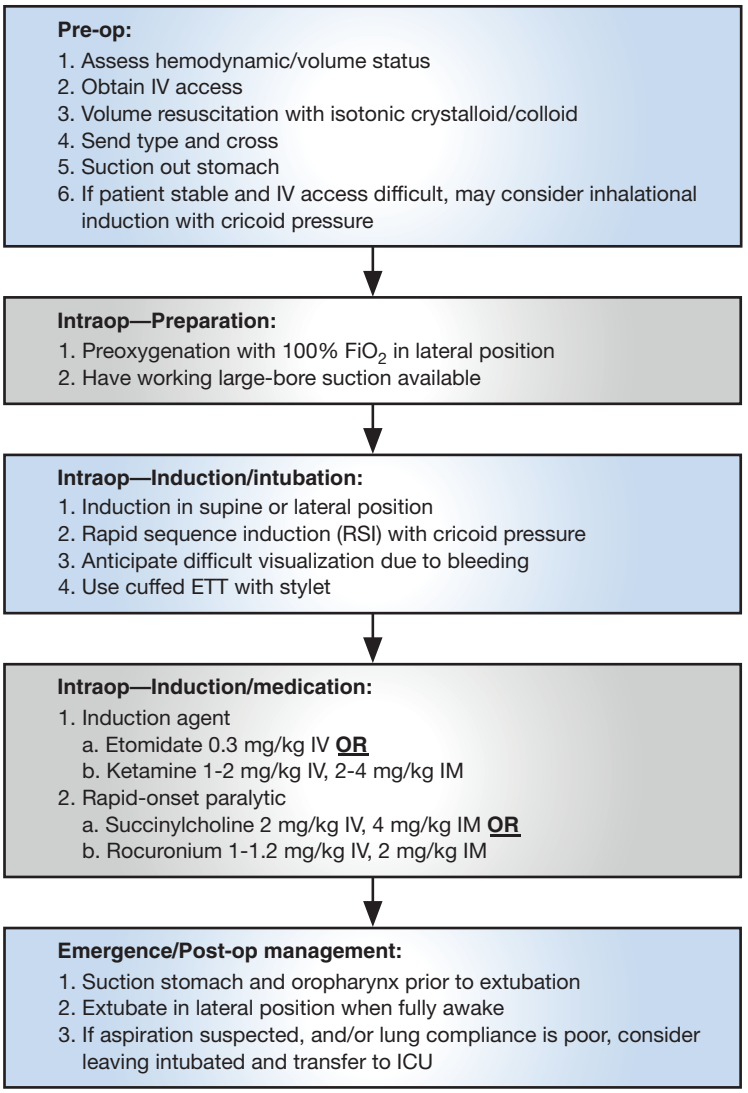
Children with severe OSA and other criteria must be admitted for observation postoperatively.

i) The need for postoperative admission and monitoring depends on the severity of preoperative illness.
ii) For OSA patients, even though the offending anatomy has been removed, the risk of obstruction may still remain and, in some instances, be exacerbated immediately postoperatively.
iii) Children with the following clinical features of OSA may require overnight admission to pediatric ward or PICU for postoperative observation.
(1) Age <3 years
(2) Severe OSA
(3) Morbid obesity
(4) Severe desaturation or obstruction on induction or postoperatively
(5) Coexisting neuromuscular, cardiac, pulmonary, or other systemic illness
(6) Pulmonary hypertension
(7) Low weight/failure to thrive

b) Other interventions that may help to avoid reintubation postoperatively include CPAP, BIPAP, and high-flow nasal cannula.
c) Though T&A can be curative for as high as 87% of OSA patients, it may not resolve OSA in all children.
i) Residual OSA has been reported to be as high as 30%.[16]
ii) Risk factors for persistent OSA are obesity and severe OSA, especially those with high AHI.

6) Posttonsillectomy bleeding is a surgical emergency (**Fig. 90-1**).
a) Approximately 75% of bleeding occurs within 6 hours of surgery with the remainder occurring 5-10 days after surgery when the scar over the surgical site retracts.[17]
b) Assess severity of hypovolemia and anemia, and initiate aggressive volume resuscitation with isotonic crystalloid and/or colloid solution before the operation.
c) Type and crossmatch (T&C) for RBC should be obtained and blood sent to the operating room.

FIGURE 90-1 Management of Posttosillectomy Bleeding



d) Patients will have likely swallowed a significant amount of blood and must be considered to have full stomachs and aspiration risk.
e) Ensure adequate large-bore suction is available.
f) **Induction**
   i) Preoxygenate in the lateral position so blood can drain out of the oral cavity.
   ii) Perform rapid sequence induction with cricoid pressure (in lateral or supine position) using reduced induction dosages of rapid-onset agents.



For posttonsillectomy bleeding, assume that patient is hypovolemic and anemic and will have a full stomach and difficult intubation.

(1) Etomidate 0.3 mg/kg IV
(2) Ketamine 1-2 mg/kg IV or 2-4 mg/kg IM
(3) Titrated doses of propofol 0.5-1 mg/kg IV

iii) Rapid-onset muscle relaxation is indicated.
(1) Succinylcholine 2 mg/kg IV or 4 mg/kg IM
(2) Rocuronium 1.2 mg/kg IV or 2 mg/kg IM

iv) Anticholinergic agents may be given simultaneously to preempt bradycardia and act as an antisialogogue.
(1) Atropine 0.02 mg/kg IV
(2) Glycopyrrolate 0.01 mg/kg IV

v) Anticipate difficulty visualizing the airway due to bleeding and full stomach.
(1) Cuffed ETT should be used to minimize aspiration risk and to avoid the need to reintubate for inappropriate size.
(2) Stylet recommended because of potentially difficult visualization.

g) **Maintenance**
i) Surgery is not as painful as initial T&A repair, so analgesic needs will likely be less.
ii) Anesthetic with volatile or TIVA supplemented with opiates as tolerated by patient hemodynamics.

h) **Emergence**
i) Request that surgeon suction the stomach and oropharynx.
ii) Fully reverse muscle relaxation.
iii) Turn patient to side for emergence.
iv) Extubate only when the child is awake with full return of airway reflexes.

i) **Postoperative management/disposition**
i) Follow up Hb/Hct.
ii) If aspiration is suspected and/or lung compliance is poor, consider leaving patient intubated and transfer to ICU.

CHAPTER SUMMARY FOR TONSILLECTOMY AND ADENOIDECTOMY

General Considerations	Indications are now limited to recurrent infection or OSA.
Preoperative Considerations	Evaluation should focus on OSA symptoms including CO_2 retention, hypoxemia, and pulmonary hypertension. Patients with severe OSA are at risk for post-op complications. Consider risk of respiratory depression with preoperative anxiolytic medications.
Intraoperative Considerations	Spontaneous breathing allows titration of narcotics to respiratory rate and $ETCO_2$. Aggressive PONV prophylaxis is recommended. Consider extubation and recovery in the lateral position. Airway fire is a sentinel event and awareness is key to prevention.
Postoperative Considerations	Severe OSA symptoms may require postoperative admission for respiratory observation.
Postoperative Bleeding	Life-threatening emergency. Patients may be severely hypovolemic and are at high risk for aspiration. Anticipate difficult airway and have adequate help available.

OSA, obstructive sleep apnea; PONV, postoperative nausea and vomiting; $ETCO_2$, end-tidal carbon dioxide.

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91 Cleft Lip/Cleft Palate

Nicholette Kasman

Cleft lip and palate are the most common craniofacial disorders. The occurrence of cleft lip or palate is 1:600 births worldwide.[1] Thirty-eight percent of the time, they are associated with additional congenital anomalies.[2] Cleft lip alone does not usually cause airway problems. Cleft palate alone is generally not problematic, but it is often associated with congenital anomalies that can make airway management difficult.[3]

1) **Surgical treatment**
   a) Usually staged, the first procedure done for repair of cleft palate closes the anterior part of the hard palate—the alveolar defect; corrects the alar base; and provides symmetrical closure of the lip.
      i) Usually occurs around 2-3 months of age.
      ii) This allows the baby to feed and demonstrate normal facial expressions.[1,4]
   b) The second procedure involves closure of the posterior hard palate and the soft palate before the development of speech.
      i) Usually occurs around 6-12 months of age.
      ii) This allows the infant to develop speech.[1,4]
   c) Surgery for lip, nose, and palatal revisions as well as alveolar bone grafts occur in early childhood.[1]
   d) Rhinoplasties and maxillary osteotomies done to complete the repair occur at 17-20 years of age.[1]



Cleft palate can result from the tongue interfering with the closure of the palatal shelves *in utero*; thus, any syndrome that has macroglossia or hypoplasia of the maxilla or mandible may be associated with cleft palate.[3]

2) **Anesthetic management**
   a) **Preoperative considerations**
      i) The most important part of the preoperative workup is evaluation of the airway. In the case of isolated cleft lip or palate, a large prospective study demonstrated the following[5]:
         (1) Failed intubation incidence of only 1%.
         (2) No occurrences of "cannot intubate and cannot mask ventilate."
         (3) Of the patients who were classified as difficult laryngoscopic views (initially grade III or IV), 45% had bilateral cleft lip, 35% had isolated cleft palate with retrognathia, and only 3% had unilateral cleft lip.
         (4) The characteristics most associated with difficult laryngoscopy were:
            (a) Extensive clefts
            (b) Retrognathia
            (c) Age <6 months



The presence of a cleft lip or a palate does not necessarily indicate that the patient will have a difficult airway.

      ii) Patients with associated syndromes may present greater challenges:
         (1) Pierre Robin sequence: Mandibular hypoplasia as well as downward displacement and retraction of the tongue.[1,3,5]

(2) Klippel-Feil syndrome: Severe limitation of flexion and extension of the neck as a result of the fusion of cervical vertebrae, spinal canal stenosis, and scoliosis.[1,3]

(3) Down syndrome: Narrowed nasopharynx and a relatively large, protuberant tongue in addition to the cleft lip and the palate.[1,3]

(4) Treacher Collins syndrome or mandibulofacial dysostosis: Maxillary, zygomatic, and mandibular hypoplasia.[1,3]



Patients with associated craniofacial syndromes may present greater challenges in part from the presence of retrognathia. There are over 300 syndromes associated with cleft lip and palate, but the most common are Pierre Robin sequence, Down syndrome, Klippel-Feil, and Treacher Collins syndrome.[1]

b) **Monitors/lines/positioning**

i) Standard ASA monitors and a single IV are generally adequate.

ii) The patient is usually positioned at 180 degrees from the anesthesiologist.

iii) Care should be taken in securing the endotracheal tube.[1,2]

c) **Intraoperative management**

i) Induction

(1) Premedication with oral midazolam decreases both patient and parental anxieties.

(2) Induction of anesthesia via face mask is usually uncomplicated. If the patient's airway is concerning, a preoperatively placed IV and induction with ketamine may be a safer approach.

(3) The tongue may fall back into the cleft, obstructing the nasal passage. This can be corrected by placement of an oral airway.[3–5]

(4) Care should be taken during laryngoscopy. Above the hard palate are the sinuses and nasal conchae. The laryngoscope blade entering the cleft and sinuses is not inherently harmful to the child, but it is important to prevent damage to the tissues that are to be repaired.

(5) Use of an oral RAE tube facilitates surgical access. Use of cuffed ETT minimizes risk of blood or secretions going into the lungs.[1]

(6) Throat packs usually impinge on the surgical field and are not generally required for cleft palate repair; however, communication between the surgeon and anesthesiologist regarding placement of throat packs must be clear.



The development of the ear is associated with that of the mandible, so patients with microtia can also be challenging.[6]



Communicate with the surgeon about placement and removal of throat packs.

ii) Maintenance

(1) Maintenance with inhaled agents or TIVA is appropriate.

(2) Muscle relaxation may be required to expedite induction or facilitate intubation but is usually not necessary during the procedure.

(3) Consider a dexmedetomidine infusion to help with emergence agitation. A bolus dose of dexmedetomidine 1 mcg/kg followed by an infusion of 0.5 mcg/kg/h helps with emergence agitation and decreases fentanyl and vapor requirement by 43% and 30%, respectively.[7]

iii) Special anesthetic considerations

(1) Vigilance should be maintained for obstruction or kinking of the ETT by surgical retractors.

(2) Allowing the patient to breathe spontaneously after induction and during the surgery allows titration of narcotics to achieve adequate analgesia prior to emergence.
  (a) Giving narcotics prior to spontaneous respirations risks apnea and delayed emergence.
  (b) Titrate opioids to keep the respiratory rate in the low normal range and the $ETCO_2$ in the high normal range.
  (c) Waiting until emergence to give narcotics risks inadequate analgesia.
(3) The incidence of postoperative emergence agitation in cleft palate surgery is significantly higher than in other types of surgery.
  (a) A dose of dexmedetomidine 0.3-0.5 mcg/kg IV prior to emergence can prevent the occurrence of emergence agitation in the postoperative period.[8]
  (b) If dexmedetomidine is unavailable, clonidine 1-2 mcg/kg IV can be given to decrease emergence delirium.[9]

iv) Emergence
(1) Prior to emergence, it is important to ensure all throat packs are removed.
(2) Ask the surgeon to place an orogastric tube to suction out the stomach under direct visualization to avoid disturbing suture lines by blind suctioning.
(3) A nasopharyngeal airway can be inserted by the surgeon prior to extubation to permit suctioning of the airway without damaging the palatal repair and to provide a patent airway.[1]
(4) An emergence without coughing or bucking is ideal to avoid bleeding and laryngospasm. Techniques that can be helpful include:
  (a) Deep extubation
  (b) Ensuring adequate analgesia
  (c) Lidocaine 1 mg/kg IV prior to extubation
(5) Some practitioners prefer to extubate the patient awake to minimize risk of laryngospasm and airway obstruction.
(6) Turning the patient on the side prior to extubation allows blood to pool out of the airway and may decrease the risk of laryngospasm.


Turning the patient on the side for extubation allows blood to pool out of the airway.

d) **Postoperative management**

i) Analgesia
(1) Postoperative pain is usually managed with opioids and acetaminophen; however, the residual respiratory depressant effect of opioids is a major concern as blood, edema, and unfamiliar suture lines can make postoperative laryngoscopy and emergency reintubation difficult.
(2) The administration of rectal acetaminophen is less effective than IV acetaminophen in the treatment of pain in children undergoing cleft palate surgery.[10,11]
(3) Placement of bilateral infraorbital nerve blocks with 0.25% bupivacaine has been shown to improve postoperative pain control as well as parental satisfaction.[12]

ii) Disposition
(1) Patients are at risk for acute upper airway obstruction due to upper airway narrowing, edema, and residual anesthetic and must be monitored in an appropriate setting.[1]
(2) Ongoing evaluation for signs of postoperative airway obstruction must continue even at home for the first 48 hours.[1]

CHAPTER SUMMARY FOR CLEFT LIP/CLEFT PALATE

General Considerations	The presence of a cleft lip or a palate is not an indication that the child will have a difficult airway, but associated anomalies such as retrognathia, mandibular hypoplasia, decreased neck flexion, or macroglossia are problematic.
Preoperative Considerations	The characteristics most associated with difficult laryngoscopy include extensive clefts, retrognathia, and age <6 months.
Intraoperative Considerations	Use of a cuffed oral RAE tube facilitates surgical access and minimizes aspiration risk of blood or secretions. The placement and the removal of throat packs need to be clearly communicated between the surgeon and anesthesiologist. Ask the surgeon to suction the stomach and the oropharynx under direct visualization to avoid disturbing suture lines by blind suctioning. A nasopharyngeal airway can be inserted by the surgeon prior to extubation to permit suctioning of the airway without damaging the palatal repair and to provide a patent airway.
Postoperative Considerations	Postoperatively, patients are at risk for acute upper airway obstruction due to upper airway narrowing, edema, and residual anesthetic and must be monitored in an appropriate setting. Ongoing evaluation for signs of postoperative airway obstruction must continue even at home for the first 48 h.

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92 Foreign Body Aspiration

Naiyi Sun

Foreign body aspiration occurs most commonly in toddlers from 1 to 3 years of age.[1] The most commonly retrieved objects are peanuts, which are problematic as they release oil, causing a chemical inflammation.[1] Nonorganic objects that are particularly challenging and require urgent removal include needles, batteries, and magnets. Removal of all aspirated items is essential due to risk of atelectasis, bronchiectasis, chronic pneumonia, and inflammatory granulation tissue formation.[2]

1) **Pathophysiology**
   a) Objects are found in the right bronchus more frequently than in the left.
   b) Less than 5% of objects are found in the trachea and hypopharynx. Two-thirds of the objects lodge in the mainstem bronchi, and the remainder are found in the distal bronchi.[3]



In patients who present with choking or refractory wheezing, there should be a high index of suspicion for aspirated foreign body.

2) **Signs/symptoms/clinical findings**
   a) Most airway foreign bodies do not cause acute respiratory distress and often have been *in situ* for days prior to seeking medical treatment. Occasionally, acute life-threatening airway obstruction may occur following an aspiration event.
   b) Presenting signs and symptoms vary depending on the location and the size of the object. Choking and wheezing following a witnessed aspiration event are the most common presentation. In patients who present with refractory wheezing, there should be a high index of suspicion for aspirated foreign body.
   c) Objects lodged in the larynx and trachea are associated with high mortality rates and are considered an airway emergency. When objects are lodged in the mainstem bronchus or more distal airway, signs and symptoms include cough, stridor, dyspnea, wheezing, unilateral decreased breath sounds, and occasionally cyanosis.
3) **Diagnosis**
   a) Chest x-ray (CXR)
      i) Often normal because most aspirated foreign bodies are organic and radiolucent.[2]
      ii) CXRs may demonstrate a localized area of hyperinflation, atelectasis, or pulmonary infiltrate. The foreign body causing obstruction to the bronchus may create a unidirectional ball-valve phenomenon leading to distal hyperinflation from air trapping. With complete bronchial obstruction by a large foreign body, distal atelectasis or collapse may occur.
      iii) Pneumothorax and pneumomediastinum are less common findings on CXR.[4]
   b) Any object causing airway obstruction with acute respiratory distress requires emergent removal. Imaging studies are usually unnecessary and should not delay surgical treatment.



Button or cell batteries lodged in the esophagus can cause severe, life threatening burns within 2 hours of ingestion, so they must be removed immediately.

4) **Treatment**
   a) Suspected foreign body aspiration requires prompt diagnostic and therapeutic rigid bronchoscopy. Removal of foreign objects with a rigid bronchoscopy has a success rate of 95%-98%.[3]

b) Button batteries (or button cell) in the esophagus can cause severe, life-threatening burns within 2 hours of ingestion and must be removed immediately.[5]

5) **Anesthetic management**

a) **Preoperative management**

i) Physical exam should focus on the degree of airway obstruction and location of the foreign body.

ii) NPO time should be established to assess the risk of aspiration. The patient's physical condition determines the timing of surgery. In acute cases, the danger of delayed removal of foreign body outweighs the risk of a full stomach.

iii) Consider administering atropine or glycopyrrolate prior to induction to dry airway secretions. This may also have the added benefit of maintaining the heart rate.

iv) Anxiolytics such as midazolam should be given with caution as it may exacerbate existing upper airway obstruction.



Anxiolytics such as midazolam should be given with caution as it may exacerbate upper airway obstruction.

b) **Monitoring**

i) A preoperative IV is useful for hydration and medication administration prior to induction. However, in an emergency or with a distressed child, IV access after inhalation induction is acceptable.

ii) A precordial stethoscope in addition to standard ASA monitors can be useful in detecting any changes in ventilation intraoperatively. Invasive monitoring is not necessary for most cases.

c) **Intraoperative management**

i) Induction

(1) Gentle inhalation induction with 100% O_2 and sevoflurane is recommended as it preserves spontaneous ventilation and may decrease the possibility of moving the foreign object more distally.

(2) Nitrous oxide should be avoided as it reduces the FiO_2 and may increase significant gas volume and pressure if significant air trapping exists.

(3) If an IV is *in situ*, careful IV induction while preserving spontaneous ventilation can be performed.

(4) Following induction of general anesthesia, the surgeon may request that the OR table be turned 90 degrees.

ii) Maintenance

(1) There are two approaches to the anesthetic management for removal of aspirated foreign bodies: spontaneous ventilation and controlled ventilation.

(a) A controlled ventilation technique relies on intermittent positive pressure breaths between apneic periods when the surgeon instruments the airway. Advantages include rapid control of the airway and no patient movement. A significant disadvantage is that positive pressure ventilation can unintentionally push the object further into the airway.

(b) Spontaneous ventilation allows for uninterrupted ventilation; however, there is higher risk of patient movement, coughing, and laryngospasm.[6]

(2) In most cases, the safest technique is to preserve spontaneous ventilation at least until the nature and the location of the foreign body have been identified by bronchoscopy.

(3) A total IV anesthetic technique using propofol plus remifentanil infusion for maintenance is preferable because it allows a steady level of anesthesia that is independent of ventilation. This can be supplemented by ketamine or



In most cases, the safest anesthetic technique for foreign body removal involves maintenance of spontaneous ventilation.

dexmedetomidine, which increases depth of anesthesia while preserving spontaneous respiration.[7] Children younger than 3 years of age can tolerate higher doses of remifentanil and still maintain spontaneous ventilation in comparison with older children.[4]

iii) Special anesthetic considerations

(1) The anesthesiologist needs to achieve sufficient depth of anesthesia during the procedure to obliterate airway reflexes and prevent patient movement and coughing during instrumentation while maintaining spontaneous ventilation and hemodynamic stability. Once the rigid bronchoscope is inserted, it is essential to avoid coughing and bucking due to risk of airway trauma and rupture.

(2) During bronchoscopy, the anesthesiologist needs to constantly monitor for chest excursion, breath sounds, and SpO_2. Constant and close communication between surgical and anesthesia personnel regarding the child's airway status and oxygenation is crucial.



Constant and close communication between surgical and anesthesia personnel is crucial.

iv) Special surgical considerations

(1) Once adequate anesthesia depth is achieved, topical lidocaine 1%-2% can be sprayed to laryngeal structures and tracheal mucosa prior to bronchoscopy. This is essential for suppression of airway reflexes and prevention of coughing and bronchospasm.

(2) Once the ventilating bronchoscope is in the subglottis, the anesthesia circuit can be connected to the side port of the scope to allow supplemental O_2 delivery and positive pressure ventilation if necessary.

(3) When a large foreign body is removed, the forceps and the bronchoscope must be removed together through immobile vocal cords. Administration of a small dose of short-acting nondepolarizing muscle relaxant may be necessary to permit safe removal of the apparatus.

(4) A partial obstruction may become a complete tracheal obstruction during removal of the foreign body. If the object cannot be immediately removed, it may be necessary to push the object down into a mainstem bronchus to allow ventilation of at least one of the lungs.



A partial obstruction may become a complete tracheal obstruction during removal of the foreign body. If the object cannot be immediately removed, it may be necessary to push the object down into a mainstem bronchus to allow ventilation of at least one of the lungs.

(5) After the foreign body is safely retrieved, a follow-up bronchoscopy is immediately performed to look for any residual foreign bodies and to rule out airway injury.

(6) Rarely, the foreign body is so disseminated that it cannot be easily removed or has caused life-threatening impairment of oxygenation and/or ventilation such that thoracotomy, bronchotomy, or cardiopulmonary bypass may be necessary.

d) **Intraoperative complications**

i) Hypoxia and hypercarbia may occur due to inadequate ventilation. Short periods of hypercarbia may be well tolerated. Hypoxia should be treated with removal of the bronchoscope and effective mask ventilation.

ii) Bronchospasm during the procedure should be treated with increasing depth of anesthesia, inhaled albuterol, or IV bronchodilators.

iii) Prophylactic steroid therapy with dexamethasone 0.5 mg/kg IV to a maximum dose of 20 mg is useful in preventing upper airway edema and secondary stridor from airway manipulation.[8]

iv) Other major complications include pneumothorax, pneumomediastinum, tracheal or bronchial laceration, and cardiac arrest.[4,9]

v) After removal of foreign body, spontaneous ventilation assisted by mask ventilation can be continued in uncomplicated cases until adequate protective reflexes have returned. In patients with marginal airway, pulmonary compromise, or intraoperative complications, postoperative ventilation may be indicated.

e) **Postoperative management**

i) The patient should be placed in the head-up position. Respiratory status should be closely monitored because of the possibility of airway obstruction secondary to edema.

ii) If stridor develops, administer humidified O_2 and racemic epinephrine inhalation (0.5 mL of 2.25% solution nebulized). Heliox (helium and oxygen mixture) may be considered if the degree of stridor is severe and the patient does not require more than 40% FiO_2.[3]

iii) Postobstructive pulmonary edema can develop in children in the recovery room. Treatment is supportive with controlled ventilation and positive end-expiratory pressure.

CHAPTER SUMMARY FOR FOREIGN BODY ASPIRATION

Diagnosis	Signs and symptoms include cough, stridor, dyspnea, wheezing, unilateral decreased breath sounds, and cyanosis. Chest radiographs are commonly normal.
Treatment	Prompt diagnostic and therapeutic rigid bronchoscopy. Button or cell batteries must be removed from the esophagus within 2 h of ingestion.
Preoperative Considerations	Consider anticholinergics to decrease secretions. Anxiolytics should be given with caution. Preinduction IV is useful but not required.
Intraoperative Considerations	Gentle inhalation induction with 100% O_2 and sevoflurane. N_2O should be avoided. Total IV anesthetic technique for maintenance with propofol plus remifentanil infusion, which can be supplemented with ketamine and/or dexmedetomidine. Preserve spontaneous ventilation until the nature and the location of the foreign body have been identified.
Postoperative Considerations	Recover in head-up position. Watch for stridor and postobstructive pulmonary edema.

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93 Epiglottitis

Naiyi Sun

Due to the small size of the airway in infants and small children, inflammation from epiglottitis can result in significant airway compromise that would otherwise not be seen in older children and adults. The management of these patients presents a unique challenge to any anesthesiologist.

1) **Pathophysiology**
   a) Epiglottitis is a life-threatening infection that causes severe edema due to inflammation of the epiglottis and supraglottic structures. The swelling can progress rapidly and result in complete airway obstruction.
   b) It can affect children in all age groups, but most commonly occurs in children between 2 and 6 years of age.
   c) Historically, the most frequent causative agent was *Haemophilus influenzae* type B (Hib).
      i) With the introduction of the Hib conjugate vaccine, epiglottitis secondary to group A *Streptococcus*, *Streptococcus pneumonia*, *Klebsiella pneumoniae*, and *Staphylococcus aureus* is becoming more prevalent.[1]



The typical presentation of epiglottitis is an acute onset with toxic appearance, high fever, respiratory distress, drooling, and sitting in tripod position with slow respiratory rate to minimize turbulence.

2) **Signs/symptoms/clinical findings**
   a) Epiglottitis is characterized by an acute and rapid onset of respiratory distress.
   b) **Symptoms can include severe sore throat, dysphagia, dysphonia, and dyspnea.**
   c) **Signs**
      i) The patient may have a toxic appearance, high fever, tachycardia, tachypnea, inspiratory stridor, and, in extreme cases, hypoxia and cyanosis.
      ii) Drooling is commonly seen due to difficulty and pain with swallowing.
      iii) The child often assumes the tripod position of sitting forward with open mouth and tongue out to try to decrease work of breathing.
   d) Respiratory pattern is often slow and quiet to minimize turbulence.



Acute epiglottitis is a clinical diagnosis. Do not delay securing the airway to obtain radiologic studies.

3) **Diagnosis**
   a) Acute epiglottitis is a clinical diagnosis. Examination of the mouth and the oropharynx should be avoided until trained experts are ready to secure the airway with an endotracheal tube (ETT) or more extreme interventions.
   b) Imaging studies are usually unnecessary and should not delay definitive airway management. If clinical presentation is inconclusive, then lateral radiograph of the neck can be useful. The thumbprint sign representing a swollen epiglottis is classically associated with epiglottitis.

4) **Anesthetic management**

a) **Preoperative considerations**

i) Manipulation of the mouth and the oropharynx should be avoided to minimize agitation of the child as it may precipitate an acute airway obstruction. For the same reason, venipuncture and painful injections should be avoided prior to securing the airway.

ii) Once the diagnosis of epiglottitis is made or strongly suspected, arrangements should be made immediately to secure the airway in the controlled setting of the operating room.

iii) The child should be transported to the operating room by personnel skilled in pediatric airway management with appropriate airway equipment.

iv) The child should be kept calm sitting on a caretaker's lap breathing spontaneously with continuous SpO_2. Supplemental O_2 should be given if tolerated by the patient. The child should remain sitting at all times and never forced into a supine position.



Manipulation of the airway should be avoided as it may precipitate an acute airway obstruction. The airway should be secured in the operating room with personnel experienced in emergency airway management.

b) **Monitors/lines**

i) Standard ASA monitors

ii) Single peripheral IV

iii) Additional monitors and invasive lines as needed after patient is anesthetized and airway secured.

c) **Intraoperative management**

i) Induction/airway

(1) A surgeon skilled in surgical airway management and rigid bronchoscopy should be present prior to induction with tracheostomy tray and bronchoscopy equipment in the operating room.

(2) Anesthesia induction should be performed by mask with sevoflurane and 100% O_2 with the child sitting up in his position of greatest comfort. Inhalation induction may be prolonged due to airway obstruction.

(3) When adequate depth of anesthesia is accomplished, the patient may be transitioned to the supine position and peripheral IV access secured. Continuous positive pressure by mask may be needed to overcome upper airway obstruction.

(4) If an IV is *in situ*, careful IV induction while preserving spontaneous ventilation can be considered.

(5) Lidocaine 1 mg/kg IV may be given on induction to minimize the risk of coughing and laryngospasm prior to laryngoscopy.[2]



If the laryngeal structures are difficult to visualize, expiratory gas bubbles during spontaneous ventilation or bubbles induced by gentle pressure on the child's chest may reveal the glottic opening.



Due to airway swelling, the endotracheal tube used should be 0.5-1 mm smaller in diameter than would be appropriate for the age and size of the patient.

ii) Special anesthetic considerations

(1) It is crucial to achieve sufficient depth of anesthesia while maintaining spontaneous ventilation prior to laryngoscopy due to the possibility of difficult intubation. The laryngeal structures may be unrecognizable due to swelling, and the glottic opening may be small or even occluded.

(2) If the laryngeal structures are difficult to visualize, expiratory gas bubbles during spontaneous ventilation or bubbles induced by gentle pressure on the child's chest may reveal the glottic opening.

(3) Due to airway swelling, use ETT 0.5-1.0 mm smaller in diameter than would be appropriate for the age and size of the patient. The ETT should be styletted to improve the ability to direct the ETT and advance through the swollen glottic opening.
(4) If the anesthesiologist is unable to intubate the trachea, then a rigid bronchoscopy should be performed. If both maneuvers are unsuccessful, then consider tracheostomy or CPB/ECMO in younger children depending on surgical expertise and facilities available.

5) **Postoperative management**
   a) Once the airway is secured, IV antibiotics should be started immediately. Empiric therapy with a third-generation cephalosporin such as ceftriaxone can be used as first line.[1]
   b) Supportive treatment includes IV hydration, sedation, and acetaminophen.
   c) Pulmonary edema can develop in 7% of children with severe epiglottitis.[3] Treatment is supportively with controlled ventilation, positive end-expiratory pressure (PEEP), and diuretics.
   d) Epiglottic abscess has been found to occur in up to 24% of patients.[4] These patients often required drainage of the abscess in addition to IV antibiotics.
   e) Flexible laryngoscopy under propofol sedation can be performed to visualize the epiglottis before extubation. The child is usually ready for extubation in 24-48 hours.[4]

CHAPTER SUMMARY FOR EPIGLOTTITIS

General Considerations	Life-threatening infection causing severe edema of the supraglottic structures. Acute onset of severe sore throat, high fever, dysphagia, dysphonia, and dyspnea.
Preoperative Considerations	It is crucial to minimize agitation of the child as it may precipitate acute airway obstruction by avoiding repeated exams and IV placement. The child should be transported to the operating room by personnel skilled in pediatric airway management with appropriate airway equipment.
Intraoperative Considerations	Mask induction with 100% O_2 and sevoflurane. Keep patient spontaneous breathing during induction. Secure critical airway with styletted ETT ½-1 size smaller. If the laryngeal structures are difficult to visualize, expiratory gas bubbles during spontaneous ventilation or bubbles induced by gentle pressure on the child's chest may reveal the glottic opening.
Postoperative Considerations	IV antibiotics, hydration, and sedation.

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94 Congenital Syndromes and Anesthetic Implications

Daniel Sisti

This chapter contains brief summaries of common and uncommon pediatric syndromes and briefly outlines considerations for safe pediatric anesthesia practice. It is meant to be a resource to the anesthesiologist who may encounter patients with a recognized syndrome with potential implications affecting anesthetic management. For each entry, a short description of the syndrome is given, along with mechanism of disease (if known), and a description of pertinent characteristics that may affect perioperative care. While the diseases and perioperative issues described here are by no means exhaustive, it is hoped that this chapter can serve as a quick and handy reference.

1) **Congenital syndromes**
   a) **A syndrome is defined as:**
      i) The concurrence of specific clusters of clinical signs and symptoms
      ii) The phenotypic expression of a genetic disorder
   b) There are over 10 000 medical syndromes recorded.
   c) Although many syndromes are becoming better understood through research and genetic characterization, anesthesiologists may yet encounter unreported challenges during anesthesia.
   d) When a particular syndrome identity is unclear, it is prudent to be prepared to encounter all possible associated disorders.
2) **Achondroplasia**
   a) **Essential characteristics**
      i) Mutations in fibroblast growth factor receptor 3 gene (FGFR3)
      ii) Rhizomelic shortening, that is, short proximal bone segments
      iii) Large head, potential foramen magnum stenosis, and narrowing of cervical vertebra
      iv) Pear-shaped chest
      v) Cupping of ribs and long bones
   b) **Anesthetic implications**
      i) High prevalence of obstructive sleep apnea
         (1) Central apnea may also be present due to foramen magnum and cervical spinal stenosis.
      ii) Large tongue and midface hypoplasia may make mask ventilation difficult.
      iii) May need smaller endotracheal tube than predicted for age
      iv) Avoid hyperextension because of risk of spinal cord compromise.[1]



Children with achondroplasia have a high prevalence of obstructive (and possibly central) sleep apnea.

3) **Alagille syndrome**
   a) **Essential characteristics**
      i) One of the most common causes of congenital chronic liver disease
      ii) Variable degree of bile hypoplasia and cholestasis
      iii) Neonatal presentation with cholestasis, dysmorphic facies, jaundice, and hepatosplenomegaly

iv) Multiorgan involvement: congenital cardiac disease (97%), vertebral anomalies (51%), and kidney malformations (40%)[2]

b) **Anesthetic implications**

i) Significant hepatosplenomegaly may increase the risk of regurgitation.

ii) Rule out significant cardiac defects. Peripheral pulmonary stenosis is the most common abnormality, but patients can also have a variety of other defects including pulmonic or aortic valve stenosis, septal defects, and tetralogy of Fallot.

iii) Patients may have liver dysfunction and/or vitamin K deficiency.

iv) Deficiencies in fat-soluble vitamins can have serious effects: coagulopathy (vitamin K), osteoporosis (vitamin D), neuropathy and myopathy (vitamin E), and eye dryness with increased risk of corneal abrasions (vitamin A).

v) Use caution with drugs that undergo hepatic metabolism.

vi) If using volatile anesthesia for maintenance, isoflurane may have the least effect on hepatic blood flow. An IV anesthetic may be the safest approach.

4) **Alport syndrome**

a) **Essential characteristics**

i) A rare cause of chronic kidney disease, affecting mostly males

ii) Most commonly an X-linked defect in the genes encoding collagen type IV; less often an autosomal dominant and autosomal recessive Alport syndrome

iii) Progressive nephropathy leading to severe hypertension and end-stage renal disease

iv) Concomitant progressive sensorineural deafness and ocular abnormalities

b) **Anesthetic implications**

i) Patients may present for kidney transplantation and ocular surgery.

ii) Complications may include uncontrolled hypertension, including cerebral or myocardial infarction, and congestive heart failure.

iii) Check electrolyte levels and adjust renally cleared drugs as necessary.

iv) Some reports of AV conduction abnormalities; consider 5-lead EKG monitoring perioperatively.[3]

5) **Angelman syndrome**

a) **Essential characteristics**

i) Developmental delay, ataxia, seizures, paroxysmal laughter

ii) Craniofacial anomalies including microcephaly and high-arched palate

iii) Possible vagal hypertonia

iv) Most cases caused by genetic defect in maternal 15q chromosome, which includes a subunit of the $GABA_A$ receptor

b) **Anesthetic implications**

i) Patients may be uncooperative.

ii) Potentially difficult airway

iii) Consider prophylactic anticholinergic in light of case reports of bradycardia and asystole with or shortly after induction.[4]



Children with Angelman syndrome may have increased vagal tone and are prone to bradycardia.

6) **Beckwith-Wiedemann syndrome**

a) **Essential characteristics**

i) Overgrowth syndrome with a range of anomalies that may include macroglossia, macrosomia, omphalocele, umbilical hernia, cardiac anomalies, neonatal hypoglycemia, and a predisposition to embryonal tumors (eg, Wilms tumor).

b) **Anesthetic implications**

i) Newborns with Beckwith-Wiedemann syndrome should have a thorough workup including echocardiogram and renal ultrasound to evaluate for associated anomalies and regular glucose checks.

ii) Expect a difficult airway due primarily to macroglossia.
iii) Maintenance of spontaneous respiration until the airway is secured is often a safe strategy, but airway obstruction may occur with deepening anesthesia due to the tongue falling back into the oropharynx.
iv) Consider placing nasopharyngeal airway prior to extubation to prevent postanesthesia obstruction.



Children with Beckwith-Wiedemann syndrome may have a difficult airway due to macroglossia.

7) **Brugada syndrome**
   a) **Essential characteristics**
      i) Autosomal dominant genetic disorder with characteristic EKG findings and increased risk of ventricular tachyarrhythmias (VT) and sudden cardiac arrest
         (1) EKG leads V_1 and V_2 show ST elevation and right bundle-branch block.
      ii) Several drugs used routinely in anesthesia may increase risk of VT in Brugada syndrome, notably propofol, bupivacaine, ketamine, and cholinergic agents (see brugadadrugs.org).
      iii) Increases in vagal tone, bradycardia, and elevated core temperature may precipitate VT in these patients.[5]
   b) **Anesthetic implications**
      i) Standard propofol induction doses are safe.[6]
      ii) Place defibrillator pads prior to induction of anesthesia.
      iii) Hyperthermia can induce Brugada syndrome VT, so maintain normothermia.
8) **Chromosome 22q11 deletion syndrome** *(CATCH 22 syndrome, Conotruncal face syndrome, DiGeorge syndrome, Shprintzen syndrome, Velocardiofacial syndrome)*
   a) **Essential characteristics**
      i) A constellation of syndromes caused by deletions of chromosome 22q11
      ii) Although there is a large range of phenotypic expression in these disorders, a common description is "CATCH 22": **C**ardiac defects, **A**bnormal facies, **T**hymic hypoplasia/aplasia, **C**left palate, **H**ypocalcemia
   b) **Anesthetic implications**
      i) Commonly associated with Pierre Robin sequence, so strong possibility of difficult airway
      ii) Cardiac defects are very common (tetralogy of Fallot, interrupted aortic arch, truncus arteriosus, hypoplastic left heart syndrome), affecting ~80% of patients.
      iii) Patients with thymic hypoplasia or aplasia have T-cell immune deficiency and require irradiated blood to prevent transfusion-associated graft vs host disease.
      iv) Hypocalcemia in neonates can present as seizures. Patients will commonly require serum calcium monitoring.[7]



Children with chromosome 22q11 deletion syndrome have increased risk of difficult airway and congenital cardiac disease.

9) **Craniosynostosis syndromes** *(Apert syndrome [acrocephalosyndactyly type I], Carpenter syndrome [acrocephalosyndactyly type II], Crouzon syndrome [craniofacial dysostosis type I], Pfeiffer syndrome [acrocephalosyndactyly type V], Saethre-Chotzen syndrome [acrocephalosyndactyly type III])*
   a) **Essential characteristics**
      i) Rare autosomal dominant or sporadic disorders usually caused by mutations in genes that encode fibroblast growth factors or a transcription factor known as TWIST[8]
      ii) Common features among these syndromes include midface hypoplasia and craniosynostosis involving multiple sutures.
      iii) Other common features of a subset of these syndromes include limb abnormalities such as syndactyly or polydactyly. High incidence of C5-C6 cervical fusion

iv) Features unique to Apert syndrome:
(1) High incidence of C5-C6 cervical fusion
(2) May have intellectual disability[9]
v) Features unique to Carpenter syndrome:
(1) Preaxial polydactyly, syndactyly, and clinodactyly
(2) Approximately 50% of patients have congenital heart disease (ASD, VSD, TOF, pulmonic stenosis, TGA).
(3) Intellectual impairment and obesity
vi) Features unique to Crouzon syndrome:
(1) Increased incidence of elevated ICP, may require VP shunting[10]
(2) May have cervical fusion
vii) Features unique to Pfeiffer syndrome:
(1) Widened thumbs and great toes
(2) Severe forms have elevated ICP, higher risk of neurologic defects, and early death.

b) **Anesthetic implications**
i) Higher likelihood of associated organ anomalies including cardiac and renal defects
ii) Likely to present for craniosynostosis repair and cranial vault reconstruction as infants
(1) Potential difficult mask ventilation due to midface hypoplasia, potentially difficult intubation[11]
(2) Expect large blood loss from scalp and cranium, with high likelihood of blood transfusion in open vs endoscopic repairs
(a) Young and small infants are at especially high risk of cardiac arrest from blood loss.
(b) Tranexamic acid (TXA) has been shown to decrease transfusion requirements.[12]
(3) Children with signs of elevated ICP require meticulous care to avoid further increases in ICP including blunting of hypertensive response to laryngoscopy and intubation, balanced maintenance anesthesia, mild hyperventilation, and administration of diuretics if needed to reduce brain volume.
(4) Venous air embolism (VAE) is a common complication of craniectomies—hemodynamically significant VAE must be rapidly recognized and treated with fluid resuscitation and vasopressors/inotropes.
(5) Large-bore peripheral IV access and arterial line are needed for open procedures.

c) **Preschool-age children often undergo maxillary advancement procedures (LeFort I/II/III osteotomies).**
i) Close communication regarding airway management between surgeon and anesthesiologist is necessary.
ii) External fixation devices may be placed, which can cause obstruction to intubation if required postoperatively.

d) Regardless of surgery, there is a high incidence of obstructive sleep apnea due to midface hypoplasia and may need higher level of monitored care postoperatively.

10) **Familial dysautonomia** (*Riley-Day syndrome*)
a) **Essential characteristics**
i) Hereditary sensory and autonomic neuropathy predominantly found in the Ashkenazi Jewish population with autosomal recessive inheritance
ii) Neonates and infants present with hypotonia, poor feeding, reflux, recurrent pneumonia, and delayed growth.
iii) Progressive degeneration of sensory and autonomic neurons

iv) Stress can induce autonomic crises with blood pressure instability, bradycardia and/or tachycardia, vomiting, profuse sweating.
v) Reduced ventilatory response and exaggerated decrease in blood pressure in response to hypoxia
vi) High incidence of QT prolongation and scoliosis

b) **Anesthetic implications**
i) Patients with FD are highly variable and may have dysrhythmias, heart block, QT prolongation, blood pressure lability, dehydration, and electrolyte abnormalities.[13]
ii) Commonly indicated surgeries include gastrostomy with fundoplication, spinal fusions, and pacemaker placement.
iii) Consider preoperative admission for IV hydration and preoperative EKG to check for QT prolongation.
iv) Rapid sequence induction and invasive blood pressure monitoring may be necessary.
v) Emergence should be smooth. Consider low dose of benzodiazepines, clonidine, or dexmedetomidine to prevent autonomic crises and paroxysmal hypertension.

11) **Glycogen storage disease type I** *(von Gierke disease)*
a) **Essential characteristics**
i) Defect of glucose-6-phosphatase or glucose-6-phosphatase transporter
ii) Patients present with fasting hypoglycemia, lactic acidosis, and hyperlipidemia.
iii) Hyperlipidemia may precipitate acute pancreatitis.
iv) Progressive renal disease due to deposition of glycogen
v) Hepatic adenomas form with risk of conversion to malignant hepatocellular carcinoma.

b) **Anesthetic implications**
i) At risk of hypoglycemia—limit fasting time or admit prior to surgery for maintenance fluid, check glucose perioperatively, and use dextrose containing fluid[14]
ii) Review renal ultrasound, and check renal function.
iii) Some patients develop acquired von Willebrand syndrome.
(1) Consider desmopressin in patients undergoing surgeries with expected major blood loss (eg, resection of hepatic adenoma).

12) **Glycogen storage disease type II** (*Pompe disease*)
a) **Characterization**
i) Defect of α-glucosidase
ii) Two forms of the disease, early (infantile) onset and late (juvenile/adult) onset
iii) Early-onset disease presents in infants with severe, generalized hypotonia; severe cardiomyopathy; and respiratory insufficiency.
iv) Late-onset disease presents with proximal muscle weakness without cardiac manifestations and may progress to respiratory failure.
v) Newborn screening and the advent of enzyme replacement therapy (ERT) has greatly improved prognosis.

b) Anesthetic implications
i) Intracardiac glycogen deposition causes hypertrophy, threatening coronary perfusion, and may affect the conduction system, leading to dysrhythmias.
ii) Untreated or late-to-treatment infants with Pompe disease are at very high risk of cardiovascular collapse with anesthetic induction. Any anesthetic should start after defibrillation pads have been placed and aim to balance myocardial oxygen demand while maintaining hemodynamic stability.
iii) Obtain preoperative EKG and echocardiogram.
iv) Assess for gastroesophageal reflux, aspiration risk, and weakness.
v) Carefully titrate muscle relaxants and monitor for adequate reversal.[15]



Pompe disease patients that are untreated or late-to-treatment are at high risk of cardiovascular collapse during induction.

13) **Glycogen storage disease type III** *(Cori disease/Forbes disease)*
    a) **Characterization**
        i) Deficiency of enzyme that catalyzes debranching of glycogen molecules
        ii) Patients have variable degrees of glycogen accumulation in skeletal muscle, cardiac muscle, and liver.
        iii) Muscle infiltration leads to weakness and hypotonia; at times, patients can develop macroglossia.
        iv) Cardiac infiltration can cause hypertrophic cardiomyopathy and outflow tract obstruction.
        v) Liver infiltration can cause hepatomegaly, low-grade hepatitis, and rarely cirrhosis. Hepatomegaly and liver function typically normalizes as age advances.[16]
    b) **Anesthetic implications**
        i) Consider preoperative EKG and echocardiogram.
        ii) Examine for possible hepatic dysfunction; consider checking coagulation function.
        iii) Prepare for potentially difficult ventilation and intubation in case of macroglossia.
        iv) Assess for gastroesophageal reflux, aspiration risk, and weakness.
        v) Carefully titrate muscle relaxants, and monitor for adequate reversal.
14) **Glycogen storage disease type IV** (*Andersen disease*)
    a) **Essential characteristics**
        i) Deficiency of enzyme that catalyzes branching of glycogen molecules
    b) **Anesthetic implications**
        i) Assess for gastroesophageal reflux, aspiration risk, and weakness.
        ii) Examine for possible hepatic dysfunction; consider checking coagulation function.
        iii) Carefully titrate muscle relaxants, and monitor for adequate reversal.[17]
15) **Glycogen storage disease type V** (*McArdle disease*)
    a) **Essential characteristics**
        i) Most common glycogen storage disease, caused by deficiency of enzyme myophosphorylase needed for catabolism of glycogen in skeletal muscle[18]
        ii) Insufficient energy for muscle function
        iii) Increased cellular stress from increased glycogen stores can lead to episodes of rhabdomyolysis.
        iv) Symptoms of exercise intolerance, myalgia, and cramping
    b) **Anesthetic implications**
        i) Assess for gastroesophageal reflux, aspiration risk, and weakness.
        ii) Carefully titrate nondepolarizing muscle relaxants, and monitor for adequate reversal.
        iii) Rhabdomyolysis with myoglobinuria and acute kidney injury can be triggered by volatile anesthetics, succinylcholine, use of tourniquet, and postoperative shivering.
        iv) Safest to use TIVA, maintain normothermia, and quickly treat shivering with meperidine as needed[19]
16) **Hereditary angioedema**
    a) **Essential characteristics**
        i) Deficient or defective C1 esterase inhibitor (C1INH) leads to unchecked complement activation and contact activation in plasma, leading to increased vascular permeability.
        ii) Patients have intermittent swelling of the skin, GI tract, and airway tract.
        iii) Skin symptoms include tightness and numbness, often involving extremities, face, and genitalia.
        iv) GI symptoms include abdominal pain, nausea, vomiting, and diarrhea.

v) Upper airway involvement is the most dangerous and potentially lethal expression of hereditary angioedema.
   (1) Laryngeal angioedema can be caused by minor trauma, dental procedures, snoring, stress, and infection.[20]
   (2) Mortality rate of 25%-40%

b) **Anesthetic implications**
   i) Anesthesiology help might be called for in cases of HAE upper airway crises.
      (1) Prophylactic intubation should be done at first sign of respiratory distress, but preparation for surgical airway should also be made.
   ii) For scheduled surgery, prevention of HAE exacerbations is critical.
   iii) Check C1-INH level preoperatively; exogenous C1INH can be given the day before surgery.[21]
   iv) Where C1-INH is unavailable, FFP can be given 6-12 hours in advance.
   v) Avoid intubation when feasible.

17) **Heterotaxy syndrome**
   a) **Essential characteristics**
      i) Failure of normal embryonic development of left-right asymmetry, resulting often in malformations in multiple organ systems such as malrotation, polysplenia or asplenia, and tracheoesophageal fistula.[22]
      ii) Nearly all patients have significant congenital heart disease.
      iii) Clinical presentation is extremely heterogeneous.
   b) **Anesthetic implications**
      i) Have a detailed understanding of the patient's pertinent anatomy including cardiac lesions and airway abnormalities.
      ii) Understanding the physiology of each will determine a safe and appropriate anesthetic.
      iii) Patients with polysplenia or asplenia are relatively immunocompromised and require great care in aseptic technique.

18) **Homocystinuria**
   a) **Essential characteristics**
      i) Autosomal recessive disorder caused by defects in methionine metabolism
      ii) Patients have marfanoid body habitus, intellectual disability, and increased risk of thromboembolism.
   b) **Anesthetic implications**
      i) Patients may present for repair of ectopia lentis.[23]
      ii) Consider preoperative admission for IV hydration to reduce risk of thromboembolism.
      iii) Some authors report giving perioperative heparin for prophylaxis.
      iv) Increased risk of hypoglycemia—check glucose intraoperatively and use dextrose containing fluids as indicated.
      v) Avoid nitrous oxide, which can increase homocysteine levels, potentially increasing risk of thromboembolism.[24]

19) **Kabuki syndrome**
   a) **Essential characteristics**
      i) Rare multisystem disease with characteristic facial dysmorphism, developmental delay, and various skeletal and visceral anomalies
      ii) Affected patients can have airway anomalies including micrognathia, arched palate.
      iii) About 50% of patients reported to have various forms of congenital heart disease, including VSD, TOF, DORV, TGA, and aortic coarctation.[25]
      iv) High incidence of sleep apnea due to both obstructive and central etiology

b) **Anesthetic implications**
   i) Careful preoperative assessment of functional status, airway examination, and previous studies and surgeries
   ii) Patients are highly variable in presentation so require tailored anesthetic based on anticipated challenges.
   iii) Consider prolonged PACU stay or overnight admission in case of positive history of sleep apnea.

20) **Kawasaki disease** (*mucocutaneous lymph node syndrome*)



Kawasaki disease is one of the most common vasculitides of childhood.

a) **Essential characteristics**
   i) One of the most common vasculitides in childhood
   ii) Unknown cause, with marked geographic and seasonal variance, increased prevalence in boys and children with Asian inheritance.
   iii) Primarily a clinical diagnosis
      (1) Criteria are fever lasting ≥5 days plus four out of five other criteria: bilateral conjunctivitis, oral mucous membrane changes (erythema, cracked lips, "strawberry" tongue), peripheral extremity swelling or redness, polymorphous rash, and cervical lymphadenopathy.
   iv) Sequelae of unrecognized Kawasaki disease (KD) are serious, with potential coronary artery aneurysms and depressed myocardial function and heart failure.[26]
   v) Early diagnosis and treatment with IVIG and aspirin decrease the incidence of coronary artery aneurysms.

b) **Anesthetic implications**
   i) In the acute phase of illness, Kawasaki disease may mimic surgical emergencies such as septic arthritis or acute abdomen.
   ii) When appropriate, the anesthesiologist must consider KD in the differential and advocate for cardiology consultation and initiation of appropriate treatment (ie, IVIG and aspirin) if KD is suspected.
   iii) Children with KD need baseline and subsequent echocardiograms to evaluate for aneurysms, function, and pericardial effusion.
   iv) Coronary artery aneurysms can lead to rupture or occlusion and myocardial infarction.[27]
   v) Older patients with coronary aneurysms from KD are at increased risk of perioperative myocardial ischemia and any anesthetic needs to balance myocardial oxygen demand and supply and maintain hemodynamic stability.

21) **Klippel-Feil syndrome**

a) **Essential characteristics**
   i) Heterogeneous syndrome featuring cervical vertebral fusion, short neck, and low posterior hairline[28]
   ii) Can be associated with multiple other anomalies, including Chiari malformation, congenital cardiac disease, genitourinary malformations, and sensorineural deafness

b) **Anesthetic implications**
   i) Patients may present for decompressive cervical laminectomy in the setting of cervical myelopathy.[29]
   ii) Perform thorough history and physical examination to evaluate for possible associated anomalies.
   iii) Prepare for airway challenges given extreme limitation of neck mobility and possible cervical instability.

22) **Klippel-Trenaunay syndrome**

a) **Essential characteristics**
   i) Rare congenital malformation characterized by triad of venous malformations, capillary malformations that can involve neurovascular structures, and limb hypertrophy

ii) Malformations are highly heterogeneous and can involve several different areas of the body, but commonly a single lower limb is affected.
iii) Important comorbidities include recurrent bleeding, deep vein thrombosis, pulmonary embolus, and recurrent cellulitis.[30]

b) **Anesthetic implications**
i) May present for laser ablation or excision of vascular malformations, or amputations and joint disarticulations for severely affected limbs
ii) History of pulmonary embolism may lead to cardiac dysfunction and elevated anesthetic risk.
iii) Adequate vascular access is paramount for surgeries that may provoke significant bleeding.
iv) Spine imaging is needed to evaluate for epidural or subdural vascular malformation if considering neuraxial anesthesia.

23) **Long QT syndrome** (*Jervell and Lange-Nielsen syndrome, Romano-Ward syndrome, Andersen-Tawil syndrome, Timothy syndrome*)

a) **Essential characteristics**
i) A heterogeneous group of disorders with various mutations of ion conduction channels resulting in prolongation of the QT interval and predisposition to ventricular dysrhythmias and sudden death[31]
ii) Syncope can be triggered by stress, intense emotion, sudden loud sounds, and exercise (especially swimming).[32]
iii) Andersen-Tawil syndrome is associated with *QU* prolongation, dysmorphism, developmental delay, and periodic paralysis.
iv) Timothy syndrome is associated with QT prolongation and syndactyly.[33]

b) **Anesthetic implications**
i) Some patients are on β-adrenergic antagonists, and these should be continued perioperatively.
ii) Check electrolytes preoperatively and replace to normal levels prior to scheduled surgery.
iii) Premedicate appropriately to avoid stress, and ensure a calm OR environment prior to induction.
iv) Some patients have pacemakers and/or ICDs. These should be managed appropriately with electrocautery, that is, pacemakers set to asynchronous mode, and ICD defibrillating function should be switched off.
v) Have defibrillator immediately available intraoperatively.
vi) Ensure adequate plane of anesthesia for laryngoscopy and intubation.
vii) Consider deep extubation if appropriate to ameliorate sympathetic response during emergence.
viii) Common anesthetic drugs that prolong the QT interval include sevoflurane, propofol, and ondansetron.

24) **Mitochondrial disease**

a) **Essential characteristics**
i) Spectrum of diseases that involve defects in oxidative phosphorylation and mitochondrial function due to genetic mutations in either mitochondrial or nuclear DNA
ii) Organ systems with highest aerobic metabolism demand—for example, brain, heart, skeletal muscle—are preferentially affected.
iii) Patients with mitochondrial disease commonly have stroke, seizure, developmental delay and regression, myalgia, weakness, hypotonia, cardiac conduction anomalies, and cardiomyopathy.



Nearly all anesthetic agents are known to affect mitochondrial energy metabolism.

b) **Anesthetic implications**
   i) Nearly all anesthetic agents are known to affect mitochondrial energy metabolism.
   ii) Proteins involved in oxidative phosphorylation are especially sensitive to volatile agents but can also be affected by propofol, etomidate, ketamine, benzodiazepines, morphine, and local anesthetics.
   iii) There are no reported problems with dexmedetomidine and fentanyl analogues.
   iv) Despite the above problems, mitochondrial disease patients appear to tolerate inhalation induction with sevoflurane and titrated boluses of propofol and other intravenous agents.[34]
   v) Patients with mitochondrial disease may be more prone to develop propofol infusion syndrome, so it may be safer to avoid propofol infusion for anesthesia maintenance.
   vi) Patients may also be at risk for perioperative hypoglycemia—fasting should be minimized as much as possible, and dextrose-containing fluids should be used, unless the patient is on a ketogenic diet for seizure control.
   vii) Lactated Ringer's should be avoided as well, since metabolism of lactate may be impaired as well, and infusion of LR can cause metabolic acidosis.[34]



Lactated Ringer's should not be used in patients with mitochondrial disease. Impaired metabolism can lead to lactic acidosis.

25) **Moyamoya disease**
   a) **Essential characteristics**
      i) Disorder featuring progressive stenosis and occlusion of internal carotid arteries and anterior and middle cerebral arteries with extensive arterial collateralization
      ii) Variable clinical presentation includes transient ischemic attacks, intracerebral hemorrhage, ischemic stroke, and seizures.
   b) **Anesthetic implications**
      i) Multiple surgical techniques for cerebral revascularization have been developed, including direct superficial temporal artery to middle cerebral artery bypass and indirect revascularization techniques, which aim to promote angiogenesis of a patent vascular network.[35]
      ii) Consider preoperative admission for IV hydration.
      iii) Consider preoperative anxiolysis as hyperventilation or crying can cause cerebral vasoconstriction and ischemia.
      iv) Ensure adequate anesthetic depth to blunt response to laryngoscopy and intubation, and aim for smooth emergence to avoid coughing and crying.
      v) Intraoperative management should include normocapnia and normothermia.
      vi) Recognize that autoregulation is often impaired, so tight control of blood pressure is imperative.[36]

26) **Mucopolysaccharidosis** (*Hurler, Hurler-Scheie, Scheie, Hunter, Sanfilippo A/B/C/D, Morquio A/B, Maroteaux-Lamy, Sly, Natowicz syndromes*)
   a) **Essential characteristics**
      i) Rare genetic disorders of metabolism of glycosaminoglycans (GAGs) that cause degradation products of GAGs to accumulate in lysosomes
      ii) All of the mucopolysaccharidoses are autosomal recessive with the exception of MPS type II, which is X-linked.
      iii) The MPS syndromes have a wide spectrum of disease.
      iv) Patients can vary from normal intellect to severe developmental delay, multilevel airway obstruction, cardiac valvular disease and cardiomyopathy, and skeletal deformities.
      v) Some types of MPS can be treated with ERT or hematopoietic stem cell transplant with amelioration but not complete cure of disease.

b) **Anesthetic implications**
   i) MPS patients present for a number of different surgeries: ENT procedures, cardiac surgery, orthopedic surgery including spine instrumentation/fixation, tracheostomy.
   ii) The primary anesthetic concerns are the presence of cardiac disease, restrictive lung disease, and above all difficult airway management.[37]
   iii) Typically the airway becomes more difficult with age.
   iv) The mainstays of safe anesthesia are maintenance of spontaneous respiration, fiberoptic intubation with an LMA as conduit, and awake intubation in older patients with more difficult airways.
   v) Older patients with cognitive impairment may not be able to cooperate with awake intubation.
   vi) Surgical airway backup should always be a consideration, especially in patients with severe forms of disease.

27) **Nager syndrome** (*acrofacial dysostosis syndrome*)
   a) **Essential characteristics**
      i) Craniofacial syndrome with autosomal dominant, autosomal recessive, and sporadic forms
      ii) Features include mandibular and midface hypoplasia, severe cleft palate, microstomia, microtia, and frequent upper limb abnormalities including shortened humeri, hypoplastic radii, hypoplastic, or absent thumbs.[38]
   b) **Anesthetic implications**
      i) Infants and children may present for craniofacial reconstructive surgery and cleft palate repair.
      ii) Anticipate difficult direct laryngoscopy and have difficult airway equipment ready.
      iii) Consider suturing ETT in place during craniofacial and intraoral surgeries.
      iv) Patients with midface hypoplasia frequently have obstructive sleep apnea—strongly consider postoperative overnight admission with higher level of monitoring.

28) **Parkes-Weber syndrome**
   a) **Essential characteristics**
      i) Rare congenital malformation characterized by single limb (typically leg) overgrowth due to AVM with multiple arteriovenous fistulae
      ii) Significant AVF burden can lead to high-output heart failure.[39]
   b) **Anesthetic implications**
      i) Treatment of large or multiple AVFs for heart failure can lead to sudden significant increase in SVR and subsequent left ventricular overload and failure.
      ii) Patients require invasive vascular monitoring including arterial and central venous catheters.
      iii) TEE is also strongly indicated for evaluation of intraoperative heart function.

29) **Pierre Robin sequence**
   a) **Essential characteristics**
      i) Triad of micrognathia (small mandible), glossoptosis (backward and downward displacement of the base of the tongue), and cleft palate
      ii) Not all patients with PRS have cleft palate, but all have upper airway obstruction.
      iii) Heterogeneous etiologies may be associated with other syndromes (eg, Treacher Collins) or may occur in isolation.
      iv) Newborns may be minimally symptomatic or may have severe obstruction with difficulty feeding, difficulty breathing, and failure to thrive.
   b) **Anesthetic implications**
      i) Patients may require a variety of procedures including CT or MRI, rigid laryngo-bronchoscopy, mandibular distraction, tracheostomy, gastrostomy, and fundoplication.

ii) Any anesthetic carries major concerns of potential difficult ventilation and intubation.[40]
iii) A key point in airway management is maintenance of spontaneous respiration until the airway is secured.
iv) Have multiple backup plans for intubation: direct laryngoscopy, video laryngoscopy, fiberoptic, possible ENT backup rigid DLB.
v) Potential airway techniques include oropharyngeal or nasopharyngeal airways, LMA, and side or prone positioning.
vi) Placement of an LMA with or without topical anesthesia as a conduit for fiberoptic intubation is a well-described technique.[41]

30) **Pseudohypoparathyroidism**
a) **Essential characteristics**
i) Rare condition of resistance to parathyroid hormone, with hypocalcemia, hyperphosphatemia, and increased levels of parathyroid hormone[42]
ii) Albright hereditary osteodystrophy (pseudohypoparathyroidism 1a), features developmental and skeletal defects including learning disabilities, basal ganglia calcifications, cataracts, and short 4th and 5th metacarpals. Other types of pseudohypoparathyroidism only have lab abnormalities of calcium homeostasis.
iii) In infants and children, hypocalcemia can present with seizures, stridor, and carpopedal spasm.
b) **Anesthetic implications**
i) Monitor EKG carefully as hypocalcemia will cause QT prolongation.
ii) Avoid respiratory alkalosis, which will exacerbate hypocalcemia.

31) **Shy-Drager syndrome** (*multiple system atrophy*)
a) **Essential characteristics**
i) Multiple system atrophy (MSA) is a neurodegenerative disorder with varying degrees of parkinsonism, ataxia, and dysautonomia. Shy-Drager syndrome typically refers to MSA with predominant autonomic features.
ii) Cause is unknown, but symptoms are due to oligodendrocyte dysfunction and demyelination.
iii) Dysautonomia can include orthostatic hypotension, paradoxical supine hypertension, impotence, bowel and bladder dysfunction, and anhidrosis.
iv) Patients have increased incidence of central sleep apnea.
b) **Anesthetic implications**
i) Consider RSI due to potential aspiration risk in the case of gastrointestinal dysmotility and laryngeal dysfunction.[43]
ii) Patients with parkinsonian features may have rigidity making intubation difficult.
iii) Extreme blood pressure lability with limited change in heart rate. Treat hypertension with short-acting agents such as nitroglycerin, nitroprusside. Hypotension is usually responsive to phenylephrine.
iv) Patients with anhidrosis can develop hyperthermia intraoperatively, so temperature monitoring is imperative.

32) **Skeletal muscle channelopathies** (*hyperkalemic periodic paralysis, hypokalemic periodic paralysis, myotonia congenita, paramyotonia congenita*)
a) **Essential characteristics**
i) A group of disorders with varying clinical presentations of weakness or abnormal muscle contraction. The uniting commonality among these disorders is mutations in genes encoding sodium, potassium, or chloride conduction in skeletal muscle cell membranes.[44]
ii) Hyperkalemic periodic paralysis is caused by a mutation in voltage-gated sodium channels in skeletal muscle.

(1) Rare autosomal dominant disease
(2) Abnormal channels discharge in response to a neuronal action potential, but they are slow to inactivate.
  (a) With high serum potassium, it is more difficult for muscle cells to repolarize as there is a prolonged influx of sodium and dampened efflux of potassium.
(3) Clinically, this presents as flaccid paralysis. Respiratory muscles are typically not affected. In between attacks of paralysis, patients may have abnormal muscle stiffness (myotonia).
(4) The disease usually begins in early childhood.
(5) Paralysis can be induced by ingestion of potassium, cold, hunger, and emotional stress.

iii) Hypokalemic periodic paralysis is caused by loss-of-function mutations in calcium channels in the T-tubules of skeletal muscle cells.
(1) Rare autosomal dominant disease
(2) When extracellular potassium is low, muscle cells repolarize quickly, and the mutated channels do not open effectively enough to allow discharge of calcium from the sarcoplasmic reticulum.
(3) Paralysis tends to affect proximal muscles, and attacks are less frequent but often more severe than hyperkalemic periodic paralysis.
(4) The disease usually begins in the second decade of life.
(5) Paralysis can be induced by intake of carbohydrates, salt, stress, and cold.

iv) Myotonia congenita is caused by mutations in chloride channels required for muscle cell repolarization.[45]
(1) Characterized by slow relaxation after muscle contraction, which improves with exercise ("warm-up phenomenon")
(2) Two forms:
  (a) Thomsen disease, autosomal dominant, onset in infancy
  (b) Becker disease, autosomal recessive, more severe, can lead to muscle wasting, onset in second decade

v) Paramyotonia congenita is caused by mutations in voltage-gated sodium channels in skeletal muscle.[46]
(1) Characterized by muscle stiffness. Unlike myotonia congenita, stiffness gets *worse* with exercise.
(2) Myotonia can also be provoked by cold temperature and fasting.

b) **Anesthetic implications**

i) Hyperkalemic periodic paralysis
(1) Succinylcholine can cause severe myotonia and masseter spasm, leading to difficult or impossible ventilation.
(2) Short-acting nondepolarizing paralytics are safe to use, but anticholinesterases can precipitate myotonia.
(3) No increased risk of malignant hyperthermia
(4) Consider premedication to avoid stress-provoked paralysis.
(5) Maintain normothermia, normokalemia, and limit fasting time. If possible, these patients should be the first case of the day.

ii) Hypokalemic periodic paralysis
(1) Consider risk for malignant hyperthermia based on case reports.
(2) Consider premedication to avoid stress-provoked paralysis.
(3) Maintain normothermia and mild hypercapnia to avoid alkalosis.
(4) Note that epinephrine can cause relative hypokalemia due to β2 stimulation.
(5) If possible, avoid dextrose-containing fluids and normal saline.

iii) Myotonia congenita
(1) Succinylcholine can cause severe myotonia, masseter spasm, opisthotonos leading to difficult or impossible ventilation.
(2) May need to use lower dose of nondepolarizing paralytics in Becker disease due to decreased muscle mass.
(3) No increased risk of malignant hyperthermia
iv) Paramyotonia congenita
(1) Succinylcholine can cause severe myotonia and masseter spasm, leading to difficult or impossible ventilation.
(2) Anticholinesterases can also cause myotonia.
(3) Maintain normothermia; limit fasting.

33) **Smith-Lemli-Opitz syndrome**
a) **Essential characteristics**
i) Autosomal recessive disease caused by deficiency of 3β-hydroxyesterol Δ^7 reductase leading to cholesterol deficiency
ii) Multiple organ systems are affected.
iii) Children can have severe developmental delay, autism, micrognathia, cleft palate, and small and abnormally hard tongues.[47]
iv) Congenital heart disease including PDA, ASD, aortic coarctation, and pulmonary stenosis
b) **Anesthetic implications**
i) Patients require careful airway examination and evaluation for potentially associated congenital heart disease.
ii) Older children with behavioral problems and aggression may benefit from child life specialist services.
iii) Unpredictable responses to anxiolytics
iv) Consider fiberoptic intubation as primary technique for airway management.

34) **Treacher Collins syndrome** (*mandibulofacial dysostosis*)
a) **Essential characteristics**
i) Autosomal dominant disorder of development of the first and second branchial arches
(1) Variable penetrance, 60% of incidence is due to fresh mutations.
ii) Patients have maxillary, zygomatic, and mandibular hypoplasia; temporomandibular joint abnormalities; microstomia; high-arched palate; and colobomas of the lateral aspect of the lower eyelids.
iii) Commonly associated with Pierre Robin sequence
b) **Anesthetic implications**
i) Infants and children may present for craniofacial reconstructive surgery and cleft palate repair.[48]
ii) Expect difficult direct laryngoscopy, with increasing difficulty with age.
iii) Fiberoptic intubation through an LMA is a commonly reported successful strategy for airway management.[49]
iv) Consider suturing ETT in place during craniofacial and intraoral surgeries.

35) **Trisomy 21** (*Down syndrome*)
a) **Essential characteristics**
i) Most common chromosomal abnormality[50]
ii) Characteristic facial features
iii) Varying degree of intellectual disability
iv) Congenital heart disease in ~50% of patients; most common lesion is AV canal defect.[51]
v) Large tongue, enlarged tonsils (secondary to chronic infection), subglottic stenosis

vi) Hypotonia and joint laxity
vii) Relative immune deficiency
viii) Congenital hypothyroidism is common.

b) **Anesthetic implications**
i) Neonates often present for repair of tracheoesophageal fistula, duodenal atresia, and congenital heart disease.
ii) Infants and young children often present for repair of congenital heart disease, tonsillectomy and adenoidectomy, tympanostomy, and tube placement.
iii) Cervical spine instability should be evaluated by a careful history (change in behavior, new weakness, gait instability, bowel/bladder problems) and a focused neurologic examination.
iv) Preoperative assessment should include evaluation for congenital heart disease.
v) Patients are at increased risk of pulmonary hypertension from either persistent left-to-right cardiac shunts or chronic obstructive sleep apnea.
vi) Potential problems during inhalation induction:
(1) Bradycardia during sevoflurane inhalation induction[52]
(a) Quickly decreasing sevoflurane concentration can often resolve bradycardia. In case of rapid drop in heart rate, children typically respond to IV or IM atropine.
(2) Airway obstruction due to large tongue and hypertrophied tonsils
vii) Smaller than predicted endotracheal tube size may be required.
viii) Down syndrome children are at increased risk of postintubation croup.
ix) Strongly consider admission for patients undergoing T&A because they are at increased risk for postoperative apnea.

36) **Turner syndrome**

a) **Essential characteristics**
i) Common chromosomal abnormality with X chromosome monosomy, X chromosome mosaicism, or X chromosome anomalies
ii) Typical features include short stature and short webbed neck.
iii) Skeletal anomalies including short neck and micrognathia
iv) About 50% of patients have cardiovascular anomalies, bicuspid aortic valve most common, and increased incidence of coronary artery disease in younger adults.[53]
v) Increased risk of autoimmune thyroiditis with hypothyroidism, metabolic syndrome, and diabetes mellitus

b) **Anesthetic implications**
i) Potential for difficult intubation in cases of micrognathia
ii) Neonates or infants may require cardiac surgery for defects such as aortic coarctation.
iii) Bicuspid aortic valve, propensity for coronary disease, diabetes mellitus raises concern for perioperative myocardial ischemia, and infarction in older patients

37) **VACTERL association**

a) **Essential characteristics**
i) A collection of associated anomalies (**V**ertebral anomalies, **A**nal atresia, **C**ardiac defects, **T**racheo**E**sophageal fistula, **R**enal defects, **L**imb defects) where two or more occur in an affected individual[54]
ii) Associated defects can fall anywhere on a spectrum of relatively mild (eg, horseshoe kidney plus a wing vertebra) to very severe (tracheoesophageal atresia plus single ventricle anatomy).

b) **Anesthetic implications**
i) The finding of one of these defects should prompt evaluation for VACTERL and trisomy 13, 18, and 21.

ii) Patients with thumb or radial defects should prompt evaluation for Fanconi anemia.
iii) Patients with TEF and concomitant large left-to-right shunt lesions should preferably undergo TEF repair in the first few days of life prior to physiologic drop in pulmonary vascular resistance.[55]

38) **von Hippel-Lindau disease**
a) **Essential characteristics**
i) Rare autosomal disorder that leads to retinal and cerebellar hemangioblastomas
ii) Hemangioblastomas are "leaky"; cerebellar hemangioblastomas can cause relatively rapid increases in ICP, and retinal lesions can cause glaucoma and blindness.
iii) Some patients may develop pheochromocytomas.
iv) Potential for worsening vascular lesions during pregnancy[56]
b) **Anesthetic implications**
i) Patients with known von Hippel-Lindau disease should undergo careful preoperative assessment.
ii) Unrecognized lesions can lead to morbidity.
(1) Agents that raise ICP in someone with already elevated ICP can precipitate Cushing reflex or cerebral herniation.
(2) Hypertensive crises with undiagnosed pheochromocytoma can be triggered by induction, intubation, surgical manipulation, and various drugs.[57]

39) **von Willebrand disease and von Willebrand syndrome**
a) **Essential characteristics**
i) von Willebrand disease (vWD) is the most common congenital bleeding disorder.[58]
ii) There are three types:
(1) Type 1: insufficient quantity of von Willebrand factor (vWF). Low bleeding risk
(2) Type 2 (subtypes A, B, M, N): abnormal function of vWF. Variable bleeding risk
(3) Type 3: extremely insufficient amount of vWF. High bleeding risk
iii) Increasingly recognized phenomenon is acquired von Willebrand syndrome (vWS),[59] with various causes including:
(1) Destruction of vWF multimers by cardiac valvular disease, ventricular assist devices, and ECMO
(2) Antibody-mediated clearance due to autoimmune and neoplastic disease
b) **Anesthetic implications**
i) Preoperative consultation with hematologist is recommended.
ii) Anticipate potential bleeding severity depending on type of disease.
iii) DDAVP increases endogenous vWF, use in type 1 and type 2A vWD.
iv) Concentrates of factor VIII and vWF are used for more severe types.
v) Intraoperatively, consider use of antifibrinolytics.
vi) Exercise caution with maneuvers that can cause mucosal trauma, such as nasogastric intubation.
vii) Exercise extreme caution with regional anesthesia after first consulting with the hematologist.
viii) Ensure availability of blood products in surgeries with potential or expected heavy bleeding.

40) **Williams syndrome**
a) **Essential characteristics**
i) Multisystem disease caused by ~28 gene deletions on chromosome 7s
ii) Williams syndrome patients may have stereotypical facial features, outgoing personalities, varying degrees of intellectual impairment, occasionally hypercalcemia, and vascular abnormalities.

iii) Vascular disease can include the following:
   (1) "Hourglass" deformity of the aortic sinotubular junction
   (2) Supravalvular aortic stenosis (SVAS), with or without aortic valve abnormalities
   (3) Coronary stenosis or dilation
   (4) Brach and/or peripheral pulmonary artery stenoses
   (5) Renal artery stenosis, leading to hypertension
iv) May have QTc prolongation

b) **Anesthetic implications**
   i) Right or left outflow tract obstruction can lead to ventricular hypertrophy with increased myocardial oxygen demand with decreased delivery.
   ii) Patients with SVAS and/or coronary abnormalities are at high risk for sudden cardiovascular collapse and death.[60]
   iii) Echocardiography and clinical assessment are insensitive to coronary abnormalities, so even children without overt signs of outflow tract obstruction are at risk of sudden perianesthetic death.
      (1) There are case reports of sudden death in Williams syndrome patients who had no autopsy findings of either outflow tract or coronary abnormalities.
   iv) Have a detailed understanding of relevant individual's anatomy including EKG, echocardiogram, angiography, CT, and MRI.
   v) Strongly consider evaluation by primary cardiologist within 1 month of proposed procedure.
   vi) For young patients, consider admission the night prior to procedure for IV access and fluid administration. Otherwise, limit NPO time and plan for first case of the day.[61]
   vii) Any anesthetic should aim to balance myocardial oxygen demand and supply and maintain hemodynamic stability.



Patients with Williams syndrome are at risk of sudden death even without any echocardiographic findings of coronary abnormalities. Vigilance during anesthesia and close postanesthetic monitoring is strongly advised.

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95 Caudal Epidural Anesthesia

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Caudal epidural anesthesia is the most commonly used regional technique in children. Both single-shot and catheter techniques may be used for intraoperative and postoperative analgesia for surgeries up to the level of the umbilicus.

1) **Indications**
   a) Analgesia alone (eg, skin grafting)
   b) Adjunct to general anesthesia (eg, abdominal surgery)
   c) Surgical anesthesia (eg, hypospadias, hernia repair)
   d) Postoperative analgesia (eg, lower extremity orthopedic cases)
   e) Treatment of chronic pain (eg, complex regional pain syndrome [CRPS])
2) **Contraindications (Table 95-1)**
3) **Risks and complications**
   a) **Risks:** According to the largest prospective database study from the Pediatric Regional Anesthesia Network (PRAN) and a similar study from Europe, there is very low rate (0.02%) of complications associated with placement of caudals in anesthetized children.
   None of the complications resulted in long-term sequelae.[1,2]
   b) **Complications**
      i) Block failure
      ii) Blood aspiration
      iii) Positive test dose
      iv) Dural puncture
      v) Cardiac arrest
      vi) Seizure
4) **Equipment**
   a) Sterilizing solution
   b) Sterile gloves and drapes
   c) Caudal needle or angiocaths: short bevel, 22-gauge, needle with stylet



Mongolian spots are NOT a contraindication to caudal block.



Caudal epidural injection is a STERILE procedure. Proper aseptic techniques must be employed.



The maximum safe dose for caudal bupivacaine is 2.5 mg/kg (equal to 1 mL/kg of 0.25% bupivacaine) or ropivacaine 2 mg/kg (equal to 1 mL/kg of 0.2% ropivacaine).

TABLE 95-1 Contraindications to Pediatric Caudal Block

Absolute Contraindications	Relative Contraindications
Patient or parent refusal	Previous caudal surgery
Lack of consent	CNS abnormalities (eg, multiple sclerosis and syringomyelia)
Coagulopathy and/or thrombocytopenia	Spina bifida/neural tube defects
Sepsis	Tethered spinal cord
Infection at site of injection	Scoliosis
Uncorrected hypovolemia	Local skin changes (eg, dimples, hairy patches, abnormal pigmentation) that may be associated with undiagnosed vertebral abnormalities

d) Connector (eg, T-piece or IV extension tubing) may be used to extend from the catheter to the syringe for easier injection.
e) Use of ultrasound guidance improves the safety of the technique in preterm babies and in infants with difficult identifiable landmarks.

5) **Drug selection**



Simple formula to determine amount of drug to give in a caudal block: 1 mL/kg will block up to umbilicus.

a) Level of block desired and drug dose is primarily dependent on the volume of local anesthetic given, rather than the concentration of the local anesthetic.
   i) Commonly used drugs and concentrations are
      (1) Bupivacaine 0.125%; 0.25%; or 0.5%
      (2) Ropivacaine 0.2%
b) The maximum safe dose for caudal bupivacaine is 2.5 mg/kg (equal to 1 mL/kg of 0.25% bupivacaine) or ropivacaine 2 mg/kg (equal to 1 mL/kg of 0.2% ropivacaine).[3]
c) There are various published formulae for calculating the appropriate dose of local anesthetic **(Table 95-2)**:
   i) The following dosing is suggested[4]:
      (1) 0.5 mL/kg will block the sacral nerve roots.
      (2) 1 mL/kg will block lower thoracic and upper lumbar nerve roots.
      (3) 1.25 mL/kg will block the midthoracic (T6) nerve roots.
   ii) An alternative formula for dermatome-based volume has been developed[5]:

      Volume of drug infused = 0.05 mL/kg/dermatome to be blocked

   iii) A simplified method of estimating volume of drug to be infused is commonly used[6]:

      1 mL/kg will block up to T6-10 level.

   iv) Maximum recommended volume for any patient is between 20 and 30 mL.[6]
   v) Use of 0.25% or greater bupivacaine may result in prolonged motor blockade, which may delay discharge for day surgeries—so use of 0.125% or 0.25% bupivacaine is recommended.

6) **Epidural adjuncts**



All additives must be preservative free.

Patients given epidural caudal opioids should be monitored for at least 12-18 hours.

a) All additives must be preservative free.
b) **Epinephrine** (2.5-5 µg/kg)
   i) May be included in the initial injection to rule out intravascular injection.
   ii) Addition of epinephrine does not prolong the duration of caudal anesthesia.
c) **Opioids**
   i) Opioids should be used judiciously because of concerns for respiratory depression, urinary retention, and nausea.
   ii) Infants and children are at special risk and should be monitored for at least 12 hours up to 18 hours after administration.
   iii) Children who also receive systemic opioids are at additional risk due to a synergistic effect.

TABLE 95-2 Formulae for Drug Dosing for Caudal Block[3–5]

Author	Amount of Local Anesthetic (Desired Level of Block)
Armitage	0.5 mL/kg (sacral) 1 mL/kg (lumbar) 1.25 mL/kg (thoracic)
Takasaki	0.05 mL/kg/dermatome
Simplified	1 mL/kg (T6-10)

d) **Clonidine** (1-2 µg/kg)
   i) May prolong duration of anesthesia.[7,8]
   ii) Sedation is an added benefit as it may reduce the incidence of emergence agitation.
e) **Ketamine** (0.5 mg/kg)
   i) May prolong the duration of anesthesia.
   ii) Concern for neurotoxicity.
   iii) Preservative-free formulation is not available in the United States.

7) **Technique**
   a) **Monitoring/lines**
      i) Standard ASA monitors should already be in place.
      ii) An IV line should be in place for fluid administration or resuscitation if necessary.
      iii) It is an accepted standard of care to administer caudal after induction of general anesthesia in children.
   b) **Positioning and preparation (Fig. 95-1)**
      i) The child is most commonly placed in the lateral decubitus position.
      ii) The posterior superior iliac spines are identified, and the sacral cornu is palpated.
      iii) Area is prepped using the sterilizing solution of choice.
   c) **Placement of block (Fig. 95-2)**
      i) Chosen needle is advanced at a 45-degree angle through the sacral hiatus.
      ii) A distinct "pop" is felt as the needle pierces the sacrococcygeal ligament.
      iii) When using angiocath, a "pop" may not occur, but the catheter should advance without resistance.



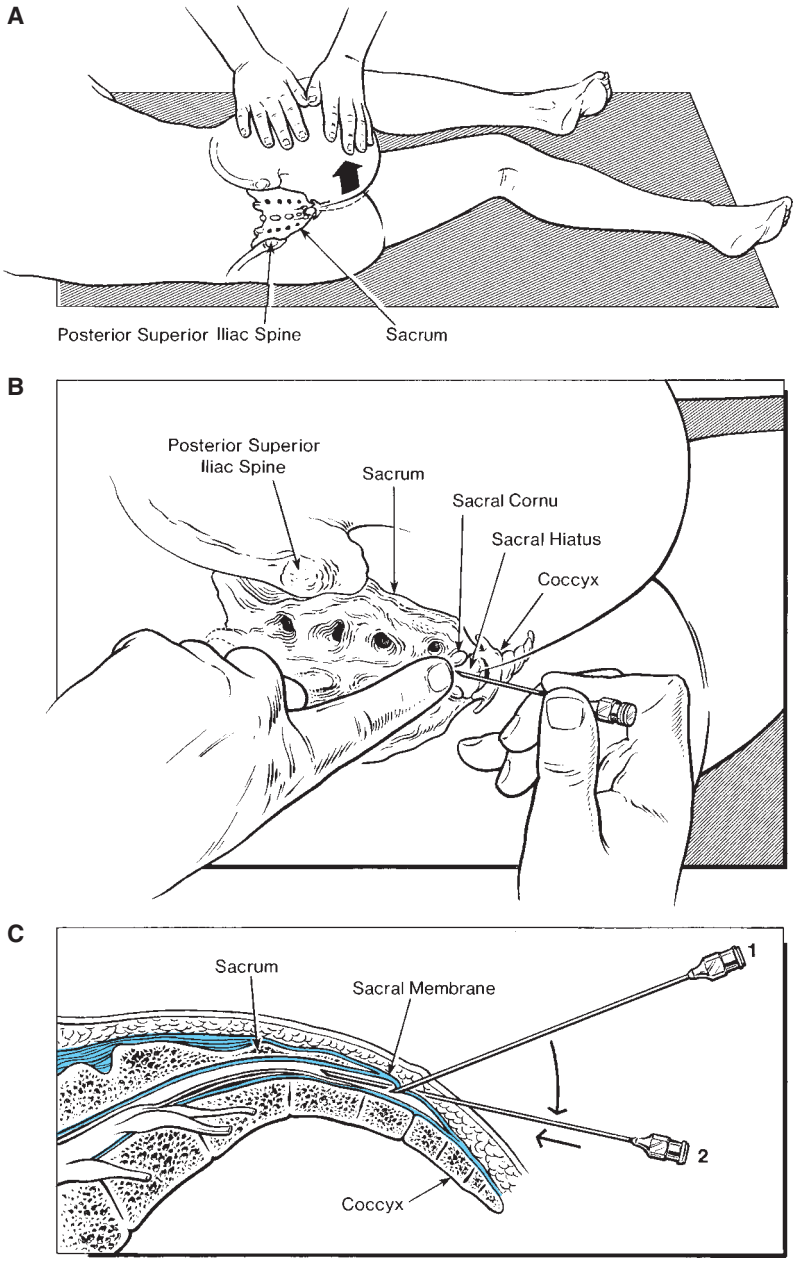
Any resistance to injection should raise concern of incorrect position of the catheter. It is a good practice to use one size syringe (eg, 10 mL syringe) for all volumes of administration to standardize the resistance inherent to the size of the syringe.

FIGURE 95-1 Positioning for Caudal Block Decubitus Position for Caudal Block



Epidural catheter or venous angiocatheter may be used. Aseptic technique with skin prep, drapes, and gloves is necessary.
Reused with permission from Long J, Suresh S. Neonatal anesthesia. In: Barash PG, Cullen BF, Stoelting RK, et al., eds. *Clinical Anesthesia*. Philadelphia, PA: Lippincott Williams & Wilkins; 2017:1199.

FIGURE 95-2 Technique for Caudal Block



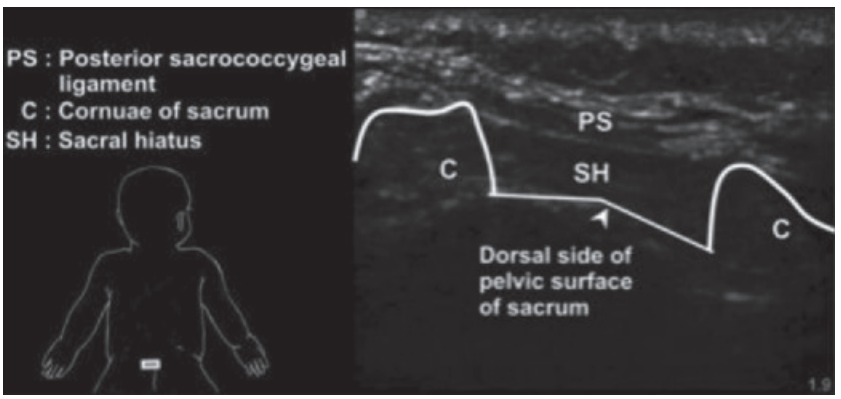
A. Lateral decubitus position for caudal block. **B.** Palpation of landmarks and insertion of catheter at sacral hiatus. **C.** Sagittal view of catheter insertion through sacral membrane and then lowering of angle to thread catheter.
Reproduced with permission from Veering BT, Cousins MJ. Epidural neural blockade. In: Cousins MJ, Carr DB, Horlocker TT, Bridenbaugh PO, eds. *Cousins & Bridenbaugh's Neural Blockade In Clinical Anesthesia and Pain Medicine*. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009:284-285.

iv) The angle of the needle is then lowered almost parallel to the skin and the catheter (but not the needle) advanced into the caudal space.
v) Verify negative aspiration of blood and cerebrospinal fluid (CSF).
vi) Administer incremental doses of the medication every 20-30 seconds over a few minutes while monitoring vital signs for evidence of IV or intrathecal administration. Stop further administration at the earliest sign of significant electrocardiogram (ECG) (>25% increase in amplitude of T waves) or vital signs changes.
vii) Ease of injection for a caudal block should be similar to that of injecting through peripheral IV. Any increased resistance to injection should raise concern of incorrect position of the catheter.
viii) It is a good practice to use one size syringe (e.g., 10 mL syringe) for all volumes of administration to standardize the resistance inherent to the size of the syringe.

d) During each medication administration, palpate for swelling/fullness at injection site to rule out subcutaneous infiltration **(Fig. 95-3)**.

8) **Continuous epidural catheter**
   a) Continuous epidural infusions may be performed in the caudal or the lumbar space by insertion of a catheter up to the lumbar or the thoracic levels.
   b) In children <9-12 months of age, a 20-gauge epidural catheter may be threaded through an 18-gauge angiocath to as high as the thoracic level.
   c) Confirmation of catheter position is recommended with chest radiograph, fluoroscopy, or ultrasound.
   d) 1 mm/kg of body weight estimates the distance from skin to epidural space in the lumbar space for children 6 months to 10 years old.[10]
9) **Anesthetic considerations**
   a) Unlike adults, pediatric patients usually do not exhibit hypotension with the onset of block.

FIGURE 95-3 Sonoanatomy of the Caudal Epidural Space at the Level of the Sacral Hiatus Using a Linear Hockey Stick Probe (SLA, 6-13 MHz, 25-mm footprint) Placed in the Transverse Plane.



Note the anechoic sacral hiatus between the hyperechoic lines of the sacrococcygeal membrane and the dorsal side of the pelvic surface of the sacrum. The sacral cornua appear as "humps" bilaterally.
Reprinted with permission from Tsui BC, Suresh S. Ultrasound imaging for regional anesthesia in infants, children, and adolescents: a review of current literature and its application in the practice of neuraxial blocks. *Anesthesiology*. 2010;112(3):719-728.

b) Hypotension should be interpreted as sign of possible intravascular injection rather than sympathectomy from block.
c) Intravascular injections may occur in spite of a negative aspiration because local anesthetics may be quickly absorbed through intraosseous channels.
d) The reliability of epinephrine causing tachycardia has also been questioned.[11]
e) The safest method to minimize risk of intravascular injection is to give incremental doses of drug.



Give incremental doses every 20-30 seconds to catch an inadvertent intravascular injection.

Hypotension should be interpreted as a sign of possible intravascular injection rather than sympathectomy from the block.

10) **Evaluation of the block**
a) Since most caudals are done in children under general anesthesia, evaluation for sensory and motor blockade may not be possible.
b) In addition, sympathectomy may also not be a reliable indicator of adequate anesthesia.
c) Therefore, lack of response to surgical stimulation proves to be the best way to assess block efficacy.
11) **Caudal and fistula after hypospadias repair**[12,13]
a) Recent studies have shown an association of caudal block with a complication of fistula formation after repair of hypospadias under caudal anesthesia.
b) The studies did not control for the severity of hypospadias.
c) While waiting for prospective randomized controlled trials (RCTs) to clarify the causality of this association, it is generally considered safe to continue to provide caudal analgesia for hypospadias repairs.
12) **Postoperative management**
a) Depending on duration of surgery, some postoperative motor blockade may be expected.
b) Motor and sensory blockade are commensurate with total dosage and type of local anesthetic and adjuvants used.
c) Parents should be warned not to allow the child to ambulate without supervision.
d) If opioids are used, patients should be monitored for at least 12 hours (and up to 18 hours) postoperatively for respiratory depression.

CHAPTER SUMMARY FOR CAUDAL EPIDURAL ANESTHESIA

General Considerations	Caudal anesthesia is one of the most useful anesthetic blocks used in children for lower extremity, genito-urological, and abdominal surgeries by blocking sacral, lumbar, and thoracic nerve roots.
Equipment Needed	Sterilizing solution Sterile gloves Sterile drapes Caudal needle or angiocath (22-gauge, short bevel, stylet) Optional connector as extension Local anesthetic Epinephrine *** all drugs must be preservative free
Intraoperative Considerations	Level of block desired and drug dose are primarily dependent on volume of local anesthetic given, rather than the concentration of the local anesthetic. Simplified formula: 1 mL/kg will block up to umbilicus. Maximal volume 20-30 mL. Give test dose, and then incremental doses of local anesthetic every 20-30 s to monitor for intravenous administration. Lack of response to surgical stimulation is the best way to assess efficacy of block.
Postoperative Considerations	Patients who have received epidural opioids should be monitored for at least 12-18 h afterward.

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96 Management of the Pediatric Difficult Airway

Samuel A. Mireles • Calvin Kuan

To safely anesthetize the pediatric patient with a difficult airway, the practitioner must be familiar with pediatric anatomy, physiology, and pharmacology and be facile with the equipment necessary to secure the pediatric airway.

1) **Introduction**
   a) Many congenital syndromes and acquired conditions are associated with having a difficult airway in children.
   b) The presence of a syndrome does not mean a patient will necessarily have a difficult airway, nor does absence of a syndrome guarantee a successful intubation.
   c) **Patient's age and progression of the syndrome may influence airway difficulty. For example:**
      i) Patients with Pierre Robin sequence tend to become easier to manage with growth of their mandible over time.
      ii) The airway management of patients with mucopolysaccharide storage diseases often becomes more difficult with age.

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The absence of a syndrome does not guarantee a successful intubation.

2) **Pediatric anatomy/physiology**
   a) **There are six clinically significant anatomic and physiologic differences between normal pediatric and adult airways. These differences all contribute to the inherent difficulty of the pediatric airway.**[1]
   b) **Tongue**
      i) Compared to adults, the infant tongue is relatively large in proportion to the oral cavity.
      ii) Significance
         (1) Airway obstruction from the tongue occurs frequently in children, especially in the supine position and with loss of pharyngeal muscle tone under anesthesia.
         (2) The tongue is more likely to obscure the view under direct laryngoscopy.
      iii) Solutions
         (1) Chin lift
         (2) Jaw thrust
         (3) Change in head position—tilt to side
         (4) Use of oral airway or supraglottic device
         (5) Manually pulling the tongue out with clamp, gauze, or a stitch to the tongue
         (6) A straight blade may be better than a curved blade to achieve a direct line of sight to the larynx in younger children.



The practitioner caring for children should understand the differences between pediatric and adult airway anatomy.



Straight blades are usually better for young children (<2-3 years old).

   c) **Position of larynx**
      i) The larynx may be as cephalad as C3 level for a premature infant, C3-C4 for a newborn infant, and C4-C5 for an adult.[1]

ii) Significance
  (1) The superior location of the larynx means that there is a more acute angle from the oral axis to the tracheal axis and may result in more difficulty visualizing the larynx under direct laryngoscopy.
iii) Solutions
  (1) External laryngeal manipulation or cricoid pressure may be employed to bring the larynx into view in order to insert the endotracheal tube (ETT).
  (2) If the above maneuver does not work, a stylet may be necessary to direct the ETT into the trachea.

d) **Epiglottis**
  i) The adult epiglottis is broad, firm, and positioned parallel to the trachea.
  ii) The neonatal/pediatric epiglottis is relatively short, narrow, floppy, and angled posteriorly relative to the trachea (**Fig. 96-1**).
  iii) Significance
    (1) Because of this posterior angle, the epiglottis may block the entrance to the larynx.
    (2) Blind techniques (eg, blind nasal intubation or insertion of an ETT through a laryngeal mask airway [LMA] without direct visualization of glottis) are likely to be less successful and more traumatic.
  iv) Solutions
    (1) Attempt to lift the epiglottis under direct laryngoscopic visualization to expose the glottis.
    (2) Use a styletted ETT to go around the epiglottis.
    (3) If there is resistance to insertion, try pulling out the stylet and sliding the ETT forward into the trachea. Without the stylet, the tip of the ETT is more malleable and less likely to hit the tracheal wall.

e) **Vocal cords**
  i) Adult vocal cords are perpendicular to the axis of the trachea.
  ii) Pediatric vocal cords are slanted such that the anterior commissure is inferior and the posterior commissure is superior (**Fig. 96-1**).
  iii) Significance
    (1) In children, the ETT may get caught at the junction of the anterior commissure and the epiglottis and not pass into the trachea.
  iv) Solutions
    (1) Directing the ETT posteriorly and twisting it with a corkscrew motion may ease insertion and allow it to pass into the trachea.

FIGURE 96-1 Orientation of Epiglottis and Vocal Cords. During direct laryngoscopy, the epiglottis may block visualization of the vocal cords. A stylet may be necessary to blindly direct the ETT underneath the epiglottis, but may still get caught at the anterior commissure.

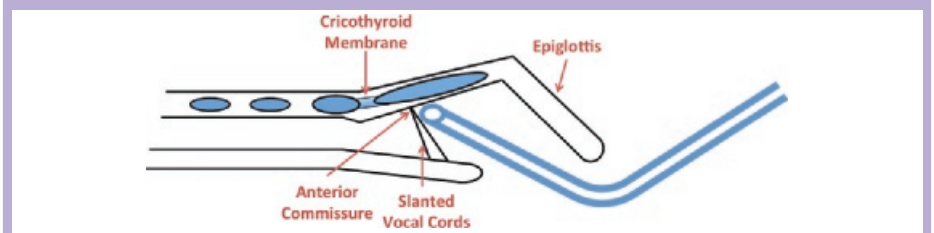


Photo by Shannon D. Schwartz.

f) **Subglottic area**
   i) The pediatric subglottic area is relatively smaller than the adult subglottic area. Multiple contemporary studies provide evidence that the narrowest point of the pediatric airway is the subglottic transverse diameter as opposed to the cricoid cartilage.[2,3] Others have pointed out that regardless of the measured diameter, the cricoid cartilage remains less distensible than more superior mucosa and therefore vulnerable.[4]
   ii) Significance
      (1) The practitioner must take care to ensure that the ETT chosen is the appropriate size and not so large that it will damage the tracheal tissue from sustained pressure.
   iii) Solution
      (1) The safest and most efficient way to accomplish this is to ensure that there is a leak around the ETT.
      (2) Ideally, this leak will occur between pressures of 15 and 25 cm of H_2O, but a leak at a higher pressure may be desired if one expects a decrease in lung compliance.
      (3) Ideally, cuff leak will be measured with a manometer, as the reliability of the traditional audible leak test has been called into question[5] (**Fig. 96-2**).

g) Oxygen consumption
   i) A healthy child has at least two to three times greater O_2 consumption at baseline compared to a healthy adult (6-8 cc/kg/min vs 3 cc/kg/min).
   ii) Significance
      (1) A healthy 70-kg adult can maintain 100% SpO_2 for almost 10 minutes after adequate preoxygenation.
      (2) A healthy 10-kg child can maintain 100% SpO_2 for only about 4 minutes—approximately the same amount of time as an obese 127-kg adult.[6]
      (3) This and other differences mean that even in normal healthy children, there is less time to secure the airway before hypoxia occurs.[6]
   iii) Solution
      (1) Adequate preoxygenation with 100% FiO_2. Ensure a closed system to avoid entraining room air.
      (2) If available, monitor expired O_2 level.
      (3) Maintain oxygenation throughout induction and intubation attempts until airway is secured (**Fig. 96-3**).



To minimize risk of tracheal injury, ensure there is a leak around the endotracheal tube.



Even in normal healthy children, there is less time to secure the airway before hypoxia occurs.

3) **Evaluation of pediatric airway**
   a) Many techniques exist to assess the airway of adults. None of them have been shown to be sensitive or specific in predicting the degree of difficulty intubating children.[7]
   b) It is essential for the practitioner to evaluate the possibility of a difficult airway in every pediatric patient with a thorough past anesthetic history, medical history, and physical exam.
   c) Investigations should include history of upper airway obstruction, difficult airway, difficulty feeding, and pediatric syndromes. Knowledge of pediatric syndromes affecting the airway and their natural history is useful.
   d) Physical exam should focus on the relative size and shape of the mandible, mouth and tongue, location and state of teeth, mouth opening, and neck mobility and include lateral assessment of the face.[8]

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FIGURE 96-2 Posey Cufflator, Endotracheal Tube Inflator, and Manometer

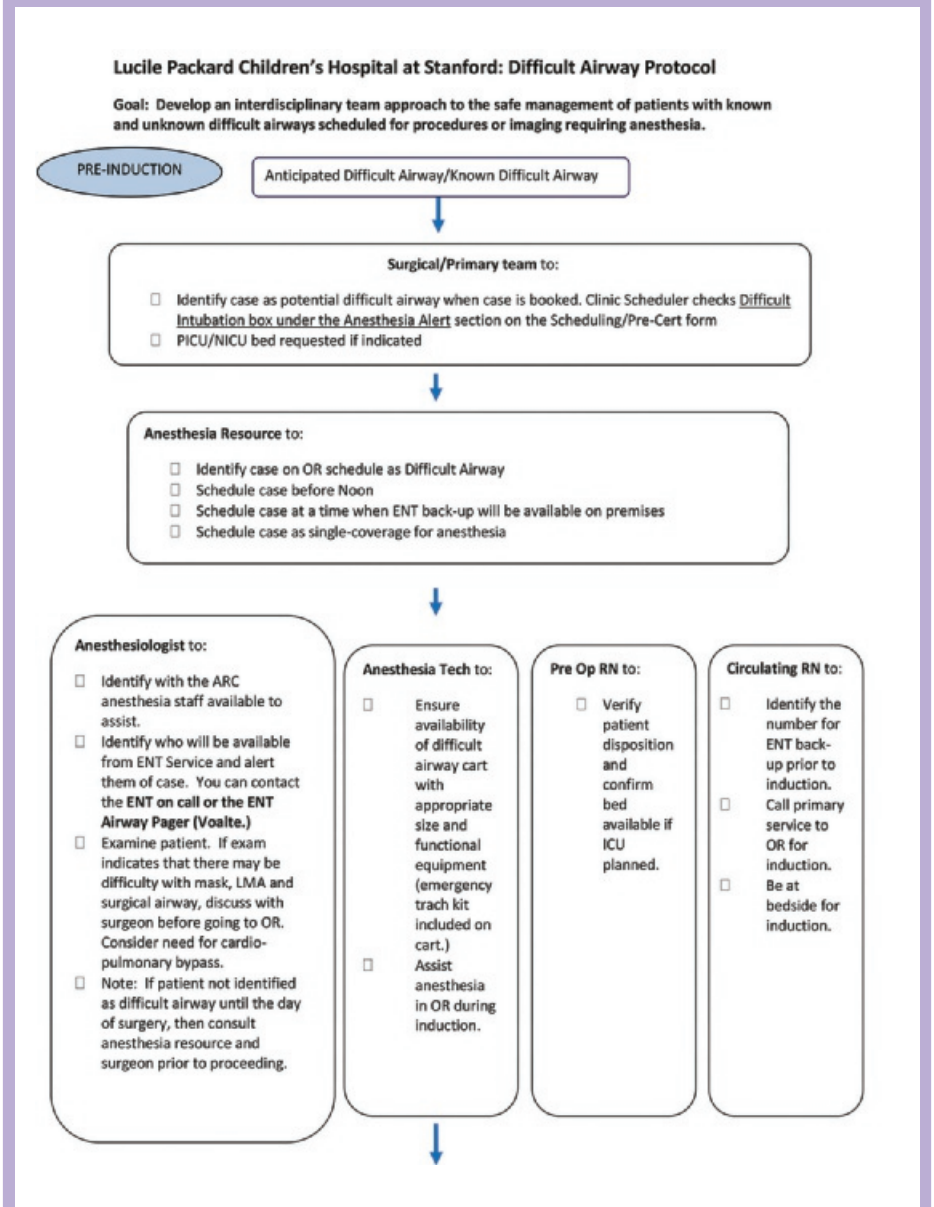


FIGURE 96-2 (Continued)

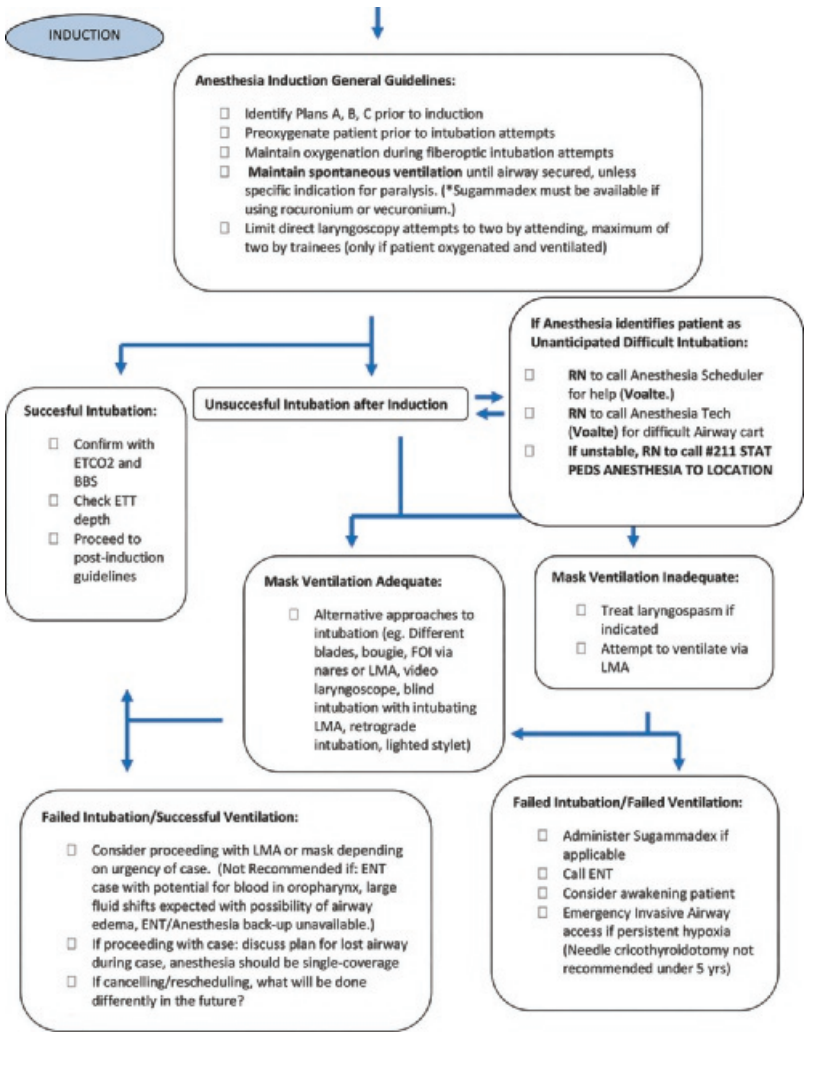
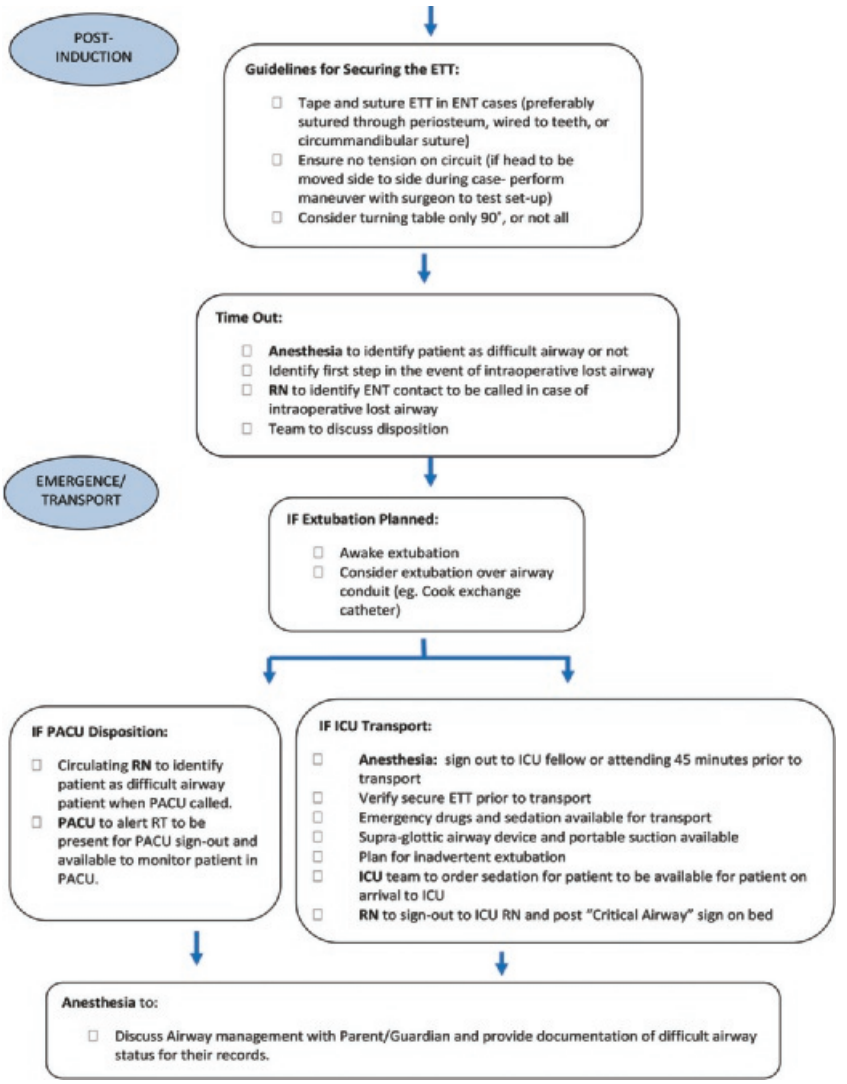


FIGURE 96-2 (Continued)



4) **Basic guidelines for management of the pediatric difficult airway**
   a) Due to the heterogeneity of causes of difficult airways in pediatrics, it is impossible to discuss specific techniques that will be applicable to all pediatric patients. However, a few important guidelines should be mentioned.
   b) **Preoxygenate and keep oxygenated.**
      i) Except for the rare clinical situations where a higher FiO_2 may be contraindicated (eg, single ventricle physiology), adequate preoxygenation and oxygenation during

intubation attempts to maximize time before desaturation in the case of a failed airway *(refer to photo Methods of Supplemental Oxygenation)* (**Fig. 96-3**).



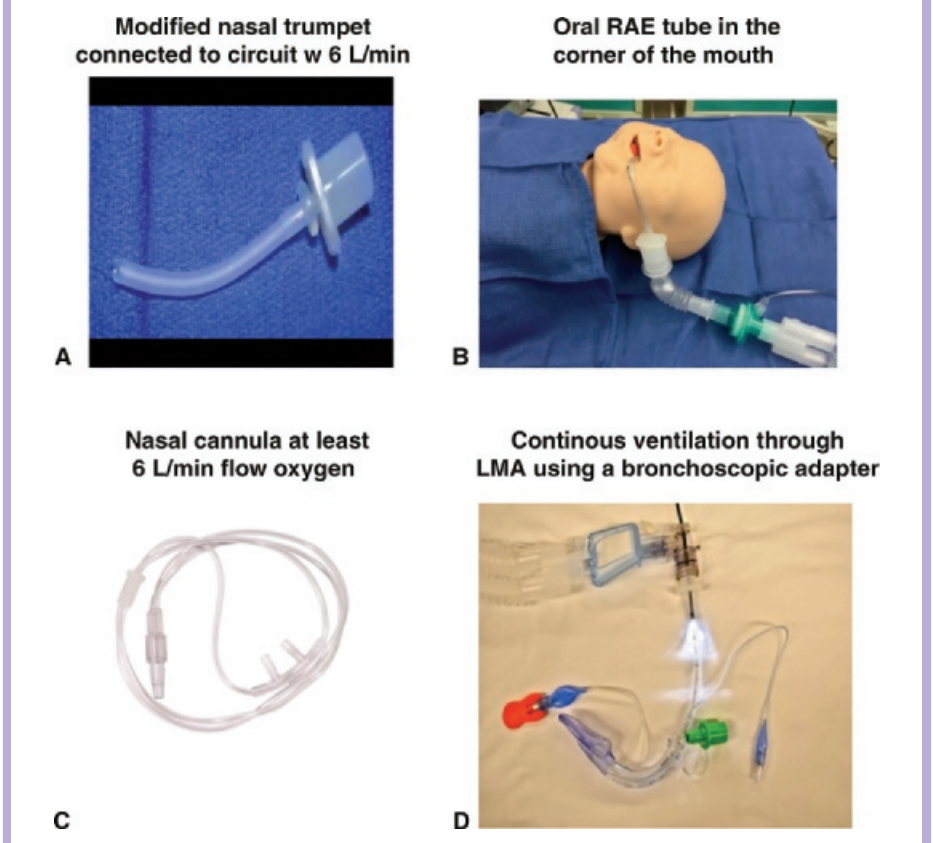
Oxygenate before and during intubation attempts to maximize time before desaturation.

c) **Consider an antisialoagogue to dry secretions prior to induction.**
   i) Glycopyrrolate (5-10 µg/kg IV) is usually adequate and can be repeated as needed.

d) **Maintain spontaneous ventilation**
   i) Allows for faster emergence if the decision is made to abort the anesthetic
   ii) Minimizes the risk of airway obstruction due to loss of tone from deeper level of anesthesia
   iii) Maintenance with some combination of ketamine, dexmedetomidine, or an inhaled anesthetic is a common technique because it is more likely to preserve respiratory drive.

e) **Always have an LMA immediately available as backup.**
   i) Even under optimal circumstances, children cannot maintain adequate SpO_2 for more than a few minutes after a failed attempt to secure the airway.

FIGURE 96-2 Posey Cufflator, Endotracheal Tube Inflator, and Manometer.



ii) There may not be enough time to attempt any technique other than an LMA. Appropriately sized LMAs should be immediately available in any location where children are anesthetized.
iii) It is worth noting that LMAs (especially those sized 2 or smaller) are more difficult to seat securely and easier to dislodge in children compared to adults.
(1) Constant vigilance is necessary to ensure that the LMA is seated properly at all times.



Complication rates are higher in cases with more than two direct laryngoscopy attempts. Minimize total direct laryngoscopy attempts by moving onto more experience providers and more advanced techniques early.

f) **Consider flexible scope laryngoscopy early in the process while the airway is still patent rather than after multiple attempts to intubate the trachea have resulted in bleeding, swelling, and unrecognizable anatomy.** Complication rates have been found to be higher in cases with more than two tracheal intubation attempts total and with more than three direct laryngoscopy attempts prior to an indirect technique.[9]
g) **Call for help early enough to make a difference!**
i) Many of the techniques that have been described for use in pediatrics require multiple steps and are most safely accomplished with experienced assistance.
h) **Know your equipment.**
i) Know what equipment is available and where to access it.
ii) Have hands-on experience setting up equipment such as intubating scopes.
iii) A "trial run" with all equipment prior to induction is strongly recommended especially when using equipment from different manufacturers or from different kits. For example, this may include checking light sources and checking that selected ETTs will fit over flexible intubating scopes and/or through intubating LMAs.



An LMA should be immediately available to be used to maintain a patent airway for oxygenation and as a conduit for fiberoptic intubation.

i) **Alternative devices**
i) Other airway devices (eg, video laryngoscopes, Trachlight, intubating LMAs, etc.) and techniques (eg, blind nasal intubation, digitally assisted intubation) are available. The decision to use these alternatives should be made on a case-by-case basis by practitioners with experience with the specific techniques.

5) **Indications for use of a paralytic agent.** Although it is generally advisable to maintain spontaneous ventilation in the case of a difficult airway, there are times when paralysis can improve ventilating or intubating conditions.
a) **Laryngospasm that does not respond to CPAP with 100% oxygen or deepening the anesthetic can be treated with succinylcholine.**
b) **Although the decrease in peripheral muscle tone caused by paralysis can lead to upper airway obstruction, the effects of neuromuscular blockers on ventilation are nuanced, and there is some evidence that paralysis can also facilitate ventilation.[10]**
c) **Paralysis can improve intubating conditions, such as in the setting of a rapid sequence induction.** However, the case of emergency surgery in a difficult airway patient with a full stomach requires careful consideration of the risks vs benefits of paralytic for rapid sequence induction (RSI). If sugammadex is available, the practitioner has a valuable tool that can change the risk-benefit analysis of paralytics.
d) **In the "cannot intubate/cannot ventilate" scenario, sugammadex can be used to reverse rocuronium or vecuronium.** As such, sugammadex should be confirmed to be immediately available prior to administration of rocuronium or vecuronium in a patient with a difficult airway.[11]

6) **Difficult airway algorithm**
   a) **Anticipated difficult airway**
      i) Use your clinical judgment. Keep in mind that children are not just little adults. They have different anatomy and physiology and therefore require individual strategies for management. Most important to consider is that there is often significantly less time to accomplish what is usually a more difficult goal.
      ii) Devise a primary plan A and have a backup plan B, C, D, etc., as needed.
      iii) Patients with clear evidence of a difficult airway and/or complex medical conditions for elective procedures should be referred to a tertiary pediatric center with multidisciplinary services that have experience caring for pediatric patients intraoperatively and postoperatively (Appendix. Stanford Children's Hospital Difficult Airway Protocol).
      iv) There may be rare clinical situations where elective use of extracorporeal membrane oxygenation (ECMO) or cardiopulmonary bypass is the safest option.
         (1) These may include severe mediastinal mass, severe anatomical distortion due to burn injury, or significant distal tracheal obstruction or disruption where even an appropriately placed airway will not provide adequate oxygenation.[12,13]



Know your equipment. Especially with advanced airway techniques, a "trial run" with all airway equipment is strongly recommended prior to induction especially when using equipment from different manufacturers or different kits.

   b) **Unanticipated difficult airway**
      i) If the first attempt at direct laryngoscopy was unsuccessful, a second attempt may be made if at least one condition has been changed including repositioning the patient, using a different blade, and changing to a more experienced practitioner.
      ii) If the second attempt is also unsuccessful, the practitioner should call for help and then weigh the risks and benefits of continued attempts.
      iii) If oxygenation and airway patency are not assured, the speediest and safest option is to insert an LMA.
      iv) Laryngospasm and gastric distention should also be considered as contributing factors in the setting of difficult ventilation.
   c) **Failed airway (cannot intubate/cannot ventilate)**
      i) In the situation of inability to mask ventilate or intubate, the most important goal of the anesthesiologist is to keep the patient oxygenated.
      ii) If the patient has received rocuronium or vecuronium, administer sugammadex for reversal.
      iii) An LMA should be placed immediately.
      iv) If the LMA is unsuccessful, or if the obstruction is at the glottis, then a surgical airway should be considered.
         (1) For children over 10-12 years of age, a surgical cricothyroidotomy or tracheostomy may be considered.
         (2) For children under 10-12 years of age, a needle cricothyroidotomy is recommended.
            a) Keep in mind that for younger children, the risk of complications with emergency surgical airway is extremely high,[1] so this should be attempted only in a true emergency.
            b) If the obstruction is located in the subglottic region or lower (ie, closer to the carina), a surgical airway at the cricothyroid membrane may not create a patent airway.

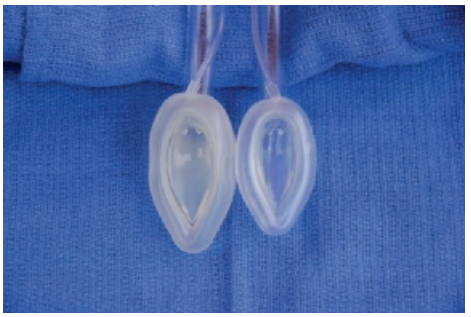


In younger children, the risk of complications with emergency surgical airway is extremely high.

READ MORE
Airway exchange catheters in practical pediatrics, Chapter 75

7) **Special techniques/situations**
   a) In cases where an appropriate pediatric size intubating LMA (such as the Air-Q LMA) is not available, it is possible to convert an LMA into an endotracheal tube.

FIGURE 96-4 Special Considerations for Pediatric Airway Equipment. LMA on the left has aperture bars removed. Smaller LMA on the right has bars intact for comparison. Note: If the bars are not cut short enough, the ETT may get caught on them during insertion.



Photos by Shannon D. Schwartz.

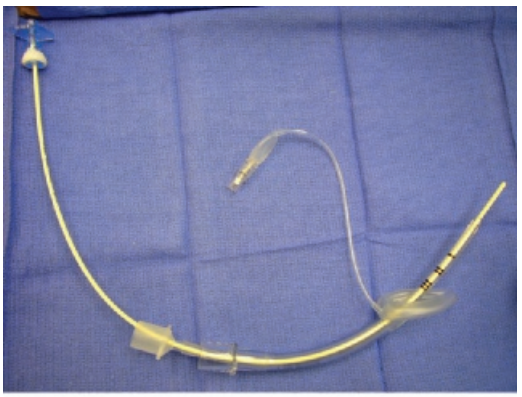
i) Blind intubation of the trachea of a smaller child through an LMA is unlikely to be successful because the epiglottis frequently obstructs the glottic opening, and fiberoptic assistance may be necessary.
ii) Additional problems should be anticipated.
(1) Problem No. 1: Aperture bars may block the advancement of the ETT.
(a) Solution No. 1-A: Cut out the aperture bars of the LMA prior to insertion (**Fig. 96-4**). The ETT may get caught on the bars if they are not cut short enough—especially the distal bars further away from the shaft of the LMA.
(b) Solution No. 1-B: An Air-Q (Mercury Medical, Clearwater, FL) intubating laryngeal airway, which does not have bars. In larger patients, a Fastrach LMA or LMA Excel, which have mobile epiglottic elevating bars, may be used (only available in sizes 3, 4, 5).
(2) Problem No. 2: The ETT cuff will not fit through the connector of the LMA **(Fig. 96-5A.)**
(a) Solution A: Use an uncuffed ETT first and then convert to a cuffed ETT with an airway exchange catheter (**Fig. 96-5B**).
(b) Solution B: Use an LMA Excel or Air-Q intubating laryngeal airway, both of which have removable connectors.
(3) Problem No. 3: The pediatric ETT is too short. The LMA cannot be removed safely without dislodging the ETT.
(a) Solution A: Elongate an ETT by connecting it end to end with an uncuffed ETT that is one half size smaller (eg, 4.0 with a 3.5 ETT) (**Fig. 96-6A**).
(b) Solution B: Alternatively, the smaller end of an ETT 15-mm connector may be cut off and used as a male-male adaptor to connect two ETTs of the same size (**Fig. 96-6B**).[14]
(c) Solution C: Leave the LMA with ETT *in situ* for the case.

b) **Fiberoptic intubation of small child with adult bronchoscope**
i) One can successfully intubate a small child or neonate even when the smallest fiberoptic scope available is an adult one.
ii) Necessary additional equipment
(1) Guidewire that will fit through the suction/working channel of the bronchoscope
(2) Guidewires smaller than 0.035 in (0.89 mm) in diameter may be too flimsy to support an ETT without kinking.

FIGURE 96-5 **A.** The cuff of the ETT will not fit through connector of LMAs smaller than size 3. **B.** An airway exchange catheter may be inserted into an uncuffed ETT through the LMA to safely exchange it for a cuffed ETT.



A



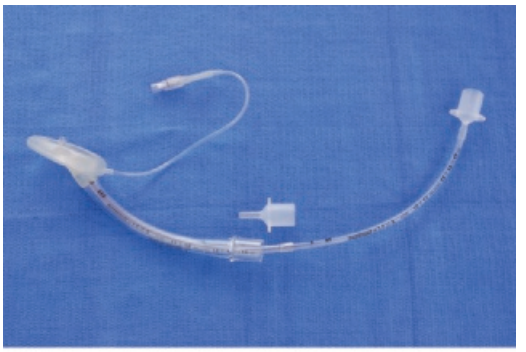
B

Photos by Shannon D. Schwartz.

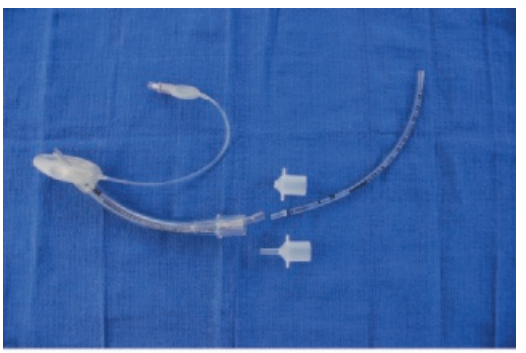
(3) It is important to verify that the wire is long enough to extend out of the bronchoscope prior to use. Cardiac catheterization or urological catheters (usually 100-150 cm long) are ideal.
(4) Airway exchange catheter that is large enough to fit over the guidewire and small enough to fit through the desired ETT

iii) Procedure
(1) First, locate the laryngeal inlet with the adult fiberoptic scope directly through the mouth.
(2) Then insert the guidewire through the working channel of the scope into the trachea under direct visualization.
(3) Similar to a Seldinger technique, remove the fiberoptic scope (but leave the guidewire).
(4) Insert the airway exchange catheter (AEC) over the guidewire to stiffen it. Without the AEC, the guidewire may not remain in the trachea and may flip out when trying to pass the ETT.

FIGURE 96-6 Special Considerations for Pediatric Airway Equipment. **A.** A 0.5 size smaller ETT may be used as a "Pusher" by fitting it into the end of an ETT with connector removed. This allows safe removal of the LMA. **B.** The smaller-diameter end of an ETT connector may be cut and used to connect two equal-sized ETT to make them long enough to safely remove an LMA.



A



B

Photos by Shannon D. Schwartz.

(5) Then pass the ETT over the AEC into the trachea.
(6) Confirm placement with $ETCO_2$ and auscultation.

c) **Emergency needle cricothyrotomy**)

i) In smaller children in whom surgical tracheostomy is not possible, insertion of an intravenous catheter may be lifesaving.

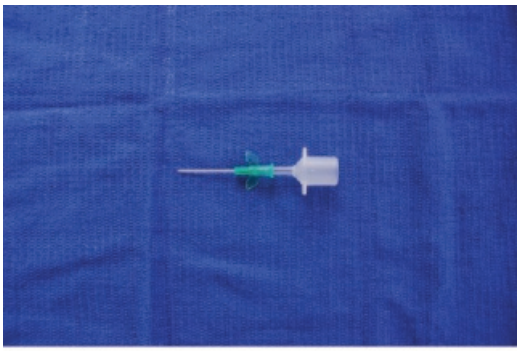
ii) One must be able to attach the 15-mm adaptor of the standard breathing circuit to the Luer-Lok end of the catheter. Two options exist here:

(1) The connectors of the standard 3.5 ETT (Sheridan CF, Hudson RCI, Durham, NC) fit directly onto the end of the intravenous catheter (**Fig. 96-7A**).

(2) A 3-mL Luer-Lok syringe (BD, Franklin Lakes, NJ) with the plunger removed can be connected to the catheter, and the connector from a 7.0 ETT (Mallinckrodt Intermediate Hi-Lo, Covidien, Hazelwood, MO) can fit directly into the syringe (**Fig. 96-7B**).

READ MORE
Scalpel-Bougie technique for emergency front of neck access (FONA), Chapter 166

FIGURE 96-7 Adapting an Intravenous Catheter for Emergency Needle Cricothyrotomy. **A.** The connector from a 3.5 ETT fits into the end of a standard intravenous catheter to convert the Luer-Lok into a 15-mm adaptor. **B.** If a 3.5 ETT connector is not available, a 3-mL syringe with the plunger removed can be attached to the connector of a 7.0 ETT.



A



B

Photos by Shannon Schwartz.

iii) For children over 10, the Scapel-Bougie technique for emergency front of neck access (FONA) can be used.
   (1) Bear in mind that the smallest ETT that can fit over an adult bougie is 6.0.

d) **Clinical pearls**
   i) Distorted or indistinguishable anatomy:
      (1) Patients may have laryngeal anatomy that is unrecognizable either by direct laryngoscopy or fiberoptic bronchoscopy.
      (2) Solution A: Ask an assistant to perform jaw thrust, which may improve visualization of the larynx.
      (3) Solution B: Place a tongue stitch and have assistant retract tongue during visualization attempts.
      (4) Solution C: Identify the trachea by looking for bubbles and directing the ETT toward them.
      (5) Solution D: Bubbles may be created by having an assistant gently squeeze the chest.



If you cannot recognize any structures during direct laryngoscopy, look for air bubbles to locate the trachea.

ii) Abdominal distention may impair ventilation.



Vigorous mask ventilation may insufflate air into the stomach and impair ventilation.

(1) Bag-mask ventilation may insufflate air into the stomach and intestines and limit the ability to ventilate or oxygenate—even to the point of causing a clinical picture like a tension pneumothorax.
(2) Solution A: Minimize inflation by using only as high-peak inspiratory pressures as necessary to achieve adequate ventilate/oxygenate during bag-mask ventilation.
(3) Solution B: Use cricoid pressure to minimize insufflation of air into the esophagus and stomach.
(4) Solution C: Place an orogastric or a nasogastric tube to release the air in the stomach. This may be repeated as necessary when high pressures are required.

CHAPTER SUMMARY FOR MANAGEMENT OF THE PEDIATRIC DIFFICULT AIRWAY

Use your clinical judgment.
Preoxygenate and keep patient well oxygenated throughout.
Use an antisialogogue to dry secretions.
Maintain spontaneous respirations.
Have an LMA immediately available.
Call for experienced assistance early enough to make a difference.
Consider fiberoptic laryngoscopy early.
Know your equipment.
If available, consider extracorporeal membrane oxygenation early.

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97 Mechanical Circulatory Support

Jason Leong • Hunter Bergamasco • Vidya K. Rao

Cardiopulmonary bypass is a form of extracorporeal circulation that provides complete circulatory and respiratory support during cardiac surgery. Effective use of CPB requires knowledge of the CPB circuit and carefully coordinated efforts by the cardiac surgeon, perfusionist, and cardiac anesthesiologist. This chapter will provide an overview of the CPB machine; the steps needed to initiate, maintain, and wean from CPB support; and relevant anesthetic considerations.

1) **Cardiopulmonary bypass**
   a) Cardiopulmonary bypass (CPB) is a form of extracorporeal support in which the physiologic functions of the heart and lungs are temporarily taken over by a machine, often referred to as "the heart-lung machine."
   b) CPB provides four primary functions:
      i) Oxygenation and elimination of carbon dioxide
      ii) Circulation of the blood throughout the body
      iii) Temperature regulation
      iv) Creation of a bloodless surgical field by diverting blood from the heart during cardiac surgery
   c) Blood is drained from the venous circulation, passed through a specialized circuit, where it is oxygenated and temperature-regulated, and returned to the arterial circulation, where it provides end-organ perfusion.
   d) CPB is most commonly utilized during cardiothoracic surgical procedures to create a bloodless, potentially motionless, surgical field, while providing end-organ perfusion and facilitating entry into cardiac chambers and great vessels without systemically entraining air.
   e) Effective use of CPB involves a clear knowledge of the bypass circuit as well as carefully coordinated efforts by the cardiac surgeon, perfusionist, and anesthesiologist.
2) **The cardiopulmonary bypass machine**
   a) The CPB circuit consists of several major components: venous and arterial cannulae, tubing, a reservoir, centrifugal pump, and oxygenator/heat exchanger (Fig. 97-1).[1]
   b) A venous cannula is a flexible plastic tube with holes, or fenestrations, that is inserted into the right atria or vena cava(e) to drain deoxygenated blood from the venous circulation into the reservoir of the pump.
      i) Venous cannulas are commonly inserted with the transesophageal echocardiography (TEE) guidance to ensure proper positioning.
      ii) Dual-stage (cavoatrial) cannulation: A single cannula is inserted into the inferior vena cava (IVC) via a single incision in the right atrium. Blood is drained from the IVC, the RA, and the coronary



The CPB circuit consists of several major components: venous and arterial cannulae, tubing, a reservoir, centrifugal pump, and oxygenator/heat exchanger **(Fig. 97-1)**.



Discussing cannulation strategy with the surgical team and perfusionist is essential for TEE guidance of proper cannula positioning.

FIGURE 97-1 General Components of the Cardiopulmonary Bypass Machine. The venous reservoir **(A)** collects blood drained via the venous cannula. The arterial pump **(B)** pushes blood from the reservoir through the oxygenator (**C**). The cardioplegia pump (**D**) allows for the administration of cardioplegia to the heart. Additional components include the pump suction, root vent, and the left ventricular vent **(E)** as well as a heat exchanger.

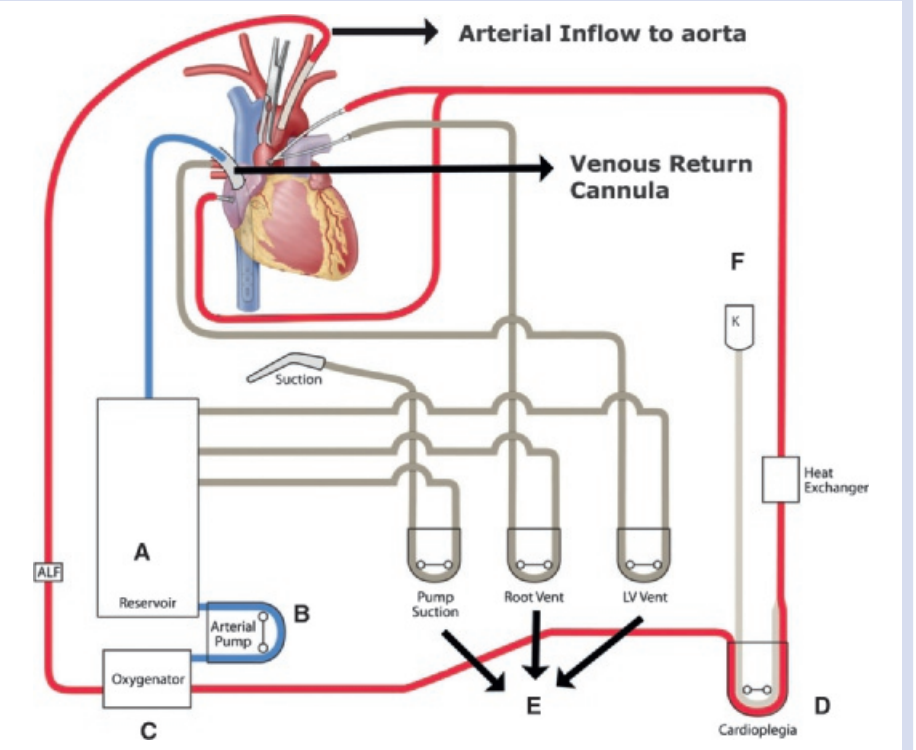


Image courtesy of Bruce Searles, SUNY Upstate Medical University, Department of Cardiovascular Perfusion.

sinus. However, drainage is incomplete, allowing some passage of blood through the cardiopulmonary tree (Fig. 97-2).[1]

iii) Bicaval cannulation: Two venous cannulas are inserted via separate incisions into the superior vena cava (SVC) and the IVC, allowing for complete systemic venous drainage, facilitating a bloodless operative field, and offering better access to the right atrium.

c) The reservoir functions as a holding tank for the deoxygenated blood drained via the venous cannula.
   i) Blood drains into the reservoir using gravity (siphon) drainage or can be augmented using vacuum or a kinetic pump.
   ii) The volume in the reservoir allows for an imbalance between venous drainage and arterial flow.
   iii) Fluids, blood, and medications may be administered via the reservoir.

d) The systemic pump propels blood forward via roller or centrifugal pumps.

FIGURE 97-2 This Schematic Depicts One Possible Cannulation Configuration. Venous drainage occurs via dual-stage (cavoatrial) cannulation **(A)**. Arterial return of oxygenated blood occurs via the aortic cannula **(B)** distal to the aortic cross clamp. Depending on the nature of the procedure, both antegrade and retrograde cardioplegia cannulas **(C)** may be utilized. An aortic root vent **(D)** allows venting of air, blood, or cardioplegia as needed.

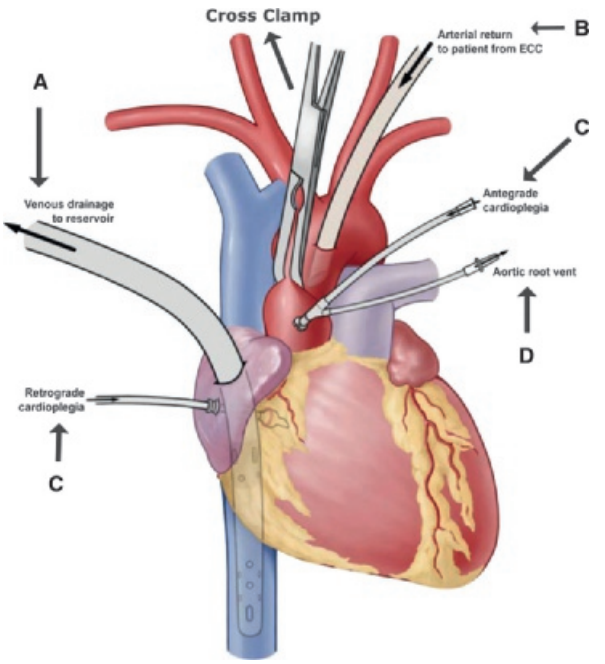


Image Courtesy of Bruce Searles, SUNY Upstate Medical University, Department of Cardiovascular Perfusion.

i) Roller pumps function by using rotating roller heads to compress tubing, producing forward flow.
   (1) Occlusion pressure, or the degree to which the tubing is compressed, must be carefully determined to avoid retrograde flow (if pressure is too low) or hemolysis (if pressure is too high).
   (2) Roller systems are afterload independent and can result in high pressure and tubing rupture in the setting of mechanical blockage.

ii) Centrifugal pumps consist of stacked cones or an impeller that rotate rapidly, generating negative pressure that pulls blood forward.
   (1) These pumps are afterload dependent and will not generate excessive pressure.
   (2) As compared to roller pumps, centrifugal pumps produce less trauma to blood cells and have a lower risk of pumping massive air emboli into the arterial circulation.

e) The oxygenator takes over the physiologic function of the lungs.

i) The oxygenator consists of a thin, gas permeable membrane of hollow microporous fibers, which facilitate gas exchange. The membrane completely separates the blood flowing outside of the fibers from the gas flowing through them.

f) The heat exchanger is essential to temperature regulation.
   i) Blood flows through a system of tubes surrounded by temperature-regulated water that may be used to either heat or cool the blood.

g) An arterial cannula is used to return oxygenated blood from the CPB machine to the patient's systemic arterial circulation.
   i) These cannulas often have metal or hard plastic tips and are most commonly inserted into the ascending aorta. Depending on the nature of the procedure, alternative arterial sites may be cannulated, including the femoral, axillary, and subclavian arteries.
   ii) The patient's cardiac output must pass through the arterial cannula, and the cannula size is determined by the patient's size and flow constraints.
   iii) Flow that is too high may result in arterial dissection or trauma to blood cells.
   iv) Arterial cannulation is commonly performed using TEE guidance to facilitate proper positioning and prevent catastrophic complications, such as flow issues and aortic injury.



In a calcified aorta, proper positioning of the arterial cannula with the tip located centrally in the vessel lumen is important to avoid dislodging calcifications or atheroma, and to avoid dissection of the vessel.

h) Tubing is made from polyvinyl chloride (PVC), which offers durability and flexibility.
   i) Tubing is required to connect the patient to the CPB circuit and to connect the components of the circuit to each other.

3) **Management of cardiopulmonary bypass**
   a) Cannulation
      i) Anticoagulation:
         (1) Anticoagulation is required prior to cannulation as clotting during any phase of CPB is catastrophic.
         (2) Most commonly, a bolus of 300 units/kg of heparin is administered and supplemented as needed until an activated clotting time (ACT) of >480 seconds is confirmed.
         (3) Risk factors for heparin resistance include AT-III ≤60%, preoperative heparin therapy, platelet count ≥300 000, and age ≥65 years.[2]



Anticoagulation is required prior to cannulation as clotting during any phase of cardiopulmonary bypass is catastrophic.

Most commonly, a bolus of 300 units/kg of heparin is administered and supplemented as needed until an activated clotting time (ACT) of >480 seconds is confirmed. ACT thresholds may vary by institution.

      ii) Cannula insertion:
         (1) Once adequate anticoagulation is confirmed, the arterial cannula is inserted in case resuscitation is required.
            (a) Central cannulation (cannula inserted into the aorta) is most common.
            (b) Depending on the procedure, a more peripheral artery may be selected.
         (2) Central cannulation is performed using TEE guidance to ensure proper placement within the lumen of the artery.
         (3) Venous cannulation is then performed. In most cases, dual-stage or bicaval cannulation is performed. Alternatively, the RA may be accessed via cannulation of the femoral vein.
         (4) Venous cannulation is also performed using TEE guidance to ensure proper positioning.



Heparin resistance, which occurs when an adequate ACT is not attained despite therapeutic doses of heparin, may be due to antithrombin deficiency.

      iii) Priming:
         (1) The CPB circuit is primed with crystalloids, colloids, and/or blood products to remove air prior to the initiation of CPB.
         (2) A crystalloid or colloid prime may result in hemodilution as the patient's circulating volume will be composed of his/her blood volume plus the priming volume.

(3) To mitigate hemodilution, retrograde or antegrade autologous priming of the CPB circuit with the patient's blood may be performed.[3]
(4) For patients with anemia and/or small body surface area, priming the circuit with blood products will prevent hemodilution.



While the aorta is cross clamped, it is important to ensure that the LV is not distended, and that there is no electrical activity.

b) **Initiation of CPB**
i) The perfusionist commences flow of oxygenated blood into the systemic circulation via the arterial cannula.
ii) A clamp on the venous limb is opened, and venous blood is gradually drained from the patient into the reservoir at a rate equal to arterial flow.
iii) The goal is to achieve "full flow," which equates to a cardiac index of ~2.4 L/min/m^2.
iv) Aortic cross clamp and cardioplegia
(1) Complete arrest of the heart is required when a motionless surgical field is required.
(2) An aortic cross clamp is applied to the aorta, and cardioplegia solution is infused antegrade into the aortic root, directly into the coronary ostia, or retrograde into the coronary sinus, to achieve electrochemical arrest of the heart. This protects the heart from ischemia.
(3) Cardioplegia solutions are usually composed of blood and concentrated potassium. The temperature of the cardioplegia solution is controlled, with cold cardioplegia providing a cardioprotective effect by decreasing the metabolic demands of the heart.
(4) Application of the aortic cross clamp and infusion of cardioplegia solution may cause left ventricular distention, particularly in patients with aortic regurgitation.
(a) Distention can increase wall tension and myocardial oxygen demand, which may result in ischemia and subendocardial necrosis.[4]
(b) Aortic root vents are inserted proximal to the cross clamp to avoid LV distention.
(5) Vents may also be inserted into left-sided chambers to drain blood and remove air.



The target MAP during CBP is 50-70m mHg but may be adjusted depending on the patient's underlying conditions.

c) **CPB maintenance**
i) Hemodynamics/perfusion
(1) Hemodynamics are monitored using mean arterial pressure (MAP) due to the loss of pulsatile flow.
(2) Vasopressor or vasodilator therapy may be required to achieve the target MAP goal of 50-70 mm Hg. Hypertension results in a decrease in flow delivered by the CPB circuit.
(3) Flows may be transiently reduced at the request of the surgeon, but prolonged flow reductions should be avoided to ensure end-organ perfusion.
(4) Mixed venous saturations, lactate levels, and/or cerebral oximetry are employed to monitor the adequacy of global and cerebral oxygen delivery.
ii) Anticoagulation
(1) Adequate levels of anticoagulation are monitored every 30 minutes on CPB by the perfusionist.
(2) Supplemental heparin is administered to maintain an ACT > 480 (threshold may vary by institution).
(3) In some centers, heparin concentration assays are also being performed every 30 minutes.

iii) Temperature
(1) Nasopharyngeal, tympanic, and/or bladder temperatures are commonly monitored.
(2) Target temperature on CPB depends on the nature of the surgical procedure and clinician preference. Hypothermia decreases metabolic demands and may be utilized for organ protection.
(3) In surgeries where circulatory arrest is required, deep hypothermia, in which temperatures are decreased to as low as 15°C-17°C, may be utilized for cerebral protection.
(4) Rewarming is performed toward the end of CPB to achieve a normothermic core temperature. Vasodilators may be utilized to facilitate rewarming by permitting higher flow rates and augmenting the distribution of blood flow.

iv) pH
(1) There are two methods of pH management while on CPB: alpha-stat and pH-stat. pH is temperature dependent, with the partial pressure of CO_2 decreasing at lower temperatures, creating a respiratory alkalosis.
(2) Alpha-stat maintains a pH of 7.4 and a $PaCO_2$ of 40 mm Hg as measured at 37°C *in vitro*. At colder temperatures, the exogenous CO_2 is not added to the system to compensate for a respiratory alkalosis. Alpha-stat is more commonly used today.
(3) pH-stat corrects for the respiratory alkalosis seen at lower temperatures by adding exogenous CO_2 into the system to achieve a pH of 7.4 and $PaCO_2$ of 40 mm Hg. There is some evidence to suggest that pH-stat promotes cerebral vasodilation, resulting in a loss of autoregulation and an increased risk of movement of microemboli into the cerebral circulation.



Trendelenberg positioning during deairing helps to prevent air embolizing to the brain.

d) **Separation from CPB**
i) During separation from CPB, the responsibility for systemic perfusion is gradually transitioned from the CPB machine to the patient's heart.
ii) In preparation for separation from CPB, air pockets that have developed in the pulmonary veins or cardiac chambers must be removed. Air may be aspirated from a vent in the aortic root, and Valsalva maneuvers may be utilized to facilitate air removal.
iii) The aortic cross clamp is removed, and the heart begins to reperfuse and contract. Pacing may be required, depending on the patient's underlying rhythm.
iv) The venous line is progressively occluded to preferentially divert blood through the cardiopulmonary tree and away from the venous cannula. This increases cardiac preload, which augments native cardiac output.
v) As the heart begins to eject, arterial pump flow is gradually decreased in 0.5-1 L increments until the heart assumes full responsibility for systemic blood flow. The venous cannulas are then clamped, and volume from the pump may be administered to the patient via the arterial cannula.
vi) If hemodynamics and surgical outcome are satisfactory, the venous cannula is first removed, to facilitate venous return to the heart. The aortic cannula is removed following partial reversal of anticoagulation with protamine.



A pre-CBP checklist is recommended to ensure safe initiation of CPB.

4) **Anesthetic considerations**
a) **Prior to CPB**
i) A pre-CPB checklist is often utilized to ensure that the patient is safely transitioned onto CPB.
ii) Anesthesia:
(1) Prior to CPB, inhalational anesthetic agents may be administered. During CPB, however, blood flow is largely diverted away from the cardiopulmonary

circulation. An alternative anesthetic strategy should be selected..

(2) Depth of anesthesia monitors may be of benefit as CPB masks many of the sympathetically mediated signs of awareness or inadequate anesthesia.

iii) Monitoring:

(1) Standard American Society of Anesthesiologists (ASA) standard monitoring modalities are required, including EKG, noninvasive blood pressure, pulse oximetry, and temperature monitoring.

(2) Invasive monitoring, including arterial pressure and central venous pressure monitoring, is required for hemodynamic management. In some cases, pulmonary artery catheterization is performed to facilitate intracardiac pressure monitoring and evaluation of cardiac output.[5]

(3) TEE is utilized to confirm cardiac function and appropriate positioning of arterial and venous cannulas and detect complications.

(4) Cerebral oximetry allows for monitoring of regional oxygen saturation and may be used during cardiac surgery. Baseline levels are obtained to allow monitoring of trends during the case. Some studies have found an association between cerebral desaturations and postoperative cognitive deficits.[6]



Evaluating for preexisting aortic insufficiency prior to initiation of bypass is important as moderate-severe aortic insufficiency predisposes the heart to LV dilation and ineffective antegrade cardioplegia unless the cardioplegia is delivered directly into the coronary arteries.

iv) Hemodynamics:

(1) Systemic pressure will need to be transiently decreased during arterial cannulation. This may be achieved via vasodilator administration as an infusion or as a bolus dose.

(2) Vasopressor support may be required to manage hypotension that occurs secondary to hemodilution associated with crystalloid/colloid circuit priming, or from hypovolemia during retrograde autologous priming.

(3) Patients with low hemoglobin or a small body surface area may require blood product transfusion to mitigate the hemodilution that occurs with crystalloid/colloid prime.

b) **Initiation of CPB**

i) Anesthesia

(1) Once "full flow" is achieved, ventilator support may be discontinued as oxygenation and carbon dioxide removal will be performed by the CPB circuit.

(2) Anesthesia must be administered either as a total intravenous anesthetic (TIVA) or as volatile agents administered by the perfusionist directly into blood flowing through the CPB circuit. Anesthetic depth must be carefully monitored during this transition.

ii) Monitoring:

(1) A pupillary examination should be performed, and the patient's face should be evaluated for asymmetric color changes or swelling. Abnormalities may indicate cannula malposition.

(2) Arterial pulsatility will be lost, so MAP must be monitored.

(3) TEE may be utilized during this process to ensure adequate decompression of the heart and to detect complications.

iii) Hemodynamics

(1) Transient hypotension and a reduction in central venous pressure may be seen as a patient is transitioned onto CPB. Transient hypotension is generally not problematic and may be managed with vasoactive infusions. Sustained hypotension should be managed aggressively and should prompt evaluation for complications, such as aortic dissection.

c) **Maintenance of CPB**

i) Anesthesia:

(1) Anesthetic requirements may decrease while on CPB due to hypothermia and increase to normal levels during the rewarming phase.

(2) Depth of anesthesia monitoring strategies may be of benefit as indicators of awareness, such as increased heart rate and elevated temperature may be masked while on CPB.

(3) Volatile agents may be administered by the perfusionist via the gas inlet to the oxygenator.

(4) TIVA may be provided, either by infusion or as bolus doses.

(5) The once-favored high-dose opiate strategy has largely fallen out of favor, with opiates now used as part of a multimodal pain regimen in order to facilitate early extubation and mobility.[7]



Left ventricular distension increases myocardial oxygen demand, which can cause ischemic injury during aortic cross clamp.

ii) Monitoring

(1) Standard ASA monitors are continued during CPB. However, pulse oximetry is of limited utility during this phase due to the lack of arterial pulsatility. The EKG is used to monitor for cardioplegic arrest; electrical activity may indicate a need for additional cardioplegic solution.

(2) MAP and central venous pressure are monitored via arterial and central lines. Trends in cerebral oximetry are also observed.

(3) TEE may be performed to monitor for cardiac decompression and avoidance of ventricular distention.

(4) Laboratory analysis from both the arterial and venous limbs of the circuit is performed at regular intervals to ensure adequate oxygen delivery; evaluate oxygen consumption and carbon dioxide removal; monitor acid-base status, electrolytes, and serum glucose levels; trend hemoglobin and hematocrit levels; and maintain appropriate levels of anticoagulation. Laboratory analysis and management should be performed in concert with the perfusionist.

(5) Urine output is closely followed throughout CPB as a marker of adequate end-organ perfusion.

(6) Depending on the surgical procedure, advanced neuromonitoring strategies, such as electroencephalography and evoked potential monitoring, may be utilized.

iii) Hemodynamics:

(1) While no hemodynamic targets have been rigidly identified, MAP is usually maintained between 50 and 70 mm Hg. Higher pressures may be desirable in patients with chronic hypertension or known severe cerebrovascular disease.

(2) Hypertension may indicate sympathetic stimulation, inadequate anesthesia, or cannula malposition.

(a) Pump flow rate may need to be transiently decreased while vasodilator therapy is initiated.

(b) Prolonged flow reduction should be avoided.

(3) Transient hypotension at the request of the surgeon is usually well tolerated, but prolonged hypotension should be avoided. Adequate flow rate should be maintained throughout CPB, and vasoconstrictor therapy may be initiated as needed.

(4) Elevations in central venous pressure, which may indicate compromised venous drainage and inadequate decompression of the heart, require prompt evaluation and management.



Circulatory arrest may be required for certain procedures. Advanced neuromonitoring and cerebral protection strategies are essential to optimize neurologic outcomes.

iv) Fluids and electrolytes
(1) Electrolytes are monitored at frequent intervals throughout CPB.
(2) Hyperkalemia may occur as a result of cardioplegia administration. Electrolyte levels may be corrected to normal levels.
(3) Hyperglycemia may develop as a result of surgical stress response, even in nondiabetic patients. A normoglycemic state should be maintained throughout CPB, with supplemental insulin administered if needed.
(4) Urine output is monitored throughout CPB as a marker of end-organ function and to monitor the response to diuretic therapy if administered.
(5) The volume level in the CPB reservoir is carefully monitored. Supplemental fluids in the form of crystalloid or colloid may be administered if the reservoir volume is low. If excess volume is noted, diuretic therapy or ultrafiltration may be utilized to remove fluids.
(6) The patient's hemoglobin is carefully monitored.
a) In cases of excessive hemodilution or concern for inadequate oxygen delivery, blood products may be administered.

v) Anticoagulation
(1) ACTs are monitored every 30 minutes, and supplemental heparin is administered as needed to maintain ACT > 400 seconds.

vi) Temperature
(1) Hypothermia is often utilized to decrease metabolic demand and protect end organs from ischemic injury. The degree of hypothermia depends on surgical preference and the surgical procedure.
(2) Rewarming occurs prior to separation from CPB to return patients to a normothermic state.

d) **Separation from CPB**

i) Anesthesia:
(1) Anesthetics are increased during rewarming as anesthetic requirements may increase during this phase.
(2) The patient's lungs are gently suctioned and re-expanded, and mechanical ventilation is resumed. Inhalational anesthetics may be re-initiated during this phase.
(3) Epicardial pacemaker wires are connected and tested. Titratable vasoactive infusions, including inotropes, vasoconstrictors, and vasodilators, are prepared and initiated to optimize hemodynamics.



Heart block or bradyarrhythmias are common after cardiac surgery. Well-positioned epicardial pacemaker wires and a functioning pacemaker controller are necessary for patient safety.

ii) Monitoring
(1) Standard ASA monitors and invasive hemodynamic monitors remain in place. Arterial pulsatility returns as the cross clamp is removed and the heart reperfuses and begins to contract.
(2) Temperature monitoring is also of particular importance during this phase as normothermia must be achieved to mitigate the risks of arrhythmia and coagulopathy.
(3) TEE evaluation is performed to assess adequacy of surgical intervention, evaluate function, identify and assist in removal of intracardiac air pockets, and guide resuscitation.

iii) Hemodynamics
(1) Vital signs (including systemic, central venous, and pulmonary artery pressures, and cardiac output), visual inspection of the heart in the surgical field, and TEE findings should be incorporated into the hemodynamic assessment.
(2) Vasoactive infusions are initiated and titrated based on global hemodynamic assessment. The patient should also be assessed for a need for advanced

therapies, including inhaled pulmonary vasodilators or mechanical circulatory support.

(3) The EKG is carefully inspected for evidence of arrhythmia or ischemia. Cardiac pacing may be initiated if needed.

(4) Volume resuscitation is initiated and may be administered via the CPB machine or directly to the patient. Post-CPB bleeding must be identified and the degree of ongoing blood loss incorporated into the resuscitation strategy.

(5) Careful coordination with the perfusionist and cardiac surgeon is essential as the patient is weaned off of CPB.



Careful coordination with the perfusionist and cardiac surgeon is essential as the patient is weaned off of CPB.

iv) Oxygenation and ventilation

(1) Re-expansion of the lungs should be carefully assessed in the field. SpO_2 and end-tidal CO_2 are closely monitored and corroborated with arterial blood gas sampling.

v) Fluids/electrolytes

(1) Acid-base status and electrolytes should be carefully reviewed for derangements and corrected to normal levels.

(2) The degree of hemodilution should be assessed as oxygen demand will increase with normothermia.

(3) Volume resuscitation may be performed with crystalloids, colloid solutions, and/or blood products as needed.

vi) Anticoagulation

(1) Following successful separation from CPB, heparin is reversed with protamine. The surgical field should be monitored for hemostasis, and blood products transfused if needed.

(2) ACT and, in some institutions, heparin concentration are evaluated to ensure return to baseline.

(3) A complete blood count, coagulation studies, and thromboelastography may be performed to identify derangements and to guide correction of coagulopathy with blood products and prothrombotic agents.

CHAPTER SUMMARY FOR CPB

General Considerations	Cardiopulmonary bypass creates a unique physiologic state that can come with a host of potential complications. Optimization of a number of hemodynamic, physiologic, and laboratory parameters are essential to safety initiating, maintaining, and separating from CPB. Ensuring proper anticoagulation, cannulation, bypass flow, and myocardial protection are vital to safely coming on and off bypass.
Pre-bypass Considerations	Adequate anticoagulation should be administered and confirmed. Cannula positioning should be assessed by TEE. Hemodilution, hypovolemia, and hypotension may occur following initiation of CPB.
Intra-bypass Considerations	MAP should be maintained between 50-70 mm Hg. Cerebral oximetry and urine output may help assess adequacy of perfusion. Hypertension should be avoided to allow for optimal circuit flow. Strategies to minimize myocardial oxygen demand during aortic cross clamp are essential.
Post-bypass Considerations	Separation from bypass requires normothermia, normalization of electrolytes and acid-base status, and optimization of hemodynamics with adequate inotropy, heart rate, and vascular tone. Evaluation of surgical repair and assessment for complications should be performed via TEE. Resuscitation will be required during the post-CPB period and should continue as the patient transitions to the intensive care unit.

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98 Ischemic Heart Disease

Lyndsey C.M. Grae • Bryan S. Ahlgren

Ischemic heart disease (IHD) is a broad term used to describe a number of closely related syndromes that share a common central pathology—an imbalance between myocardial oxygen supply and demand. Over 90% of myocardial ischemia is due to atherosclerotic obstruction of coronary artery blood flow.

1) **Epidemiology**
   a) Coronary artery disease (CAD) is the nation's leading killer of both men and women.
   b) About 790 000 people in the United States suffer a myocardial infarction (MI) annually.[1] Both modifiable and nonmodifiable risk factors have been implicated.
      i) Modifiable risk factors
         (1) Include obesity, physical inactivity, high carbohydrate intake, smoking, excessive alcohol consumption, high trans-unsaturated fat intake, stress, hypertension, hyperlipidemia, hyperlipoproteinemia, postmenopausal estrogen deficiency, and diabetes
      ii) Nonmodifiable risk factors
         (1) Include advancing age, family history, and genetic abnormalities in lipid metabolism along with genetic predisposition.
         (2) The extent of impact of a positive family history is yet to be determined, however, a patient's risk for MI is estimated to double if they have a first-degree male relative diagnosed with an MI before age 55 or female relative before age 65.
         (3) With new capabilities of genome-wide association studies over 160 genetic loci have been identified that may contribute as modest risk factors for CAD. The caveat being, these risk loci are extremely common in the general population and further studies are needed to qualify their significance.[2]
2) **Pathophysiology**
   a) **Determinants of myocardial O_2 supply/demand**
      i) Myocardial O_2 supply is dependent on coronary artery diameter, aortic diastolic pressure, left ventricular (LV) diastolic pressure, and arterial O_2 content.
      ii) Myocardial O_2 demand is determined by ventricular wall tension (often described by the Law of LaPlace), heart rate, and contractility.
         (1) Law of LaPlace: Wall tension (T) is proportional to intraventricular pressure (P) × ventricular radius (r); ($T \propto P \cdot r$)
         (2) Tachycardia increases myocardial O_2 demand through increased myocardial work. Tachycardia also shortens the diastolic filling period, thus reducing the time for optimal coronary perfusion.[3]
         (3) Hypervolemia increases ventricular wall tension and myocardial O_2 demand. At the same time, coronary perfusion (O_2 supply) is reduced in the distended ventricle because the increased LV end diastolic pressure limits coronary flow.[3]



Myocardial O_2 demand is dependent on ventricular wall tension, heart rate, and contractility.

      iii) Anemia can upset both sides of the supply-and-demand equation.
         (1) Anemia decreases O_2 carrying capacity. This decreases O_2 content which reduces O_2 supply.

(2) Increased heart rate and cardiac output secondary to anemia can increase demand.[3]

b) **Atherosclerosis (Fig. 98-1)**

i) Atherosclerosis is the deposition of atheromatous plaques in the intimal layer of arteries.

ii) This process begins early in life and gradually narrows the lumen of coronary arteries.

iii) The risk of an individual developing detectable IHD depends in part on the number, distribution, and structure of atheromatous plaques, and the degree of narrowing they cause.

iv) A fixed obstructive lesion of 75% or greater generally causes symptomatic ischemia induced by exercise; with this degree of obstruction, the augmented coronary flow provided by compensatory vasodilation is no longer sufficient to meet even moderate increases in myocardial demand.

v) A 90% stenosis can lead to inadequate coronary blood flow even at rest.[3]

vi) Slowly developing occlusions may stimulate collateral vessels over time, which can protect against distal myocardial ischemia and infarction even with an eventual high-grade stenosis.

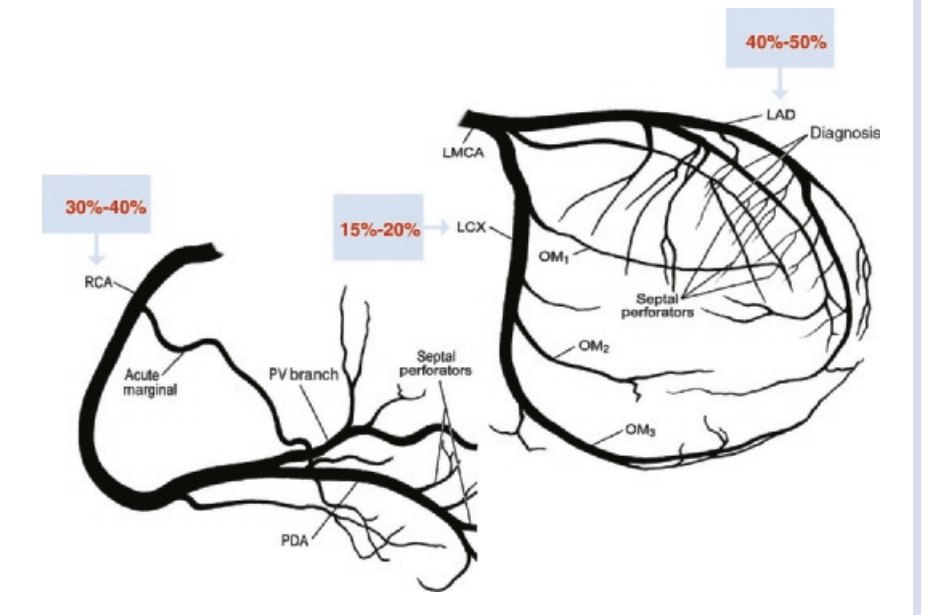
(1) Common distribution patterns of stenotic lesions and frequency of involvement[3]:

(a) Left anterior descending (LAD) 40%-50%

(b) Left circumflex (LCX) 15%-20%

(c) Right coronary artery (RCA) 30%-40%

FIGURE 98-1 Typical Left and Right Coronary Artery Anatomy and Frequency of Common Stenotic Coronary Lesions by Location



(2) Clinically significant stenotic plaques may be located anywhere within these vessels but tend to predominate within the first several centimeters of the LAD and LCX and along the entire length of the RCA.[3]

c) **Atherosclerotic plaque changes**

i) In most patients experiencing acute coronary syndrome (unstable angina, MI, or sudden cardiac death [SCD]), a previously stable plaque has undergone one of the following acute changes followed by thrombosis and compromised distal coronary vessel flow:

(1) Rupture/fissuring, exposing the highly thrombogenic plaque constituents

(2) Erosion/ulceration, exposing the thrombogenic subendothelial basement membrane to blood

(3) Hemorrhage into the atheroma, expanding its volume

(4) Causes of abrupt changes in plaque configuration and superimposed thrombosis are complex and poorly understood. Influences, both intrinsic (eg, plaque structure and composition) and extrinsic (eg, blood pressure, platelet reactivity), are significant factors.[3]

3) **Clinical syndromes of IHD**

a) **Angina pectoris**

i) Angina pectoris is a symptom complex of IHD characterized by paroxysmal and usually recurrent attacks of substernal or precordial chest discomfort (variably described as constricting, squeezing, choking, pressure, or knifelike).

ii) All angina is caused by varying combinations of increased myocardial O_2 demand and decreased myocardial perfusion, owing to disrupted plaques, fixed stenotic plaques, vasospasm, thrombosis, platelet aggregation, or embolization.

iii) Reduced blood flow causes transient (15 seconds to 15 minutes) myocardial ischemia that falls short of inducing the cellular necrosis that defines infarction. Three overlapping patterns of angina pectoris exist:[2]

(1) Stable or typical angina

(2) Prinzmetal or variant angina

(3) Unstable or crescendo angina

b) **Myocardial infarction (MI)**

i) MI, also known as "heart attack," is the death of cardiac muscle resulting from ischemia and is described as transmural or subendocardial infarction.

(1) Transmural

(a) Ischemic necrosis involves the full or nearly full thickness of the ventricular wall in the distribution of a particular coronary artery, often associated with ST segment elevation on ECG.

(b) This pattern of infarction is usually associated with coronary atherosclerosis, acute plaque change, and superimposed thrombosis.

(2) Subendocardial (nontransmural)

(a) Infarct constitutes an area of ischemic necrosis limited to the inner one-third to one-half of the ventricular wall. The subendocardial zone is the least well-perfused region of myocardium and therefore most vulnerable to any reduction in coronary flow. Common teaching is that lesion-associated ECG changes include ST segment depression; however, alternate tracing abnormalities may also be present.

(b) This type may extend laterally beyond the perfusion territory of a single coronary artery.

(c) These infarcts are typically due to a plaque disruption leading to coronary thrombus which is lysed before myocardial necrosis extends across the major thickness of the wall.

(d) Another possible mechanism is decreased coronary perfusion due to sufficiently prolonged and severe reduction in systemic blood pressure, as in shock. In cases of global hypotension, resulting subendocardial infarcts can be global.[3]



Sudden cardiac death may be the first manifestation of IHD, and the mechanism is most often a lethal arrhythmia.

c) **Sudden Cardiac Death (SCD)**
   i) SCD is defined as unexpected death from cardiac causes within minutes after symptom onset or without the onset of symptoms.
   ii) Mechanism of SCD is most often a lethal arrhythmia (eg, asystole, ventricular fibrillation).
   iii) SCD due to IHD is thought to be caused by ischemic injury encompassing the conduction system itself, or electrical irritability of myocardium that may be distant from the conduction system, induced by ischemia or other cellular abnormalities.

d) **Chronic IHD or ischemic cardiomyopathy**
   i) Chronic IHD is typically characterized by prior MI and often previous coronary arterial bypass graft (CABG) surgery or percutaneous interventions with ischemic insult accumulation and progressive damage.
   ii) This process usually constitutes postinfarction cardiac decompensation and failure owing to exhaustion of the compensatory hypertrophy of viable myocardium that is in jeopardy of ischemic injury. Onset and severity of symptoms are highly variable depending on extent of disease.[3]

4) Severity or disease burden of MI depends on a number of factors including:
   a) **Location, severity, and rate of development of coronary atherosclerotic obstructions**
   b) **Size of the vascular bed perfused by the obstructed vessels**
   c) **Duration of the occlusion**
      i) Loss of contractility can occur in <2 minutes, irreversible cell injury in 20-40 minutes, and microvascular injury typically takes over 1 hour to develop.
   d) **Metabolic needs of the myocardium at risk**
   e) **Extent of collateral blood vessels**
   f) **Presence, site, and severity of coronary arterial spasm**
   g) Other clinical factors including alterations in blood pressure, heart rate, and cardiac rhythm

5) **Complications secondary to MI:** Nearly three-fourths of patients have one or more complications following acute MI, which include the following[3]:
   a) **Contractile dysfunction**
      i) MI produces abnormalities in LV function approximately proportional to the size of infarct. This can range from mild dysfunction to cardiogenic shock.
   b) **Arrhythmias**
      i) Many patients will have MI-associated arrhythmias that include sinus bradycardia, heart block (including asystole), tachycardia, premature ventricular contractions or ventricular tachycardia, and ventricular fibrillation.
   c) **Myocardial rupture**
      i) Cardiac rupture syndromes result from mechanical weakening that occurs in necrotic and subsequently inflamed myocardium. These include:
         (1) Rupture of the ventricular free wall (most common), with hemopericardium and cardiac tamponade, usually fatal. Typically occurs 3-7 days postinfarction.
         (2) Rupture of the ventricular septum (less common), leading to a left-to-right shunt.
         (3) Papillary muscle rupture (least common) can be partial or complete. Complete papillary muscle rupture more commonly results in the acute onset of severe mitral regurgitation.

(a) Both the anterolateral and the posteromedial papillary muscles are at risk for rupture.
(b) Posteromedial papillary muscle rupture is more common due to its single blood supply compared to the dual supply of the anterolateral papillary muscle.

d) **Right ventricular dysfunction**
   i) Rare, though may accompany septal or posterior infarct. A right ventricular infarct of either type can yield serious functional impairment.
e) **Infarct extension**
   i) New necrosis may occur adjacent to an existing infarct.
f) **Pericarditis**
   i) A fibrinous or fibrohemorrhagic pericarditis can develop the second or third day following a transmural infarct and usually will resolve.
   ii) Dressler syndrome is a secondary pericarditis where myocardial and/or pericardial injury related to an MI initiates an inflammatory immune response resulting in pericarditis with or without pericardial effusion.
g) **Mural thrombus**
   i) The combination of local myocardial contractile abnormalities (causing stasis) and endocardial damage (causing a thrombogenic surface) can lead to mural thrombosis formation and potentially to thromboembolism.
h) **Ventricular aneurysm**
   i) True aneurysms of the ventricular wall are bounded by myocardium that has become scarred.
   ii) Aneurysms of the ventricular wall are a late complication and most commonly result from a large transmural infarct (often one that has undergone expansion) that heals into a large region of thin scar tissue, which paradoxically bulges during systole.
   iii) Aneurysm formation as demonstrated in **Figure 98-2** can lead to mural thrombus, arrhythmias, and heart failure, but rupture of the fibrotic wall is infrequent (unlike ventricular pseudoaneurysm).
i) **Papillary muscle dysfunction**
   i) As described previously, on rare occasions after MI, early dysfunction of a papillary muscle occurs due to its rupture.
   ii) More frequently, postinfarct mitral regurgitation results from early ischemic dysfunction of a papillary muscle and underlying myocardium and later from papillary muscle fibrosis and shortening or ventricular dilation.

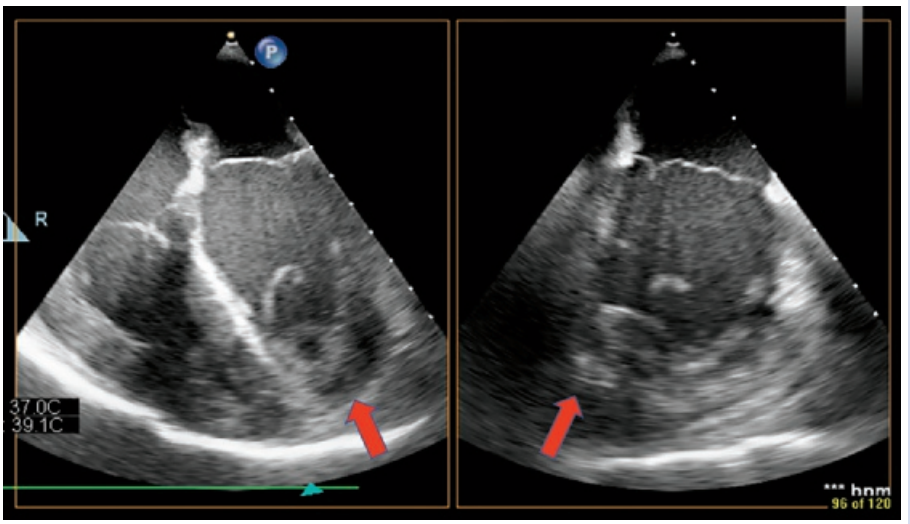
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Congenital heart disease, Chapter 85.

6) **Treatment modalities**
   a) **Reperfusion (Fig. 98-3)**
      i) The purpose of reperfusion therapy is to restore coronary blood flow to the area at risk for infarction and possibly rescue the ischemic (but not yet necrotic) heart muscle.
      ii) Removal of thrombus reestablishes flow through the occluded coronary artery in most cases; early reperfusion can salvage myocardium and thereby limit infarct size, with consequent improvement in both short- and long-term function and survival.
      iii) Restoration of coronary flow (reperfusion) is accomplished via
         (1) Thrombolysis
            (a) IV administration of fibrinolytic agents, which activate plasminogen (tPA, streptokinase, alteplase, etc.)
            (b) Reserved for situations where timely access to percutaneous coronary intervention (PCI) is not possible



The most effective way to salvage ischemic myocardium threatened by infarction is to restore tissue perfusion as rapidly as possible. Figure 98-3 has post-MI reperfusion timeline goals.[4]

FIGURE 98-2 Example of an Apical Aneurysm Seen on Transesophageal Echocardiogram After Prior MI With Subsequent Left Ventricular Clot Formation Due to Abnormal Flow and Contractility in Area of the Aneurysm



Images generously contributed by Dr. Tamas Seres, MD, Division of Cardiothoracic Anesthesia, University of Colorado SOM Department of Anesthesiology.

iv) Percutaneous coronary intervention (PCI) with angioplasty
(1) Primary PCI is preferred for acute ST elevation MI, if performed in a timely fashion by an expert operator.[4]
(2) PCI-related delay >60-120 minutes appears to eliminate the survival benefit of primary PCI over fibrinolysis.[4]
v) Coronary artery bypass grafting (CABG)
(1) In general, primary indications for CABG include[5]:
(a) Three-vessel disease with proximal LAD stenosis
(b) Single proximal left main stenosis with ischemia
(c) Multivessel disease with ejection fraction (EF) > 50%
(d) Failed PCI with ongoing ischemia
(e) Unfavorable anatomy for PCI
(f) Significant angina despite alternative therapy

b) **Medical therapy**
i) Medical therapy is critical for the postinfarct patient to optimize myocardial O_2 supply/demand, stabilize atherosclerotic plaques, and inhibit platelet function.
ii) In almost all instances (with the exception of antiplatelet), these medications should be continued up until the time of surgery[6] (**Table 98-1**).

7) **Anesthetic considerations for the patient with CAD**
a) **Preoperative evaluation**
i) History
(1) Preoperative history should be tailored to elucidate history of symptoms that may indicate CAD.
(2) History of known risk factors as well as chest pain with or without radiation to the inner aspect of the arm or neck; dyspnea on exertion, orthopnea;

FIGURE 98-3 Reperfusion Time Goals and Standards Based on Intervention Type After Acute MI

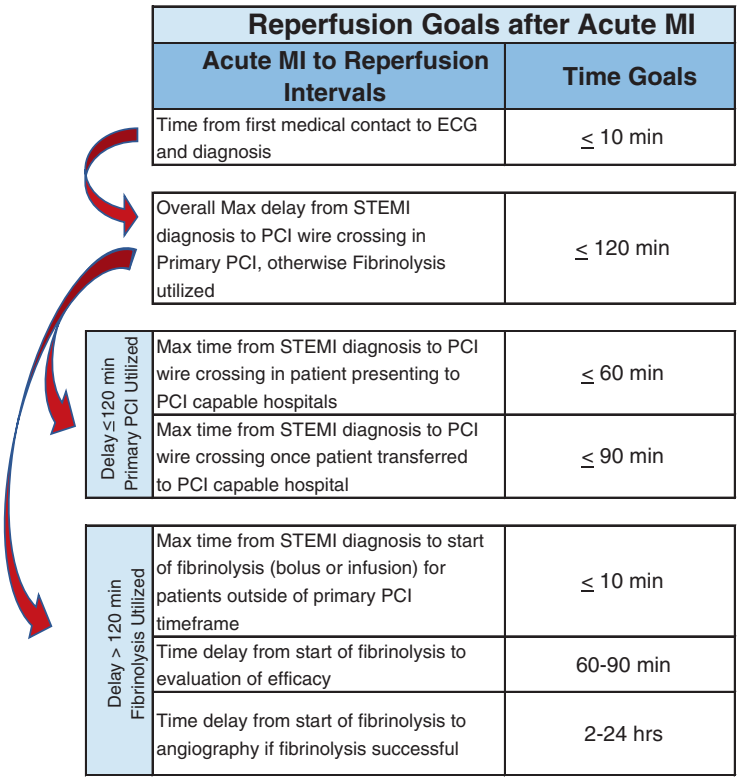


*Figure Adapted from 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. Derived from Ibanez B, James S, Agewall S, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: the task force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). *Eur Heart J*. 2018;39(2):119-177.

paroxysmal nocturnal dyspnea; nocturnal coughing; previous or current peripheral or pulmonary edema; and history of MI should all be queried.

(3) Ability to perform moderate physical activity equal to or greater than four metabolic equivalents (walking up a flight of stairs or up a hill, walking flat at 4 mph, run a short distance) usually infers a low perioperative risk for cardiac ischemia.

TABLE 98-1 Medical Therapy Aimed at Limiting Cardiac Ischemic Episode Progression and Recurrence

β-Adrenergic antagonists	Vasodilators: coronary or systemic
Statins	ACE inhibitors
Antiplatelet medications: (aspirin vs. clopidogrel)	Calcium channel blockers
Anticoagulant therapies	Nitrates

TABLE 98-2 Physical Examination in a Patient at Risk for CAD

Head and neck	Examination for evidence of jugular venous distension, or carotid bruits
Chest	Auscultation/percussion for evaluation of rales, rhonchi, wheeze, or effusions
Cardiac	Auscultation/palpation for evaluation of heaves, thrills, murmurs, rubs, arrhythmia
Abdominal	Examination for pulsatile mass, hepatomegaly, hepatojugular reflex
Extremity	Examination for the presence of cyanosis, clubbing, edema, capillary refill. Blood pressure should be checked in both arms.

ii) Physical examination (**Table 98-2**)

iii) Laboratory examination—Laboratory examination should be tailored to patient and type of surgery per American Society of Anesthesia practice guidelines.[7]

(1) Baseline hemoglobin or hematocrit is not routinely indicated. Measurement is suggested for patients with liver disease, extremes of age, or with history of bleeding, anemia or hematologic diseases. Measurement should be considered for younger patients undergoing major surgery that is expected to result in significant blood loss.

(2) Coagulation studies may be indicated if any history of bleeding disorders, anticoagulant use, and liver or renal dysfunction.

(3) Consider chemistry panel based on medication regimen, history of endocrine disorders, and risk of renal or liver dysfunction.



The decision to obtain further noninvasive studies in a patient with possible CAD should be instituted when suspicion is high, and the outcome of these tests would alter patient care. Emergency surgeries and life-saving procedures should not be delayed for further cardiac testing regardless of the patient's CAD risk/status.

iv) Noninvasive studies

(1) Consider a pre-op ECG in patients with known CAD, significant arrhythmias, peripheral vascular disease, cerebrovascular disease or other significant heart disease unless they are undergoing only low-risk surgery.[8]

(2) Although this is not mandatory, it may provide additional information regarding their cardiac conduction and any previous ischemic events.

(3) The predictive value of noninvasive stress imaging abnormalities for perioperative ischemic events is comparable between available techniques but the accuracy varies with the prevalence of CAD.

(a) For elevated-risk patients, exercise stress testing and pharmacologic stress testing may not be necessary if they demonstrate excellent functional capacity. Conversely, those with moderate-to-good or poor functional capacity may require stress testing procedures if the results will alter patient management or discussions.[8]

(4) The American College of Cardiology/American Heart Association has a recommended risk stratification algorithm and an algorithm for perioperative cardiac assessment in order to aid clinicians in the management of patients with CAD or significant risk factors for CAD undergoing noncardiac surgery.[8]

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Preanesthesia assessment, Chapter 4.

b) **Intraoperative management**

i) Type of anesthetic

(1) There is no convincing evidence that either general or regional anesthesia is superior in reducing cardiac events in the perioperative period.[6,8]

(2) Decision on type of anesthesia should be based on type of surgery, patient preference, and skill of practitioner.

(3) In either case, maintaining tight hemodynamic control close to patient's baseline vitals will yield the best long-term results.

ii) Premedication

(1) Premedication of the patient with CAD may alleviate sympathetic activation and its detrimental effect on myocardial O_2 demand. Careful titration is needed in order to prevent respiratory depression with respiratory acidosis, hypoxia, and hypotension.

iii) Monitoring

(1) Intra-arterial pressure monitoring

(a) With few exceptions, the use of intra-arterial pressure monitoring is advisable in patients with known severe CAD or significant risk factors, especially for high-risk procedures.

(2) Central venous pressure (CVP)

(a) CVP monitoring may be useful in prolonged procedures involving significant blood loss or fluid shifts.

(3) Pulmonary artery catheter (PAC) or transesophageal echocardiography (TEE)

(a) May provide valuable information in select patients with reduced EF (<40%), moderate or severe pulmonary hypertension, or severe valvular disease

(b) TEE allows detection of wall motion abnormalities and evaluation of chamber size and contractility.

(4) Neither PAC or TEE has been shown to improve outcome in most clinical studies, therefore routine use is not indicated.[6,8]

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Central venous monitoring and pulmonary artery catheter, Chapter 13

(5) Detection of ischemia

(a) TEE recognition of wall motion abnormalities may be the most sensitive monitor for intraoperative ischemia.[9]

(b) ECG changes may be subtle, including changes in T-wave morphology before less subtle ST segments become depressed.

(c) Initial hemodynamic changes are commonly due to hypertension and tachycardia, with hypotension representing a later and more ominous finding.

(d) If a Swan-Ganz catheter is in place, the pulmonary capillary wedge pressure may elevate in the presence of acute MR due to papillary muscle dysfunction or acute LV dilation or dysfunction.

READ MORE
Transesophageal echocardiography basic views, Chapter 201

c) **Induction and maintenance**

i) Induction and maintenance of anesthesia are aimed at optimizing myocardial O_2 supply and demand variables. Choice of agent is not as critical as the manner in which they are delivered.

ii) Induction goals are to maintain hemodynamic parameters as close to patient's baseline as possible.

iii) Special attention must be paid to the adrenergic stimulation caused by endotracheal intubation.

iv) General anesthesia is typically maintained with a combination of opioid and volatile techniques.

v) Past studies touted that volatile and opioid anesthetics have cardioprotective mechanisms, likely through preconditioning mechanisms. It was thought that protection was greatest with isoflurane then less so with sevoflurane and desflurane in sequence. Newer data suggest that all volatile anesthetics may confer similar cardioprotective levels and that propofol may also be their equal for protecting at risk myocardium.[10,11]

TABLE 98-3 Approach to the Postoperative Patient With Myocardial Ischemia

- Assess and stabilize airway, breathing, and circulation.
- Provide oxygen; attach cardiac and oxygen saturation monitors; establish/maintain IV access.
- If hemodynamically unstable treat according to ACLS protocols.
- Attain 12-lead EKG.
- Consider giving IV β-adrenergic antagonist (metoprolol 5 mg IV) if hemodynamically stable.
- Treat pain/anxiety with IV opioid medications (morphine 2-4 mg).
- Obtain blood for cardiac biomarkers (troponin preferred), electrolytes, hematocrit/hemoglobin.
- Obtain emergent cardiology consultation for patients with cardiogenic shock, left heart failure, or sustained ventricular tachyarrhythmia.
- Consider indications and risk/benefit ratio of giving antiplatelet medications (aspirin, Clopidogrel) to the postoperative patient.
- Further intervention (PCI, anticoagulation) based on clinical scenario.

d) **Postoperative management**
   i) Emergence from anesthesia can put further stress on the myocardium.
   ii) Supplemental O_2 should be given until adequate oxygenation is confirmed.
   iii) Pain should be treated liberally in order to alleviate its sympathetic output, which will increase myocardial O_2 consumption. Control of pain with a fast-onset, short-acting opioid such as fentanyl followed by administration of a longer acting opioid such as hydromorphone is recommended.
   iv) Postoperative shivering should be treated with warming and meperidine.
   v) Hypothermia must be corrected with the use of a forced air warming blanket.



Shivering can greatly increase overall oxygen consumption and should be treated once recognized.

e) **Postoperative ischemia**
   i) The majority of perioperative ischemic episodes occur in the first 3 days following surgery, usually 24-48 hours after surgery.[12,13] The use of ECG as well as laboratory tests may aid in the diagnosis (Table 98-3).

CHAPTER SUMMARY FOR ISCHEMIC HEART DISEASE

Epidemiology	Leading killer of both men and women; about 790 000 MIs each year in the United States
Anesthetic Management	**Preoperative:** Thorough history can be invaluable. Follow current ACC/AHA guidelines for testing specific to CAD severity and surgical risk. Invasive testing should only be considered for patients with active cardiac conditions or if the outcome will significantly alter medical management. **Intraoperative:** No clear evidence of superiority between regional or general. Tight hemodynamic control critical in patients with significant CAD regardless of anesthetic choice geared to optimize myocardial oxygen supply and minimize demand. **Postoperative:** Smooth emergence minimizes stress. Supplemental oxygen should be used. Avoid elevations in heart rate (pain, shivering, etc.). Pain control is critical.
Monitoring	Consider arterial line for high-risk procedures. Consider central line, PAC or TEE as indicated by surgical risk and degree of CAD.

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99 Mitral Valve Disease

Benjamin A. Abrams • Nathaen Weitzel

The mitral valve (MV) is a bileaflet, saddle-shaped structure that promotes unidirectional flow from the left atrium (LA) into the left ventricle (LV). A variety of pathologic processes, involving either the leaflets or the submitral apparatus, can impair normal function of the valve. Lesions can result in either mitral stenosis (MS) or mitral regurgitation (MR). This chapter will discuss the pathophysiology and anesthetic considerations for both.

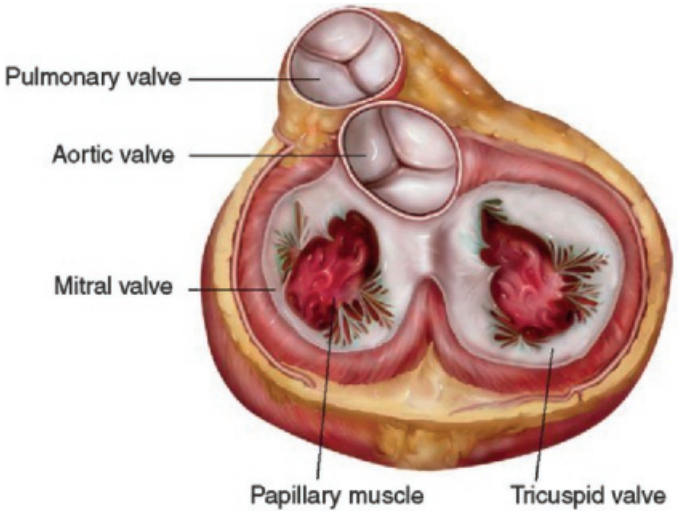
1) **Normal mitral valve anatomy and function**
   a) Composed of anterior and posterior leaflets (Fig. 99-1)
      i) The posterior leaflet is relatively long and thin compared to the anterior and composed of three separate scallops (anterolateral, middle, posteromedial).
      ii) Each leaflet covers approximately the same surface area with respect to the MV orifice.
      iii) Separate regions of the anterior leaflet are defined by the corresponding scallop with which it coapts during systole (Fig. 99-2).
   b) The bases of both leaflets insert onto the mitral annulus, which is a saddle-shaped, dynamic structure that decreases in size as it approaches the LV apex during systole.
   c) A normal MV orifice area is ~4-6 cm^2 in adults.[1,2]
   d) The subvalvular apparatus consists of numerous thin, fibrous structures called chordae tendineae that attach the MV leaflets to two papillary muscles: the anterolateral and posteromedial.
      i) These muscles lie along the endocardium of the LV, and contract during systole to help support coaptation of the mitral leaflets.
      ii) Myocardial ischemia involving the papillary muscles may compromise this function.
      iii) During diastole, the chordal attachments to the papillary muscles support LV function by preventing dilation and increased wall strain; as such, an attempt is made to preserve the submitral apparatus during MV replacement.[1]
2) **Mitral stenosis (MS)**
   a) **Etiology**
      i) MS is most frequently rheumatic in origin.[3]
         (1) Rheumatic fever (RF) involves an immune response that is triggered by cross-reactivity between group A hemolytic streptococcal bacterial antigens and normal MV tissue.[4]
         (2) Recurrent episodes of RF, for example, multiple episodes of untreated streptococcal pharyngitis, increase the risk of later developing rheumatic heart disease (RHD).[5]



The most common cause of mitral stenosis is rheumatic fever.

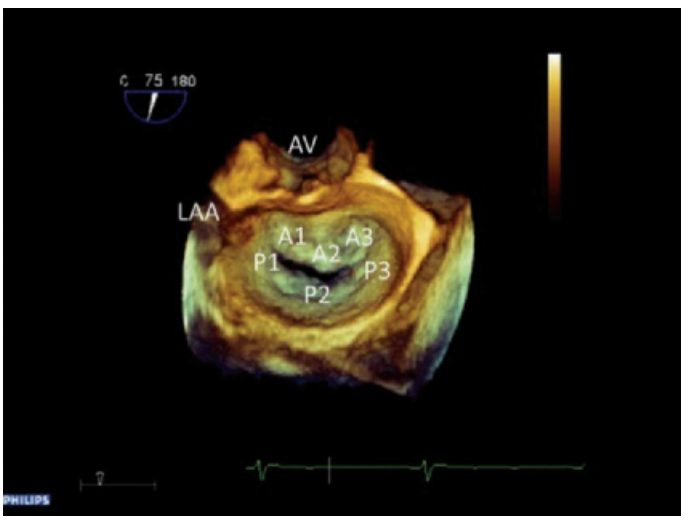
      ii) Senile, calcific MS
         (1) Increasing in incidence in North America
         (2) Calcification of the mitral annulus extends onto the leaflets, causing annular narrowing and leaflet restriction.[6]
      iii) Other forms of nonrheumatic MS
         (1) Congenital causes, such as parachute MV[6]
         (2) MV obstruction due to left atrial masses or tumors also occurs, for example, atrial myxomas.

FIGURE 99-1 The Mitral Valve During Diastole.



The posteromedial papillary muscle within the LV is labeled, and the associated chordae tendineae are visible. Note the close proximity of the aortic valve. See text for details. Reprinted with permission from Perrino Jr. AC, Reeves ST. *A Practical Approach to Transesophageal Echocardiography*. 4th ed. Philadelphia, PA: Wolters Kluwer; 2019.

FIGURE 99-2 Three-dimensional TEE Image of a Normal Mitral Valve from the Left Atrial Perspective, Also Known as the "Surgeon's View."



The MV leaflet segments are labeled according to the Carpentier nomenclature. The left atrial appendage (LAA) and aortic valve (AV) are also visible. Reprinted with permission from Perrino Jr. AC, Reeves ST. *A Practical Approach to Transesophageal Echocardiography*. 4th ed. Philadelphia, PA: Wolters Kluwer; 2019.

TABLE 99-1 Hemodynamic Parameters for Grading MS Severity

	Mean Valve Gradient (mm Hg)[a]	MV Area (cm^2)	Systolic Pulmonary Artery Pressure (mm Hg)
Normal	Negligible	4-6	
Mild	<5	1.5-2.5	<30
Moderate	5-10	1.0-1.5	30-50
Severe	>10	<1.0	>50

[a]For sinus rhythm at heart rate between 60 and 80 beats per minute. Higher heart rates will increase the mean gradient for a given MV area.
Adapted from Baumgartner H, Hung J, Bermejo J, et al. Echocardiographic assessment of valve stenosis: EAE/ASE recommendations for clinical practice. *J Am Soc Echocardiogr*. 2009;22(1):1-23, with permission from Elsevier.

(3) Radiation valvulitis secondary to mediastinal radiation is another possible etiology.[5]

iv) Iatrogenic MS may develop as a complication of MV repair.

b) **Grading MS severity**

i) Echocardiography is an important diagnostic and surveillance tool for managing MS. Parameters for grading severity are listed in Tables 99-1 and 99-2.

READ MORE
TEE evaluation of the mitral valve, Chapter 203.

c) **Pathophysiology**

i) Left atrial pressure (LAP) overload

(1) Progressive narrowing of the mitral orifice leads to increased LAP and compensatory LA enlargement.

(2) This LA remodeling predisposes these patients to the development of atrial fibrillation (AF).

(a) AF is particularly detrimental in MS because atrial contraction can contribute as much as 30% of LV preload.[2]

(b) Rapid ventricular rate (RVR) associated AF decreases diastolic filling time, further reducing LV preload.

TABLE 99-2 General Parameters in Grading MR

	Mild	Moderate	Severe
Regurgitation volume (mL/beat)	<30	30-59	≥60[a]
Regurgitant fraction (%)	<30	30-49	≥50
Estimated regurgitant orifice area (cm^2)	<0.2	0.2-0.39	≥0.4
Doppler vena contracta width (cm)	<0.3	0.3-0.69	≥0.7
Pulmonary vein flow	Systolic dominance	Normal or systolic blunting	Systolic flow reversal
Associated findings			LAE, LVE

Note that the severity of MR can vary depending on the SVR, which is an important consideration for assessment in the setting of general anesthesia. Other qualitative Doppler variables are also relevant. For example, and eccentric, well-hugging jet is usually considered severe until proven otherwise.
[a]May be less in the setting of LV dysfunction.
LAE, left atrial enlargement; LVE, left ventricular enlargement.
Adapted from Zoghbi WA, Adams D, Bonow RO, et al. Recommendations for noninvasive evaluation of native valvular regurgitation: a report from the American Society of Echocardiography Developed in Collaboration with the Society for Cardiovascular Magnetic Resonance. *J Am Soc Echocardiogr*. 2017;30(4):303-371, with permission from Elsevier.

(3) Increased LAP leads to pulmonary venous congestion.
(a) Gradual increases in pulmonary venous pressure results in a compensatory increase in pulmonary lymphatic drainage and may delay the onset of pulmonary edema.[4,7]
(b) Elevated pressures lead to remodeling of the pulmonary vasculature, including pulmonary arteriolar hyperplasia and vasoconstriction.
(c) Manifests as right ventricular (RV) pressure overload over time

ii) Biventricular dysfunction
(1) MS results in a chronically underfilled LV.
(2) Despite normal LV contractile function, low left ventricular end diastolic volume (LVEDV) limits stroke volume through impairment of Starling forces.
(3) LVEDV may be further reduced by RV dysfunction (described below).
(4) Cardiac output (CO) cannot be effectively augmented by increasing heart rate as the concomitant reduction in diastolic filling time will further reduce LVEDV.
(5) Over time, patients may develop LV systolic dysfunction from myocardial deconditioning.[2]
(6) Alterations in pulmonary vasculature from chronically elevated LAP lead to pulmonary hypertension and RV pressure overload.
(a) The RV hypertrophies and dilates, which may lead to tricuspid regurgitation.
(b) Eventually, RV dysfunction ensues.
(c) Signs of right-sided overload develop, including congestive hepatopathy and peripheral edema.[7]
(d) Worsening RV function results in paradoxical motion of the interventricular septum, further impairing LV preload.

d) **Clinical presentation** (Table 99-3)
i) Despite normal LV function, the combination of pulmonary venous congestion with reduced CO presents a similar clinical picture to left-sided congestive heart failure.
(1) Patients may report dyspnea on exertion and orthopnea secondary to elevated LAP, or generalized fatigue from reduced CO.
(2) Hemoptysis may develop from pulmonary venous hypertension causing rupture of bronchial veins.
(3) LA enlargement can lead to hoarseness from compression of the left recurrent laryngeal nerve (Ortner syndrome).[4]


The onset of atrial fibrillation secondary often coincides with new or worsening symptoms of MS.

ii) The onset of AF often coincides with new or worsening symptoms of MS.[4]
(1) Thirty to forty percent of patients with severe MS will develop AF.[6]
(2) Loss of atrial contraction significantly reduces LV preload, which can be further worsened by AF with RVR.
(3) Thromboembolic complications of AF, that is, stroke, may represent the initial presenting symptoms of MS.

iii) Patients present with sequelae of infective endocarditis (IE).[8]
iv) Symptoms of MS may also be unmasked or worsened by physiologic stressors associated with compensatory increases in CO, for example, sepsis, pregnancy, anemia, thyrotoxicosis, and perioperative surgical stress.[2,6]

e) **Anesthetic considerations**
i) General hemodynamic goals for MS
(1) Heart rate and rhythm
(a) Prevention and rapid treatment of tachycardia is paramount in order to maintain adequate LV and diastolic filling time.

TABLE 99-3 Clinical Findings in MS and MR

	MS	Acute MR	Chronic MR
Physical exam	• Auscultation: opening snap, mid-diastolic "rumble" • Narrow pulse pressure	• Auscultation: systolic murmur may be limited by rapid equilibration of LA and LV pressure	• Auscultation: systolic murmur radiating to the axilla, third heart sound (S3) • Displaced apical pulse
ECG	• *P mitrale* ("M-shaped" P wave in lead II) • Biphasic P wave in lead V1 • Atrial fibrillation	• Myocardial ischemia	• Atrial fibrillation
Pulmonary artery catheter	• Large A wave (if sinus rhythm) • PCWP and PAD will *overestimate* LVEDP	• Large V wave	• Small V wave
CXR	• LA enlargement • Pulmonary edema	• Pulmonary edema	• LA enlargement, cardiomegaly • Pulmonary edema
Echocardiography	• See Table 99-1	• See Table 99-2	• See Table 99-2

ECG, electrocardiogram; CXR, chest x-ray; RVH, right ventricular hypertrophy; JVD, jugular venous distension; PCWP, pulmonary capillary wedge pressure; PAD, pulmonary artery diastolic pressure; LVEDP, left ventricular end-diastolic pressure.
Adapted from: Conti CR. Mitral stenosis: a review. *Cardiovascular Innovations and Applications*. 2018;2(4):459-465; Townsley M, Martin D. Anesthetic management for the surgical treatment of valvular heart disease. In: Hensley F, Martin D, Gravlee G, eds. *A Practical Approach to Cardiac Anesthesia*. Philadelphia, PA: Lippincott Williams & Wilkins; 2013; Enriquez-Sarano M, Akins CW, Vahanian A. Mitral regurgitation. *Lancet*. 2009;373(9672):1382-1394.

(b) Maintenance of sinus rhythm because the atrial kick contributes a significant portion of LV preload
(c) Atrial tachyarrhythmias with RVR reduce diastolic filling time.
(d) Equipment for synchronized electrical cardioversion should be immediately available.
(e) Transesophageal echocardiography (TEE) to assess for LA appendage thrombus should be considered prior to cardioversion.



In patients with mitral stenosis TEE should be performed to assess for LA appendage thrombus prior to cardioversion.

(i) Bradycardia may be detrimental to CO and should also be avoided because stroke volume is fixed by the stenotic valve and therefore cannot be augmented to maintain CO in the setting of slower HR.[2]

(2) Preload
(a) Sufficient LA filling generates LAPs capable of driving forward flow across the stenotic valve and is important for maintaining LV preload.
(b) Caution must be taken to not administer excessive intravascular volume, which may exacerbate pulmonary venous congestion leading to pulmonary edema and/or RV failure.
(c) Note that pulmonary capillary wedge pressure (PCWP) and pulmonary artery diastolic (PAD) pressure will *overestimate* LVEDP in the setting of MS.
(d) Left-sided preload is also dependent on RV function and right-sided CO.
(e) Excessive volume loading in an effort to maintain LAP may be detrimental to RV function, which may in fact reduce left-sided preload.

(3) Afterload
  (a) In the setting of relatively fixed CO, mean arterial pressure (MAP) is highly dependent on systemic vascular resistance (SVR).
  (b) The reduction in SVR associated with many anesthetic techniques must be anticipated and avoided when possible.
(4) Contractility
  (a) RV dysfunction may develop as a long-term consequence of MS. Inotropic agents may be necessary in order to maintain right-sided CO.
  (b) LV dysfunction as a consequence of chronic underfilling may lead to myocardial deconditioning and systolic dysfunction.
  (c) Inotropes must be titrated carefully in the setting of MS, as most agents have chronotropic and arrhythmogenic effects as well.
(5) Pulmonary vascular resistance (PVR)
  (a) Many patients with MS have baseline PH and are therefore sensitive to acute increases in PVR, which are common during the perioperative period.
  (b) Care must be taken to avoid hypoxemia, acidosis, excessive positive pressure ventilation, hypothermia, and elevated sympathetic tone (eg, secondary to pain) before RV failure ensues.



Prevention and treatment of tachycardia is essential for maintaining hemodynamic stability in patients with MS.

ii) Preoperative assessment
(1) Obtain a thorough history of symptoms, associated comorbidities, valve lesion severity, and assessment of LV and RV function.
(2) Mild premedication to prevent anxiety-induced tachycardia may be necessary, while avoiding excessive sedation and hypoventilation.
(3) Medication reconciliation, including antiarrhythmics and anticoagulants for those patients with AF is important to consider perioperatively.



Hypoxia, hypercapnia, acidosis, hypothermia, and pain (sympathetic activation) all increase PVR and must be avoided in both MS and MR.

iii) Monitoring
(1) In addition to standard ASA monitors, the placement of an arterial line prior to the induction of anesthesia should be considered for close blood pressure monitoring.
(2) A pulmonary artery catheter (PAC) and TEE provide useful information regarding intravascular volume status and biventricular function.
(3) Consider placement of external cardioversion/defibrillation pads prior to induction, so that atrial dysrhythmias can be treated without delay.
iv) Anesthetic technique
(1) General anesthesia is preferred over neuraxial, as significant decreases in SVR secondary to sympathectomy will not be well tolerated by MS physiology.
(2) For general anesthesia, no single induction technique is clearly the safest.
  (a) Multiple combinations of medications have the potential to maintain ideal physiology for MS patients: avoidance of both tachycardia and decreases in SVR.
(3) It is important to ensure an adequate depth of anesthesia in order to avoid sympathetic activation with increases in PVR and tachycardia.
(4) When indicated, monitored anesthesia care (MAC) must be carefully titrated to avoid hypoventilation and secondary increases in PVR.
  (a) In addition to its lack of respiratory depression, dexmedetomidine may also be of benefit through its tendency to promote slower heart rates.

(5) Careful management of intravascular volume is paramount, as both hypovolemia and hypervolemia are poorly tolerated.
(a) A PAC and/or TEE can provide critical information.
(6) Vigilant ventilator management, and aggressive correction of both respiratory and metabolic acidosis is important to prevent increases in PVR and RV dysfunction.
(7) See section 4 for considerations specific to MV surgery.

f) **Long-term management**

i) Pharmacologic therapy

(1) The goals of long-term medical management include controlling heart rate to promote forward flow and maintain CO, as well as decreasing LAP to prevent pulmonary edema and pressure overload of the RV.
(a) β-adrenergic antagonists and diuretics are the mainstays of medical management.[8]
(2) Patients with AF need appropriate anticoagulation to reduce the risk of thromboembolic events.
(a) The American Heart Association specifically recommends vitamin K antagonists over direct oral anticoagulants for patients with AF and RHD.[9]
(3) Antibiotic therapy is an important consideration for patients with MS.
(a) Primary prevention of RHD has improved as a result of diagnostic advancements that allow the timely diagnosis and treatment of streptococcal pharyngitis.
(b) Most patients do not require infective endocarditis (IE) prophylaxis prior to dental procedures, unless they have had a history of IE.[4,6,9]

ii) Percutaneous mitral balloon commissurotomy

(1) This procedure is performed by advancing one or more balloon catheters percutaneously across the MV.
(a) The balloons are inflated in an effort to fracture the fused portions of the commissures.[4]
(2) This technique is recommended for symptomatic patients with severe MS (MV area $\leq$1.0 cm^2).
(a) Patients must have favorable valve morphology, including leaflets that are mobile, relatively thin, and with limited calcification.
(b) They must also not have significant coexisting MR (less than moderate-to-severe).
(c) TEE is performed prior to the procedure to rule out thrombus and avoid the risk of embolic complications.[4,6]

iii) Surgical management

(1) Surgical correction of MS can involve either repair or replacement of the damaged valve.
(2) Surgery is indicated for symptomatic patients with severe MS who are not considered high risk and who are either not candidates for or have failed previous percutaneous interventions.
(3) Surgery may be favored in patients with concomitant moderate or severe tricuspid regurgitation that can be repaired at the same time.[6]
(4) Replacement options include both mechanical and bioprosthetic valves, depending on patient age, risks associated with anticoagulation, and surgeon and patient preference.[4]



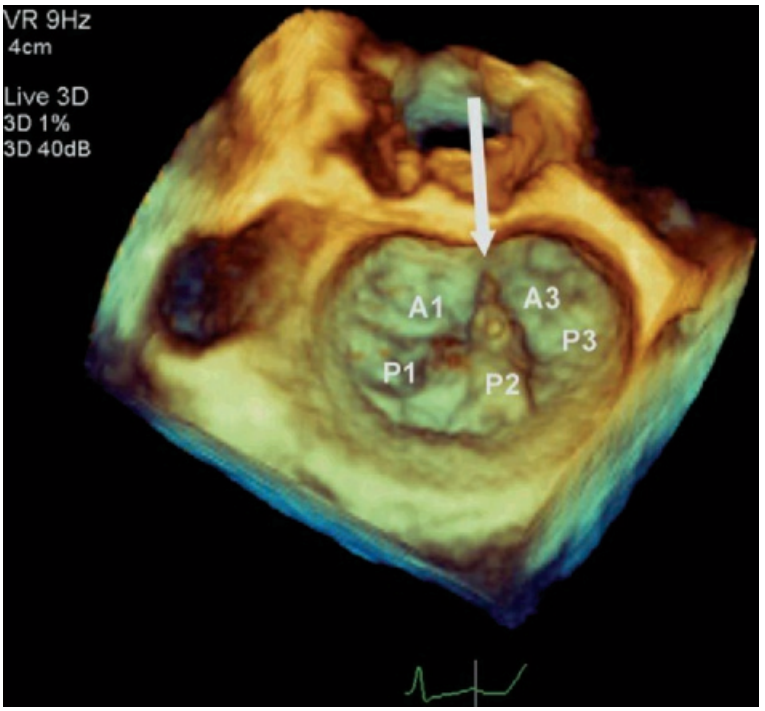
Atrial fibrillation is particularly detrimental in MS because atrial contraction can contribute as much as 30% of LV preload.

3) **Mitral regurgitation**

a) **Etiology**

i) The mechanisms of MR can be subdivided into primary and secondary pathologies.
(1) Primary pathologies involve defects of the mitral apparatus, including the leaflets, valve annulus, chordae tendineae, and papillary muscles.

FIGURE 99-3 Three-dimensional TEE Image of the MV from the Left Atrial Perspective, Also Known as the "Surgeon's View."



The patient has mitral regurgitation secondary to flail of the P2 scallop (labeled by the *large arrow*). A ruptured segment of chordae tendineae can be seen attached to the P2 scallop. Reproduced with permission from Lambert A. Mitral Regurgitation. In: Perrino A, Reeves S, eds. *A Practical Approach to Transesophageal Echocardiography*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2013.

(2) Secondary forms are found in the setting of altered LV geometry, which prevents normal function of the mitral apparatus.

ii) Myxomatous degeneration represents the most common cause of MR in industrialized countries (Fig. 99-3).[1]
(1) Thickening and elongation of the mitral leaflets result in leaflet prolapse or flail.

iii) Other primary leaflet pathologies include congenital lesions (eg, mitral cleft associated with atrial septal defects), IE, mitral annular calcification, and RHD (especially in developing nations).[10,11]

iv) Ischemic MR results from impaired papillary muscle function during systole.

v) In extreme cases of myocardial infarction, papillary muscle rupture is possible.
(1) The posteromedial papillary muscle is more susceptible to ischemia because, unlike the anterolateral papillary muscle, it is dependent upon a single source of blood supply from the posterior descending artery.
(2) Ischemic MR tends to be found in the setting of inferior wall myocardial infarctions.

vi) Functional MR results from pathologic changes in LV geometry, such that alterations in the orientation of the submitral apparatus prevent leaflet coaptation.

(1) These changes may be due to either regional or global LV dysfunction.[12]
(2) Functional MR can be seen in both ischemic and nonischemic forms of cardiomyopathy.

vii) Systolic anterior motion (SAM) results from displacement of the anterior leaflet of the MV into the left ventricular outflow tract (LVOT) during systole.
(1) SAM was originally considered a unique feature of hypertrophic obstructive cardiomyopathy, but is now known to be associated with a variety of other clinical conditions.[13]
(2) Conditions associated with low LVEDV, high contractility, and low SVR may predispose patients to SAM, in addition to anatomic changes associated with MV repair.[14]
(3) SAM is a dynamic pathology, in which Venturi forces may pull the anterior leaflet of the MV into the LVOT leading to both MR and LVOT obstruction.

b) **Grading MR severity**
i) Echocardiography is an important diagnostic and surveillance tool for managing MR (**Table 99-2**).
(1) The reduction in SVR related to general anesthesia often lessens the severity of MR found on intraoperative TEE compared to preoperative studies.[15]

c) **Pathophysiology**
i) The clinical presentation of MR is largely dependent on the time course of the disease, namely acute vs chronic onset.
(1) Compensatory changes in LA and LV size in the setting of chronic MR help maintain both LAP and forward stroke volume (FSV).

ii) Acute MR
(1) Acute MR may develop in the setting of papillary muscle rupture, chordal rupture with flail leaflet, or IE.
(2) Acute MR typically presents with more profound cardiopulmonary instability than chronic MR as LAP increases quite rapidly.
(a) Elevated LAP is transmitted to the pulmonary vasculature, leading to pulmonary venous congestion, pulmonary hypertension, pulmonary edema, and possibly RV failure.
(b) Acute MR may present as biventricular failure with concomitant respiratory failure.
(3) Compensatory increases in sympathetic tone eventually lead to a downward spiral in hemodynamics.
(a) Increased myocardial oxygen demand leads to ventricular ischemia.
(b) Increased SVR can worsen the severity of MR and decrease forward CO.
(4) While the LV ejection fraction (LVEF) may initially increase above baseline, by the Frank-Starling mechanism as the LV works at a higher LVEDV, forward CO is typically insufficient to provide adequate end-organ perfusion.
(a) A significant portion of the LV stroke volume contributing to the "normal" EF is actually passing retrograde back into the LA.
(b) This portion of stroke volume is known as the regurgitant volume, which can represent over half of the total stroke volume in the case of severe MR.[12]

iii) Chronic MR
(1) Chronic MR may result from a variety of primary or secondary pathologies.
(2) Both the LA and LV dilate over time.
(a) LA dilation allows accommodation of the regurgitant volume to prevent pulmonary venous congestion.
(b) LV dilation prevents elevations in LVEDP and preserves FSV by increasing total stroke volume.

(3) The above compensatory changes often lead to progression of MR over time.
(a) LV remodeling and dilation may worsen functional MR, or add a component of functional MR to other primary pathologies.

Normal LVEF in the setting of MR is not indicative of adequate CO because a significant portion of the stroke volume may be passing back into the LA. An LVEF < 60% in the setting of severe mitral regurgitation is suggestive of LV systolic dysfunction.[16] A "normal" LVEF in the setting of MR is ~70%.[6]

d) **Clinical presentation (Table 99-3)**
i) Acute MR
(1) Without time for compensatory LA and LV remodeling, acute MR often presents with profound hemodynamic instability and respiratory failure.
(a) Acute MR may lead to biventricular dysfunction.
(2) Without compensatory changes in LV size, FSV is not maintained and systemic perfusion is compromised.
(a) The development of end-organ injury from hypoperfusion will ensue.
(3) Clinical features associated with the inciting pathology are often present.
(a) For example, endocarditis may present with signs of sepsis or embolic complications such as stroke.
ii) Chronic MR
(1) With time for LA and LV remodeling, patients with chronic MR are able to prevent sequelae of pulmonary venous congestion and also maintain FSV.
(a) Symptoms typically do not develop until the point that LV function begins to decline.[17]
(2) Without intervention, these compensatory changes eventually lead to worsening MR because of annular dilation and alterations in LV geometry.
(3) At a certain point, LV remodeling can no longer keep up with increasing regurgitation to maintain forward flow, and LV function may begin to suffer.
(a) These patients will report signs of left-sided heart failure, such as generalized fatigue and orthopnea.
(b) As LAP continues to rise, pulmonary hypertension and RV dysfunction will develop, as well as pulmonary edema.
(4) LA remodeling predisposes these patients to atrial arrhythmias, particularly AF.
(a) AF may be better tolerated in MR than MS because the former is less dependent on atrial contraction to maintain LV preload.
(b) The onset of AF in MR may present with thromboembolic complications, as opposed to symptoms of a hemodynamic nature.

e) **Anesthetic considerations**
i) General hemodynamic goals for MR
(1) Heart rate and rhythm
(a) Bradycardia must be avoided in patients with MR, as slower heart rates will increase LVEDV, increase RF, and decrease forward CO.
(b) Heart rate should be maintained in the normal to high range.
(c) Ischemic MR is one exception to this rule as slower heart rates may promote coronary perfusion.
(d) Similarly, SAM may improve or even resolve with slower heart rates because of an increase in LVEDV.
(2) Preload
(a) Hypovolemia is poorly tolerated because preload is essential for adequate forward flow.
(b) However, excessive fluid administration may dilate both the LV and mitral annulus and worsen the degree of MR.

(c) Prevention and treatment of SAM is particularly responsive to preload augmentation, in order to maintain higher LVEDV.

(3) Afterload
(a) Maintaining lower SVR will decrease RF and promote forward flow.
(b) Lower SVR will also protect against further LV decompensation in the setting of LV dysfunction.
(c) Ischemic MR is again an exception, as maintenance of adequate SVR to promote coronary perfusion is essential.
(d) Similarly, low SVR in the setting of SAM may decrease LV end-systolic volume (LVESV), thus worsening the degree of MR and LVOT obstruction.



Slow heart rates increase diastolic time and LV filling, which may cause LV distension and worsen MR.

(4) Contractility
(a) Biventricular dysfunction is possible in both acute and chronic MR.
(b) Worsening LV systolic function may lead to ventricular and annular distention, increasing the degree of functional MR.
(c) Elevated LAP will lead to pulmonary congestion, with pulmonary hypertension and RV dysfunction.
(d) RV dysfunction will limit LV preload, leading to a cycle of worsening FSV.
(e) Inotropic agents may be required to support biventricular function, particularly in the setting of acute MR.



The general hemodynamic goals for managing patients with MR are "FAST, FULL, and FORWARD." SAM is an important exception to this rule.

(5) Pulmonary vascular resistance
(a) Many patients with MR have baseline pH and are therefore sensitive to acute increases in PVR.
(b) Conditions that further increase PVR are common in the perioperative period, including hypoxemia, acidosis, positive pressure ventilation, hypothermia, and elevated sympathetic tone (eg, secondary to pain).

ii) Preoperative assessment
(1) Obtain a thorough history of symptoms, associated comorbidities, valve lesion severity, and assessment of LV and RV function.
(2) Premedication with benzodiazepines may be helpful in preventing anxiety-induced elevation in SVR and PVR.
(a) Premedication must be carefully titrated as hypoventilation from oversedation may increase PVR.
(3) Medication reconciliation, including antiarrhythmics and anticoagulants for those patients with AF is important to consider perioperatively.
(4) Prophylactic antibiotic against IE is not indicated, unless the patient has a prior history of IE.[16]

iii) Monitoring
(1) In addition to standard ASA monitors, the placement of an arterial line prior to the induction of anesthesia should be considered for close blood pressure monitoring.
(2) A PAC and TEE provide invaluable information regarding intravascular volume status and biventricular function.

iv) Anesthetic Technique
(1) Unlike MS, neuraxial anesthesia may prove beneficial in optimizing the hemodynamics of MR.
(a) The sympathectomy associated with neuraxial techniques can decrease SVR, and promote forward flow.

(b) However, significant venodilation and reduced preload may not be well tolerated.

(2) General anesthesia tends to have similar beneficial effects on SVR.

(3) Large doses of opioids with induction may lead to bradycardia, which may be attenuated by the chronotropic effects of adding ketamine.

(4) It is important to ensure an adequate depth of anesthesia in order to avoid sympathetic activation with increases in PVR and SVR.

(5) Careful management of intravascular volume is paramount, as both hypovolemia and hypervolemia are poorly tolerated.

(6) Vigilant ventilator management and aggressive correction of both respiratory and metabolic acidosis is important to prevent increases in PVR and RV dysfunction.

(7) See section 4 for considerations specific to MV surgery.

f) **Long-term management**

i) Medical management

(1) Acute MR

(a) In many cases, medical stabilization may only serve as a temporizing measure until emergent cardiac surgery can be performed, particularly for primary etiologies of MR.

(b) Inotropes and vasodilators should be titrated to promote forward flow, although profound hypotension may require vasopressors to maintain MAP.

(c) Ensure simultaneous treatment of the underlying mechanism for MR, for example myocardial ischemia and IE.

(d) The placement of an intraaortic balloon pump may be beneficial to reduce afterload, while supporting coronary perfusion.

(e) SAM should be treated with volume resuscitation, AV nodal blocking agents (eg, β-adrenergic antagonists), and vasopressors.

READ MORE
Aortic valvular disease, Chapter 100.

(2) Chronic MR

(a) An important aspect of medical management in chronic, primary MR is to provide close surveillance of symptoms and LV function so that surgical intervention can be performed before irreversible LV dysfunction occurs.

(b) The onset of symptoms, LV dysfunction, or pulmonary hypertension worsens the prognosis for patients with MR.[6]

(c) For patients who are not surgical candidates, standard medical therapy for systolic heart failure should be instituted for symptomatic MR in patients with LVEF <60%.[6]

(d) The nature of chronic, functional MR highlights the importance of medical management of the underlying disease process, for example, appropriate management of coronary artery disease is essential in preventing further progression of chronic, ischemic MR.

(e) Due to the high incidence of AF in patients with chronic MR, appropriate anticoagulation and antiarrhythmic therapies are important to ensure.

ii) Surgical management

(1) Surgical options for MR include both repair and replacement techniques.

(a) Surgical intervention is generally indicated for asymptomatic patients with chronic, *primary* severe MR if LVEF is 30%-60% and/or LVEDV is ≥40 mm.

(b) If symptomatic, these patients should undergo surgical correction as long as the LVEF is at least 30%.

(c) The benefit of restoring MV competence in the setting of chronic, *functional* MR does not address the underlying pathology, so indications for intervention are less clear.[6]

(2) Compared with MS, many patients with primary MR have favorable valve anatomy for repair.

(a) Pathologies with favorable outcomes for repair include degenerative changes of the posterior leaflet, clefts, IE, and ruptured chordae or papillary muscles.[6,14]

(3) When feasible, MV repair is preferred over replacement.

(a) Repair is associated with lower operative mortality compared to replacement.

(b) Improved preservation of the submitral apparatus after repair is associated with better LV function.

(c) Repair avoids thromboembolic and anticoagulation risks associated with mechanical replacement, as well as structural deterioration of bioprosthetic valves.[18]

(4) Percutaneous interventions are an emerging technology for high-risk surgical patients.

(a) Devices such as the MitraClip can be placed transseptally across the MV to serve as an "edge-to-edge" repair.

(b) The anterior and posterior leaflets are tethered together in order to limit systolic leaflet motion, resulting in a dual-orifice MV.[17]

(c) Early results from studies such as the EVEREST II trial remain promising.[19]

4) **Anesthetic considerations for MV surgery**

a) In addition to the above anesthetic considerations, there are specific anesthetic considerations for MV surgery.

b) TEE is an essential intraoperative monitor in this setting.

c) LV dysfunction is relatively common and inotropic support is often required.

i) In the setting of MS, a chronically underfilled, deconditioned LV may become overloaded after flow through the MV is restored.

ii) An LV that is accustomed to the low-resistance path of retrograde flow through a regurgitant MV may not tolerate the increased afterload from removing this "pop off" valve.

d) After MV repair, it is important to assess for iatrogenic MS, residual MR, iatrogenic SAM, and myocardial ischemia due to circumflex artery damage from annuloplasty ring sutures.

e) Complications specific to MV replacement are similar for both MS and MR.

i) Transvalvular and paravalvular regurgitation are both possible.

ii) Similar to annuloplasty rings, posterior stitches can obstruct blood flow through the circumflex artery and cause myocardial ischemia.

iii) The aortic valve annulus can be disrupted, leading to iatrogenic aortic regurgitation.

f) It is important to ensure adequate de-airing of the LV prior to weaning from cardiopulmonary bypass in order to limit the risks of air embolism, including neurologic complications and myocardial ischemia.

CHAPTER SUMMARY FOR MV DISEASE

	Mitral Stenosis	Mitral Regurgitation
General Considerations	Primarily the result of rheumatic heart disease Can be effectively treated with balloon valvuloplasty in certain cases	Acute MR is poorly tolerated and usually results in cardiogenic shock. Chronic severe MR may progress into irreversible LV dysfunction. MV repair is preferable to replacement.
Hemodynamic goals	Avoid tachycardia and decreases in SVR. Maintain sinus rhythm. Maintain adequate preload. Maintain contractility. Avoid factors that increase PVR.	"FAST, FULL, and FORWARD" Avoid bradycardia and increases in SVR. Maintain adequate preload. Maintain contractility. Avoid factors that increase PVR. Neuraxial and general anesthesia are better tolerated than in MS. SAM is an important exception to these rules.
Monitoring	Arterial line, central line ± PAC, ±TEE	

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100 Aortic Valvular Disease

Lindsay Raleigh • Daryl Oakes

Aortic valve (AV) disease accounts for a major portion of cardiac disease in the United States. The presence of either symptomatic aortic stenosis (AS) or severe aortic regurgitation (AR) carries a significantly increased morbidity and mortality risk compared to the general population.[1,2] It is essential to understand the pathophysiology and hemodynamic consequences of aortic valvular diseases in order to anticipate problems and safely manage these patients. This chapter presents an overview of pathophysiology of aortic valvular heart disease.

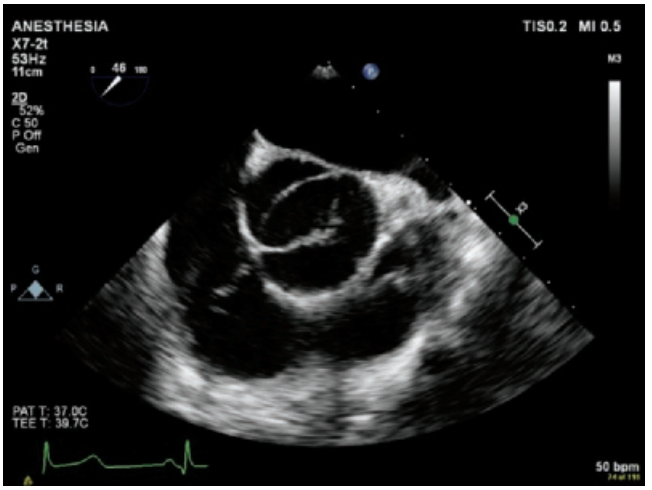
Aortic Stenosis (AS)

1) **Etiology**
   a) Progressive calcific degeneration of the trileaflet aortic valve is the most common cause of AS, with symptoms presenting in the 6th-8th decades of life.[3]
   b) Bicuspid aortic valve (BAV) disease is the most common congenital anomaly, occurring in about 1%-2% of the general population, and is associated with early valvular stenosis.
      i) Degenerative changes occur at an earlier age due to the abnormal hemodynamics across the valve leaflets, usually in the 4th-5th decades of life[3] (Fig. 100-1).
   c) Rheumatic heart disease, infection, and systemic lupus erythematosus are other rare causes of AS.
   d) Subvalvular or supravalvular lesions can also cause AS. These lesions are very uncommon and will not be discussed here.
2) **Pathophysiology**
   a) Calcification of the aortic leaflets leads to their thickening and restricted mobility, and eventual stenosis.
   b) Stenosis is a fixed obstruction to the left ventricle (LV) emptying, which leads to the following changes:

Hemodynamic Calculations; Laplace's Law
Wall stress = Pressure × Radius/(2 × Wall thickness)

      i) Pressure overload
         (1) The high ventricular afterload (pressure) associated with the stenotic valve directly increases myocardial wall stress (or tension) in accordance with Law of Laplace.
         (2) Leads to LV concentric hypertrophy as a compensatory response. Increased wall thickness and associated decreased intracavitary chamber size (radius) offsets the increased ventricular wall tension caused by elevated intracavitary.
      ii) LV hypertrophy
         (1) Consequences include a decrease in diastolic compliance and an increase in myocardial O_2 consumption.
         (2) Inability of coronary artery perforator vessels to deliver adequate blood flow to inner portions of this thickened myocardium results in an imbalance in myocardial O_2 supply and demand.

FIGURE 100-1 Transesophageal Midesophageal Short Axis View of a Bicuspid Aortic Valve



(3) This imbalance leads to subendocardial ischemia and, long-term, subendocardial scar formation.
(4) Wall thickening often results in decreased intracavitary chamber size and decreased stroke volume.

iii) Decreased LV compliance/increased diastolic dysfunction
(1) Necessitates higher LV filling pressures and an increased reliance on atrial contraction ("atrial kick") for LV filling.
(2) Increased LV end-diastolic pressures (LVEDP) decreases the coronary perfusion pressure gradient (see equation below).
(3) High LVEDP results in elevated left atrial pressure and LA enlargement. LA dilation increases risk of atrial dysrhythmias, which further reduce LV filling and stroke volume.

Hemodynamic Calculations
Coronary Perfusion Pressure = Diastolic Blood Pressure — LVEDP

3) **Signs and symptoms**
a) **Early stages**
i) Patients are asymptomatic due to adequate physiologic compensation, primarily through LV hypertrophy.
b) **Intermediate and late stages**
i) Patients develop a triad of symptoms:
(1) Angina: occurs when oxygen demand exceeds oxygen supply to the myocardium.
(2) Syncope: usually exertional; occurs
(a) Due to the inability of the heart to increase stroke volume in response to increased exertion or decreased peripheral vascular resistance
(b) Due to atrial or ventricular tachydysrhythmias

(3) Dyspnea: related to congestive heart failure (both systolic and diastolic) that develops as a result of
   (a) Compromised stroke volume from diminished intracavitary chamber size
   (b) High diastolic filling pressures from decreased LV diastolic compliance (high diastolic filling pressures are transmitted backward to the pulmonary vasculature causing pulmonary edema)



All AS patients are at risk of sudden death, with higher risk in symptomatic AS. Sudden death is typically from ventricular fibrillation.

ii) All AS patients are at risk of sudden death, with higher risk in symptomatic AS. Sudden death is typically from ventricular fibrillation.

iii) With onset of CHF, the average life expectancy is 1-2 years.[2]

iv) AS patients have a nearly normal survival rate up until symptom onset. Once symptoms develop, prognosis is dismal with a life expectancy of <5 years (**Fig. 100-2**).[2]

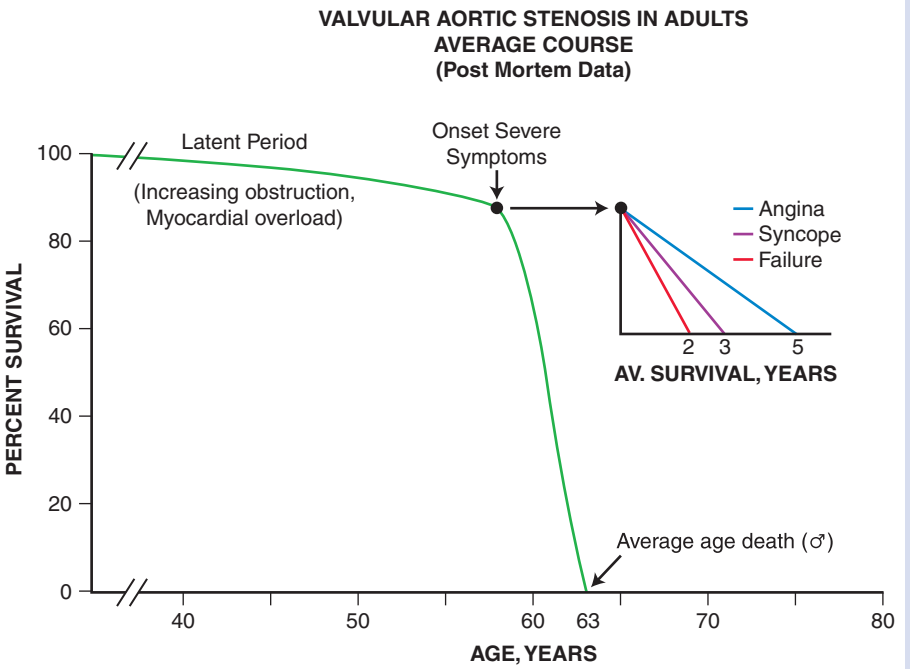
c) **Associated findings**

i) Physical findings

(1) A loud crescendo-decrescendo systolic murmur at the right upper sternal border, radiating to the neck bilaterally

(2) A late-peaking, narrow pulse pressure (<50 mm Hg) may be noted in severe AS ("pulsus tardus et parvus").

FIGURE 100-2 Average Course of Valvular Aortic Stenosis in Adults.



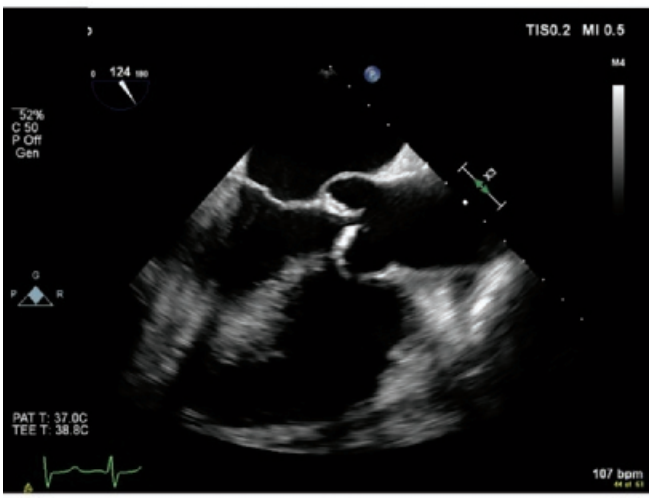
Data assembled from postmortem studies.
Reproduced from Lester SJ, Heiboron B, Gin K, et al. The natural history and rate of progression of aortic stenosis. *Chest*. 1998;113:1109-1114, with permission from Elsevier.

ii) Diagnostic studies
   (1) CXR may show signs of pulmonary congestion and calcification of aortic cusps.
   (2) ECG frequently demonstrates LVH and occasionally atrial fibrillation.
   (3) Echocardiographic examination allows for a noninvasive assessment of the severity of stenosis and the adequacy of contractile function (Fig. 100-3).
   (4) Cardiac catheterization allows direct measurement of transaortic pressures.
   (5) Coronary angiography will assess coexisting coronary artery disease.

FIGURE 100-3 **A.** Transesophageal midesophageal short axis view of a calcified trileaflet aortic valve. **B.** Transesophageal midesophageal long axis view of a calcified trileaflet aortic valve.



A



B

TABLE 100-1 Aortic Stenosis Grading

	Max velocity (m/s)	Mean Pressure (mm Hg)	AV Area (cm^2)	Indexed AVA AVA/BSA (cm^2/m^2)
Normal	<2	–	2.5-3.5	–
Sclerosis	2-2.5	–	–	–
Mild	2.6-2.9	<20	>1.5	>0.85
Moderate	3-3.9	30-39	1.0-1.5	0.6-0.85
Severe	≥4	≥40	<1.0	<0.6

Adapted from Baumgartner H, Hung J, Bermejo J, et al. Recommendations on the echocardiographic assessment of aortic valve stenosis: a focused update from the European Association of Cardiovascular Imaging and the American Society of Echocardiography. *J Am Soc Echocardiogr*. 2017;30(4):372-392, with permission from Elsevier.

4) **Classification and severity grading**
   a) *Grading of AS*: Based on the severity of obstruction (Table 100-1)[4]

Echocardiography Calculations
Continuity Equation
AVA (cm^2) = CSA (LVOT) × VTI (LVOT)/VTI (AV)

CSA, cross-sectional area; VTI, velocity time integral; LVOT, left ventricular outflow track.

   b) *Staging of AS*: Determined by degree of stenosis, valve hemodynamics, hemodynamic consequences of AS, and the absence or presence of symptoms (Table 100-2)[5]
      i) Two subsets of severe AS (stages D2 and D3) are defined by low-flow and low-gradient valvular hemodynamics (AVA ≤ 1.0 cm^2, but aortic velocity <4.0 m/s or mean pressure gradient ≤40 mm Hg) as a result of low flow rates across the valve due to one of the following:

TABLE 100-2 Staging of Aortic Stenosis (AS)

Stage	Definition (Valve Anatomy and Hemodynamics)	Hemodynamic Consequences
A	**At risk of AS** (BAV or valve sclerosis with V_{max} < 2 m/s)	None
B	**Progressive AS** (Mild or Moderate AS)	+/– Early diastolic dysfunction Normal LVEF
C C1 C2	**Asymptomatic severe AS** No LV dysfunction LV dysfunction	 LV diastolic dysfunction, normal LVEF LV diastolic dysfunction, LVEF <50%
D D1 D2 D3	**Symptomatic severe AS** Severe symptomatic AS Low-flow/low-gradient severe AS with reduced EF Low-gradient AS with normal EF or paradoxical low-flow severe AS	 LV diastolic dysfunction, LV hypertrophy, +/– Pulmonary HTN LV diastolic dysfunction, LV Hypertrophy, LVEF < 50% Increased LV wall thickness, Small LV chamber with low SV, LVEF > 50%

Adapted from Nishimura RA, Otto CM, Bonow RO, et al. 2014 AHA/ACC guideline for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Thorac Cardiovasc Surg*. 2014;148(1):e1-e132, with permission from Elsevier.

TABLE 100-3 Perioperative Complications in Noncardiac Surgery Patients with AS

Myocardial infarction	Heart failure
Hypotension	Stroke
Arrhythmias	Death

(1) Poor LV systolic function (reduced LVEF) secondary to ventricular remodeling caused by long-term subendocardial ischemia and scarring
(2) LV hypertrophy resulting in small ventricular volumes and small stroke volumes and low cardiac output despite a normal or even supra-normal LVEF



In the setting of low cardiac output, severe AS may be present despite a "low" pressure gradient.

c) *Pseudostenosis*: Patients with a low valvular gradient due to the reduced forward flow, which results in limited valve opening and a mistakenly low calculated valve area.
i) A dobutamine stress echocardiogram can differentiate between patients with pseudostenosis and those with true AS by augmenting cardiac output.
ii) Patients with true AS will have an increase in the mean gradient, but no increase in valve area; patients with pseudostenosis will have no increase in gradient, but an increase in valve area.

5) **Preoperative risk stratification of patients with severe AS for noncardiac surgery**
a) Several studies suggest that severe aortic stenosis is associated with increased morbidity and mortality after noncardiac surgery (Table 100-3).[6-8]
b) Significant predictors of 30-day mortality and postoperative myocardial infarction after noncardiac surgery are high-risk surgery, symptomatic severe aortic stenosis, coexisting mitral regurgitation, and preexisting coronary artery disease.[9]
c) The AHA/ACC Guidelines on perioperative cardiovascular evaluation and management for noncardiac surgery[10] provide the following statements and recommendations (Table 100-4).

6) **Preoperative management**
a) A detailed preoperative assessment and risk stratification are essential.
i) Identify the urgency and risk of surgery, the degree of stenosis, the presence of symptoms related to valve pathology, the systolic function of the left ventricle, and the presence of other valvular lesions.
b) Diagnostic studies:
i) Echocardiography allows for the grading of stenosis and assessment of other cardiac comorbidities.

It is recommended that patients with clinically suspected moderate or greater degrees of valvular stenosis or regurgitation undergo preoperative echocardiography if there has been either (1) no prior echocardiography within 1 year or (2) a significant change in clinical status or physical examination since last evaluation. (Level of Evidence: C)[10]

7) **Intraoperative management**
a) The perioperative risk of noncardiac surgery in the aortic stenosis patient can be mitigated with careful preoperative planning and vigilant intraoperative management.
b) **General hemodynamic goals (Table 100-5)**
i) Heart rate/rhythm
(1) Extremes of heart rate are not well tolerated.

TABLE 100-4 AHA/ACC Guidelines on Perioperative Cardiovascular Evaluation and Management of Noncardiac Surgery

Emergency noncardiac surgery may occur in the presence of severe aortic stenosis.
For adults who meet indications for valvular intervention on the basis of symptoms and severity of stenosis, surgical aortic valve replacement is recommended before elective noncardiac surgery to reduce perioperative risk. (Class I, Level of Evidence C)
Elevated risk elective noncardiac surgery with appropriate intraoperative and postoperative hemodynamic monitoring is reasonable to perform in patients with asymptomatic severe aortic stenosis. (Class IIa Level of Evidence B)

From Fleisher LA, Fleischmann KE, Auerbach AD, et al. 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: a report of the American College of Cardiology/American Heart Association Task Force on practice guidelines. *J Am Coll Cardiol*. 2014;64(22):e77-e137. doi:10.1016/j.jacc.2014.07.944; Baumgartner H, Hung J, Bermejo J, et al. Recommendations on the echocardiographic assessment of aortic valve stenosis: a focused update from the European Association of Cardiovascular Imaging and the American Society of Echocardiography. *J Am Soc Echocardiogr*. 2017;30(4):372-392. doi:10.1016/j.echo.2017.02.009.

(2) Goal is to maintain a low normal heart rate, giving time for adequate systolic ejection and adequate coronary blood flow.
(3) Maintaining sinus rhythm is crucial. Any dysrhythmia should be treated aggressively.
(4) Loss of sinus rhythm can lead to rapid clinical deterioration; atrial systole may account for up to 40% of LV end-diastolic volume.
(5) Tachycardia limits the time in systole and diastole, which limits stroke volume and decreases diastolic coronary artery perfusion time.



Loss of sinus rhythm can lead to rapid clinical deterioration; atrial systole may account for up to 40% of LV end-diastolic volume.

ii) Preload
(1) Poor ventricular compliance necessitates preload augmentation.
(2) Hypovolemia may result in a decreased stroke volume.

TABLE 100-5 Staging of AR

Stage	Definition (Valve Anatomy and Hemodynamics)	Hemodynamic Consequences
A	**At risk of AR** (BAV/congenital valve abnormality, valve sclerosis, diseases of the aortic sinuses, ascending aorta, history rheumatic fever/known rheumatic heart disease, infective endocarditis)	None
B	**Progressive AR** (Mild or Moderate AR)	Normal LVEF Normal LV volume or mild dilation
C C1 C2	**Asymptomatic severe AR** No LV dysfunction LV dysfunction	 Normal LVEF (>50%), mild-to-moderate LV dilation (LVESD < 50 mm) LVEF <50%, severe LV dilatation (>50 mm)
D	**Symptomatic severe AR**	May occur with normal LVEF (>50%), mild-to-moderate LV dysfunction (40%-50%), or severe LV dysfunction (<40%) Moderate-to-severe LV dilation is present

Adapted from Nishimura RA, Otto CM, Bonow RO, et al. 2014 AHA/ACC guideline for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Thorac Cardiovasc Surg*. 2014;148(1):e1-e132, with permission from Elsevier.

iii) Afterload
  (1) Preserving systemic vascular resistance (SVR) and adequate diastolic pressure are critical for coronary perfusion.
  (2) Hypotension can compromise the tenuous balance of O_2 supply and demand and lead to myocardial ischemia.
  (3) A relatively fixed stroke volume exists in severe AS so excessive changes in SVR are not tolerated. Medications causing arterial dilation should be used cautiously.
  (4) Hypotension from any cause should be avoided.
  (5) Phenylephrine is the agent of choice to treat hypotension.



Phenylephrine is the vasopressor of choice for patients with aortic stenosis. It is an alpha agonist that increases SVR, maintains CPP, and does not increase chronotropy.

iv) Contractility
  (1) Administer inotropic agents with caution.
    (a) Primarily useful in patients with reduced left ventricular ejection fraction.



Use of large bolus doses of venodilators such as nitroglycerin may seriously decrease LV filling and drop cardiac output in patients with AS.

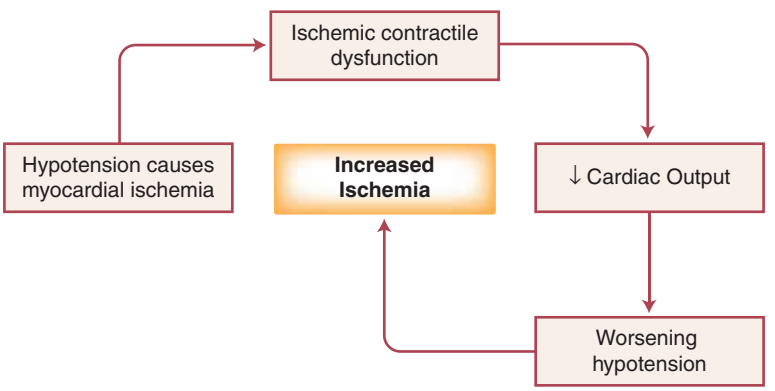
v) "Death spiral"
  (1) In severe or critical AS, a precipitous drop in systemic blood pressure from any reason can lead to a vicious cycle of events (Fig. 100-4).
  (2) Chest compressions are often ineffective as it is difficult to generate enough mechanical force to create adequate stroke volume across the stenotic valve. Sudden death may result.
  (3) Consider an intra-aortic balloon pump or other forms of mechanical circulatory support if medical therapies fail to stabilize the patient.



Whether from vasodilation, hypovolemia, tachycardia, or atrial fibrillation, all causes of hypotension need to be treated aggressively in patients with aortic stenosis.

c) **Monitoring**
  i) American Society of Anesthesiologists (ASA) standard monitors
    (1) ECG—detects myocardial ischemia and identifies arrhythmias
  ii) In patients with severe stenosis and those undergoing moderate to high-risk surgeries, more advanced monitoring should be considered.

FIGURE 100-4 Consequences of Decreased Arterial Blood Pressure in AS.



(1) Invasive arterial monitoring allows continuous blood pressure measurement.
(a) Enables rapid intervention to changes in afterload and optimization of coronary perfusion pressure.
(b) Placement prior to induction is strongly recommended for moderate to severe lesions.
(2) Central venous pressure monitoring allows estimation of right atrial filling pressures.
(a) Placement of a triple lumen catheter allows administration of vasopressor or inotropic medications that may be needed to support cardiac function.
(3) TTE or TEE monitors ventricular systolic and diastolic function, provides assessment of wall motion and ventricular filling, and aids in diagnosis of causes of intraoperative hypotension.
(4) Pulmonary artery catheterization provides information about cardiac function and adequacy of cardiac output and perfusion.
(a) May be beneficial in patients with reduced left ventricular systolic function, as a means to trend mixed venous oxygen saturation and cardiac output.

d) **Induction**
i) Preinduction arterial lines are indicated continuously monitor blood pressure and allow for early hemodynamic interventions.
ii) Induction agents with minimal affects on SVR and HR are preferred.
(1) Many clinicians prefer an opioid-based technique; alternatively, ketamine offers a favorable hemodynamic profile.
(2) Phenylephrine or vasopressin is frequently utilized to augment systemic vascular tone and avoid significant hypotension with induction.
(3) Consider mild preinduction volume loading, especially in patients who have been NPO for prolonged periods or are clinically hypovolemic.
iii) A defibrillator should be readily available for immediate cardioversion if necessary.

e) **Maintenance**
i) General anesthesia or neuraxial/regional techniques can be performed, as long as close hemodynamic monitoring occurs and adverse hemodynamic effects are minimized.
(1) Neuraxial anesthesia is relatively contraindicated in severe AS due to concerns of sympathectomy-induced hypotension.
ii) Hemodynamically significant arrhythmias should be treated with electrical cardioversion.

f) **Emergence**
i) At the conclusion of surgery, the decision to extubate will be dependent on the patient's baseline condition, intraoperative course, and type of surgery.
ii) If extubation is deemed appropriate, preparation needs to be made to control the sympathetic activation that is inevitable with emergence, avoiding tachycardia and arrhythmias.
iii) Volume shifts and hypervolemia are poorly tolerated given high diastolic pressures and risk of pulmonary edema.
(1) Carefully evaluate hypoxemia after extubation to determine if volume status is appropriate.

8) **Postoperative management**
a) Depending on the patient's comorbidities, intraoperative course, and expected postoperative hemodynamic changes, it may be necessary to arrange for postoperative recovery in a location that allows for more intensive hemodynamic monitoring, such as an intensive care or telemetry unit.
b) Maintenance of adequate blood pressure and preload and avoiding tachycardia and arrhythmias are of the upmost importance.
c) Adequate pain control will reduce the risk of postoperative tachycardia and myocardial ischemia.

9) **Treatment of aortic stenosis**
   a) There is no medical therapy to prevent disease progression.
   b) Definitive treatment is aortic valve replacement, either surgical (SAVR) or transcatheter (TAVR).
      i) Once symptoms are present, outcomes are extremely poor unless intervention occurs.
      ii) AHA/ACC guidelines for the management of patients with valvular heart disease[5] recommend aortic valve replacement (AVR) for the following patients:
         (1) Patients with symptomatic severe AS (stage D) promptly after the onset of symptoms (Class I, Level of Evidence B)
         (2) Asymptomatic patients with severe AS and a left ventricular ejection fraction <50% (stage C2) or stage C patients undergoing other cardiac surgery (Class I, Level of Evidence B)
      iii) For patients who are considered ineligible or high risk for surgical aortic valve replacement, transcatheter aortic valve replacement (TAVR) has emerged as a valuable alternative, with outcomes superior to medical management alone and noninferior to surgical aortic valve replacement (SAVR).[11]
      iv) Recent data from the Partner Trials suggest that SAVR and TAVR have similar outcomes in intermediate-risk surgical patients, as well.[12] Thus, TAVR may be a reasonable alternative for these patients as well.
   c) **Balloon valvotomy**
      i) Temporarily relieves symptoms but does not prolong survival.
      ii) Valvotomy is never recommended as an alternative to AVR. However, it may be considered in certain clinical situations.
      iii) The rate of serious complication including death, stroke, aortic rupture, and aortic regurgitation (AR) exceeds 10%.[5]

Aortic Regurgitation (AR)

1) **Etiology**
   a) AR results from the inability of the aortic leaflets to close tightly, which may be due to disease of the leaflets themselves and/or the aortic root or ascending aorta.
   b) **Acute AR**
      i) Causes include infective endocarditis, aortic dissection, or blunt chest trauma.
      ii) Iatrogenic causes include balloon valvotomy or TAVR.
   c) **Chronic AR**
      i) Most common causes are bicuspid aortic valves and calcific valve disease.
      ii) Other causes include primary diseases of the aorta, including aortic root or ascending aortic dilation.
         (1) Dilation can be idiopathic, or associated with connective tissue disorders (ie, Marfan syndrome, Ehlers-Danlos syndrome, Loeys-Dietz syndrome).
      iii) Rheumatic heart disease is the leading cause of chronic AR in developing countries.
         (1) May clinically present in the 2nd or 3rd decade of life.[13]
2) **Pathophysiology**
   a) **Acute AR**
      i) Sudden reflux of a large regurgitant volume back into the LV imposes a tremendous volume burden on the ventricle and causes acute decompensated systolic heart failure.
      ii) LVEDP and left atrial pressure increase precipitously leading to abrupt pulmonary congestion/edema.
      iii) Severe systemic hypotension from decreased cardiac output can occur.

iv) Tachycardia, peripheral vasoconstriction, and increased myocardial contractility all strive to improve forward flow and are mediated through release of catecholamines.
v) Severe regurgitation causes decreased systemic diastolic pressures, which in the setting of severely elevated LVEDP, significantly decreases coronary perfusion pressure, leading to ischemia even in the setting of normal (patent) coronary arteries.
vi) Myocardial ischemia and sudden death are common in acute AR.

b) **Chronic AR**
i) Disease progression is slowly progressive.
ii) Volume overload:
(1) The degree of volume overload is dependent on three factors: regurgitant orifice area, pressure gradient across the AV, and diastolic time.
(2) Chronic volume overload leads to LV dilation through eccentric hypertrophy.
(3) A dilated LV results in high diastolic compliance thus allowing large increases in LVEDV with minimal elevation in filling pressure.
iii) Pressure overload:
(1) Ejection of a large total stroke volume creates:
(a) An increased pulse pressure with systolic hypertension, which imposes an elevated pressure load on the ventricle
(b) A decreased diastolic pressure, which results in low coronary perfusion pressure
(2) According to Laplace law, LV dilation increases systolic wall tension.
(a) Increased myocardial O_2 consumption occurs due to the presence of LV hypertrophy and an elevated wall tension.
iv) Progressive volume and pressure overload overcome the compensatory effect of LV dilation and hypertrophy leading to systolic dysfunction. Diastolic dysfunction is also common.

3) **Signs and symptoms**

a) **Acute AR**
i) Presentation is rare but dramatic.
ii) Can present in cardiogenic shock.
iii) Typical signs include shortness of breath and hypoxemia from severe pulmonary edema and hypotension refractory to medical management.
iv) Physical findings may be minimal. Diastolic murmur, widened pulse pressure, and cardiomegaly may be all missing.

b) **Chronic AR**
i) Patients typically are asymptomatic until late in the disease process.
(1) Rate of progression of asymptomatic AR to either symptomatic AR or LV dysfunction is <6% per year.
ii) Mortality is <0.2% per year if asymptomatic with normal LV function.[10]
iii) Symptoms appear when regurgitant volume exceeds 60% of stroke volume.
(1) Fatigue, dyspnea on exertion, orthopnea, and paroxysmal nocturnal dyspnea
(2) Angina or CHF portend a mortality >10% per year and >20% per year, respectively.[10]
iv) Patients with severe symptoms (NYHA Class III or IV) are at especially high risk with about 25% per year mortality rate without surgery. Even mild symptoms (Class II) have a 6.3% per year mortality rate[13] (**Table 100-6**).
v) Signs include
(1) Classic decrescendo early diastolic murmur at upper left sternal boarder
(2) Widened pulse pressure, marked by high systolic and low diastolic pressures



"Fast, full, and forward"—phrase summarizes the pertinent hemodynamic goals for AR.

TABLE 100-6 New York Heart Association Classification of Cardiovascular Disease

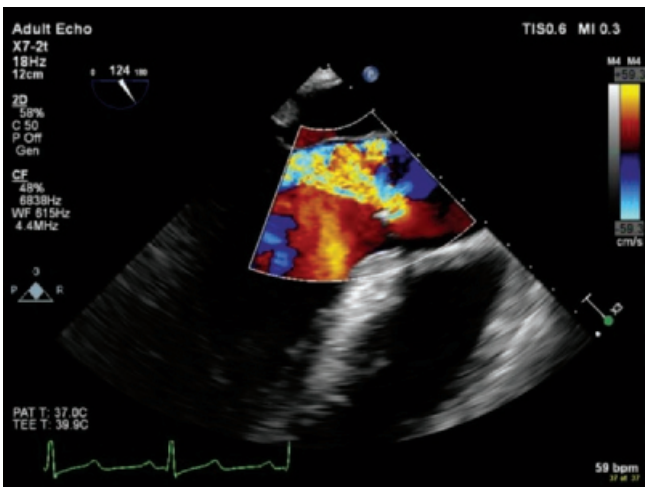
Class I Patients with cardiac disease but no limitation to their physical activity. No symptoms of cardiac decompensation (angina, palpitations, dyspnea, fatigue) occurs with activity.
Class II Patients who have mild limitations in physical activity due to cardiac disease. Patients are comfortable at rest but develop symptoms with mild-to-moderate exertion.
Class III Patients with marked impairment in physical activity due to cardiac disease. Typically asymptomatic at rest, but minimal exertion will result in symptoms of cardiac decompensation.
Class IV Patients with severe functional limitation with minimal exertion due to cardiac disease. These patients may also be symptomatic at rest.

NYHA Criteria: Adapted from The Criteria Committee of the New York Heart Association. *Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Great Vessels*. 9th ed. Boston, MA: Little Brown & Co., 1994:253-256.

(3) Bounding carotid pulse, head bobbing, and pulsation of the uvula
(4) Presence of S3 and leftward displacement of apical cardiac impulse, due to LV enlargement

vi) Diagnostic studies
(1) CXR may show pulmonary congestion and cardiomegaly.
(2) Echocardiography is useful for classifying AR severity, assessing LV function and evaluating the mechanism for AR.
(3) Cardiac catheterization may confirm echocardiography findings or diagnose coexisting coronary artery disease.

FIGURE 100-5 Transesophageal Midesophageal Long Axis View Valve Demonstrating Severe Aortic Regurgitation by Color Flow Doppler.



4) **Classification and severity grading**

Doppler echocardiography is the mainstay for severity assessment (Fig. 100-5).

a) *Grading of AR*: Based on the severity of regurgitation (Table 100-7)[14]

b) AR stages are defined by symptomatic status, severity of regurgitation, and LV volume and systolic function (Table 100-5).

5) **Preoperative risk stratification**

a) **Acute AR**

i) Early intervention is associated with improved survival.

b) **Chronic AR**

i) The AHA/ACC Guidelines on perioperative cardiovascular evaluation and management for noncardiac surgery[10] provide the following statements and recommendations:

Emergency noncardiac surgery may occur in the presence of severe aortic regurgitation.

For adults who meet indications for valvular intervention on the basis of symptoms and severity of stenosis, surgical aortic valve replacement is recommended before elective noncardiac surgery to reduce perioperative risk (Class I, Level of Evidence C).

Elevated risk elective noncardiac surgery with appropriate intraoperative and postoperative hemodynamic monitoring is reasonable to perform in patients with asymptomatic severe aortic regurgitation and normal LV ejection fraction (Class IIa, Level of Evidence C).

6) **Preoperative management**

a) Obtain a thorough history of symptoms and associated comorbidities.

b) Symptomatic severe AR is an active cardiac conditions—all elective noncardiac surgeries should be deferred until valve replacement completed.

7) **Intraoperative management**

a) **General hemodynamic goals (Table 100-6)**

i) Heart rate/rhythm

(1) Elevated HR (~80-90 bpm), improves forward flow by decreasing diastolic duration → ↓regurgitant volume, and ↑EF.

(2) Improved coronary perfusion is also seen due to a higher mean diastolic pressure and a lower LVEDP.

ii) Preload

(1) Adequate preload is necessary to maintain forward flow.

iii) Afterload

(1) Afterload reduction through peripheral arteriolar dilation improves forward flow by reducing ventricular stroke work.

(2) When initiating vasodilator therapy, additional preload is usually needed.

TABLE 100-7 General Parameters in Grading AR

	Mild	Moderate	Severe
Regurgitant fraction (%)	30-39	40-49	≥50
Estimated regurgitant orifice area (cm^2)	0.1-0.19	0.2-0.29	≥0.3
Color Doppler jet width (as a % of LVOT)	25-45	46-64	≥65
Vena contracta width (cm)	<0.3	0.3-0.6	>0.6
Pressure half-time (msec)	>500	50-200	<200
Diastolic flow in the descending thoracic aorta			Holodiastolic reversal

Adapted from Zogh bi WA, Adams D, Bonow RO, et al. Recommendations for noninvasive evaluation of native valvular regurgitation: a report from the American Society of Echocardiography Developed in Collaboration with the Society for Cardiovascular Magnetic Resonance. *J Am Soc Echocardiogr*. 2017;30(4):303-371, with permission from Elsevier. Ref.[15]

iv) Contractility
  (1) If ventricular systolic dysfunction is present, consider "inodilators" such as dobutamine or milrinone.

b) **Monitoring**
  i) American Society of Anesthesiologists (ASA) standard monitors
    (1) ECG—monitors for myocardial ischemia and identifies arrhythmias
  ii) In patients with severe regurgitation and those undergoing moderate- to high-risk surgeries, more advanced monitoring should be considered.
    (1) Invasive arterial catheterization—allows for continuous blood pressure monitoring, enabling quick interventions to changes in afterload and to optimize coronary perfusion pressure
      (a) Placement prior to induction is strongly recommended for moderate to severe lesions.
    (2) Central venous pressure—allows for estimation of right atrial filling pressures
      (a) Placement of triple lumen catheter allows for administration of vasopressor or inotropic medications that may be needed to support cardiac function.
    (3) TTE or TEE—monitors ventricular systolic and diastolic function, provides assessment of wall motion and ventricular filling; aids in diagnosis of intraoperative hypotension
    (4) Pulmonary artery catheterization—provides information on cardiac function in addition to adequacy of cardiac output and perfusion
      (a) May be beneficial in patients with reduced left ventricular systolic function, as a means to trend mixed venous oxygen saturation and cardiac output

c) **Induction**
  i) Preinduction arterial lines are indicated to continuously monitor blood pressure and allow for early hemodynamic interventions.

d) **Maintenance**
  i) GETA or neuraxial/regional techniques can be performed, as long as close hemodynamic monitoring occurs and adverse hemodynamic effects are minimized.
    (1) Neuraxial anesthesia may also increase venous capacitance, which can severely decrease preload and cardiac output.
      (a) If a high block occurs due to local anesthetic blockade of cardiac accelerator fibers, bradycardia and reduced forward flow may result.
    (2) General anesthesia
      (a) May be accomplished via a combination of volatiles and opioids
  ii) Blood loss must be promptly replaced in order to maintain adequate intravascular volume.
  iii) Atropine or glycopyrrolate may be considered for the treatment of significant bradycardia or junctional rhythm but used with extreme caution in AS because the goal HR is 70-80.
  iv) Hemodynamically significant arrhythmias should be treated with electrical cardioversion.
  v) Phenylephrine should be used cautiously, as this can elevate the regurgitant fraction. A mixed agonist such as ephedrine may be a better choice.
  vi) In cases of acute AR, inotropic support may be required.
  vii) Intra-aortic balloon pump is contraindicated in AR patients since diastolic augmentation will worsen the regurgitant volume.

e) **Emergence**
  i) At the conclusion of surgery, the decision to extubate will be dependent on the patient's baseline condition, intraoperative course, and type of surgery.
    (1) In acute AR, presence of pulmonary edema may delay extubation.

ii) If extubation is deemed appropriate, maintain the hemodynamic goals, as discussed above.

8) **Postoperative management**
   a) Depending on the patient's comorbidities, intraoperative course, and expected postoperative hemodynamic changes, it may be necessary to arrange for postoperative recovery in a location that allows for more intensive hemodynamic monitoring, such as an intensive care or telemetry unit.
   b) Maintenance of adequate heart rate and preload, and avoiding increases in afterload are of the upmost importance.

9) **Treatment of aortic regurgitation**
   a) **Acute AR**
      i) Emergent valvular surgery is often indicated, and early intervention is associated with improved survival.
      ii) Afterload reduction is imperative to avoid increasing regurgitant volumes. Nitroprusside and IV hydralazine have been shown to ↓ regurgitant volume, ↓ LVEDV, and improve ventricular function.
      iii) Inotropes such as epinephrine, dobutamine, or milrinone may also be helpful in temporarily managing cardiogenic shock prior to valve surgery.
   b) **Chronic AR**
      i) Since patients remain clinically compensated for years, they can be safely followed until symptom onset, LV dilation (end-systolic diameter >50 mm), or LV dysfunction (EF < 50%).[5]
         (1) Increases in LVESD and decrease in LVEF are associated with the development of heart failure symptoms
      ii) Medical management:
         (1) Treatment of hypertension is recommended in patients with chronic AR (stages B and C)
            (a) Goal SBP < 140 mm Hg
            (b) Dihydropyridine calcium channel blockers and ACE inhibitors/ARBS are the preferred agent.[5]
            (c) Beta-blockers should not be first-line agents, given the reduction in HR, which leads to an increased diastolic filling time and a higher stroke volume.
         (2) Medical therapy can help alleviate symptoms in patients who have symptomatic AR, but who are considered high risk for surgery.
      iii) Surgical management:
         (1) In some instances where the valve leaflets are without pathology, valve sparing aortic root replacement or ascending aorta replacement are a possible strategy for the treatment of severe AR.
         (2) Valve replacement is a more common option, especially when the valve leaflets are calcified or stenotic.
            (a) AHA/ACC guidelines for the management of patients with valvular heart disease[5] recommend AVR for the following patients:
               (i) Patients with symptomatic, severe AR (stage D) promptly after the onset of symptoms (Class I, Level of Evidence B)
               (ii) Asymptomatic patients with severe AR and a left ventricular ejection fraction <50% (stage C2) (Class I, Level of Evidence B) or stage C patients undergoing other cardiac surgery (Class I, Level of Evidence C).

SUMMARY FOR AORTIC VALVULAR DISEASE

	Aortic Stenosis	Aortic Regurgitation
General Considerations	Long latent period without symptoms/complications Onset of symptoms results in large increase in mortality—surgery is the only effective therapy.	Acute AR may present with severe hemodynamic instability. Emergent cardiac surgery is usually necessary Chronic AR imposes both components of pressure and volume overload on the left ventricle
Indications for Surgery	Symptomatic severe AS Asymptomatic severe AS + reduced systolic function Severe AS when undergoing other cardiac surgery	Symptomatic AR Asymptomatic AR + LV dysfunction (EF< 50%) Severe AR when undergoing other cardiac surgery

Anesthetic Management

Preoperative	Complete cardiac history focusing on symptoms, current exercise tolerance, and evaluation of comorbid conditions. Review all relevant cardiac studies (ECG, TTE). For noncardiac surgery—review of valve disease severity, presence of symptoms, and relevant comorbidities should guide decision to proceed with elective surgery				
Intraoperative	**Hemodynamic Goals**				
		Heart Rate	**Preload**	**Afterload**	**Contractility**
	AS	Sinus Low normal HR	↑	Normal to slightly elevated	Maintain
	AR	↑	↑	↓	Maintain
Postoperative	For cardiac surgery—postoperative resuscitation and monitoring in an intensive care unit For noncardiac surgery, maintain hemodynamic goals in the postoperative period, with particular attention to emergence from GA				
Monitoring	Arterial line, central line ± PAC, +/− TEE				

LV, left ventricle; PAC, pulmonary artery catheter; SVR, TEE, transesophageal echocardiography.

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101 Right Ventricle, Tricuspid Valve, and Pericardiectomy

Sheela Pai Cole

Recent advances in medical science have improved the clinician's understanding of right ventricular and tricuspid valvular anatomy. These advances, combined with the early diagnosis of pericardial disease and intervention, have improved clinical outcomes. This chapter will review the relevant anatomy of the right ventricle (RV) and tricuspid valve (TV) and will provide detailed descriptions of the preoperative assessment, intraoperative management, and postoperative challenges of anesthesia for pericardiectomy.

1) **Anatomy of the right heart**
   a) Right ventricle
      i) The right ventricle (RV) in contrast to the left ventricle (LV) is a low-pressure high-volume system.
      ii) The RV muscle mass comprises one-sixth that of the LV.
      iii) RV forms an accordion-like crescent around the LV.
      iv) The blood supply to the LV occurs during diastole as the concentric muscle mass of the LV contracts during systole to stop blood flow. In contrast, RV blood supply is continuously occurring during systole and diastole.
      v) Due to the difference in the timing of blood supply, it is important to maintain both systolic and diastolic pressure to adequately perfuse the RV.



Maintaining RV perfusion
The RV is perfused during systole and diastole; hence, it is important to maintain systolic and diastolic pressure.

   b) Tricuspid valve
      i) The TV is a trileaflet atrioventricular valve located between the right atrium and ventricle.
      ii) The septal leaflet is inserted into the TV annulus at a point lower than the mitral leaflet insertion in the mitral annulus.
      iii) The apical displacement of the tricuspid valve allows for differentiation between the right and left sides of the heart on imaging studies.
      iv) The systolic movement of the lateral annulus of the TV known as tricuspid annular plane systolic excursion (TAPSE) is used as a surrogate of adequate RV contractility.
      v) Normal values for TAPSE are >18 mm.
2) **Pericardiectomy**
   a) Definition
      i) Surgical removal of part or most of the pericardium
      ii) This operation is most commonly performed to treat constrictive pericarditis or to remove a pericardium that has become calcified and fibrous.
3) **Pericarditis and pericardial disease**
   a) Definition of pericarditis
      i) Pericarditis is inflammation of the pericardium.
   b) Incidence and prevalence of pericarditis
      i) Pericarditis is the most common form of pericardial disease.
      ii) The incidence is 25-35 per 100 000 person-years.

iii) There is a preponderance among males.
iv) Accounts for ~0.2% of all cardiovascular hospital admissions.[1]
v) May be idiopathic (80%), followed by neoplastic (5%-10%); systemic inflammatory process and pericardial syndromes (2%-7%); tuberculous (4%) and purulent pericarditis (1%)
vi) Pericardiectomy is recommended for symptomatic patients with chronic relapsing pericarditis.
vii) Symptomatic pericarditis commonly manifests as constrictive pericarditis or chronic relapsing pericardial effusions.[1]


Pericardiectomy is recommended for symptomatic patients with chronic relapsing pericarditis.

c) Signs and symptoms of pericardial disease
i) Frequently, pericarditis presents with sharp pleuritic chest pain, which is improved upon sitting up.
ii) A friction rub maybe heard in one-third of patients due to inflammation between the layers of the pericardium.
iii) Nonspecific ST elevation in all leads may be seen in 60% of patients.
iv) In about 60% of patients with pericarditis, a small pericardial effusion may be visualized.
v) Poor prognostic indicators include fever (temperature >100.4°F), subacute course, large pericardial effusion, tamponade physiology, and lack of response to nonsteroidal anti-inflammatory agents.
vi) Chronic constrictive pericarditis presents with heart failure symptoms along with echocardiographic evidence of severe diastolic dysfunction.
vii) Recurrent pericarditis is defined by symptom-free interval of 4-6 weeks between episodes; recurrent pain combined with a friction rub; presence of EKG and echocardiographic features; presence of inflammatory markers such as elevated white blood cell count, sedimentation rate, and increased C-reactive protein.[2]


Poor LV function, preexisting end-organ dysfunction portend a poor prognosis.

d) Therapy for pericarditis
i) Medical therapy with anti-inflammatory agents such as nonsteroidal anti-inflammatory drugs (NSAIDs) and colchicine are the first-line therapy.
ii) Second-line agents such as steroids may be administered in cases that do not respond to first-line therapy.[1]
iii) Surgical treatment is reserved for chronic relapsing pericarditis.

4) **Preoperative assessment for pericardiectomy**
a) A preoperative assessment for pericardiectomy should include a complete history and physical.
b) Surgical planning
i) The etiology of pericarditis is an important consideration in surgical planning.
ii) Surgical intervention in neoplastic disease should be avoided, whereas active infections should be treated and stabilized prior to surgery.
c) Perioperative risk predictors
i) Presence of decompensated heart failure and LV dysfunction are poor prognostic indicators for perioperative outcomes. Medical stabilization should be attempted in these conditions prior to elective surgical pericardiectomy.
ii) Evidence of end-organ dysfunction signifies chronic decompensated heart failure and would likely portend a poor perioperative outcome.
d) Functional studies for preoperative cardiac evaluation
i) Preoperative evaluation of ventricular function allows for proper intraoperative hemodynamic and inotropic planning.[3]

ii) Echocardiography is the first-line imaging modality used to determine the anatomical and physiologic characteristics of pericardial disease.
(1) A detailed examination of biventricular function should be performed with echocardiography by a combination of 2-dimensional, 3-dimensional techniques as well as newer techniques such as strain and tissue Doppler modalities.

e) Imaging studies for preoperative pericardial evaluation
i) Imaging strategies often can further elucidate the nature and extent of pericardial disease to aid in surgical planning.
ii) Computerized tomography (CT) and magnetic resonance imaging (MRI) provide information on calcific deposits and tissue characteristics of pericardial disease, both of which help with surgical decision-making.[4]
iii) 3D echocardiography (3D echo) has clear-cut advantages in studying the echodensities involving the pericardium.
(1) 3D echo visualizes these densities from different angles and provides information about possible nonhomogeneity within the pericardial lesions.
(2) For example, a hypoechoic center is characteristic of granulomatous disease, while the presence of fibrin strands and pericardial exudate are helpful findings to differentiate tuberculous pericardial effusion from other chronic pericardial effusions.[3]

5) **Intraoperative concerns (Table 101-1)**
a) Procedure risk
i) Isolated pericardiectomy historically carried a very high mortality and morbidity rate (~14%).
ii) In the current era, early diagnosis of heart failure in pericardial disease and mitigation of end-organ damage have resulted in improved postoperative outcomes.[5,6]

b) Surgical approach
i) Overview
(1) Multiple single-center studies report the benefit of pericardiectomy; there has been no consensus on a standardized surgical technique.
(a) Techniques vary widely from surgeon to surgeon and institution to institution.
ii) Left-sided anterolateral thoracotomy
(1) The approach historically performed by surgeons
(2) Less commonly performed because the right-sided pericardium cannot be effectively removed
iii) Bilateral anterior thoracotomy
(1) The approach performed in many surgical centers
(2) Consists of anterior phrenic to phrenic resection of the pericardium.
(3) Cardiopulmonary bypass (CPB) is not used due to difficulties in resecting and exposing below the phrenic nerve.
(4) The anterior pericardium is resected, leaving behind the posterior pericardium.
iv) Median sternotomy with CPB
(1) This approach allows for complete pericardiectomy.
(2) An oscillating saw is used to enter the sternum.
(3) Systemic heparin is administered, and the aorta and SVC are cannulated, and partial bypass is initiated.
(4) The pericardium is resected while carefully preserving the phrenic nerves.

TABLE 101-1 Perioperative System-Based Checklist

	Monitoring Equipment	Interventional Equipment/Pharmacology
Neurologic	• Cerebral oximetry • Electroencephalogram • Temperature	• Anesthetic agents (inhalational agents, propofol, etomidate) • Analgesic agents (fentanyl, sufentanil, ketamine, dexmedetomidine, Tylenol) • Muscle relaxant agents (rocuronium, vecuronium) • Fluid warmer, Bair Hugger
Cardiovascular	• 5 lead EKG • Large-bore peripheral IV • Arterial line • Central line • PAC • TEE • CCO/SvO_2 monitor	• Inotropes: Beta agonists: epinephrine, dobutamine, other: milrinone • Vasopressors: phenylephrine, norepinephrine, vasopressin • Vasodilators: nitroglycerin, nitroprusside, clevidipine • Antiarrhythmics: amiodarone, lidocaine, atropine, glycopyrrolate • Rapid transfusion device (level 1, Belmont) • Temporary pacer box, (ICD device deactivation) • IABP, ECMO, VAD (in case of failure of separation from CPB)
Respiratory	• Pulse oximetry • Capnography	• Subglottic endotracheal tube • Respiratory parameters to allow adequate venous return • Inhaled pulmonary vasodilators
Gastroenterology	• OGT	
Renal	• Foley	• Diuretics
Hematology/ infectious disease	• ACT • ABG • TEG	• Antibiotics • Anticoagulants: heparin • Procoagulants: protamine, antifibrinolytics (eg, TXA), vitamin K • Blood products: blood products
Metabolic/ endocrine	• ABG	• Electrolytes: calcium, magnesium, potassium, and acid base • Insulin, dextrose

EKG, electrocardiogram; PAC, pulmonary artery catheter; TEE, transesophageal echocardiography; CCO, continuous cardiac output; SvO_2, systemic venous oxygenation; ICD, implantable cardiac defibrillator; IABP, intra-aortic balloon pump; ECMO, extracorporeal membrane oxygenation; VAD, ventricular assist device; CPB, cardiopulmonary bypass; OGT, oral gastric tube; ACT, activated clotting time; ABG, arterial blood gas; TEG, thromboelastography.

v) Surgical approach considerations
   (1) There continues to be an ongoing controversy whether a complete pericardiectomy should be performed or whether an anterior pericardiectomy would suffice.
   (2) An anterior pericardiectomy removes only the anterior pericardium between both phrenic nerves.
   (3) A complete pericardiectomy removes not only the anterior pericardium as described but also the inferior (diaphragmatic) and left lateral (posterior to left phrenic nerve). During the procedure, care should be taken to preserve the phrenic nerves neuromuscular monitoring intraoperatively. [7]

(4) The pericardial "peel" in constrictive pericarditis may be densely adherent and calcified necessitating CPB for safe conduct of the procedure.
(5) A limited anterior pericardiectomy via an anterior thoracotomy maybe able to be performed safely without bypass especially in the setting of chronic relapsing effusions.[5]


Anterior pericardiectomy—removal of anterior pericardium; complete pericardiectomy—anterior, inferior, and left lateral.

c) Monitoring
   i) Routine American Society of Anesthesiologist monitors are recommended.
   ii) Invasive monitoring should include a preinduction arterial line, central venous pressure monitoring, and transesophageal echocardiography. Presence of significant pulmonary hypertension or RV dysfunction should prompt placement of a pulmonary artery catheter. **Table 101-1** demonstrates the monitoring and preparation required for these cases.
   iii) Use of cerebral oximetry and bispectral index monitors should be individualized based on patient disease, institutional practice, and provider experience. These monitors lower the incidence of stroke, tissue malperfusion, and delirium in cardiac surgical patients. [8,9]
d) Induction of anesthesia
   i) Presence of significant decompensated heart failure requires judicious induction with careful maintenance of hemodynamics with short-acting vasoactive drugs to preempt shock.
   ii) Presence of significant diastolic dysfunction should warrant careful preload management to minimize risk of cardiovascular collapse.
e) Maintenance of anesthesia
   i) General anesthesia and positive pressure ventilation will potentially worsen venous return by increasing intrathoracic pressure.
   ii) Close monitoring of airway pressures as well as watching for "auto-PEEP" will help restore preoperative intrathoracic milieu while mitigating hypotension and cardiovascular collapse.
   iii) A balanced technique with inhalational and intravenous agents should be used carefully to maintain end-organ perfusion.


Look out for auto-PEEP during institution of positive pressure in patients undergoing pericardiectomy.

f) Cardiopulmonary bypass
   i) Use of CPB will allow for extensive pericardial resection with hemodynamic stability.
   ii) When CPB is not planned, close observation for malignant arrhythmias and constant communication with the surgical team is required to maintain hemodynamic stability.
   iii) Use of antiarrhythmic agents such as amiodarone and lidocaine may be required depending on the type of arrhythmia generated.
g) Intraoperative blood component therapy
   i) Based on the extent of pericardial adhesions, protracted dissection to separate the pericardial sac may generate many raw bleeding surfaces.
   ii) Blood and blood products may be required to support ongoing losses and coagulopathy if CPB is used.
      (1) Thromboelastography and related coagulation monitors along with routine parameters of coagulation such as prothrombin time (PT), partial thromboplastin time (PTT), and international normalized ratio (INR) may help guide transfusion practices.

h) Intraoperative TEE evaluation
   i) Intraoperative TEE should be utilized not just for overall biventricular function assessment but should be focused on assessment of the RV and tricuspid valve to promptly diagnose and treat RV dysfunction.[10]



Invasive monitoring with TEE monitoring is required for safe conduct of these cases.

i) Tricuspid regurgitation
   i) Decortication of the pericardium often results in RV release and concomitant dysfunction but in some cases will "liberalize" the tricuspid annulus and worsen tricuspid regurgitation.
   ii) Some experts recommend tricuspid annuloplasty if pericardiectomy results in >2+ tricuspid regurgitation.[11]

6) **Postoperative challenges**
   a) A successful pericardiectomy results in reduction in the central venous pressure and improvement in New York Heart Association (NYHA) classification from NYHA III to NYHA II are most often seen in the long term.[12]
   b) In the immediate postoperative period, release of the long-standing constrictive pericardial sac may result in transient worsening of biventricular function necessitating prolonged inotropic, pressor, and hemodynamic support.[13]



Postoperative LV dysfunction is common after pericardiectomy due to changes in LV geometry from release of cytokines and the pericardial constraint.

   c) Ventilatory support
      i) Tracheal extubation and liberation from mechanical ventilation should be performed expeditiously to relieve the role of positive pressure ventilation on preload.
      ii) This needs to be balanced with the tenuous hemodynamic state of the patient. Presence of ongoing bleeding and hemodynamic instability must be corrected prior to extubation.
   d) Arrhythmia and pacemaker support
      i) Antiarrhythmic therapy should be continued till electrophysiologic evidence of their cessation. Transvenous pacing should be considered for significant bradyarrhythmias.
      ii) Decisions for permanent pacemaker placement should be deferred for 7-10 days after the surgery to allow abatement of the inflammatory postsurgical state.
   e) Right ventricular failure
      i) Postoperative morbidity and mortality after a pericardiectomy is most commonly associated with the development of RV failure.
      ii) RV failure presents with elevated CVP, which results in back pressure on hepatic and renal tissue beds resulting in hepatic congestion and renal failure.
      iii) RV dysfunction and failure are difficult problems to treat and remain a major challenge and an important cause of postoperative death.[14]
      iv) Maintenance of adequate systolic and diastolic blood pressure while maintaining a low CVP with aggressive diuresis is the mainstay of treatment of RV failure.
      v) It is important to remember that "time is muscle," and every hour that the CVP remains elevated, there is RV muscle injury.[15]
      vi) Beta agonists such as epinephrine that augment contractility while maintaining blood pressure are pivotal in treatment of RV failure.
         (1) The chronotropic effect of heart rate augmentation improves systolic blood flow and aids in RV perfusion.
   f) Renal failure

i) Postoperative morbidity and mortality after a pericardiectomy is also associated with the development of renal failure.
ii) Institution of continuous renal replacement therapy or ultrafiltration may be required for fluid volume removal if aggressive diuresis fails.

g) Pulmonary hypertension
i) Use of inhaled pulmonary vasodilators may be considered in the setting of elevated pulmonary vascular resistance (PVR).
ii) Long-acting prostacyclin analogues, nitric oxide (NO) donors, phosphodiesterase type 3 and 4 inhibitors, and type A–specific endothelin receptor antagonists are available pulmonary vasodilators.[16]
iii) It is important to remember the differences in various inhaled agents especially during the weaning process.
(1) Inhaled nitric oxide (INO) generates methemoglobin, and long-standing use requires routine monitoring.
(2) Weaning INO should be done in a slow, deliberate, manner to avoid rebound pulmonary vasoconstriction.
(3) Inhaled prostacyclin analogues such as epoprostenol have platelet inhibition properties, which may provoke pulmonary hemorrhage, although this remains a very rare complication.
(4) There is minimal rebound pulmonary vasoconstriction, but use of the oral agents may help in the weaning process.[16]
(a) There are some data to advocate starting oral pulmonary vasodilators (eg, sildenafil) to help in the weaning process.
(5) Nebulized phosphodiesterase inhibitors have been used with mixed success in PVR reduction.
(a) Challenges with delivery and formulation along with tachyphylaxis remain the main problems with this therapy.
iv) Systemic pulmonary arterial vasodilators such as phosphodiesterase inhibitors (eg, milrinone) work well to reduce PVR.
(1) Use is limited to patients who have adequate systemic blood pressure.
(2) Systemic pulmonary vasodilators are nonspecific vasodilators and may cause hypotension, which worsens RV perfusion.



Maintaining SBP and MAP is important for prevention of RV failure. Furthermore, it is important to maintain a low normal CVP in RV dysfunction.

7) **Outcomes**
a) Outcomes after pericardiectomy have improved with early diagnosis and intervention.
b) Early adoption of surgery after failed medical therapy can conclusively mitigate symptoms and improve quality of life.
c) Procedural outcomes depend on the choice of the procedure. Some centers advocate complete pericardiectomy as procedure of choice, but this needs to be balanced with duration of CPB, degree of inflammatory response, and overall duration of the procedure.
d) Another series suggests that better patient selection for anterior pericardiectomy may change the outcomes for that procedure.
e) Overall pericardiectomy is a therapeutic option at curing select patients with symptomatic recurring pericardial disease.
f) RV failure and renal failure remain the main causes of morbidity and mortality after this procedure.

CHAPTER SUMMARY FOR RIGHT VENTRICLE, TRICUSPID VALVE, AND PERICARDIECTOMY

Anatomy	RV is a low-pressure high-volume system. It is important to maintain both systolic and diastolic pressure to adequately perfuse the RV. The TV is a trileaflet atrioventricular valve located between the right atrium and ventricle.
Pericarditis and Pericardial Disease	Pericarditis is the most common form of pericardial disease. Frequently, pericarditis presents with sharp pleuritic chest pain, which is positionally improved upon sitting up. Pericardiectomy is recommended for symptomatic patients with chronic relapsing pericarditis.
Preoperative	The etiology of pericarditis is an important consideration in surgical planning. Presence of decompensated heart failure and LV dysfunction are poor prognostic indicators for perioperative outcomes. Echocardiography is the first-line imaging modality used to determine the anatomical and physiologic characteristics of pericardial disease.
Intraoperative	Isolated pericardiectomy historically carried a very high mortality and morbidity rate (~14%). There is no consensus on a standardized surgical technique. Invasive monitoring with TEE monitoring is required.
Postoperative	In the immediate postoperative period, release of the long-standing constrictive pericardial sac may result in transient worsening of biventricular function necessitating prolonged inotropic, pressor, and hemodynamic support.[13] Tracheal extubation and liberation from mechanical ventilation should be performed as soon as possible to relieve the role of positive pressure ventilation on preload. RV failure remains a difficult postoperative problem and a major cause of mortality and morbidity in the perioperative period.
Outcomes	Outcomes of pericardiectomy have improved after better understanding of RV and TV anatomy and improvements in postoperative management. Early adoption of surgery after failed medical therapy can conclusively mitigate symptoms and improve quality of life. Overall pericardiectomy is a therapeutic option at curing select patients with symptomatic recurring pericardial disease. RV failure and renal failure remain the main causes of morbidity and mortality after this procedure.

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102 Aortic Disease and Procedures

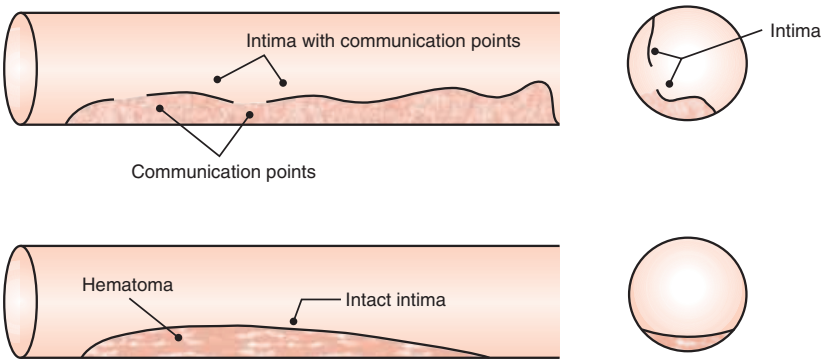
Sarah Sarcona • Gregory K. Kim

The volume of cases of thoracic pathology continues to grow due to an aging population and earlier diagnosis of pathology. Some aortic diseases can be treated medically; however, surgery remains a mainstay of treatment for certain aortic pathology. Surgical techniques continue to improve as do surgical outcomes; however, the surgical procedures for repair are complex. Much of the repair is performed utilizing cardio pulmonary bypass and may even include full hypothermic circulatory arrest. Ischemia of major organs, especially the spinal cord and brain, is a major concern of the anesthesiologist during these surgeries. The anesthetic management for these cases is challenging, and a detailed knowledge of the disease process as well as the more unique aspects of these operations is required.

I. Aortic dissection

1) **Introduction**
   a) The aortic wall contains three layers (intima, media, and adventitia).
   b) Aortic dissection results from the separation of these layers, most frequently between the intima and media (**Fig. 102-1**).
      i) The blood tracks within the wall of the aorta can progress rapidly and involve any area of the aorta.
      ii) These dissections may progress proximately to involve the coronary arteries and the aortic valve compromising its function and also propagate distally to involve the carotid, renal, and iliac arteries.
   c) Acute aortic dissection is one of the most common aortic emergencies, and without prompt care can be fatal.[1]
2) **Epidemiology**
   a) Acute aortic dissection incidence is estimated at around 3 per 100 000 yearly, although this is likely an underestimation.[2]
   b) Mortality from aortic dissection is estimated at 1% per hour initially, with half the patients not surviving to the 3rd day.[3]
   c) There is not a single cause, but many risk factors can be responsible.[4,5]
      i) Long-standing arterial hypertension
      ii) Advanced age
      iii) Smoking
      iv) Dyslipidemia
      v) Cocaine
      vi) Connective tissue diseases
         (1) Marfan syndrome, Ehlers-Danlos syndrome, Turner syndrome, bicuspid aortic valve, coarctation, giant cell arteritis, Takayasu arteritis, syphilis
      vii) Aortic aneurysm
      viii) Pregnancy
      ix) Trauma
         (1) Fall or catheter intervention
      x) Aortic surgery
3) **Diagnosis**
   a) The most common symptom is sharp pain, usually in the chest or back.[1]

FIGURE 102-1 The two forms of aortic dissection: (*upper*) tear of the intima into the media causing the column of blood to propagate with potentially multiple points of communication between the lumen and the intima/media space; (*lower*) the spontaneous intramural hematoma variant in which there is rupture of the smaller vasculature within the wall of the vessel resulting in hematoma in the medial space.



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i) May also present with syncope, hypertension, and pulse deficits
ii) May have a variation in pulse or BP between two arms depending on location and propagation of the dissection
iii) While hypertension is a common symptom, hypotension may occur.
iv) Symptoms may vary depending on the involvement of the coronary arteries or aortic valve—aortic valve involvement may be diagnosed with a new-onset murmur.
v) Cardiac tamponade may occur, especially with dissection into the aortic valve.[4]

b) Imaging is often used to make the diagnosis.
i) Chest CT is usually the initial imaging modality (Fig. 102-2).
(1) Sensitivity of 83%-95% and specificity of 87%-100% for diagnosis of acute aortic dissection[4]
ii) Patients usually under more than one imaging modality to characterize the dissection.
iii) Chest x-ray may show widened mediastinum as the significant radiologic finding but is not sufficient to rule out dissection (Fig. 102-3).[4]
iv) TTE can also be used to diagnose but cannot rule out a dissection in an emergency setting (Fig. 102-4).[4,6]
v) TEE is comparable to CT scan for diagnosis with a sensitivity as high as 98% and a specificity range from 63% to 96%.[5]

4) **Types of aortic dissection**

a) The thoracic aorta has four segments:
i) The aortic root—further divided into the aortic annulus, the sinuses of Valsalva, and the sinotubular junction
ii) The ascending aorta—from sinotubular junction to innominate artery



Aortic dissections do not always present with hypertension. Hypotension is often found in patients with dissections propagating into the coronary arteries causing myocardial ischemia, aortic valve causing acute aortic insufficiency, or causing pericardial tamponade.

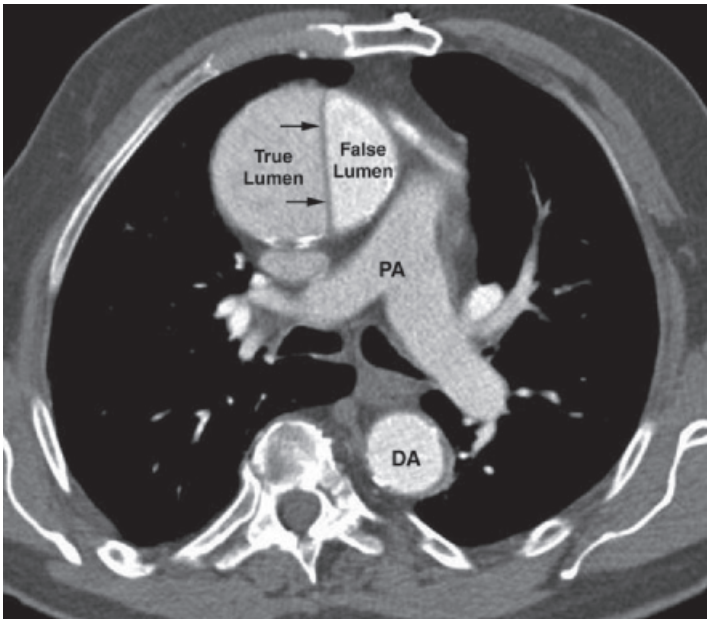


Best imaging modalities to confirm aortic dissections are CT scans and TEE.



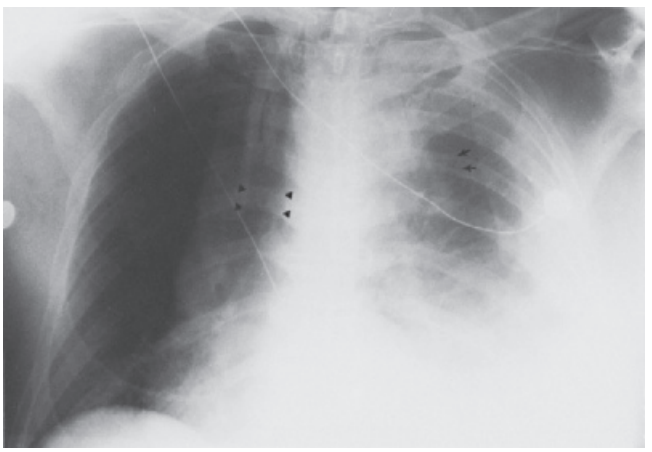
Chest x-ray and TTE cannot rule out aortic dissections.

FIGURE 102-2 CT Scan Showing Ascending Aortic Aneurysm With the True and False Lumen, Pulmonary Artery (PA), Descending Aorta (DA).



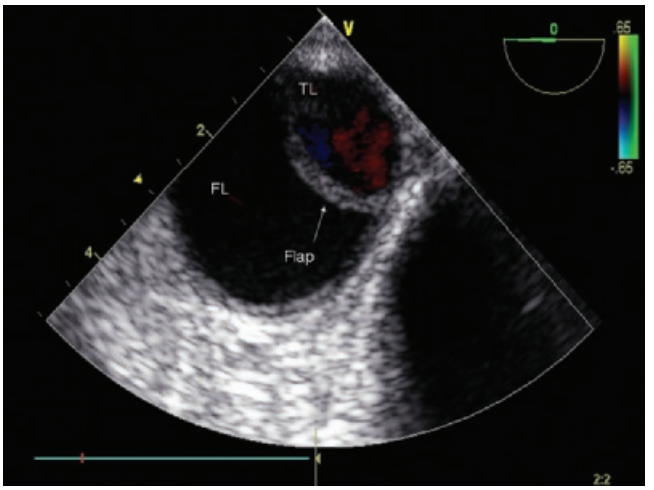
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FIGURE 102-3 Chest X-Ray of a Type A Aortic Dissection. Note the indistinct aortic knob (*arrows*), tracheal deviation (*triangles*), and left pleural effusion.



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FIGURE 102-4 Transesophageal Echocardiogram of the Aorta in Short Axis. The true lumen (TL) is shown along with the dissection flap creating the false lumen (FL).



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iii) Transverse aortic arch—from the innominate artery to the left subclavian artery
iv) Descending aorta—from the left subclavian to the diaphragm

b) Two classifications for aortic dissection have been established **(Fig. 102-5)**.
i) The Stanford classification for aortic dissection—more commonly used[4]
(1) Type A (DeBakey types I and II)—any involvement of the ascending aorta
(2) Type B (DeBakey type III)—dissection confined to descending aorta
ii) DeBakey classification[4]
(1) Type I—originates in the ascending aorta and propagates to at least the aortic arch
(2) Type II—originating in and confined to the ascending aorta
(3) Type III—originating in the descending aorta and extending distally or proximally

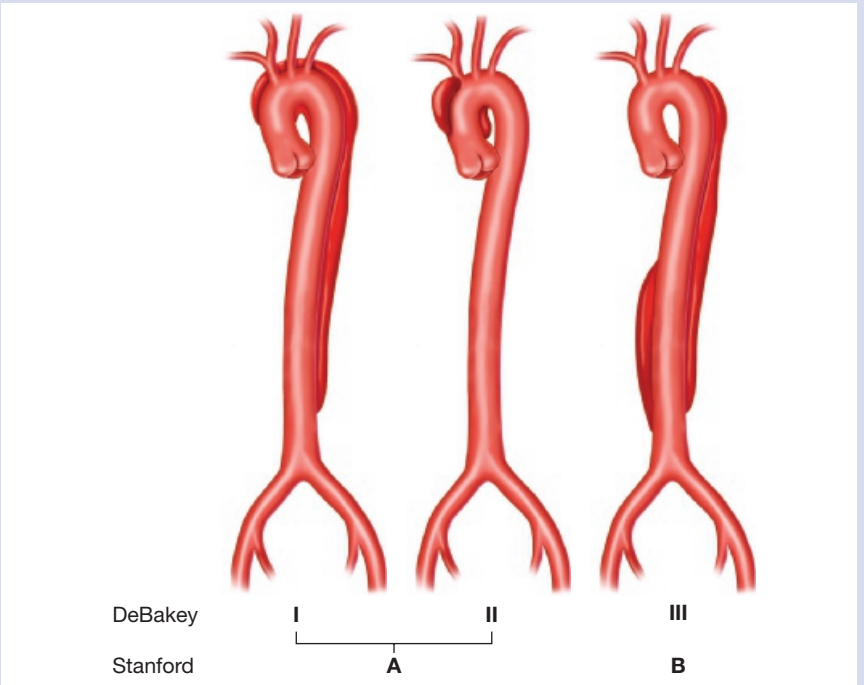


Medical management of type A dissections carries a mortality risk as high as 50%!

c) The decision to proceed with surgical vs medical management is determined, among other things, by the location of the dissection.
i) Surgical treatment is indicated for all type A aortic dissections, while medical management can be used for uncomplicated type B dissections.[7]
ii) Mortality for surgical management of acute type A aortic dissection is 7%-36%, while medical management carries a 50% mortality rate.[2]
iii) The main goal for surgical management in type A dissection is to prevent pericardial effusion leading to tamponade as well as aortic rupture.
iv) The main goal in type B dissection is preventing rupture.

5) **Surgical management**
a) In emergency cases, the coordination of a safely delivered anesthetic and rapid IV access is essential.
b) The primary goal of surgical management is to correct ascending and transverse aortic pathology.
i) Descending aortic pathology can be managed medically.

FIGURE 102-5 Stanford and DeBakey Classifications.



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c) The key portions of the surgery are as follows:
   i) Peripheral cannulation
      (1) Bypass access is first obtained peripherally, often in the femoral and axillary vessels.
      (2) Arterial cannulation can be accomplished for antegrade perfusion via the distal aortic arch (if it is not involved), right subclavian artery, innominate artery, or true lumen of the dissected ascending aorta.
      (3) Retrograde aortic perfusion can occur using either femoral artery at the risk of extension of the dissection.
      (4) Venous cannulation can be provided via femoral or central veins.
   ii) Chest opening
      (1) Once peripheral bypass cannulas are placed, the chest can now be opened for access to the aorta.
      (2) Care must be taken at this stage as the aneurysmal or dissected vessel can be dilated such that injury could occur during chest opening.
      (3) The perfusion team must be on standby for quick transition to bypass should injury to the aorta occur.
   iii) Cardiopulmonary bypass (CPB)



Large-bore IV access is essential **prior** to start of the surgical procedure.



Anesthetic and perfusion teams must be available for immediate transition to cardiopulmonary bypass should injury to the aorta occur.

(1) Central cannulation can be obtained once the chest is opened; however, this decision depends on the extent of the aneurysm or dissection to be repaired as well as the current stability of the patient.
(a) Otherwise, bypass can be initiated with previously obtained peripheral cannulation.
(2) Transition onto bypass is a concerted effort between the surgeon, anesthesiologist, and perfusionist.
(3) The patient is administered heparin IV prior to CPB to obtain an activated clotting time (ACT) >400.

READ MORE
Mechanical circulatory support, Chapter 97.

iv) Deep hypothermic circulatory arrest
(1) Often a necessary step for thoracic aneurysm repair to allow for optimal surgical conditions
(2) The patient is cooled to a temperature of 18°C-20°C using cooled fluid through the CPB circuit.[7,8]
(3) Additionally, the patient's head is packed in ice to ensure cooling of cerebral tissue.
(4) Once the patient is adequately hypothermic, cardiac arrest occurs and circulatory arrest begins with temporary cessation of CPB.
(5) Most patients can tolerate 30 minutes of hypothermic circulatory arrest, but increase in the incidence of brain injury is higher after 40 minutes.[8]



The incidence of brain injury is higher after >40 minutes of deep hypothermic circulatory arrest.

v) Selective cerebral perfusion
(1) The brain can be selectively perfused during circulatory arrest through perfusion cannulas placed retrograde or anterograde.
(2) Antegrade cerebral perfusion can be achieved either bilaterally or unilaterally.
(a) With unilateral selective cerebral perfusion, blood flow to the contralateral side depends on collateral flow in the circle of Willis.
(b) Unilateral perfusion is successful for short (<40 minutes) amounts of time, but if longer times are needed, bilateral perfusion is preferable.[9]
(3) An superior vena cava (SVC) cannula can be placed for retrograde perfusion and maintenance of cerebral hypothermia.

vi) Cessation of CPB
(1) At the end of the full repair, the patient is weaned off CPB.
(2) The patient must slowly be warmed back to physiologic temperature.
(3) This is a coordinated effort between the surgeons, perfusionists, and anesthesiologist.

6) **Anesthetic management**

a) A preanesthetic assessment is done on all patients prior to delivery of anesthesia.
i) Assess cardiac risk, preoperative medications, and preexisting conditions.
ii) Depending on urgency of case, additional testing can be done.

b) Standard American Society of Anesthesiologists (ASA) monitors with additional hemodynamic and neuromonitoring
i) Invasive blood pressure monitoring with arterial line, sometimes bilaterally
(1) Arterial line placement is dependent on the location of the cross clamp and bypass cannulation.
ii) Temperature monitoring for the hypothermic protocol as well as rewarming
iii) Neuromonitoring may be used and is a decision made based on the urgency of the case, patient factors, and surgical technique.



Have perfusion and surgical teams present during induction in the event that patient needs to emergently undergo CPB.

c) Large potential for massive blood loss
i) Large-bore IV access is required.

ii) Must have rapid availability of packed red blood cells, platelets, and plasma
iii) Central venous access is essential.
(1) Potential requirement for pulmonary artery catheterization to aid in resuscitation management and cardiac function

d) Intraoperative TEE is indicated in thoracic aortic surgery for hemodynamic and cardiac function monitoring, as well as to assist in surgical guidance.

e) Induction is a critical step in anesthesia management.
i) All anesthetic agents should be titrated slowly to effect to maintain hemodynamic instability while simultaneously blunting any sympathetic response to endotracheal intubation.[10]
(1) Etomidate is often the anesthetic of choice due to its hemodynamic stability with the addition of fentanyl and midazolam.
(2) Induction should include doses of narcotics to help blunt response to endotracheal intubation.
ii) Vasodilatory drugs as well as vasoconstricting drugs should be readily available.



Induction goals for patients with aortic dissection:
- Avoid hypertension.
- Avoid tachycardia.
- Reduce contractility.
- Maintain adequate preload.
- Smooth induction with minimal stimulation on placement of ETT

f) Maintenance of anesthesia is achieved with a balanced anesthesia approach using inhalational anesthetics, neuromuscular blockade, and narcotics.[10]
i) Blood pressure should be maintained at systolic blood pressure (SBP) 100-120 to prevent further propagation of the dissection.
ii) Vasodilators can be used for this purpose.
iii) Maintenance of a slower HR, possibly with the use of β adrenergic antagonists, is important as well as decreased contractility to reduce intimal wall stress.

g) Patients often remain intubated and require postoperative ICU care.

6) **Cerebral protection**

a) There is a high risk of stroke due to the cerebral ischemia during aortic arch procedures.[9]

b) The brain is highly susceptible to ischemia during circulatory arrest.
i) Profound hypothermic circulatory arrest and cooling to 18°C prevents neurologic injury.[8]
ii) Measurement of cerebral oximetry may be helpful for these procedures.
(1) Cerebral oximetry estimates cerebral oxygenation utilizing venous and arterial blood.
(2) Provides an estimate of cerebral oxygenation in the frontal cerebral cortex
(3) Not dependent upon pulsatile flow
iii) Rewarming should happen gradually in order to minimize the increase in cerebral metabolic rate and subsequent reperfusion injuries.



Cerebral oximeter can assess cerebral oxygenation during periods of prolonged hypotension such as circulatory arrest.

c) **Retrograde cerebral perfusion**
i) An SVC cannula can be placed for retrograde perfusion and maintenance of cerebral hypothermia.

d) **Selective antegrade cerebral perfusion**
i) The brain can be perfused selectively during brief periods of surgery through perfusion cannulas in the cerebral vessels.
ii) Shown to possibly reduce the incidence of temporary neurological dysfunction, however, may not reduce the risk of stroke.[9]

e) **Pharmacologic neuroprotection**

i) Thiopental and propofol have been used for neurologic protection after deep hypothermic circulatory arrest, but there is no evidence that either improves neurologic outcomes.[8]
ii) Steroids have been shown to decrease proinflammatory cytokines, but there is no evidence that they are neuroprotective after deep circulatory arrest.[8]
(1) Dexamethasone and methylprednisolone have been used.

II. Aortic aneurysms

1) Introduction

a) An aneurysm is a permanent dilation of an artery to 1.5 times its normal diameter.
b) Often results from cystic medial degeneration, which leads to weakening of the aortic wall

2) Epidemiology

a) There are multiple causes of aortic aneurysms, including:
i) Marfan syndrome, familial aortic aneurysm syndrome, bicuspid aortic valve, atherosclerosis, syphilis, Turner syndrome, aortic arteritis, aortic dissection, trauma[11]
b) The mean rate of growth for thoracic aneurysms is 0.1 cm/y.[7]

3) Diagnosis

a) **Most patients are asymptomatic.**
i) The aneurysm is discovered incidentally by imaging studies that were done for another medical reason.[11]
ii) Patients may have a diastolic murmur discovered on examination.
b) **Symptoms may be present.**
i) Patients may have symptoms of heart failure or mass effect symptoms from compression of the trachea or bronchus.[7]
ii) Chest or back pain is rare.
iii) If rupture or dissection has occurred, the typical presenting symptom is the sudden onset of severe pain often in the chest, back, neck, or abdomen.[11]
c) The best methods to image the aorta are CT with contrast and MR angiography.[11]
i) Basic chest x-ray can also lead to the diagnosis if showing specific features sure as widened mediastinum, enlarged aortic knob, or leftward tracheal deviation (**Fig. 102-6**).

4) Types of aneurysm

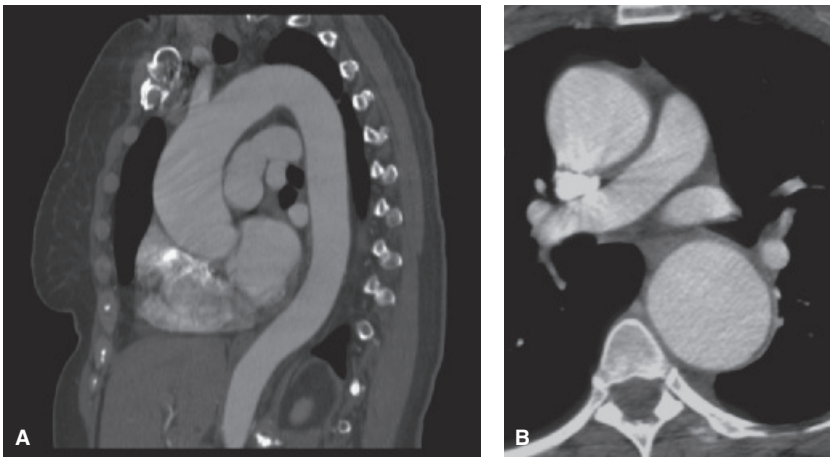
a) **The Crawford classification is used.**[12]
i) Type I—from left subclavian artery to the diaphragm
ii) Type II—from left subclavian artery to below the renal arteries
iii) Type III—from midthoracic descending aorta to below the renal arteries
iv) Type IV—from diaphragm to below the renal arteries

5) Surgical management

a) Surgery is indicated for thoracic aortic aneurysms measuring a diameter of ≥5.5 cm.[11]
i) This cutoff can be 6 cm for patients with an increased operative risk.
ii) Conversely, operative repair may occur at 5 cm or less in those with increased risk of dissection or rupture (eg, Marfan syndrome).[7]
b) **Open repair**
i) Surgical repair of ascending aortic aneurysms has a mortality rate of 3%-5%.[11]
ii) For descending aortic repair, the mortality rate is 5%-14%.
iii) The patient is placed on CPB, and the aneurysmal portion of the aorta is resected and replaced with a prosthetic tube graft.
c) **Aortic root and valve involvement**
i) If the aneurysm involves the aortic root with aortic regurgitation, the aortic valve can also be replaced at the same time.
(1) Surgery is challenging and carries a risk of stroke and neurologic damage.

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Aortic valvular disease, Chapter 100

FIGURE 102-6 CT Scan of a Thoracic Aortic Aneurysm.



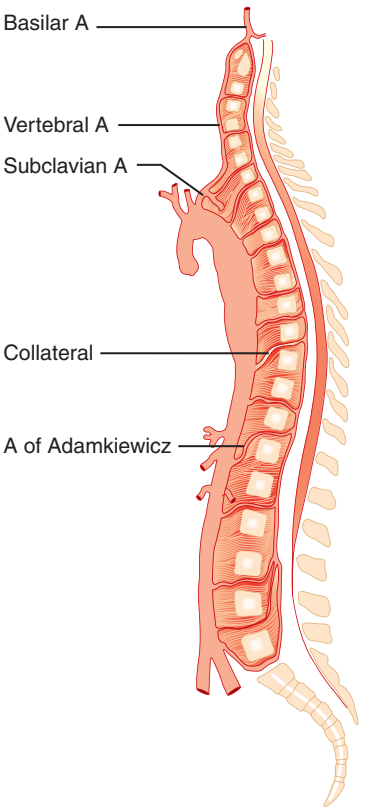
Reprinted with permission from Ghanta RK, Ailawadi G. Thoracic Aortic Aneurysms and Aortic Dissection. In: Greenfield LJ, Mulholland MW, et al. *Greenfield's Surgery: Scientific Principles and Practice*. 6th ed. Philadelphia, PA: Wolters Kluwer; 2017:1563.

ii) A valve-sparing repair can be done in which the sinuses of Valsalva are excised, sparing the aortic leaflets. The graft is then sewn to the base of the aortic annulus, and the aortic valve leaflets are reimplanted in the graft.[7]

d) Endovascular repair
   i) Descending thoracic aortic aneurysm repair can be achieved using an endovascular stent graft.[11]
   ii) This is a less invasive method with potentially lower morbidity, reserved for those with favorable anatomy or who are poor surgical candidates.
e) Staged procedures can be performed where one aortic segment is repaired first and the second segment repaired at a later date.

6) **Anesthetic management**
   a) The same management considerations occur as with aortic dissections.
   b) Risks that occur with aortic cross clamping include:
      i) Renal failure
         (1) Usually from acute tubular necrosis and ischemia-reperfusion injuries to the kidney
         (2) Mannitol can be given to increase renal blood flow during cross clamping.[13]
         (3) Dopamine does not aid with renal protection.[13]
         (4) Adequate fluid replacement remains crucial in minimizing renal risk.[13]
      ii) Visceral ischemia
      iii) Spinal cord ischemia with resulting paraplegia (**Fig. 102-7**)
         (1) The artery of Adamkiewicz supplies blood to the anterior two-thirds of the spinal cord and arises from the lower thoracic or upper lumbar aorta (often originating between T8 and T12).
         (2) Cross clamping can affect this blood supply.
         (3) Methods have been employed to reduce the risk of spinal cord ischemic injury.
            (a) Epidural cooling for hypothermic protection of the spinal cord
            (b) Cerebrospinal fluid (CSF) drainage via lumbar drain

FIGURE 102-7 The Vascular Supply of the Spinal Cord.



From Valentine EA, Ochroch EA. Anesthesia for vascular and endovascular. In: Barash PG, et al., eds. *Clinical Anesthesia*. 8th ed. Philadelphia, PA: Wolters Kluwer; 2017:1131 and Adapted from Piccone W, DeLaria GA, Najafi H. Descending thoracic aneurysms. In: Bergan JJ, Yao JST, eds. *Aortic Surgery*. Philadelphia, PA: WB Saunders; 1989:249.

(c) Reimplantation of important intercostal arteries
(d) Using intraoperative somatosensory evoked potential monitoring
(e) Using atriofemoral bypass to allow for distal aortic perfusion

7) **Lumbar drains**
   a) Spinal cord ischemia can result in paraplegia that may or may not resolve.
   b) Spinal cord perfusion pressure is the difference between the distal aortic pressure and the CSF pressure.
      i) *Spinal cord perfusion pressure = mean arterial pressure (MAP) – intrathecal or cerebrospinal pressure*
         (1) *CVP can be used as a surrogate for intrathecal pressure.*
      ii) Aortic cross cramping decreases anterior spinal artery pressure and increases CSF pressure.
      iii) Spinal cord perfusion pressure can therefore be increased either by decreasing CSF pressure with a spinal drain or increasing distal aortic pressure.

c) A lumbar drain can be introduced into the subarachnoid space at the L3-L4 or L4-L5 intervertebral space.
   i) This catheter can be set to drain when the CSF pressure increases above 10-12 mm Hg.[14]
   ii) Generally no more than 10 mL/h of CSF should be drained.[14]
   iii) This catheter is placed prior to surgery and left in for 24-72 hours following the procedure.
   iv) The catheter can be removed after a period of 12-24 hours with stopped drainage, monitoring for any neuro-defecits.[14]

CHAPTER SUMMARY OF THORACIC AORTA SURGERIES

Prior to Induction	Focused anesthetic assessment (if emergent) Two large-bore IV access Lumbar drain (optional depending on procedure) Arterial line
Induction of Anesthesia	Perfusion and surgical teams must be available in the event the patient needs to emergently undergo cardiopulmonary bypass Anesthetic drugs must be titrated slowly to minimize hemodynamic instability Induction dose must prevent the sympathetic response to endotracheal intubation
Lines/Monitors	Central line with pulmonary artery catheter Possible second arterial line (optional) Transesophageal echocardiogram Cerebral oximeter (optional)
Maintenance of Anesthesia	Balanced anesthetic approach making sure to avoid wide swings in hemodynamics Maintenance of a slower heart rate
Circulatory Arrest/ Cerebral Protection	Minimizing total circulatory arrest time Ice on the head may decrease oxygen utilization Selective cerebral perfusion may aid in perfusion of the head while allowing for appropriate surgical conditions
Postprocedure	All patients will require close monitoring in the intensive care unit All patients remain intubated until they are stable enough to be extubated Sedation may be required for clean and stable transition of care from the anesthesiologist to the intensivists

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103 Anesthesia for Minimally Invasive Cardiac Surgery

Jessica Brodt • Lindsay Raleigh

The past 10 years have seen significant changes in transcatheter and endovascular procedural techniques. Evolution in vascular access, valve technology, graft materials, and deployment techniques allow definitive management of specific severe valvular and aortic lesions without a surgical incision in select patients. Understanding of pathophysiology, perioperative steps, and hemodynamic management is essential in order to anticipate problems and safely manage patients undergoing these procedures.

This chapter is presented in 2 sections:

Section 1: Perioperative considerations for transcatheter valve therapies, including aortic valve replacement, mitral valve repair and replacement, and pulmonary valve replacement. Patients presenting for transcatheter valve procedures have severe valvular disease, most commonly aortic stenosis. Non–aortic valve procedures, including pulmonary and mitral valve therapies, are less common and require different considerations and approaches. Patients are often ineligible for traditional surgical procedures due to high surgical risk. Specific planning is required to manage severe valvular lesions along with possible risk of conversion to open surgical repair if complications occur.

Section 2: Perioperative considerations for abdominal and thoracic endovascular aortic replacement. Endovascular aortic repair has emerged as a treatment for pathologies of the aorta, including aneurysm or dissection. Compared to open surgical repair, endovascular therapy has lower perioperative morbidity and mortality and is a less invasive approach, which is especially important for patients at high surgical risk.

Section 1: Transcatheter Valve Therapies

1) **Transcatheter aortic valve replacement (TAVR)**
   a) **Aortic stenosis (AS)**
      i) Pathophysiology
         (1) AS leads to left ventricular (LV) pressure overload, concentric hypertrophy, diastolic dysfunction, and increased myocardial O_2 consumption.
         (2) With end-stage AS, or severe AS with concurrent coronary artery disease (CAD) or other valvular lesions, LV systolic function may decline with dilation of left ventricle.
         (3) For a complete review of AS see (Chapter 102, Aortic Disease and Procedures).
      ii) Hemodynamic goals
         (1) Heart rate and rhythm: Maintain at low normal sinus rhythm.
         (2) Preload: Preserve normal preload.
         (3) Afterload: Preserve afterload and avoid hypotension.
      iii) Indications for TAVR[1] (**Table 103-1**)
         (1) TAVR is indicated for patients with severe symptomatic AS with high or prohibitive surgical risk.
         (2) TAVR is considered reasonable for patients who are intermediate surgical risk.



Hemodynamic goals in AS

Heart rate—low normal
Rhythm—sinus
Preload—normal, preserved
Afterload—preserved to high
Contractility—preserved

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Aortic disease and procedures, Chapter 102

TABLE 103-1 Definition of Surgical Risk for Open Aortic Valve Procedures

Prohibitive surgical risk is defined as:
Predicted risk of death or major morbidity >50% at 30 d
Disease affecting ≥ 3 major organ systems not expected to improve postoperatively
Anatomic factors such as porcelain aorta, prior radiation, or previous arterial bypass graft adherent to chest wall
Society of Thoracic Surgeons Predicted Rate of Mortality (STS-PROM) >8%
Intermediate surgical risk is considered STS-PROM 4%-8%
See www.riskcalc.sts.org for online risk calculator

b) **Preoperative assessment**
   i) Multidisciplinary team evaluation
      (1) Specific recommendations for patient workup and evaluation at TAVR centers include assessment by a multidisciplinary team consisting of interventional cardiologists, cardiac surgeons, anesthesiologists, and imaging physicians.[2]
      (2) Preoperative workup includes imaging of vascular access options (eg, diameter of femoral and iliac arteries, abdominal aorta) and aortic valve annular sizing.
   ii) Detailed anesthetic assessment and risk stratification are essential.
      (1) Cardiac disease
         (a) Identify symptoms related to AS, presence of other valvular lesions, systolic function of the left ventricle, and recent or previous decompensated heart failure.
      (2) Airway assessment
         (a) Necessary equipment must be immediately available for elective or emergent airway management, including advanced airway equipment for any anticipated difficulty with ventilation or intubation.
   iii) Assess suitability for monitored anesthesia care (MAC) vs GA-based anesthetic.[3] (Table 103-2)
      (1) General anesthetic is preferred for:



Orthopnea, musculoskeletal pain, and movement disorders may limit ability of patients to lie supine and still during procedure, necessitating general anesthesia.

TABLE 103-2 Considerations for GA vs MAC-Based Anesthetic

	General Anesthesia	Monitored Anesthesia Care
Philosophy	Balanced anesthetic with secure airway via ETT, maintained at appropriate depth for age, organ function, and level of surgical stimulation	Age- and organ function–appropriate titration of sedative agents, maintaining spontaneous ventilation at all times with minimal respiratory depression
Procedural needs	Surgical or nonfemoral access sites Need for intraoperative TEE guidance	Percutaneous access No procedural need for intraoperative TEE
Patient needs	TEE for management of other valve lesions or heart failure Orthopnea Movement or musculoskeletal disorders limiting ability to lie still Communication barrier Significant anxiety	Well-compensated heart failure, no other significant valve lesions No orthopnea Cooperative, comfortable when supine Appropriate resources to support emergent conversion to GA if perioperative complication occurs (~5%-6% MAC to GA conversion rate)
Anesthesia specific	Anticipated difficult airway—assess on an individual case basis for specific challenges in ventilation and/or intubation	

GA, general anesthesia; MAC, monitored anesthesia care; TEE, transesophageal echocardiography; ETT, endotracheal tube.

(a) Procedural considerations
(i) Need for surgical access including cutdown at femoral access sites
(ii) Nonfemoral access sites including transapical or transaortic approaches
(b) Monitoring considerations
(i) Intraoperative TEE for valve sizing or proceduralist requesting TEE for guidance of deployment
(c) Patient comorbidities
(i) Aspiration risk
(ii) Inability to lie flat and still due to orthopnea, musculoskeletal pain, and symptomatic movement disorders including restless leg syndrome and Parkinson disease

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Anesthesia for the aging patient, Chapter 128

(2) Sedation-based anesthetic is preferred at many high-volume centers.
(a) Associated with faster handoff and turnover times and less vasoactive support required during procedures[4,5]
(b) Specific considerations for MAC include the following:
(i) Planned percutaneous transfemoral access
(ii) Need to secure extremities to avoid risk of life-threatening complications if movement occurs during case
(iii) Selection of sedative agents to minimize risk of hypercarbia, respiratory arrest, and airway obstruction

iv) Geriatric considerations
(1) TAVR patients are frequently in their 9th or 10th decade of life.
(2) Perioperative risk is generally increased in geriatric patients, but distinction between "physiologic age" vs "chronologic age" is important.
(3) Anesthetic requirements, and liver and kidney function are reduced.



Frailty is a reduced physiologic reserve with increased vulnerability to stressors and contributes to physiologic aging. Perioperative management of frail patients is centered on:

- Prehabilitation
- Strict adherence to NPO times and nutritional management
- Early mobilization
- Advanced discharge planning

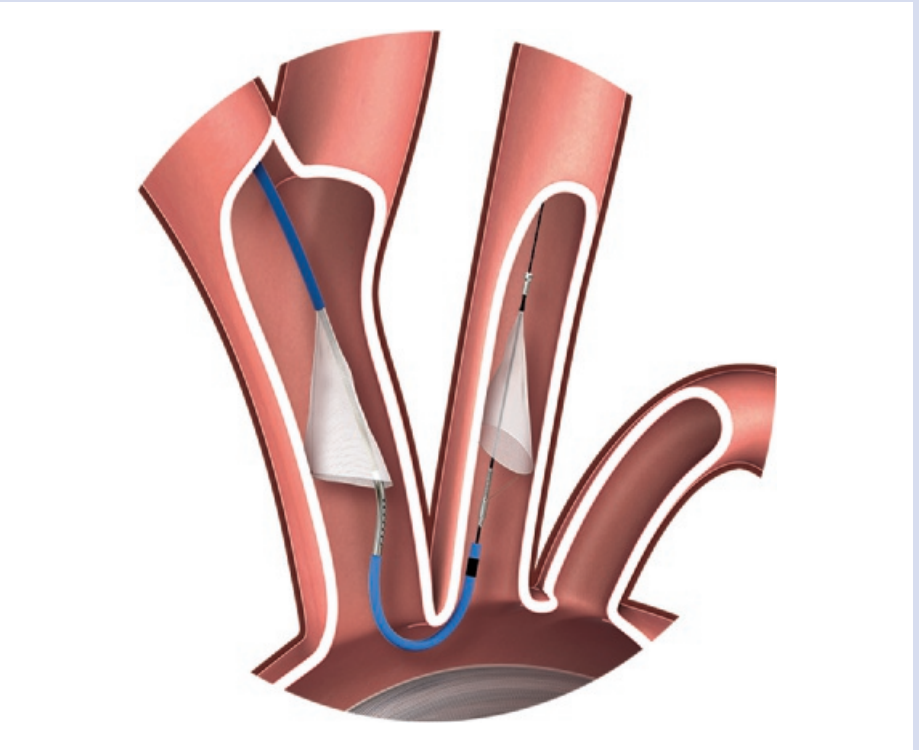
v) Cerebral embolic protection devices may be used in specific patients (eg, Sentinel device, Claret Medical Inc., Santa Rosa, CA).
(1) Temporary filters inserted from right radial artery using an articulating sheath, sitting in left internal carotid artery and brachiocephalic artery, and fully retrieved after valve deployment (Fig. 103-1)
(2) Consider specific risk factors for cerebral emboli including previous history of stroke and heavy atheromatous burden in aortic arch.



Self-expanding valves have significantly higher risk of delayed conduction blockade including complete heart block (10%-30% depending on device). Patients should have temporary transvenous pacemaker (generally via right internal jugular vein) and be monitored continuously for at least 24 hours after valve deployment.

c) **TAVR valves**
i) Two different deployment mechanisms exist in current generation of TAVR valves: balloon expandable and self-expanding (Table 103-3).
ii) Valve size and diameter of delivery systems differ between manufacturers.
(1) Final valve choice depends on native annular sizing, access vessel diameter, and tortuosity of arterial system.
iii) Access site choices
(1) Transfemoral: most common, may be percutaneous or surgical cutdown. Amenable to MAC in appropriate candidates
(2) Transapical: through LV apex via mini left thoracotomy. Consider regional analgesia in addition to GETA

FIGURE 103-1 Sentinel Device With Temporary Filters in Bilateral Internal Carotid Arteries.



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(3) Transaortic: ascending aorta via mini-sternotomy
(4) Transcaval: vascular shunt access between inferior vena cava and descending aorta
(5) Subclavian artery

d) **Intraoperative management**
i) Room preparation
(1) Perfusionist and cardiopulmonary bypass (CPB) machine on standby in room throughout procedure

TABLE 103-3 Balloon Expandable vs Self-Expanding TAVR Valves

Type of Valve	Balloon Expandable Valve	Self-Expanding Valve
Example	Sapien S3 or XT valves (Edwards Lifesciences, Irvine, CA)	CoreValve or Evolut R valves (Medtronic, Minneapolis, MN) Lotus Edge valve (Boston Scientific, Marlborough, MA)
Deployment technique	Rapid ventricular pacing to stop cardiac output during balloon expansion (Fig. 103-2)	Gradual release from delivery system; no rapid ventricular pacing for primary deployment
Risk of heart block	Generally <10%, occurs at time of valve deployment	Between 10% and 30% depending on system. Risk of delayed complete heart block

(2) Blood products: minimal bleeding expected, but risk of sudden catastrophic vascular injury and/or need to institute CPB for procedural complications
  (a) Consider 2 units cross-matched PRBCs in room.
(3) Rapid infusion/fluid warming system
(4) Ultrasound and central line kits in case escalation of support needed
(5) Defibrillation pads

ii) Monitoring
(1) Arterial line for continuous hemodynamic monitoring and intermittent blood gas analysis
(2) Transthoracic echocardiography (TTE)
(3) Consider pulmonary arterial catheterization and/or intraoperative TEE if active decompensated heart failure or cardiogenic shock.
(4) TEE may be required for intraoperative sizing of valve annulus if preoperative size estimates are inadequate (generally by CT or 3D CT), or to guide valve deployment.
(5) Consider cerebral oximetry and awareness monitors.

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Transthoracic echocardiography, Chapter 14

iii) Hemodynamic management
(1) Immediate access to potent vasoconstrictor infusions (eg, phenylephrine, norepinephrine) for all patients, and inotropes (eg, epinephrine) for patients with depressed ventricular function.
(2) Goal perfusion pressure (MAP) >65 mm Hg, higher if needed for cerebrovascular disease or suspected cerebral dysautoregulation

iv) Valve deployment
(1) Balloon expandable valves require rapid ventricular pacing (RVP), usually via temporary femoral venous pacemaker. RVP results in loss of pulsatility and systemic hypotension (**Fig. 103-2**).
(2) In patients with tenuous hemodynamics or depressed ventricular function prior to valve deployment, preemptive vasoactive support prior to RVP is recommended.
(3) Self-expanding valves do not use RVP for deployment but often have a brief (10-15 seconds) period with relative hypotension and decreased pulsatility while valve is partially obstructing LV outflow.

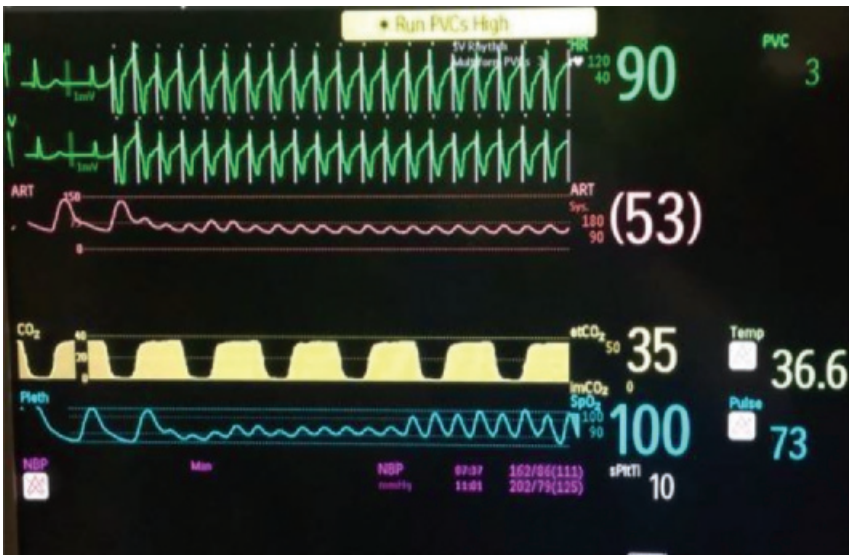
v) Post valve deployment
(1) Immediately assess rhythm and hemodynamic recovery. Be prepared to support rhythm and hemodynamics if there is any delay in recovery.
(2) Assess for paravalvular leak via fluoroscopy and TTE. Valve may require balloon dilation (uses RVP, see **Fig. 103-2**).
(3) Vasodilation is often required after valve deployment; maintain MAP 70-80 mm Hg until surgical hemostasis.
  (a) Avoid vasodilators and antihypertensives with A-V nodal blocking actions (eg, nicardipine, beta-blockade).
  (b) Appropriate vasodilators include nitroglycerin, clevidipine, nitroprusside, nitroglycerin, and hydralazine.
(4) Ensure distal arterial pulses are present after access sheath is removed; monitor post-op until discharge.

vi) Postoperative management
(1) Postanesthesia care unit (PACU) and telemetry floor admission is appropriate in uncomplicated cases.
(2) ICU admission required in certain cases (eg, ongoing mechanical ventilation, inotropic support, hemodynamic instability, transvenous pacing, or after open surgical repair on CPB)
(3) Specialized post-op teams trained in management of TAVR patients
(4) Venous thromboembolism prophylaxis according to established guidelines



Capacity to immediately convert to GA with sternotomy and CPB is necessary for all patients undergoing TAVR. A primed CPB machine and perfusionist should be on standby throughout the procedure.

FIGURE 103-2 Hemodynamics During Rapid Ventricular Pacing (RVP) for Balloon-Expandable Valve Deployment. Note rapid cessation of normal pulsatility in arterial and pulse oximetry waveforms during transvenous pacing (180 bpm).



Courtesy of Dr. Jessica Brodt.

e) **TAVR complications**
   i) Complications of TAVR are shown in Table 103-4.
   ii) The majority of TAVRs completed with experienced teams proceed uneventfully.

2) **Non–aortic transcatheter valve therapies**
   a) Preoperative assessment and perioperative management
      i) Please see above for management of TAVR patients as the same principles apply to procedures on other valves.
      ii) Discussion with proceduralist regarding specific access site requirements
      iii) Planning for cardiopulmonary support depending on valve pathology
      iv) Need for invasive monitors including TEE and transvenous pacing capabilities
      v) Transcatheter tricuspid therapies are still considered experimental.
   b) **Mitral valve therapies**
      i) Transcatheter mitral valve repair (TMVr)
         (1) Current device is the MitraClip (Abbott, Abbott Park, IL)
         (2) Indicated for patients with severe degenerative mitral regurgitation
         (3) Femoral venous access with transseptal puncture into left atrium



Perioperative support may range from light sedation with a single vasoactive infusion to GA with mechanical circulatory support. Consult an experienced colleague for assistance in perioperative planning if you do not have experience managing the procedure.

TABLE 103-4 Periprocedural Complications of TAVR

Etiology	Mild	Severe
Access sites	• Pain • Minor bleeding • Ecchymosis	• Vascular injury • Hemorrhage • Retroperitoneal hematoma • Limb ischemia
Guidewire and/or delivery system	• Arrhythmias • Hypotension or hypertension • Transient MR	• Mitral valve trauma • LV perforation • Pericardial effusion • Pericardial tamponade • Aortic dissection • Aortic rupture
Valve deployment	• Transient conduction block • PVCs, nonsustained VT • Hypotension • Trace to mild paravalvular leak (PVL)	• Complete heart block • Asystole, PEA • Ventricular fibrillation • Coronary obstruction • Annular rupture • Moderate to severe PVL • Cerebral emboli

LV, left ventricle; MR, mitral regurgitation; PEA, pulseless electrical activity; PVCs, premature ventricular contractions; PVL= paravalvular leak; TAVR, transcatheter aortic valve replacement; VT, ventricular tachycardia.

(4) TEE guided placement of clip onto anterior and posterior mitral leaflets. More than one clip may be used during procedure.
(5) Evaluate for mitral stenosis and residual MR after deployment.
(6) GA with hemodynamic support for severe mitral regurgitation is often required.
(7) Frequently patients have comorbid pulmonary venous hypertension, and sequelae of aging and heart disease.

ii) Transcatheter mitral valve replacement (TMVR)
(1) Currently reserved for patients with existing bioprosthetic mitral valve
(2) Transfemoral or transapical access
(3) General anesthesia with TEE guidance and appropriate hemodynamic support

iii) Complications of TMV therapies include residual mitral regurgitation, mitral valve perforation or injury, device embolization, partial clip detachment (TMVr), vascular injury and hemorrhage, LVOT obstruction, pericardial effusion and tamponade, intracardiac shunt (atrial septal access).

c) **Pulmonary valve replacement**

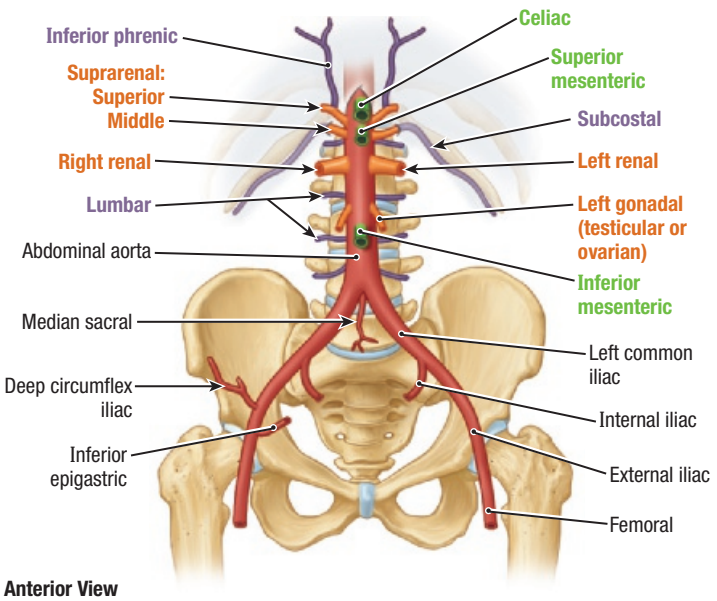
i) Typically patients have right ventricular outflow tract (RVOT) conduit stenosis or degeneration of bioprosthetic valve.
ii) May have complex congenital heart disease
iii) Currently available valves are Melody (Medtronic, Minneapolis, MN) and Sapien (Edwards Lifesciences, Irvine, CA)
iv) Vascular access via venous system (femoral or internal jugular)
v) GA is typically used with standard monitors, arterial pressure monitoring, and large bore IV access.
vi) TEE or intracardiac echocardiography at team's discretion
vii) RVP may be used especially in cases with severe pulmonary regurgitation to reduce risk of valve migration during deployment.
viii) Complications include coronary artery compression (especially if aberrant coronary anatomy), valve embolization, rupture of RVOT, conduit or PA, tricuspid valve injury, ventricular perforation, pulmonary artery branch occlusion, hemoptysis, pulmonary edema, stent fracture, and aortopulmonary fistula (presents late).

Section 2: Endovascular Procedures of the Aorta

Endovascular aortic repair has emerged as a treatment modality for pathologies of the aorta, including aneurysm or dissection. Compared to open surgical repair, endovascular therapy is associated with lower perioperative morbidity and mortality and offers a less invasive approach, especially important for patients at high surgical risk.

1) **Endovascular abdominal aortic repair (EVAR)**
   a) **Abdominal aorta physiology**
      i) A retroperitoneal structure beginning at the hiatus of the diaphragm and ending at its bifurcation into the common iliac arteries at L4)
      ii) Major branches from superior to inferior (Fig. 103-3)
         (1) Celiac artery
         (2) Superior mesenteric artery (SMA)
         (3) Bilateral renal arteries
         (4) Inferior mesenteric artery (IMA)
         (5) Bilateral iliac arteries
   b) **Abdominal aortic aneurysm (AAA) etiology and pathophysiology**[6]
      i) Weakening of the aortic walls leads to enlarging of the abdominal aorta and aneurysm formation.
         (1) Infrarenal—aneurysm originates below the renal arteries (85% of cases)
         (2) Juxtarenal—aneurysm originates at the level of the renal arteries

FIGURE 103-3 Abdominal Aortic Anatomy



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(3) Suprarenal—aneurysm originates above the renal arteries

ii) Risk factors include tobacco usage, male sex, age >65, family history, hyperlipidemia, peripheral vascular disease, or connective tissue disorders. Rare causes include infection, trauma, and arteritis.

iii) Complications include rupture, acute aortic occlusion, or peripheral embolism.

(1) Risk factors for rupture: aneurysm diameter, presence of symptoms, positive family history, and rate of expansion

(2) Grey Turner sign: flank ecchymosis due to retroperitoneal bleeding



AAA diameter is the best predictor of rupture[6] **(Table 103-5)**.

c) **AAA signs and symptoms**

i) Largely asymptomatic, increasingly painful as aneurysm enlarges

ii) Pain in the chest, abdomen, or lower back; pulsing sensation in the abdomen

iii) A pulsatile abdominal mass may be noted on physical examination.

iv) Screening and diagnosis

v) Abdominal ultrasound is the screening test of choice.

(1) Presence of bowel gas or obesity may limit its effectiveness.

(2) CT scan is another imaging modality option, with ~100% sensitivity.



The US Preventative Screening Task Force (USPSTF) recommends screening for AAA in males ages 65-75 with a history of smoking.[7]

d) **AAA management**[8]

i) Elective AAA repair is not recommended in asymptomatic patients until the risk of rupture exceeds the risks associated with repair (Table 103-5).

(1) In the majority of patients, this occurs with aneurysm diameter >5.5 cm.

(2) Risk of rupture increases in aneurysms with rapid expansion.

(a) Defined as an increase in maximal aortic diameter ≥5 mm over a 6-month period of time or >10 mm over 1 year

ii) Medical management is recommended and includes control of blood pressure and lipid levels.

(1) Smoking cessation is recommended to reduce the risk of aneurysm growth and rupture.

(2) AAA is regarded as a coronary risk equivalent; therefore, aspirin and statin therapy are recommended to reduce the risk of a future cardiovascular event.[9]

(3) No pharmacologic therapy has been proven to prevent AAA expansion or prevent rupture.

iii) Surgical repair (open or endovascular) should be considered for symptomatic aneurysms, for ruptured aneurysms or for aneurysms at risk of rupture.

TABLE 103-5 AAA Diameter and Risk of Rupture

AAA Diameter (cm)	Risk of Rupture (%/y)
4-5	1-5
5-6	3-15
6-7	10-20
7-8	20-40

AAA, abdominal aortic aneurysm.
Modified from Brewster DC, Cronenwett JL, Hallett JW, et al. Guidelines for the treatment of abdominal aortic aneurysms. Report of a subcommittee of the Joint Council of the American Association for Vascular Surgery and Society for Vascular Surgery. *J Vasc Surg*. 2003;37(5):1106-1117, with permission from Elsevier.

(1) A door-to-intervention time of 90 minutes is recommended in patients with a ruptured aneurysm.
(2) Considerations for elective surgical repair include the size and location of the aneurysm, the rate of growth, aneurysm morphology, involvement of other vasculature, ongoing symptoms (ie, pain, visceral ischemia, impending rupture), and the patient's overall health.
(3) Open repair involves replacement of the diseased aortic segment with a prosthetic tube graft through a midline abdominal or retroperitoneal incision.
(4) Endovascular repair involves the placement of graft material delivered via the iliac or femoral arteries to line the intimal aortic wall and exclude the aneurysm sac from the circulation.
(a) EVAR is a feasible option for older patients, or those at high or prohibitive risk for open repair.
(b) Requires adequate distance (1.5-2 cm) for graft deployment (known as the "landing zone") in addition to adequate vascular diameter for access.
(c) EVAR has lower rates of morbidity and mortality when compared with open surgical AAA repair in the early perioperative period, but is associated with a higher reintervention rate. [10,11]
(d) Long-term aneurysm sac enlargement remains a problem and highlights the importance of long-term imaging surveillance with reintervention, if necessary.

e) **EVAR procedural steps**
i) Vascular access is obtained via percutaneous approach or via surgical cutdown.
(1) Unilateral or bilateral arterial access needs depend on the number and location of grafts to be deployed
(2) Occasionally, arterial access via the upper extremity (typically left axillary) may be required.
ii) Arterial sheath is placed and the guidewire is advanced into the aorta to position the stent.
iii) Stent is advanced under fluoroscopic imaging and deployed in the aorta, contacting the aortic wall and excluding the aneurysm sac from aortic blood flow and pressure.
(1) To ensure the aneurysm sac is excluded, the endograft must contact the arterial wall 1.5-2 cm proximally and distally to the aneurysm (known as "landing zones").
(2) The graft generates a radial force of the aortic wall.
iv) With involvement of visceral vessels, special grafts may be necessary, which allow grafts to align with branching arteries or allow for placement of separate grafts into the visceral vessels.
(1) Fenestrated grafts—have openings in the endograft to allow for flow into the visceral arteries
(2) Branched grafts—have small side-arm grafts sutured to the main endograft body for deployment into a visceral vessel to preserve its flow (ie, hypogastric or renal arteries)
(3) Chimney grafts—a graft that is deployed alongside the main endograft to allow for flow into a branch vessel (typically suprarenal vessels), usually used cases of more extensive or complex aneurysms
(a) Associated with increased risk of Type 1 endoleaks from leakage at either end of the graft (see **Table 103-7**)
v) With successful graft placement, the aneurysm sac will eventually thrombose and may progressively shrink.

f) **Preoperative considerations**[8,9]
   i) Comorbidities include hypertension, hyperlipidemia, tobacco usage/chronic obstructive pulmonary disease (COPD), peripheral vascular disease, CAD, and advanced age.
      (1) Continue beta-blockers in the perioperative period.
      (2) Smoking cessation 2 weeks before surgery is recommended.
      (3) Hold ACE-inhibitor or angiotensin receptor antagonist therapy the morning of the procedure.
      (4) Cardiac evaluation is guided by the American College of Cardiology/American Heart Association guidelines regarding evaluation and management of cardiac risk in patients undergoing intermediate-risk noncardiac surgery
      (5) A 12-lead EKG should be obtained in the 30 days prior to elective procedure.
   ii) Risk of renal injury due to contrast-load, renal artery occlusion by graft, emboli to the renal arteries, or preexisting renal dysfunction
      (1) Baseline creatinine should be obtained prior to the procedure.
      (2) Preprocedural hydration should be considered in patients with renal insufficiency or those at risk of contrast induced nephropathy.
   iii) Pre-op imaging includes CT angiography of the aorta and iliac arteries to define vascular anatomy and choose the size and configuration of the endograft components.

g) **Intraoperative management**
   i) Anesthetic choice
      (1) EVAR can be performed under local anesthesia (LA), MAC, neuraxial anesthesia, or general anesthesia (GA).
      (2) Choice of anesthetic approach depends on patient comorbidities (including the ability to lie flat or follow commands), method of surgical access (cutdown vs percutaneous), patient's preference (ie, anxiety), and the expected degree of difficulty with graft positioning (which may include the need for cessation of respiration or "breath holding").
      (3) No randomized control trials have compared anesthetic techniques for EVAR.
         (a) A meta-analysis showed no mortality benefit between local anesthesia and GA.[12]
   ii) Monitoring
      (1) Standard ASA monitors should be utilized.
      (2) Arterial pressure monitoring via an arterial catheter will allow for close hemodynamic monitoring and quick management of hemodynamic fluctuations.
   iii) Considerations by organ system **(Table 103-6)**

h) **Postprocedure management**
   i) Monitoring can occur in the PACU unless comorbidities or procedural complications necessitate a higher level of care (ie, intensive care unit).
   ii) Neurovascular monitoring, including monitoring of peripheral pulses, is important in the postprocedure period.
      (1) Risks of EVAR include access site complications and acute arterial thrombosis.
   iii) Home medications should be resumed as appropriate, especially cardiovascular medications.
   iv) Prophylaxis for venous thromboembolism should be considered according to guidelines.[11]
      (1) Early ambulation and intermittent pneumatic compression are recommended in all patients.

TABLE 103-6 Intraoperative Considerations by Organ System

Neurologic	• Immobility required for graft positioning; ensure patient comfort in supine position for several hours • Local anesthetic administered at the sites of vascular access can decrease periprocedural pain
Cardiovascular	• Blood pressure control is of the upmost importance. ○ Avoid hypertension prior to stent placement, especially at time of induction, due to risk of aneurysm rupture. • Short-acting vasodilators and vasoconstrictors should be available to maintain hemodynamic goals. • Arterial line placement is recommended (right radial or brachial given possible need for vascular access via left upper extremity).
Respiratory	• MAC may be preferred due to avoidance of intubation and mechanical ventilation; use end-tidal CO_2 monitoring. • Suspension of respiration (or "breath holding") may be necessary during stent deployment.
Renal	• Risk of renal injury, especially contrast-induced nephropathy. • Avoid hypovolemia; fluid administration must balance renal perfusion with other comorbidities (ie, heart failure with reduced ejection fraction). • Administration of perioperative fluids (balanced salt solutions of isotonic sodium bicarbonate fluids) in patients with renal insufficiency • Close monitoring of urine output • Stop metformin 48 h before contrast administration; avoid NSAIDs in the postoperative period.
Hematologic	• Systemic anticoagulation is necessary during procedure (heparin 80-100 units/kg, goal ACT >200 s) • Anticoagulation reversal at end of procedure with protamine • Risk of large-volume blood loss due to vascular injury; large-bore IV access prudent • Ensure availability of blood products.
Other	• Antibiotic prophylaxis, per standard protocols • Avoidance of hypothermia (goal temperature >36°C) • Ensure full reversal of muscle paralysis, if applicable.

ACT, activated clotting time; COPD, chronic obstructive pulmonary disease; GA, general anesthesia; IV, intravenous; LA, local anesthesia; MAC, monitored anesthesia care; NSAIDs, nonsteroidal anti-inflammatory drugs.

v) Acute ischemic colitis is a rare complication after EVAR, due to iatrogenic ischemia or emboli to visceral arteries; the presence of new abdominal pain should be evaluated urgently.

vi) Postimplantation syndrome—a self-limited inflammatory state related to new thrombus formation in the aneurysm sac; may present with fever and elevated inflammatory markers

i) EVAR complications

i) Procedural complications include

(1) Vascular injury due to obtaining vascular access or arterial thrombosis

(2) Renal injury due to contrast-induced nephropathy

ii) Stent-related complications include endoleak (persistent flow into the aneurysm sac), aneurysm sac enlargement, stent migration, and separation of the device components.

(1) Failure of the endografts can lead to continued expansion of the aneurysm sac and continued risk of rupture (Table 103-7).

(2) Changes in the aneurysm sac may result in graft kinking, migration, or endoleak; long-term surveillance is also recommended.

iii) The risk of endoleaks is a long-term complication (>5 years) following EVAR; post-op surveillance is recommended[8]

TABLE 103-7 Endoleak Classification

Type of Endoleak	Location of Leak
Type 1 (A and B)	Leak at graft ends A—proximal B—distal
Type 2	Branch vessel filling aneurysm sac
Type 3	Leak through defect in graft fabric
Type 4	Leak via porous nature of graft (not a defect)
Type 5	Continued expansion of the aneurysm sac, without clear leak ("endotension")

2) **Thoracic endovascular aortic repair (TEVAR)**

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Aortic valvular disease, Chapter 100

a) **Descending thoracic aorta physiology**

i) Located in the posterior mediastinal cavity, extending from the left subclavian artery to the diaphragm

ii) Thoracic aortic pathologies that may be indications for endovascular repair include

(1) Thoracoabdominal aortic aneurysm

(2) Thoracoabdominal aortic dissection

(a) Also known as a "Stanford type B" dissection; can be acute or chronic

(3) Thoracoabdominal aortic intramural hematoma or penetrating ulcer

(4) Blunt thoracic aorta trauma (especially acceleration-deceleration injuries, usually located at the aortic isthmus distal to the left subclavian artery)

(5) Aortoesophageal fistula



Branches of the thoracic aorta include the segmental (or medullary) arteries, which branch off the aorta from T9 to L1 and perfuse the spinal cord (via the anterior spinal artery).

b) **Thoracoabdominal aortic disease etiology and pathophysiology**

i) Thoracoabdominal aortic aneurysm (TAAA)

(1) Weakening of the aortic walls leads to enlarging of the thoracic aorta and aneurysm formation

(2) TAAAs are classified according to location of aneurysm under the Crawford Extent Classification[13] (**Table 103-8**)

ii) Thoracoabdominal aortic dissection ("Type B")

(1) Intimal tear resulting in separation of layers of the aorta, with blood flow between the intimal and medial walls creating a "false lumen"

(2) Dissections are often described by location, timing, and presence of complications:

(a) Stanford classification by dissection location:

(i) Type B dissection is confined to the descending thoracoabdominal aorta, distal to the left subclavian artery.

TABLE 103-8 Crawford Classification of TAAA

Extent I	From the origin of the left subclavian artery to the suprarenal abdominal aorta
Extent II	From the origin of the left subclavian artery to the aortoiliac bifurcation
Extent III	From the distal thoracic aorta to the aortoiliac bifurcation
Extent IV	Limited to the abdominal aorta (below the diaphragm)
Extent V	From the distal thoracic aorta to the suprarenal abdominal aorta

(ii) Type A dissection is any dissection involving the ascending aorta; can include dissection of the thoracoabdominal aorta

(b) Timing: hyperacute (<24 hours), acute (2-7 days), subacute (8-30 days), and chronic (>30 days)[14]

(c) Dissections are designated as "complicated" if there is rupture, impending rupture, evidence of end-organ malperfusion, limb ischemia, or unrelenting pain.

c) **Thoracoabdominal aortic disease signs and symptoms**

i) TAAA

(1) Largely asymptomatic, but become increasingly painful as aneurysm enlarges

(2) Symptoms include pain or pressure in the chest or back.

(3) Aneurysm can cause compressive symptoms on adjacent structures.

(a) Hoarseness due to recurrent laryngeal nerve stretching

(b) Stridor due to tracheal or bronchial compression

(c) Dyspnea due to lung compression

(d) Dysphagia due to esophageal compression

ii) Thoracoabdominal aortic dissection

(1) Acute dissection presents most commonly with chest or back pain, radiating; tearing sensation.

(a) May also present with end-organ or peripheral ischemia due to disruption of blood flow by the dissection flap

(2) Chronic dissections are often asymptomatic, with aneurysmal dilation.

iii) Diagnosis of thoracoabdominal aortic disease

(1) CT angiography (chest and abdomen) is the most commonly used imaging modality.

(2) Cardiac ultrasound—transthoracic or transesophageal echocardiography (TTE or TEE)

(a) TEE has sensitivity of 98% and specificity of 95% in diagnosing aortic dissection.

(b) Visualization of aorta by TTE is usually limited due to inadequate acoustic windows

(3) Magnetic resonance imaging (MRI)

(a) Least frequently used

(b) Sensitivities and specificities are similar to CT.

(c) Length of study limits application in emergent situations.

(4) Chest x-ray

(a) May identify abnormalities in aortic size or contour (widened mediastinum)

(b) Limited ability to further characterize aortic pathology (dissection vs aneurysm) size, location/extent

(c) Cannot rule out aortic dissection

d) **Thoracoabdominal aortic disease (TAAA) management**

i) Medical management[15]

(1) Elective repair is not recommended in asymptomatic patients until the risk of rupture exceeds the risks associated with repair.

(a) In the majority of patients, this occurs with aneurysm diameters >5.5-6.0 cm.

(i) Repair is indicated at smaller aortic diameters in patients with connective tissue disorders (Marfan syndrome, Loeys-Dietz, Ehlers-Danlos), Turner syndrome, or bicuspid aortic valves.

(b) Risk of rupture also increases in aneurysms with rapid expansion.

(2) Medical management includes

(a) Control of blood pressure (antiimpulse control)

(b) Smoking cessation

(c) Treatment of dyslipidemia

(d) Screening for associated genetic conditions
(e) Surveillance imaging (timing depending on underlying comorbidities and genetic predispositions)

ii) Surgical repair
(1) Considered in the following situations:
(a) Symptomatic patients
(b) Aneurysms or dissection at risk of rupture
(c) Aneurysms or dissections with complications (rupture, pending rupture, or evidence of malperfusion with organ or limb ischemia)
(2) Choice between open vs endovascular repair: considerations include aneurysm size, location, morphology, and the involvement of other vasculature, and the patient's comorbidities and overall health.
(a) Open repair involves replacement of the diseased aortic segment with a prosthetic tube graft through a thoracotomy approach, often with CPB support, in addition to a retroperitoneal incision.
(b) Endovascular repair involves the placement of graft material delivered via the iliac or femoral arteries to line the intimal aortic wall and exclude the aneurysm sac from the circulation.
(i) TEVAR is a feasible option for older patients, or those at high or prohibitive risk for open repair.
(ii) TEVAR is not recommended in patients with connective tissue disorders, except in emergencies, as a bridge to definitive open surgical repair, or as a reintervention repair of a prior graft (when stent graft would land within prior prosthetic graft).
(iii) Requires adequate distance for the graft to deploy ("landing zone") in addition to adequate vascular diameter for access
(iv) Guidelines support endovascular stenting for a variety of aortic pathologies (Table 103-9).[16]



No randomized control trials have compared elective open surgical repair with endovascular repair for TAAA.

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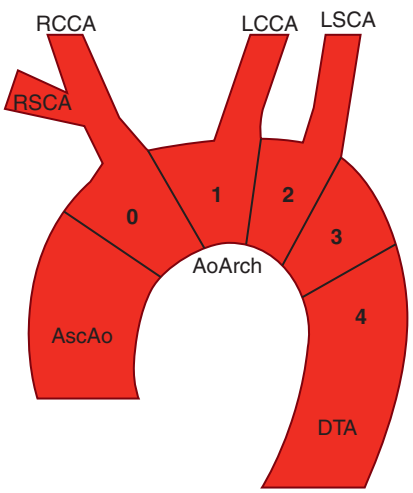
TABLE 103-9 Guidelines on the Treatment of Descending Thoracic Aortic Disease Using Endovascular Stent-Grafts[16]

Thoracoabdominal Aortic Pathology	Recommendation for TEVAR
Penetrating ulcer/intramural hematoma	
Asymptomatic	SHOULD NOT be performed
Symptomatic	REASONABLE to consider
Traumatic injury	
Acute	SHOULD be considered
Chronic	REASONABLE to consider
Acute Type B dissection	
Uncomplicated	MAY BE considered
Complicated	SHOULD be considered
Subacute of chronic Type B dissection	MAY BE considered
Thoracoabdominal aortic aneurysm	
>5.5 cm, comorbidity	REASONABLE to consider
>5.5 cm, no comorbidity	MAY BE considered
<5.5 cm	SHOULD NOT be performed

Reprinted from Rizvi AZ, Sullivan TM. Incidence, prevention, and management in spinal cord protection during TEVAR. *J Vasc Surg*. 2010;52(4 Suppl):86S-90S, with permission from Elsevier.

e) **TEVAR procedural steps**
   i) Vascular access is obtained via percutaneous approach or via surgical cutdown.
      (1) Unilateral or bilateral arterial access needs depend on the number and location of grafts to be deployed.
      (2) Occasionally, arterial access via the upper extremity (typically left axillary) may be required.
   ii) Arterial sheath is placed and the guidewire is advanced into the aorta to position the stent.
   iii) Stent is advanced under fluoroscopic imaging and deployed in the aorta, contacting the aortic wall and excluding the aneurysm sac from aortic blood flow and pressure.
      (1) To ensure the aneurysm sac is excluded, the endograft must contact the arterial wall 1.5-2 cm proximally and distally to the aneurysm (known as "landing zones", Fig. 103-4).
      (2) The graft generates a radial force of the aortic wall.
   iv) Development of fenestrated and branched endografts has allowed for the complete endovascular repair of complex thoracoabdominal aortic aneurysms. These graft techniques are customized for the patient's unique anatomy, allowing for perfusion of branch vessels.
      (1) Fenestrated grafts—have openings in the endograft to allow for flow into the visceral arteries
      (2) Branched grafts—have small side-arm grafts sutured to the main endograft body for deployment into a visceral vessel to preserve its flow (ie, hypogastric or renal arteries)

FIGURE 103-4 Landing Zones of the Thoracic Aorta. AoArch, aortic arch; AscAo, ascending aorta; DTA, descending thoracic aorta; LCCA, left common carotid artery; LSCA, left subclavian artery; RCCA, right common carotid artery; RSCA, right subclavian artery.



Courtesy of Dr. Jessica Brodt.

(3) Chimney grafts—a graft that is deployed alongside the main endograft to allow for flow into a branch vessel (typically suprarenal vessels), usually used in cases of more extensive aneurysms
(a) Associated with an increased risk of type 1 endoleaks (Table 103-7)

v) With successful graft placement, the aneurysm sac will eventually thrombose and may progressively shrink.

vi) Other special circumstances: occasionally, a fenestration is created between the true and false lumen of an aortic dissection to equalize pressures, relieve occlusion of branch vessels by the dissection flap, and restore perfusion.

f) **Preoperative considerations**

i) Comorbidities include hypertension, hyperlipidemia, tobacco usage/COPD, peripheral vascular disease, CAD, advanced age.
(1) Continue beta-blockers in the perioperative period.
(2) Smoking cessation is recommended.
(3) Cardiac evaluation guided by the 2014 American College of Cardiology/American Heart Association guidelines regarding evaluation and management of cardiac risk in patients undergoing intermediate-risk noncardiac surgery[9]

ii) Risk of renal injury due to contrast-load, renal artery occlusion by graft, emboli to the renal arteries, or preexisting renal dysfunction
(1) Baseline creatinine should be obtained prior to the procedure.
(2) Consider preprocedural hydration in patients with renal insufficiency or those at risk of contrast-induced nephropathy.[15]

iii) Risk of stroke and spinal cord ischemia (due to graft occlusion of spinal cord arterial blood supply)
(1) Obtain baseline neuro examination.
(2) Hold anticoagulation and antiplatelet agents per American Society of Regional Anesthesia guidelines for anticoagulation an interventional spine and pain procedures.[17]
(3) Assess baseline platelets and coagulations parameters (PT, PTT, INR) prior to placement.


Spinal cord ischemia and paralysis is a risk of TEVAR procedures.

iv) Preoperative imaging includes CT angiography of the aorta and iliac arteries to define vascular anatomy and choose the size and configuration of the endograft components, as well as the vascular access sites.

g) **Intraoperative management**

i) Anesthetic type
(1) TEVAR is generally performed under general anesthesia (GA) due to the complexity of stent placement during TAAA repair and the need for cessation of respiration.

ii) Monitoring
(1) Standard ASA monitors should be applied.
(2) Arterial pressure monitoring allows close hemodynamic monitoring and quick management of hemodynamic fluctuations.
(3) Placement of a central venous catheter should be considered because intraoperative or postoperative vasoactive infusions are often required to augment mean arterial pressure (MAP) to support spinal cord perfusion.
(a) Monitoring of central venous pressure (CVP) is recommended when central line is placed.

(4) TEE can be used as an intraoperative monitor of cardiac function or volume status, to guide and evaluate surgical repair, and to evaluate for iatrogenic aortic injury (ie, retrograde type A dissection) due to wires placed proximally in the DTA.
(5) Cerebral spinal fluid (CSF) pressure (via a lumbar drain catheter) is recommended for patients at risk of spinal cord ischemia.[15]

iii) Considerations by organ system (Table 103-10)

h) **Postprocedure management**

i) Monitoring in the ICU is recommended for the following reasons:
(1) Management of lumbar drain
(2) Hemodynamic monitoring and management such as
(a) Augmentation of MAP with vasopressors
(b) Support of cardiac output with inotropic agents
(3) Frequent postprocedure neuro checks

TABLE 103-10 TEVAR Intraoperative Considerations by Organ System

Neurologic	• Risk of spinal cord ischemia; consider cerebral spinal fluid (CSF) drainage for spinal cord protection; management of lumbar drain (see below) • Consider neuromonitoring with evoked potentials in high-risk patients; tailor anesthetic choice to avoid effects on neuromonitoring signals. • Stroke risk associated with atheromatous disease in the arch or proximal DTA and instrumentation of proximal DTA or arch
Cardiovascular	• Blood pressure control is of the upmost importance. ○ Avoid hypertension prior to stent placement, especially at time of induction, due to risk of aneurysm rupture. • Short-acting vasodilators and vasoconstrictors should be available to maintain hemodynamic goals. • Arterial line placement is recommended (right radial or brachial given possible need for vascular access via left upper extremity, and the possibility of the stent covering the left subclavian artery). • Consider TEE when indicated by patient comorbidities or procedural need (confirmation of guidewire location with complex aortic anatomy, assess repair, evaluate for iatrogenic aortic injury).
Respiratory	• Most cases performed under GA; management of airway and mechanical ventilation per usual • Suspension of respiration (or "breath holding") may be necessary during stent deployment.
Renal	• Risk of renal injury, especially contrast-induced nephropathy • Avoid hypovolemia; fluid administration must balance renal perfusion with other comorbidities (ie, heart failure with reduced ejection fraction). • Administration of perioperative fluids (balanced salt solutions of isotonic sodium bicarbonate fluids) in patients with renal insufficiency • Close monitoring of urine output • Stop metformin 48 h before contrast administration. • Avoid NSAIDs in the postoperative period.
Hematologic	• Systemic anticoagulation is necessary during procedure (heparin 80-100 units/kg, goal ACT >200 s). • Anticoagulation reversal at end of procedure with protamine • Risk of large-volume blood loss due to vascular injury; large-bore IV access prudent • Ensure availability of blood products.
Other	• Antibiotic prophylaxis, per standard protocols • Avoidance of hypothermia (goal temperature >36°C) • Ensure full reversal of muscle paralysis prior to extubation.

ACT, activated clotting time; DTA, descending thoracic aorta; GA, general anesthesia; IV, intravenous; MAC, monitored anesthesia care; NSAIDs, nonsteroidal anti-inflammatory drugs; TEE, transesophageal echocardiography.

ii) Spinal cord ischemia remains a devastating risk of TEVAR.
iii) Neurovascular monitoring, including monitoring of peripheral pulses, is important in the postprocedure period.
  (1) Risks of EVAR include access site complications and acute arterial thrombosis
iv) Continue blood pressure augmentation in the immediate postprocedural period (usually for 24 hours)
  (1) Delay restarting home antihypertensives until risk of spinal cord ischemia is low.
v) Prophylaxis for venous thromboembolism should be considered according to guidelines.
  (1) Early ambulation and intermittent pneumatic compression recommended in all patients
vi) Thromboembolism to the visceral vessels or stent migration may present as new abdominal pain or distention and should be evaluated urgently.
vii) New chest pain should be evaluated to assess for an acute cardiac etiology or endoleak.
viii) Postimplantation syndrome
  (1) A self-limited inflammatory state related to new thrombus formation in the aneurysm sac
  (2) May present in the first 24 hours with fever and elevated inflammatory markers
  (3) Managed with analgesics and anti-inflammatory agents

i) **Spinal cord ischemia (SCI)**
  i) Arterial supply to the spinal cord is complex. Major branches include
    (1) Vertebral arteries (arising from the subclavian arteries) → anterior spinal artery
    (2) Thoracic segmental (or meduallary) arteries → join the anterior spinal artery
      (a) Artery of Adamkiewicz—arteria magna radicularis
    (3) Hypogastric arterial network: composed of the lumbar segmental arteries, middle sacral artery, lateral sacral arteries, iliolumbar arteries
  ii) Risk of SCI after TEVAR is between 3% and 10%.[18,19]
  iii) Risk of SCI related to sacrifice of arterial vascular supply to the spinal cord by the stent graft with the following associated with increased risk:
    (1) Extent of repair: stents >20 cm in length
    (2) Stents covering T8-L2
    (3) Compromised collateral blood flow:
      (a) Hypogastric iliolumbar—that is, prior AAA repair, arterial occlusions, or injury
      (b) Segmental arteries—stent placement covering T8-L2
      (c) Vertebral arteries—Left subclavian arterial occlusion by stent
  iv) Other risk factors for SCI include perioperative hypotension, renal failure, and aortic atheromatous disease.
  v) SCI may present as paraparesis or paraplegia; can present immediately or delayed (hours or days after surgery)
    (1) Can be associated with hypotension, autonomic dysfunction, or neurogenic shock
    (2) Can be temporary or result in permanent impairment
  vi) Associated with increased morbidity and mortality[20]
  vii) Risk is mitigated by the placement of a CSF catheter (or "lumbar drain") to drain CSF and decrease the CSF pressure.[21]

j) **CSF drain**
  i) Catheter and drainage system
    (1) A multiorificed catheter is placed 5-10 cm into the subarachnoid space at the L3-L4 or L4-L5 intervertebral interspace under sterile conditions.

(2) Catheter attached to a sterile, closed-circuit reservoir to collect CSF
(3) CSF pressure measured via a transducer zero-referenced at the level of the right atrium

ii) Contraindications to placement include coagulopathy and patient refusal.
iii) Complications of drain placement include post–dural puncture headache, infection/meningitis, epidural hematoma, nerve root injuries, catheter fracture, subdural hematoma, or intracranial hemorrhage (related to excessive CSF drainage).
iv) Drain management
(1) Monitor CSF pressure continuously throughout the procedure (zero-reference at the level of the right atrium).
(2) Drain CSF for goal ICP <10 mm Hg.
(a) Limit CSF drainage to <10 mL/h.
(b) Monitor CSF color (blood may signify subdural hematoma or intracranial hemorrhage).
v) Postoperative hemodynamic goals and neurologic monitoring
(1) Obtain neurological examination as soon as possible after examination, preferably at end of procedure prior to leaving cath lab.
(a) Continue q1h neurologic checks while drain in place, particularly hip flexion.
(2) Hemodynamic goals
(a) MAP 80-100 mm Hg
(b) CSF pressure <10 mm Hg
(c) Spinal cord perfusion pressure (SCPP) 60-80 mm Hg
(i) SCPP = MAP – CSF pressure (or CVP, whichever is higher)



Spinal Cord Perfusion Pressure (SCPP) = MAP — CSF pressure (or CVP, if higher).

(3) Standard procedure at many institutions is to drain CSF for 24 hours, clamp the drain at 48 hours, and remove drain at 72 hours unless signs of SCI develop.
(a) If postoperative weakness or paralysis is present:
(i) Continue to drain CSF.
(ii) Augment MAP to >100 mm Hg.
(iii) Optimize oxygen delivery to spinal cord.
1. Maintain cardiac output.
2. Optimize O_2 content in the blood.
3. Transfuse PRBC for goal Hgb > 10.
4. Increase arterial partial pressure of oxygen (PaO_2).

CHAPTER SUMMARY

Indications	Patients with severe symptomatic valvular lesions with high or prohibitive surgical risk
Preoperative Assessment	Multidisciplinary team evaluation assesses vascular access options and valvular anatomy using a variety of imaging modalities, most commonly CT angiography and echocardiography. Detailed anesthetic assessment and risk stratification are essential
Intraoperative Management	Depending on the type of procedure, perfusionist and CPB machine should be on standby. Ensure appropriate equipment, blood products, and monitoring devices (lines and TEE) are available
Postoperative Considerations	Monitor for postoperative complications, which can vary depending upon procedure. They can include spinal cord injury, arrhythmias and bleeding (TAVR), vascular and renal injury (EVAR), spinal cord injury (TEVAR)

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104 Heart, Lung, and Heart-Lung Transplantation

Sameer Khan • Charles C. Hill

Patients with end-stage heart disease (ESHD) and end-stage lung disease (ESLD) requiring transplantations offer unique challenges in anesthetic selection and management in the perioperative period. Careful perioperative planning with an in-depth understanding of the patient's pathophysiology can help prevent many complications. Heart transplantation has advanced considerably since the first heart transplant in 1971 by Dr. Shumway. With the advancement of antirejection drugs, heart failure management, and extracorporeal devices, as well as ventricular assist devices, considerations for both the anesthetic and the patient population have drastically changed. The first successful lung transplantation occurred in 1963. Bronchial dehiscence limited the success of lung transplantation, until the availability of cyclosporine, the use of heart-lung machines and antibiotic therapies. Over the years, outcomes have improved as donor and recipient selection, medical therapy, and surgical techniques have been refined. Nevertheless, chronic rejection remains the leading obstacle to better long-term survival.

1) **Heart transplantation**
   a) **General considerations** (Table 104-1)
      i) Donor criteria for heart transplantation include the following:
         (1) Age <55
         (2) No history of chest trauma or cardiac disease
         (3) No prolonged hypotension or hypoxemia
         (4) Appropriate hemodynamics (MAP > 60, CVP 8-12)
         (5) Inotropic support <10 mg/kg/min of dopamine or dobutamine
         (6) Normal studies including ECG, echocardiogram, cardiac angiography
         (7) Negative serology (hepatitis B, hepatitis C, HIV)[2]
      ii) Over the years, the time from transplant listing to organ transplantation has been reduced.
      iii) Organ ischemia time should be less than 6 hours to prevent organ damage.
      iv) Immunosuppressive agents have improved the success of transplantation.
      v) Nonischemic cardiomyopathy is the most common primary diagnosis for heart transplantation.
      vi) 54% of left ventricular assist devices (LVADs) are placed as a bridge to transplant.[3]



Organ ischemia time should be less than 6 hours to prevent organ damage.

   b) **Preoperative considesrations**
      i) Pathophysiology:
         (1) Patients presenting for heart transplant often have decreased stroke volume (SV), increased left ventricular end-diastolic volume (LVEDV), left ventricular end-diastolic pressure (LVEDP), pulmonary vascular pressures, pulmonary congestion and edema, increased sympathetic vascular tone, and increased salt/water retention.
         (2) Patients may have previous sternotomies and mechanical circulatory support (MCS), including intra-aortic balloon pump (IABP), VADs, and intracardiac devices (ICD).[4]

TABLE 104-1 Heart Transplantation Indications and Contraindications[1]

Indications	Contraindications
• Cardiogenic shock requiring continuous IV support or MCS • Persistent NYHA class IV HF symptoms refractory to maximal medical therapy LVEF < 20%, VO_2 max < 10-12 • Intractable angina symptoms in patients with CAD not amenable to revascularization therapy • Intractable life-threatening arrhythmias unresponsive to medical therapy	Absolute • Systemic illness with life expectancy <2 years, including active malignancy, AIDS with opportunistic infections, active systemic lupus erythematosus, or sarcoid • Amyloidosis with multisystem involvement • Irreversible hepatic or renal dysfunction • Fixed pulmonary HTN with PASP > 60 mm Hg • Mean transpulmonary gradient >15 mm Hg • PVR > 6 Wood units Relative • Age > 72 years • Active infection • Severe peripheral or cerebral vascular disease (symptomatic carotid stenosis, AAA > 6 cm) • Severe DM with end-organ damage (retinopathy, nephropathy, neuropathy) • Morbid obesity with BMI > 35 • Recent pulmonary infection • Irreversible neurological/neuromuscular disease • Drug/tobacco/alcohol abuse • Active mental illness/psychosocial instability • Active peptic ulcer disease • Heparin-induced thrombocytopenia • Abnormal lab values: Cr > 2.5, CrCl < 25, TBili > 2.5, AST/ALT > 3×, INR > 1.5 off Coumadin

HF, heart failure; LVEF, left ventricular ejection fraction; VO_2max, oxygen consumption; CAD, coronary artery disease; PASP, pulmonary artery systolic pressure; PVR, pulmonary vascular resistance; AAA, abdominal aortic aneurysm; DM, diabetes mellitus; BMI, body mass index; Cr, creatinine; CrCl, creatinine clearance; TBili, total bilirubin; AST, aspartate transaminase; ALT, alanine transaminase; MCS, mechanical circulatory support.
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ii) Preoperative assessment (Tables 104-2 to 104-4)
   (1) It is important to assess the patient for changes in functional capacity, including decreased exercise tolerance or inability to perform activities of daily living.
   (2) A thorough history should include last oral and anticoagulation intake, medications, laboratory, and imaging evaluation.
   (3) The physical exam should include assessment of volume status, including peripheral edema or weight gain.
      (a) Cerebral dysfunction can result from chronically decreased CO and perfusion pressures.
      (b) Pulmonary dysfunction can result from fluid retention and elevated pulmonary arterial pressure (PAP).
         (i) Increased PVR is predictive of early graft dysfunction and increased mortality because of a high incidence of right ventricular (RV) failure.
   (4) Hepatorenal dysfunction can result from chronic systemic hypoperfusion and venous congestion.
iii) Donor characteristics: Successful transplantation depends on a quality assessment of the donor, with matching ABO blood group compatibility, reactive antibodies, and size/gender matching.[3]



Emergent timing and coordination is needed to minimize the ischemic time of the transplanted organ.



64% of HF patients require some sort of MCS prior to heart transplantation. It is important to interrogate such devices and be familiar with their functions, settings, and common complications.

TABLE 104-2 Common Medications for Patients Presenting for Heart Transplantation and Their Perioperative Implications

Clinical Indication	Perioperative Implication
Anticoagulants	Low cardiac outputs and a high incidence of atrial fibrillation • Maintained on warfarin, heparin, and aspirin therapy • May contribute to perioperative bleeding
Beta-adrenergic antagonists	Chronically stimulated autonomic sympathetic tone, progressively leading to LV remodeling • Maintained on metoprolol or carvedilol to improve hemodynamics, exercise tolerance, and mortality in patients awaiting heart transplantation • May contribute to fluid retention, fatigue, bradycardia, heart block, and hypotension
Diuretics	Fluid retention • Maintained on loop and thiazide diuretics • May contribute to electrolyte disturbances, hypotension, intravascular volume depletion, and azotemia
Inotropes	High levels of catecholamines, decreased myocardial stores of norepinephrine, down-regulation of receptors • May require escalation of inotropic doses to achieve therapeutic effect • Digoxin is a weak inotrope. Its usage is limited by its toxic side effects • Phosphodiesterase inhibitors are effective, but chronic therapy can increase mortality • Patients dependent on inotropic therapy are often maintained with infusions of beta-adrenergic agonists (dopamine, dobutamine, epinephrine, and norepinephrine)
Vasodilators	Use vasodilators with caution: HF patients have a fixed stroke volume and require adequate preload, elevated systemic vascular resistance, and heart rate to maintain cardiac output • Acute treatment: nitroglycerine, sodium nitroprusside, and nesiritide reduce myocardial work and oxygen demands by decreasing preload and afterload • Nesiritide has dose-dependent reduction in RAP, PAP, and PCWP • Chronic treatment: ACE-I and ARB increases natriuresis and decreases ventricular remodeling but can cause hypotension requiring vasopressin and methylene blue

HF, heart failure; RAP, right atrial pressure; PAP, pulmonary arterial pressure; PCWP, pulmonary capillary wedge pressure; ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker.

(1) Donor age is an independent risk factor for recipient mortality (average age = 35).
(2) Matching gender improves survival.[7]
(3) Donor-recipient size mismatching of >30% is contraindicated.
  (a) An undersized donor heart in the setting of recipient pulmonary hypertension (pHTN) increases the risk of posttransplant RV failure.
(4) Donor assessment includes ECG, CXR, lab data including cardiac enzymes, and echocardiography (ECHO).
  (a) ECHO is used to evaluate left ventricular hypertrophy (LVH), regional wall motion abnormalities (RMWAs), valve dysfunction, diastolic function, and congenital abnormalities.
    (i) Donor LVH > 14 mm is associated with poor outcomes.[8]
  (b) Coronary angiography is recommended in males >45 and females >55.
  (c) Lab data include viral serologies (CMV, hepatitis B, hepatitis C, HIV).
  (d) Modest increase in troponin is not correlated with posttransplant mortality.[9]
  (e) The upper limit of *ex vivo* human heart storage is <6 hours.
    (i) Cardioplegia and hypothermia are effective in maintaining cardiac function.



The goals of induction are to avoid decreases in contractility, vascular tone, or venous capacitance.

c) **Intraoperative considerations for recipient**
  i) Induction

TABLE 104-3 Mechanical Circulatory Support for Patients Presenting for Heart Transplantation[5]

IABP	Patients in cardiogenic shock refractory to medical therapy may require counter pulsation for coronary perfusion via an intra-aortic balloon pump • Can lead to vascular complications and hemolysis • The device immobilizes the patient
VAD	About half of transplant recipients had VADs, and over half of VADs placed are used as a bridge to transplantation • No difference in transplantation outcomes for VAD patients, including length of VAD care[6]
Antiarrhythmic ICD	The patency, sterility, and access lines of ICDs should be assessed. After external defibrillation pads are placed, they should be reprogrammed/suspended to avoid misinterpretation of electrocautery • Magnets should be readily available • After most heart transplantation cases, the antiarrhythmic device will be removed

HF, heart failure; MCS, mechanical circulatory support; IABP, intra-aortic balloon pump; VAD, ventricular assist device; ICD, intracardiac device. From Vega JD, Moore J, Murray S, et al. HT in the United States, 1998-2007. *Am J Transplant*. 2009;9(4 Pt 2):932-941. Reprinted by permission of John Wiley & Sons, Inc.

TABLE 104-4 Heart Transplantation Preoperative Checklist by Systems

	Monitoring Equipment	Interventional Equipment/Pharmacology
Neurologic	• Cerebral oximetry • Electroencephalogram • Temperature (PAC, Foley)	• Anesthetic agents (inhalational agents, propofol, etomidate) • Analgesic agents (fentanyl, sufentanil, ketamine, dexmedetomidine, acetaminophen) • Muscle relaxant agents (rocuronium, vecuronium) • Fluid warmer, forced air warming device
Cardiovascular	• Five lead ECG • Large-bore peripheral IV • Arterial line • Central line • PAC • TEE • CCO/SvO_2 monitor	• Inotropes: epinephrine, milrinone, dopamine, dobutamine, ephedrine • Vasopressors: phenylephrine, norepinephrine, vasopressin • Vasodilators: nitroglycerin, nitroprusside, clevidipine • Antiarrhythmic: amiodarone, lidocaine, atropine, glycopyrrolate, • Rapid transfusion device (rapid infusion device) • Pacing box (ICD device deactivation) • IABP, ECMO, VAD
Respiratory	• Pulse oximetry • Capnography	• Subglottic ETT • Inhalational beta agonists • iNO, inhaled epoprostenol
Gastroenterology	OGT	Prophylaxis (PPI, H2 blocker)
Renal	Foley	Diuretics: furosemide, nesiritide
Hematology/ infectious disease	• ACT • POCT, ABG • TEG	• Antibiotics: (cefepime, vancomycin, piperacillin, and tazobactam) • Immunosuppressant, antirejection medications • Anticoagulants: heparin • Procoagulants: protamine, antifibrinolytics (tranexamic acid, aminocaproic acid), vitamin K • Blood products: pRBC, FFP, platelets, cryoprecipitate, factor concentrate • Anti-inflammatory: dexamethasone, methylprednisolone
Metabolic/ endocrine	POCT	• Electrolytes: calcium, magnesium, potassium, sodium bicarbonate • Insulin, dextrose

PAC, pulmonary artery catheter; EKG, electrocardiogram; TEE, transesophageal echocardiography; CCO/SvO_2, continuous cardiac output/venous oxygen saturation; ICD, intracardiac device; IABP, intra-aortic balloon pump; VAD, ventricular assist device; ETT, endotracheal tube; iNO, inhaled nitric oxide; OGT, oral gastric tube; PPI, proton pump inhibitor; ACT, activated clotting time; POCT, point-of-care test; ABG, arterial blood gas; TEG, thromboelastogram; pRBC, packed red blood cells; FFP, fresh frozen plasma.

(1) Full stomach and aspiration precautions are imperative.
(2) Use caution when administering premedication with sedatives as it is poorly tolerated in this patient population and can lead to hemodynamic collapse.
(3) Place external defibrillator pads prior to induction.
(4) Place hemodynamic monitors prior to induction.
  (a) This patient population has slow circulation times and delayed response to administered anesthetics.
(5) Inotropic support is almost always necessary during induction.
  (a) The hypokinetic and noncompliant heart is sensitive to alterations in afterload and preload.
  (b) It is important to maintain preload and continue all preoperative inotropic and vasoconstrictive agents.



Use caution when administering premedication with sedatives as it is poorly tolerated in this patient population and can lead to hemodynamic collapse.



It is important to maintain preload and continue all preoperative inotropic and vasoconstrictive agents.

ii) Pretransplant maintenance and management strategies
(1) The prevention of infection and rejection requires aseptic technique with invasive line placement, in addition to specific and appropriately timed antibiotic and immunosuppressive therapy.
(2) Irradiated *pRBCs* should be available in the OR prior to incision especially in cases with repeat sternotomy.
(3) Antifibrinolytics should be administered to minimize bleeding.
(4) Ventilation strategies should be directed to improve RV function and reduce PAP.
  (a) Avoid hypoxia and hypercarbia.
  (b) Ventilate with high FiO_2 concentrations and low tidal volumes (TVs) (6 mL/kg).
  (c) Achieve optimal positive end expiratory pressure (PEEP) since excessive PEEP can increase PVR by compression of small intra-alveolar vessels and reduce preload, while insufficient PEEP may not facilitate adequate oxygenation.
(5) Early detection and treatment of RV failure with inotropic support and reduction of PAP is critical.

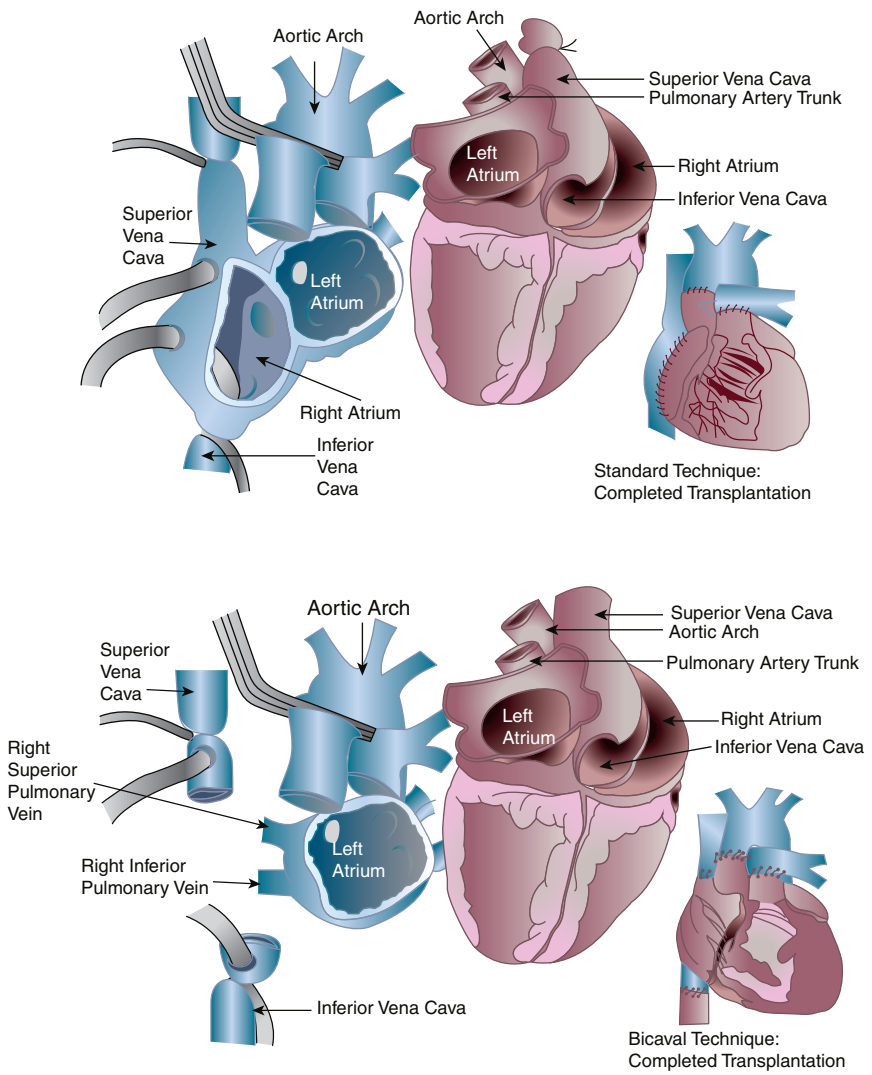
iii) Surgical approach
(1) A common surgical approach involves a median sternotomy using cardiopulmonary bypass (CPB).
(2) The bicaval technique improves perioperative mortality, maintains sinus rhythm, improves valvular function, and results in fewer pacemaker insertions.[10] (Fig. 104-1)
  (a) Bicaval technique involves five anastomosis sites: left atrial cuff, superior vena cava (SVC), inferior vena cava (IVC), pulmonary artery (PA), and aorta.
(3) During CPB
  (a) Maintain hypothermic environment for the donor organ for cardiac protection.
  (b) Monitor hemofiltration and administer mannitol.
  (c) Glucocorticoids are frequently administered to reduce the risk of rejection.



Cardiopulmonary bypass

iv) Transesophageal echocardiography (TEE)
(1) Pre-CPB: TEE is used to identify intracavitary thrombus, intracardiac shunts, aortic atherosclerotic disease, assess volume status, identify effusions, and evaluate biventricular function.
(2) Post-CPB: TEE is used for deairing maneuvers, assessment of surgical anastomosis sites, turbulent flow across the caval veins, PA, pulmonary veins, aorta can suggest stenosis of vessels. Evaluation of atrial sizes, biventricular systolic and diastolic function, particularly RV function, volume status, valvular function (particularly tricuspid regurgitation [TR] and mitral regurgitation [MR]), effusions/tamponade, and intracardiac shunts.

FIGURE 104-1 Surgical Approach to Heart Transplantation



(a) Up to one-third of patients can have RV enlargement and TR posttransplant that usually resolves over time.
(b) Excess donor tissue can obstruct the mitral valve causing pulmonary HTN and RV failure.

v) Posttransplant maintenance and management strategies (Table 104-5)
(1) Primary graft dysfunction (PGD) is the most common cause of early mortality (Table 104-6). PGD is based on several factors, including donor risks, procedural risks, and recipient risks.[13]

TABLE 104-5 Post-Heart Transplantation Maintenance and Management Strategies by Systems

	Goals	Common Problems and Interventions
Neurologic	• Amnesia • Analgesia • Normothermia	• Awareness: sedation (dexmedetomidine, propofol) • Pain: (fentanyl, sufentanil) • Hypothermia: fluid warmers, warming blankets • Stroke prevention: thorough deairing, maintain adequate perfusion pressures
Cardiovascular	• HR 90-110 • MAP > 65 • CVP 12-16 • PCWP 14-18 • CO/CI: 4-6/2-4	• Low cardiac output: maintain adequate preload, inotropes, IABP, ECMO • Diastolic dysfunction: calcium, inotropes (usually resolves after 1 mo) • Arrhythmias: external pacing, amiodarone • Pulmonary HTN: milrinone, pulmonary vasodilators—iEpo, iNO • RV failure: maintain SR, inotropes, milrinone, pulmonary vasodilators—iEpo, iNO—correct acid-base and electrolyte abnormalities, minimize blood transfusions, optimize ventilation settings, place RV assist device • Hemorrhage: volume repletion, vasoconstrictors • Anastomosis obstruction: TEE evaluation and surgical intervention • Primary graft dysfunction: inotropic support, early use of ECMO
Mechanical circulatory support: patients with CI < 2 L/min/m^2, SvO_2 < 60%, MAP < 50 mm Hg, SaO_2 < 85%, and pH < 7 in the setting of high-dose inotropic and vasopressor support ECMO provides more effective hemodynamic support compared to ventricular assist devices[11]		
Respiratory	• PaO_2 > 100 • $PaCO_2$ < 50 • TV 6 mL/kg • PEEP 5-10	• Hypoxia: FiO_2, PEEP, ECMO • Hypercarbia: TV, RR, ECMO • Pulmonary edema: antisialagogues, diuretics, BAL, chest physiotherapy • TRALI: diuretics, lung protective ventilation, supportive care
Renal	UOP > 0.5 mL/kg/h	• Anuria/dysuria: adequate perfusion pressures, diuretics, HD • Hematuria: check Foley placement, adequate perfusion pressures
Hematologic and metabolic	• Hgb 8-12 • Plt > 100 • PT/PTT < 15/40 • INR < 1.5 • Fibrinogen > 200	• Anemia: cell saver, autologous blood, pRBC transfusion • Thrombocytopenia: platelet transfusion • Coagulopathy: protamine, DDAVP, FFP, cryoprecipitate, factor concentrate, vitamin K • Electrolyte, acid/base abnormalities: replete to maintain pH: 7.35-7.45; Na: 135-145; K: 4-5; Ca: 1.0-1.6; HCO_3: 22-26; Mg: 2-4
Endocrine	Glu 100-150	Hyperglycemia: insulin

HR, heart rate; MAP, mean arterial pressure; CVP, central venous pressure; PCWP, pulmonary capillary wedge pressure; CO/CI, cardiac output/cardiac index; IABP, intra-aortic balloon pump; ECMO, extracorporeal membrane oxygenation; HTN, hypertension; iEpo, inhaled epoprostenol; iNO, inhaled nitric oxide; RV, right ventricle; SR, sinus rhythm; TEE, transesophageal echocardiography; SvO_2, venous oxygen saturation; SaO2, arterial oxygen saturation; PaO_2, partial pressure of arterial oxygen; $PaCO_2$, partial pressure of arterial carbon dioxide; FiO_2, fraction of inspired oxygen; PEEP, positive end-expiratory pressure; TV, tidal volume; RR, respiratory rate; BAL, bronchoalveolar lavage; TRALI, transfusion-related acute lung injury; UOP, urine output; HD, hemodialysis; Hgb, hemoglobin; Plt, platelet count; PT/PTT, prothrombin time/partial thromboplastin time; INR, international normalized ratio; pRBC, packed red blood cells; DDAVP, desmopressin; FFP, fresh frozen plasma; Na, sodium; K, potassium; Ca, calcium; HCO_3, bicarbonate; Mg, magnesium; Glu, glucose.

(a) Donor risks include old age, cause of death (intracranial hemorrhage, carbon monoxide poisoning), heart dysfunction, LVH, high inotrope requirement, diabetes mellitus (DM), illicit drug use.
(b) Procedural risks include prolonged ischemic time, donor/recipient mismatch, gender mismatch.

TABLE 104-6 Primary Graft Dysfunction after Heart Transplantation[12]

Grade	Left or Biventricular	Right Ventricular	Support
Mild	RA > 15 mm Hg, PCWP > 20 mm Hg, CI < 2.0 L/min/m^2 (lasting > 1 h) Or LVEF 40%-50%	RA > 15 mm Hg, PCWP < 15 mm Hg, CI < 2.0 L/min/m^2 *Plus* TPG < 15 mm Hg and/or PASP < 50 mm Hg	Low-dose inotropic infusion
Moderate	RA > 15 mm Hg, PAWP > 20 mm Hg, CI < 2.0 L/min/m^2, MAP < 70 mm Hg (lasting > 1 h) *Or* LVEF < 40%	RA > 15 mm Hg, PCWP < 15 mm Hg, CI < 2.0 L/min/m^2, MAP < 70 mm Hg (lasting > 1 h) *Plus* TPG < 15 mm Hg and/or PASP < 50 mm Hg	High-dose inotropic support *Or* IABP
Severe	As above *Plus* LVAD, BiVAD, or ECMO	As above *Plus* RVAD	ECMO

RA, right atrium; PCWP, pulmonary capillary wedge pressure; CI, cardiac index; LVEF, left ventricular ejection fraction; TPG, transpulmonary gradient; PASP, pulmonary artery systolic pressure; PAWP, pulmonary artery wedge pressure; MAP, mean arterial pressure; IABP, intra-aortic balloon pump; LVAD, left ventricular assist device; BiVAD, biventricular assist device; ECMO, extracorporeal membrane oxygenation; RVAD, right ventricular assist device. Reprinted from Kobashigawa J, Zuckermann A, Macdonald P, et al. Report from a consensus conference on primary graft dysfunction after cardiac transplantation. *J Heart and Lung Transplantation*. 2014;33(4):327-340, with permission from Elsevier.

(c) Recipient risks include old age, inotrope dependence, ventilator support, MCS, pulmonary HTN, DM, obesity, kidney disease.
(d) High-risk heart donors when combined with complicated recipients can lead to severe PGD.[14]
(i) Early extracorporeal membrane oxygenation (ECMO) and MCS devices can be used to improve survival.[15]

(2) Mechanical circulatory support:
(a) 4%-12% of patients require a permanent pacemaker after heart transplant from loss of sinoatrial node function.[16]
(b) In severe HF, RV assist devices may be necessary to support cardiac output.
(c) Early use of IABP optimizes coronary perfusion and decreases afterload.
(d) ECMO should be instituted early on if there is a high suspicion for development of multiorgan failure after transplant.
(i) When ECMO is instituted early, it has promising outcomes: 60% of patients were able to be weaned from support, with 46% surviving to discharge and 39% surviving to 1-year posttransplant.[17]

d) **Postoperative considerations[18]**

i) The denervated heart
(1) Has a normal resting HR of 90-110
(2) Has no response to manipulations of the autonomic nervous system, including anticholinergic medications, manipulations of cardiac acceleration fibers, or afferent pain from angina
(3) Has no response to hypovolemia, vasodilation, or exercise
(a) Has a decreased response to changes in cardiac filling pressures
(b) Has a decreased response to the renin-angiotensin-aldosterone system
(4) Has decreased adrenergic stimulation and consequently up-regulated beta-receptors
(a) Use direct-acting agents to obtain a gradual response.
(5) Is prone to arrhythmias from the native and donor SA node



The denervated posttransplant heart does not respond to manipulations of the autonomic nervous system, including anticholinergic medications. Direct-acting agents will obtain a gradual response.

ii) Rejection:
(1) Accounts for 11% of deaths within 1 month.
(2) 30% of patients experience rejection within 1 year.
(3) Females are at increased risk of rejection.

(4) Endomyocardial biopsy is used to confirm rejection.
(a) TR is a risk of repeated endomyocardial biopsies.
(5) Rejection requires aggressive up-regulation in immunosuppressive agents.

iii) Cardiac allograft vasculopathy (CAV):
(1) This presents as diffuse hyperplasia involving the all coronary arterial segments.
(2) Risk factors include HTN, hyperlipidemia (HLD), DM, hyperhomocysteinuria, and increased donor age.
(3) Cotransplanted liver can modify the recipient's immunological risk factors and improve the natural history of CAV.[19]

iv) Infection:
(1) Accounts for 11% of heart transplant deaths within 1 month and 29% of deaths within 1 year.

2) **Lung transplantation**

a) **General considerations** (Table 104-7)

i) The lung allocation score (LAS) is a numerical value used to assign relative priority for the distribution of donated lungs for transplantation. It considers the statistical probability of patient survival in the next year without transplant and the projected length of survival posttransplant.

ii) The complexity of patients has increased, including patients with pulmonary HTN and RV dysfunction.

iii) Survival rates: 1 year, 84%; 3 years, 67%; 5 years, 55%

b) **Preoperative considerations**

i) Pathophysiology: end-stage lung disease
(1) Obstructive disease: risk of hyperinflation, auto-PEEP, and pneumothorax
(2) Restrictive disease: risk for severe pulmonary HTN and RV failure
(3) Parenchymal disease (cystic fibrosis): thick secretions and severe hypercapnia

TABLE 104-7 Lung Transplantation Indications and Contraindications[20]

Indications	Contraindications
• Patients with end-stage pulmonary, parenchymal, and/or vascular disease who have a projected life expectancy of <50% within 2 y • In addition to patient factors, the LAS also consider the best utilization of donor organs. Since lung transplantation, itself also risks mortality, patients are prioritized based on specific disease-related mortality with an expected > 80% chance of 5-year survival after transplantation	Absolute: • Recent malignancy (need 5-y disease-free period for any major malignancy) • Untreatable major organ dysfunction (that is not paired with another transplant) • Atherosclerotic disease with end-organ dysfunction/CAD (not amenable to revascularization) • Any acute medical instability • Chronic infection (including mycobacterium tuberculosis) • Significant chest wall deformity that would be expected to cause severe restriction • BMI > 35 • Psychiatric/psychological condition that results in an inability to cooperate with medical care • Absence of social support system • Severely limited functional status with poor rehabilitation potential
	Relative: • Age > 65 (with low physiologic reserve) • BMI 30-35 • Severe malnutrition • Extensive prior chest surgery • Mechanical ventilation or mechanical circulatory support

LAS, lung allocation score; CAD, coronary artery disease; BMI, body mass index. Reprinted from Weill D, Benden C, Corris PA, et al. A consensus document for the selection of lung transplant candidates: 2014–an update from the pulmonary transplantation council of the international society for heart and lung transplantation. *J Heart Lung Transplant*. 2015;34(1):1-15, with permission from Elsevier.

ii) Risk factors for increased posttransplant mortality include:
  (1) Right ventricular dysfunction
  (2) Presence of pHTN
  (3) Prior extensive thoracic procedures



The goals of induction for lung transplant are to protect the airway, maintain SVR, and avoid hypoxia and hypercapnia.

iii) Assessment:
  (1) Emergent timing and coordination are essential in order to minimize organ ischemia time.
  (2) Evaluate for changes in functional capacity, NPO status, anxiety, medications, and recent laboratory and imaging.

iv) Preoperative checklist by systems (see **Table 104-4**)
  (1) Cardiopulmonary assessment includes a thorough history and physical exam evaluating oxygen requirements, the ability of the patient to tolerate one lung ventilation (V/Q scan), current hemodynamic support, history of pHTN and RV dysfunction, or any recent swelling or secretions.
  (2) Adequate oxygenation and ventilation in addition to adequate lung isolation are mandatory.[21]
    (a) Ensure availability of a left-sided double-lumen tube (L-DLT): 39 Fr to fit adult sized bronchoscope or bronchial blocker and fiberoptic scope for visualization of positioning.
  (3) Mechanical support: Some young patients without multiorgan dysfunction and a good potential for recovery require extracorporeal life support (ECLS) as a bridge to transplantation.
    (a) CPB and ECMO should be readily available prior to induction.
    (b) PA clamping: In pulmonary HTN/RV dysfunction, clamping the PA may not be tolerated.
      (i) RVEF and CO should be assessed to predict which patients will require CPB (**Table 104-8**).



Patients presenting for lung transplant have decreased cardiovascular reserve and are catecholamine depleted. Initiation of positive pressure ventilation, combined with anesthetic vasodilation, hypotension, and myocardial depression can cause cardiovascular collapse.

c) **Intraoperative considerations**
  i) *Induction*
    (1) The prevention of infection and rejection requires aseptic technique with invasive line placement, a specific antibiotic administration and appropriate timing, as well as care for administering required immunosuppressive therapy.
    (2) Patients are chronically hypoxic or hypercapnic, placing them at high risk of respiratory depression from sedatives.
    (3) Preoxygenation is vital in this patient population.
    (4) Inhaled beta-adrenergic agonists should be considered.
    (5) Rapid sequence intubation with a double-lumen tube or a bronchial blocker with a single-lumen tube in anticipation of one-lung ventilation (OLV)
    (6) Inotropic agents prior to induction may be necessary to support the right ventricle.
      (a) Systemic hypotension in the setting of increased right ventricular end-diastolic pressure (RVEDP) reduces coronary perfusion.
      (b) ECMO/CPB should be ready in case of emergency.[23]
  ii) Pretransplant maintenance and management strategies
    (1) Pretransplant ventilation strategy[21]:
      (a) Consider pressure control ventilation (PCV) for lung transplant patients at risk for lung injury.
      (b) Keep peak inspiratory pressures (PIP) <35 cm H_2O.

TABLE 104-8 Lung Transplantation TEE Evaluation Guidelines[22]

Pretransplant	**Interatrial septum/PFO assessment** Tissue flap in interatrial septum in ME bicaval view. CFD across interatrial septum. Saline contrast study **Right ventricle** *Size*: minor axis ME4C view (normal <3.3 cm in diastole). *Fractional area change*: ME4C view (normal >35%). *Wall thickness*: inferior wall in TG SAX of RV (normal <5 mm in diastole). *TAPSE*: M-mode deep TG view (normal >16 mm). *RIMP* using PWD (normal <0.4). *Septal position and motion*: TG SAX view (flattening, bulging into LV during systole and/or diastole). *RVSP*: CWD of TR jet in ME RV I-O view or modified bicaval view (normal <2.8 m/s) **Left ventricle** Systolic function: ME4C, ME2C, ME LAX, TG mid papillary SAX view **Pulmonary veins** Baseline diameter (normal >5 mm). Baseline velocity (normal <100 cm/s)
Intraprocedural	• Assess RV response to pulmonary artery clamping • Reassess size for chamber dilation, TAPSE for contractility, TR jet for estimation of PA systolic pressures • Hemodynamic instability • Hypovolemia, MI, RV failure, or tamponade • If PFO present • CFD assessment of reversibility of shunt
Posttransplant	**Pulmonary veins** Diameter (normal >5 mm). Laminar vs turbulent flow via CFD. Velocity via PWD (normal <100 cm/s). Thrombus, torsion, air **Pulmonary artery anastomosis (RPA, LPA)** Diameter (normal >75% proximal PA). Laminar vs turbulent flow via CFD in UE AA SAX view **Right ventricle** Dilation: RVEDP. TAPSE. Septal position. RVSP. RIMP. **Left ventricle** Preload. Systolic function. Air **If PFO present** Direction of flow

PFO, patent foramen ovale; ME, midesophageal; CFD, color flow Doppler; ME4C, midesophageal four chamber; TG SAX, transgastric short axis; RV, right ventricle; TAPSE, tricuspid annular plane systolic excursion; TG, transgastric; RIMP, right ventricular index of myocardial performance; PWD, pulse wave Doppler; RVSP, right ventricular systolic pressure; TR, tricuspid regurgitation; ME RV I-O, midesophageal right ventricular inflow-outflow; ME2C, midesophageal two chamber; ME LAX, midesophageal long axis; PASP, pulmonary artery systolic pressure; MI, myocardial infarction; RPA, right pulmonary artery; LPA, left pulmonary artery; UE AA SAX, upper esophageal ascending aorta short axis; RVEDP, right ventricular end diastolic pressure. Reprinted with permission from Evans A, Dwarakanath S, Hogue C, et al. Intraoperative Echocardiography for Patients Undergoing Lung Transplantation. *Anesthesia and Analgesia*. 2014;118(4):725-730.

(c) Keep plateau airway pressures <25 cm H_2O.
(d) RR adjustment: expect $PaCO_2$-$ETCO_2$ gradient to increase by 3 mm Hg during OLV.
  (i) Permissive hypercapnia can cause pulmonary HTN and RV dysfunction.
(e) Use a TV 5-6 mL/kg of ideal body weight (IBW).
(f) FiO_2 should be guided by oxygen saturation.
(g) Keep PEEP around 5 cm H2O. Avoid added PEEP in patients with moderate-severe chronic obstructive pulmonary disease (COPD).
  (i) Note that excessive PEEP may shunt blood to the nonventilated lung (Table 104-9).

(2) RV failure
(a) Increases in afterload and decreases in SV make the right ventricle prone to failure.

TABLE 104-9 Pre-Lung Transplantation Maintenance and Management of Different Lung Pathologies[21]

Pathology	Presentation	Problems	Interventions
Obstructive disease	Bronchiolitis obliterans COPD Emphysema	Hyperinflation Tension Pneumothorax	• Pressure-controlled ventilation • Maximizing exhalation time (I:E ratio 1:3-1:4) • Minimize auto-PEEP • No or low extrinsic PEEP (3-4 cm H_2O)
Parenchymal diseases	Bronchiectasis Cystic fibrosis[24]	Hypercapnia Secretions	• BAL and suctioning • Higher airway pressures • Higher PEEP
Pulmonary vascular disease	Pulmonary HTN	RV failure	• Inhaled prostaglandins, iNO • Central venous access and monitoring prior to induction • Inotropic/vasopressor/inhaled pulmonary vasodilators • Extracorporeal mechanical circulatory support
Restrictive disease	Interstitial lung disease Sarcoidosis	Hypoxia Pulmonary HTN Trauma	• Higher peak inspiratory pressures (40 cm H_2O) • Higher extrinsic PEEP (8-10 cm H_2O) • Maximizing inspiratory time (I:E= 1:1-1:2)

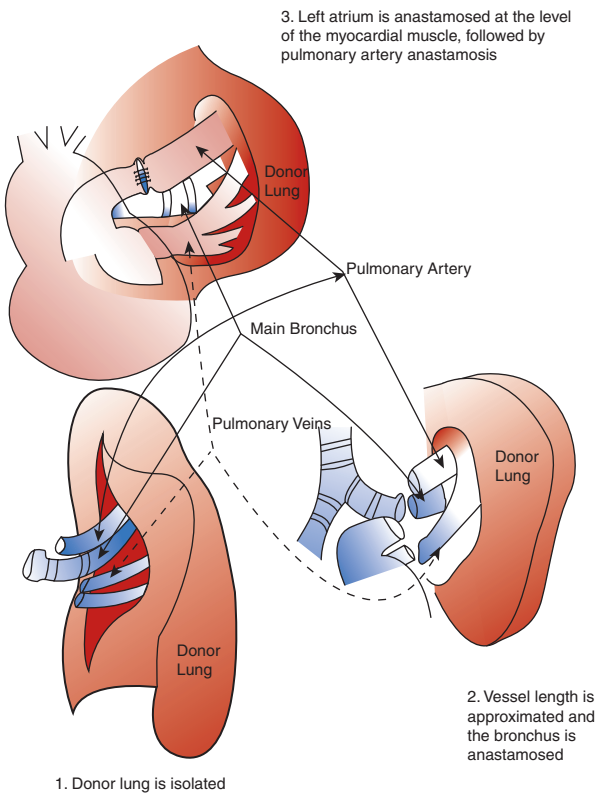
I:E, inspiratory to expiratory; PEEP, positive end-expiratory pressure; BAL, bronchoalveolar lavage; HTN, hypertension; RV, right ventricular; iNO, inhaled nitric oxide. Reprinted from Nicoara A, Anderson-Dam J. Anesthesia for Lung Transplantation. *Anesthesiology Clin*. 2017;35(3):473-489, with permission from Elsevier.

(b) Treatment includes maintaining RV coronary perfusion pressure and avoiding large TVs or high PEEP.
  (i) If PVR normal: increase preload
  (ii) If PVR increased: inotropes and pulmonary vasodilators

iii) Surgical approach
  (1) Positioning: supine, lateral decubitus
  (2) Incision: thoracosternotomy, thoracotomy, sternotomy
  (3) If bilateral lung transplant, first, transplant the side that receives less perfusion (**Fig. 104-2**)

iv) Procedure Summary:
  (1) Bronchus is anastomosed
  (2) Left atrium is clamped intrapericardially, opened, and anastomosed at a level at which myocardial muscle tissue is present, because at the level of the veins, the tissue is too fragile to allow a safe anastomosis
  (3) Pulmonary artery is centrally clamped, opened, and anastomosed
  (4) The vessels should be kept short enough to avoid kinking
  (5) Initial dose of immunosuppressant is administered (methylprednisolone, mannitol)
  (6) Retro- and antegrade flushing is performed to deair the vasculature and flush out the preservative solution
  (7) The sutures of the vessels are knotted
  (8) Protective ventilation without a manual recruitment maneuver started
  (9) If the procedure is done without extracorporeal support, controlled reperfusion should be performed for 10 min with partial manual compression of the PA to avoid initial volume overload of the implanted lung
  (10) Meticulous hemostasis is performed with special attention to the donor pulmonary ligament and pericardium, which may be the source of significant bleeding

FIGURE 104-2 Lung Transplantation: Surgical Approach



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v) Posttransplant maintenance and management strategies
   (1) Posttransplant ventilation strategy (**Table 104-10**):[29]
      (a) The goal is to avoid barotrauma, volutrauma, and PGD.
      (b) Single-lumen endotracheal tube (ETT) exchange at the end of the case (the airway is likely to be edematous)
      (c) Use a TV of 6 mL/kg IBW (adjust for OLV if needed).
         (i) Consider using donor body weight if the allograft is undersized.
         (ii) Use careful recruitment maneuvers.
         (iii) Keep PIP < 30 cm H_2O.
      (d) Keep PEEP between 6 and 8 cm H_2O.
      (e) Use the lowest FiO_2 to maintain a PaO_2 > 70 mm Hg.
      (f) Adjust the RR to maintain $PaCO_2$ < 50.
   (2) Primary graft dysfunction (PGD): PGD is based on several factors, including donor risks, procedural risks, and recipient risks.
      (a) Presents as hypoxemia, poor lung compliance, pulmonary HTN, RV failure, diffuse pulmonary edema, and infiltrates on chest radiograph.



Early veno-venous (VV) ECMO in the setting of PGD improved 30-day, 1-year, and 5-year survival of 82%, 64%, and 49%, respectively.[28]

TABLE 104-10 Post-Lung Transplant Maintenance and Management Strategies by Systems

Neurologic	• Awareness: sedation (dexmedetomidine, propofol) • Pain: analgesia (fentanyl, sufentanil) • Hypothermia: fluid warmers, Bair Hugger, blankets • Stroke prevention: thorough deairing, maintain adequate perfusion pressures
Cardiovascular	• Hypotension (due to reperfusion, pneumoplegia, metabolite washout, RCA air entrainment): ↓ fluid administration (associated with pulmonary edema and PGD), use vasopressors • Hemorrhage: blood product transfusion >1 L associated with PGD • Pulmonary HTN: milrinone, pulmonary vasodilators—inhaled epoprostenol, iNO (decreases ischemia-reperfusion injury) • RV failure: inotropes, milrinone, decrease preload (NTG), pulmonary vasodilators—inhaled epoprostenol, iNO • Anastomosis obstruction: TEE evaluation and surgical intervention
Respiratory	• Hypoxia: optimal FiO_2 and PEEP, use lowest FiO_2 to maintain $PaO_2 > 70$ • Hypercarbia: optimal TV and RR, maintain $PaCO_2 < 50$ • Pulmonary edema/secretions: limit fluids, suction, antisialagogues, diuretics, bronchoscopy, BAL, chest physio • PGD: lung protective ventilation strategies (see ventilation strategy below), pulmonary vasodilator therapy (iNO, prostacyclin), inotropic support, volume restriction, diuretics, ECMO[26,27]
Renal	Anuria/dysuria: adequate perfusion pressures, diuretics, HD/UF, RRT
Hematologic and metabolic	• Coagulopathy: protamine, DDAVP, FFP, cryoprecipitate, factor concentrate, vitamin K • Electrolyte, acid/base abnormalities: replete to maintain pH: 7.35-7.45; Na: 135-145; K: 4-5; Ca: 1.0-1.6; HCO_3: 22-26; Mg: 2-4
Endocrine	Hyperglycemia: insulin

RCA, right coronary artery; PGD, primary graft dysfunction; HTN, hypertension; iNO, inhaled nitric oxide; RV, right ventricle; NTG, nitroglycerin; TEE, transesophageal echocardiography; FiO_2, fraction of inspired oxygen; PEEP, positive end-expiratory pressure; PaO_2, partial pressure of arterial oxygen; TV, tidal volume; RR, respiratory rate; $PaCO_2$, partial pressure of arterial carbon dioxide; BAL, bronchoalveolar lavage; ECMO, extracorporeal membrane oxygenation; HD/UF, hemodialysis/ultrafiltration; RRT, renal replacement therapy; DDAVP, desmopressin; FFP, fresh frozen plasma; Na, sodium; K, potassium; Ca, calcium; HCO_3, bicarb; Mg, magnesium.

(b) Donor risks include age <21, age> 45, African American, female, and a history of smoking.
(c) Recipient risks include obesity, female, pulmonary HTN, fibrosis, and sarcoidosis.
(d) Operative risks include single-lung transplant, prolonged ischemia, CPB use, transfusion >1 liter, and a $FiO_2 > 0.4$ at reperfusion (Table 104-11).[30]

(3) Mechanical circulatory support:
(a) Over one-third of lung transplant procedures require CPB or ECMO.
(i) Advantages include hemodynamic stability and ease of surgical manipulation.
(ii) Disadvantages include higher incidence of PGD.
(b) ECMO has a lower incidence of transfusion, pulmonary edema, reoperation, and renal complications as compared to CPB.[31]

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d) **Postoperative considerations**
i) Transplanted lung
(1) Has no response to parasympathetic stimulation, poor compliance, and interstitial infiltrates
(2) Often has pulmonary HTN due to decreased ability for smooth muscle relaxation, pulmonary vasoconstriction, changes in V/Q mismatch, and a blunted response to pulmonary vasodilators



The transplanted lung has no response to parasympathetic stimulation, poor compliance, and interstitial infiltrates.

TABLE 104-11 Primary Graft Dysfunction after Lung Transplantation[33]

Grade	Diffuse Pulmonary Edema on Radiograph	PaO_2/FiO_2	Exceptions
0	–	Any	
1	+	>300	On nasal cannula or $FiO_2 < 0.3$
2	+	200-300	
3	+	<200	Any patients on ECMO or on iNO with $FiO_2 > 0.5$

PaO_2/FiO_2, partial pressure of arterial oxygen/fraction of inspired oxygen; ECMO, extracorporeal membrane oxygenation; iNO, inhaled nitric oxide. Reprinted from Christie JD, Carby M, Bag R, et al. Report of the ISHLT Working Group on Primary Lung Graft Dysfunction part II: definition. A consensus statement of the International Society for Heart and Lung Transplantation. *J Heart Lung Transplant*. 2005;24(10):1454-1459, with permission from Elsevier.

(3) Often has pulmonary edema due to reperfusion injury, endothelial damage, leaky capillaries, depressed mucociliary function, and an inability to clear secretions (Table 104-12)

3) **Combined heart-lung transplantation**

a) **General considerations**

i) Patients with primary pHTN and congenital heart diseases (Eisenmenger syndrome)

ii) Critically ill patients with idiopathic pHTN may have improved outcomes, particularly reduced PGD, after combined heart-lung transplant as compared to double-lung transplant alone.[32]

b) **Preoperative considerations**

i) Patients have end-stage biventricular failure with pulmonary HTN.

ii) Cardiac anatomy may involve complex congenital abnormalities.

c) **Intraoperative considerations**

i) TEE is essential for perioperative cardiopulmonary evaluation and thorough deairing.

TABLE 104-12 Post-Lung Transplantation Common Complications by Systems[34]

Neurological	Tremor, seizures, posterior leukoencephalopathy, headache, paresthesias, peripheral neuropathy
Cardiovascular	Air embolism, postoperative pericarditis, thromboembolism, supraventricular tachycardia, arterial hypertension
Respiratory	Allograft: Primary transplant dysfunction, necrosis and obstruction of anastomotic region, acute rejection, BOS, drug-induced pneumonitis (sirolimus, everolimus) Thoracic: Diaphragm paralysis (phrenic n. lesion), vocal cord paralysis (recurrent laryngeal n. lesion), gastroparesis or delayed gastric emptying (vagus n. lesion), chylothorax, pneumothorax, pleural effusion
Gastrointestinal	Hepatobiliary: Hepatitis (CMV, EBV, HEV, HBV, HCV, drug-induced toxicity) Esophagitis (*Candida* or CMV), gastroparesis, GERD, diarrhea or pseudomembranous colitis (*C diff.*), diverticulitis, colon perforation, distal intestinal obstruction
Renal	Renal failure (acute/chronic), calcineurin inhibitor–induced nephropathy
Hematological/ oncological	Anemia, leukopenia, thrombocytopenia, hypogammaglobulinemia, thrombotic microangiopathy Posttransplantation lymphoma, skin tumors, other malignant tumors
Infections	Bacteria, fungi (esp. *Aspergillus* spp.), viruses (esp. CMV, HSV, RSV)
Musculoskeletal	Steroid myopathy, rhabdomyolysis (cyclosporine + HMG-CoA inhibitor), osteoporosis, aseptic bone necrosis
Endocrine	Obesity, diabetes mellitus, hyperlipidemia, hyperlipoproteinemia, hyperammonemia

BOS, bronchiolitis obliterans syndrome; CMV, cytomegalovirus; EBV, Epstein-Barr virus; HEV, hepatitis E virus; HBV, hepatitis B virus; HCV, hepatitis C virus; GERD, gastroesophageal reflux disease; *C diff.*, *Clostridium difficile*; HSV, herpes simplex virus; RSV, respiratory syncytial virus. Reprinted by permission from Springer: Dierich M, Fuehneret T, Welte T, et al. Lung transplant: Indications, long-term results and special impact of follow-up care. *Internist (Berl)*. 2009;50(5):561-71.

ii) Ventilation management: (see pre- and posttransplantation ventilation strategies)
  (1) Avoid hypoxia and hypercapnia
iii) Fluid management
  (1) Balance adequate preload for the transplanted heart with limited crystalloid administration to avoid pulmonary edema and PGD in the transplanted lungs.
  (2) Denervation:
    (a) Transplanted heart: lack of autonomic response requires adequate preload and direct-acting inotropic agents.
    (b) Transplanted lungs: limited vascular and airway smooth muscle response as well as altered pulmonary lymphatic drainage and impaired mucociliary clearance.
  (3) Cardiopulmonary dysfunction due to ischemia times, cardioplegia/pneumoplegia, hypothermia, reperfusion injury, and immunosuppressive agents

d) **Postoperative considerations**
i) Invasive monitoring, continued cardiopulmonary support is similar to heart transplant and lung transplant.
ii) Infection: antibiotics, antivirals, antifungals
iii) Rejection: immunosuppressants
  (1) Heart-lung transplant recipients commonly develop accelerated cardiac allograft vasculopathy and/or bronchiolitis obliterans syndrome.

CHAPTER SUMMARY

Anesthesia Considerations for Heart and Lung Transplantation

Preoperative	The emergent nature of the surgery requires efficient communication with all health care providers and thorough investigation of both patient and donor. Successful operations depend on appropriate patient selection and unique management of specific heart and lung pathologies.
Intraoperative	End-stage heart disease pathophysiology often requires pharmacologic and mechanical support and presenting patients have often had previous sternotomies. For end-stage lung disease, minimize fluid administration, utilize lung-protective ventilation strategies and have a low threshold to initiate early extracorporeal membrane oxygenation.
Postoperative	Posttransplant denervated hearts have a unique physiology, requiring vigilance and early action to support the heart and avoid dysfunction. While patients can develop a myriad of posttransplant complications, the mainstay of management is the avoidance of primary graft dysfunction, rejection, and infection.

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105 Ventricular Assist Devices

Wolf B. Kratzert • Charles C. Hill

Ventricular assist devices (VADs) are pumps that can partially or fully take over the function of the ventricles of the heart. Mechanical ventricular support has become an increasingly employed adjunct in the medical treatment of acute cardiogenic shock, as well as short- and long-term support of the patient awaiting orthotopic heart transplantation. Devices exist for left, right, and biventricular support. With rapidly improving technology and an increasing number of long-term VADs in use, the need to provide care to the stable VAD patient undergoing elective or semielective surgery will continue to grow. This chapter focuses on the anatomy, physiology, and perioperative management of short- and long-term VADs.

1) **General considerations**
   a) **Short-term support**[1]
      i) Short-term VADs are being used as a bridge-to-recovery in patients with conditions expected to improve over time, such as acute viral myocarditis, acute cardiogenic shock, postcardiotomy cardiogenic shock, and left ventricular assist device (LVAD)-associated right ventricular (RV) failure.
      ii) These devices are extracorporeal (pump is located outside the body), less technically difficult to implant, and less operator dependent.
      iii) Cumbersome external control and power components that require the patient to be hospitalized.
      iv) Short-term devices may be placed percutaneously or via thoracotomy with direct cannulation of the heart.
      v) If the ventricle does not recover, these devices become the bridge-to-bridge and need to be replaced by a long-term device.



Currently there are four indications for VADs: bridge to recovery, bridge to bridge, bridge to transplant, and destination therapy.[1]

   b) **Long-term support**[1]
      i) VAD therapy as a bridge-to-heart transplantation has marked survival benefits and improvement in quality of life in end-stage heart failure.
      ii) Additionally, improvements in size, durability, and mobility are leading to an increase in the use of the VAD as destination therapy.
      iii) Most of these devices are intracorporeal (pump implanted in pre-peritoneal cavity). The only extracorporeal component is the subcutaneously tunneled driveline that connects to a control device and a wearable battery (Table 105-1).
   c) **VAD anatomy**
      The mechanical principles of all devices are similar and consist of VAD inflow cannula, flow generating chamber, VAD outflow cannula, and the driveline.
      i) Cannulas: The inflow and outflow cannulas, most commonly placed in the LV apex and the ascending aorta, respectively, bring blood from the heart into the pumping chamber of the device and back into the systemic circulation.
      ii) Flow generating chamber: Consists of either pusher plates or a diaphragm to create pulsatile flow; in an axial flow device, a rotating impeller provides continuous nonpulsatile blood flow.
      iii) External parts: The pumping chamber connects via a driveline to external controls/power source.

TABLE 105-1 Commonly Used VADs

Location		Device	Type	rpm	Max. Flow	Systemic Anticoagulation
Short term	External	VA ECMO	Centrifugal	1500-3500	7 L/min	Yes (heparin)
		Thoratec CENTRIMAG	Centrifugal	2000-5500	10 L/min	Yes (heparin)
		CardiacAssist TANDEM HEART	Centrifugal	3000-7500	4 L/min	Yes (heparin)
		CardiacAssist PROTEK DUO	Centrifugal	3000-7500	4 L/min	Yes (heparin)
	Internal	AbioMed IMPELLA 2.5/5.0/RP	Axial	10 000-50 000	2.5/5.0 L/min	No
Long term	Internal	Thoratec HEARTMATE II	Axial	6000-15 000	10 L/min	Yes (aspirin + warfarin)
		Jarvic JARVIK 2000	Axial	8000-12 000	5.5 L/min	Yes (aspirin + warfarin)
		BERLIN HEART INCOR	Axial	5000-10 000	7 L/min	Yes (aspirin + warfarin)
		HEARTWARE HVAD	Centrifugal	16 000-28 000	10 L/min	Yes (aspirin + warfarin)
		Thoratec HEARTMATE III	Centrifugal	2000-5500	10 L/min	Yes (aspirin + warfarin)

rpm, revolutions per minute; SV, stroke volume.

d) **VAD histology**[2]
   i) A VAD is a mechanical pump that depends on adequate preload. A left VAD therefore relies on the native right ventricle to pump venous blood through the lungs and into the left heart circulation.
   ii) Most first-generation VADs were pulsatile devices and functioned similarly to the native ventricle by filling and emptying the pumping chamber sequentially.
   iii) Currently used second- and third-generation VADs are continuous flow devices with the advantage of higher durability, smaller size, decreased disruption of RV function, and increased long-term hemodynamic stability.



RV failure can be catastrophic as there is not adequate filling pressure for the LVAD pump.

e) **Continuous flow devices**[3]
   i) Continuous flow devices function by axial or centrifugal flow and produce nonpulsatile blood flow. These include the HeartMate II, HeartMate 3, and HeartWare devices.
   ii) Driven by a centrally rotating element, blood is accelerated through the device into the systemic circulation.
   iii) Nonpulsatile flow may be adjusted by changing the device rotation speed given in revolutions per minute (rpm). As flow rates are increased, so is the amount of support by promoting ventricular unloading and augmenting systemic blood flow.
   iv) Native ventricular function is indicated by residual pulsatility seen on the arterial line tracing, as well as the pulsatility index (PI) in some devices.

v) Axial flow devices will cycle off 8-10 seconds per minute to allow ejection through the aortic valve and movement of blood antegrade through the aortic root to prevent thrombosis of the aortic root.

vi) Continuous flow devices are dependent on preload as well as afterload. Hypovolemia and cannula malposition will cause decrease in device output and can lead to suctioning events. Increased afterload has a slowing effect on forward flow and can impair VAD output. Both thereby result in decreased systemic perfusion.



VAD patients commonly have chronic systemic disease secondary to long-standing heart failure.

2) **Preoperative considerations**[4]

a) **Neurologic**

With increased risk of thromboembolic events and hypoperfusion, a comprehensive baseline neurologic exam should be obtained.

i) Pulmonary

Signs of pulmonary congestion on preoperative clinical and radiographic exam can indicate possible device malfunction with suboptimal drainage of the left cardiac chamber by the LVAD.

ii) Cardiovascular

(1) Evaluate for symptoms and signs of LV and RV failure.

(2) Backward failure with pulmonary edema, elevated JVD, hepatosplenomegaly, and pedal edema, or forward failure, with hypoperfusion and worsening end-organ function, could be signs of suboptimal device function.

(3) The presence of intracardiac or device-associated thrombi should be assessed as these patients are at significant risk for intraoperative cerebrovascular accidents, ischemic bowel, and vaso-occlusive damage to the peripheral vasculature.

(4) Assessment of RV contractility and volume status is critically important, as this represents the ability to fill the left VAD in cases of left ventricular support.

b) **Hematologic**[5]

i) Patients are at increased risk of perioperative bleeding.

ii) Mechanical destruction of cells leads to anemia and thrombocytopenia.

iii) Hepatic and splenic congestion, chronic renal dysfunction, and poor nutritional status all lead to impairment of the coagulation cascade.

iv) Systemic anticoagulation is required with the use of VADs to prevent thrombus formation.

v) Anticoagulation with warfarin should be held with a transition to heparin infusion.

c) **Endocrine**

i) In patients with chronic illness, on preoperative steroids, or exhibiting insensitivity to vasopressor agents, stress dose steroids should be considered.

d) **Gastrointestinal**

i) VAD patients should be considered a "full stomach," secondary to the intraperitoneal or preperitoneal placement of the assist device.

ii) VAD patients have an increased risk of gastrointestinal bleeding due to higher incidence of arteriovenous malformation, acquired platelet dysfunction, and systemic anticoagulation.

e) **Hepatic**

i) Chronic heart failure can cause hepatic congestion with impaired synthetic function, most commonly exhibited by mild coagulopathy and hypoalbuminemia.

ii) Patients are at high risk for intraoperative and postoperative hepatic dysfunction.

f) **Renal**

i) Chronic low cardiac output states lead to chronic kidney disease with electrolyte imbalances, platelet dysfunction, and hypervolemia.

3) **Intraoperative considerations**[6] **(Table 105-2)**

a) **VAD coordination**

i) Preoperative coordination with the LVAD team is imperative.

ii) The cardiothoracic surgery team, perfusionists and cardiac anesthesiologist, should all be aware of the case and available in the event of an emergency.

iii) LVAD-trained nursing personnel should be available at all times.

iv) Patient transport should be undertaken carefully and with adequate personal to assure hemodynamic stability and safety.

v) Adequate device battery life and power supply in the OR must be guaranteed.

vi) Anticoagulation with warfarin should be held prior to surgery, with a transition to heparin infusion to maintain therapeutic levels of anticoagulation.

vii) Availability of blood products should be assured prior to incision.

viii) With the risk of VAD infection, perioperative antibiotic prophylaxis is vital and care should be taken to minimize contamination.



In a patient with a VAD, careful transportation and positioning is crucial to avoid kinking of cannulas, hemodynamic instability, and interruption of power supply.

b) **Device management**

i) Optimization of device function is important for hemodynamic stability.

ii) Electromagnetic interference should be avoided.

iii) Bipolar cautery should be used and the grounding pad placed away from the VAD.

iv) The VAD device console should be clearly visible and in close proximity.

c) **Monitoring**[7]

i) Type and extent of surgery determine the need for invasive intraoperative monitoring.

ii) In patients with continuous flow VADs and minimal pulsatility, an arterial line is optimal to adequately assess blood pressure. Ultrasound can facilitate placement.

iii) Central venous access to monitor CVP and/or pulmonary artery catheter monitoring depends upon the type of surgery, comorbidities, risk of blood loss, and possibility of fluid shifts.

iv) Due to the lack of pulsatility, arterial waveforms show no or only narrow pulse pressures, and mean arterial pressures (MAPs) should be used as guide for systemic blood flow.

v) Pulse rate as measured by pulse oximetry or the arterial catheter reflects the VAD ejection rate and may differ from the native heart rate measured by the ECG.

TABLE 105-2 Preoperative Checklist

- VAD team notified and emergency contact available
- VAD-trained nursing personnel available
- Anticoagulation management discussed
- Plan for transport discussed with OR personnel
- Adequate perioperative power supply assured
- Defibrillator in the OR
- Emergency drugs available
- Blood products available
- Adequate vascular access and monitoring
- Place electric cautery away from device

vi) Transesophageal echocardiography (TEE) provides significant value for monitoring and management of VAD patients during the intraoperative period. Key imaging includes biventricular size and systolic function, intraventricular septal position, position and flow of VAD inflow- and outflow cannulas, AV opening, and search for intracardiac clot formation or device thrombosis.

vii) Monitoring of adequate end-organ perfusion by laboratory marker such as serum lactates is useful during long operative cases with significant fluid shifts.



Intraoperative echocardiographic monitoring should focus on RV size and systolic function, intraventricular septal position, and intermittent opening of the AV.

d) **Anesthetic plan**

i) Careful preoperative and intraoperative positioning should be done to avoid changes in VAD preload or compromise of inflow and outflow cannulas.

ii) Rapid sequence induction recommended due to increased aspiration risk.

iii) Induction goal should be maximal hemodynamic stability maintaining MAPs between 60 and 90 mm Hg.

iv) Rapid alterations in afterload during induction and maintenance of anesthesia should be avoided.

v) Judicious fluid resuscitation should be based on intraoperative fluid losses. Avoidance of volume overload is imperative due to the risk of acute RV failure.

vi) Criteria for postoperative extubation are similar to patients without VADs.

vii) The patient should be then closely monitored in the postanesthesia care unit (PACU) or the intensive care unit (ICU) by LVAD-trained personnel.

4) **Intraoperative complications**[8–10] **(see Table 105-3)**

a) **Hemodynamic instability**

i) Hemodynamic instability manifested with low or fluctuating device flows is a frequently encountered intraoperative complication.

TABLE 105-3 Intraoperative Complications

Problem		Etiology		Treatment
Low device flow	Normotensive/ hypertensive	↑ SVR	→	↓ SVR (deepen anesthesia, vasodilators)
	Hypotensive	Arrhythmias Hypovolemia RV failure	→ → →	ACLS protocol Give volume Increase RV function
Arrhythmias	Stable	Supraventricular	→	Rate control + pharmacologic conversion
	Instable	Ventricular	→	Cardioversion
Hypoxemia		Volume overload Inadequate LV Unloading intracardiac shunt	→ → →	↑ FiO_2 + diuresis + ↑ peep ↑ FiO_2 + ↓SVR + ↑ peep ↑ FiO_2 + ↑ RV function + ↑ device flow
Device failure		Power supply Pump failure	→ →	Battery backup Pneumatic hand pump Optimize native heart function
Bleeding			→	Blood products DDAVP

SVR, systemic vascular resistance; ACLS, advanced cardiac life support; DDAVP, 1-deamino-8-D-arginine vasopressin.

ii) Most common causes of hypotension are anesthesia-associated systemic vasodilation and decreased VAD output as a result of reduced VAD preload.
iii) Loss of vascular tone from sympathectomy should be counteracted by judicious use of vasopressors.
iv) Etiologies resulting in decreased VAD preload are hypovolemia, arrhythmias, and RV dysfunction.
   (1) "Chattering" is the clinically observed pulsation of inflow and outflow lines. This is the result of fluctuating device flow from insufficient VAD preload and intermittent suction events.
   (2) Hypotension as a result of hypovolemia should resolve with careful volume resuscitation.
v) If RV failure is the cause of instability, treatment becomes a challenging task.
   (1) Conservative maneuvers to lower pulmonary vascular resistance (PVR) and an increase in native heart rate promote RV forward flow. Avoidance of hypoxemia, hypercarbia, and acidosis is pertinent, and decreasing intrathoracic pressures by optimizing ventilatory settings and paralysis should be considered.
   (2) Maintenance of systemic blood pressures for RV perfusion is vital and vasopressors may be needed.
   (3) Chemical RV support is best achieved with inodilators such as phosphodiesterase inhibitors and β-adrenergic agonists, if hemodynamics allow.
   (4) Inhaled nitric oxide (iNO) is a potent selective pulmonary vasodilator and may be useful in cases of elevated PVR.
   (5) TEE for RV assessment and management guidance is very helpful in the acute setting.
vi) Less commonly encountered causes, such as inflow cannula obstruction, outflow graft kinking, or cardiac tamponade, should always be ruled out. These mechanical causes usually require surgical intervention.
vii) With continuous flow devices, significant increases in MAP secondary to sympathetic stimulation or iatrogenic drug administration can lead to decreased device output with subsequent end-organ malperfusion and intracardiac or device thrombus formation.

b) **Arrhythmias**
i) VAD patients are at high risk to develop supraventricular and ventricular arrhythmias.
ii) Preoperative consultation with the LVAD team should address risk of perioperative arrhythmias and management strategies.
iii) Intermittent arrhythmias are often tolerated in continuous-flow VADs. Hemodynamic instability with arrhythmias are the result of decrease in VAD preload and compromise of RV systolic function.
iv) Hemodynamic compromising arrhythmias should be treated by pharmacological or electrical termination. If arrhythmias are well tolerated, consultation with the primary VAD team prior intervention is recommended.
v) The VAD should not be stopped for electrical defibrillation or synchronized cardioversion. Both intervention may cause temporary fluctuating device flows, and device assessment afterward by the VAD team is recommended.



Mean arterial pressure does not always equal end-organ perfusion—differentiation of etiology and specific treatment of hypotension is key for the hemodynamic management of VAD patients.

c) **Hypoxemia**
i) VADs pose a unique risk for hypoxemia due to disruption of the natural cardiovascular anatomy and physiology.

ii) Pulmonary edema may result from hypervolemia or inadequate unloading of the left side of the heart.
(1) Hypervolemia should be treated with medical interventions such as diuresis or dialysis if needed.
(2) Inadequate ventricular unloading often does not respond to device output adjustments and treatment usually requires involvement of a cardiac anesthesiologist and cardiac surgeon.
iii) Admixture of oxygenated and deoxygenated blood secondary to a newly formed or previously unrecognized intracardiac right to left shunt may also lead to hypoxemia.
(1) Diagnosis of intracardiac shunts usually requires TEE for confirmation and intraoperative management is limited and often temporary.
(2) Immediate interventions include optimizing right-sided filling pressures by decreasing RV preload and decreasing RV afterload (PVR) to improve forward flow and mitigate right to left shunting.
(3) Advanced management options, such as adjustment of VAD settings in certain devices and interventional cardiac procedures to address intracardiac shunts, have less value in the acute perioperative setting and should be deferred to the primary VAD and cardiology team.

d) **Bleeding**
i) Hemodynamically significant bleeding should be treated as in the non-VAD patient.
ii) Recombinant factor VIIa/pharmacologic clotting adjuncts should be avoided unless discussed with the VAD team, due to thrombosis risk.

e) **ACLS**
i) Standard pharmacologic ACLS protocol measurements need to account for the unique VAD physiology; defibrillation is acceptable in most devices in case of emergencies.


Chest compression should be strictly avoided due to the risk of cannula dislodgement or disruption.

f) **Device failure**[11]
i) VAD failure can occur in any of the device components including controller, peripheral components such as driveline or batteries, and implantable pump.
ii) Current continuous flow devices lack a backup option, and the patient will be dependent on their native heart function until the failure can be fixed.
iii) In any case of device failure, needed circulatory support has to be provided by inotropes and prompt contact of the LVAD team is warranted to minimize the time without mechanical support.

5) **Postoperative considerations**
a) Decision on the location of immediate postoperative care should be made based on patients' comorbidities, surgical procedure, intraoperative course, and hemodynamic stability, and the provider should err on the safe side when deciding between floor and ICU needs.
b) Early postoperative complications are quite similar to the above-described intraoperative problems and may appear in the immediate perioperative period in the PACU or ICU.
c) Late complications include thromboembolic events, infection, and multiorgan system failure.

CHAPTER SUMMARY FOR VAD	
Definitions	Mechanical circulatory support (MCS) devices assume the role of the left, right, or both ventricles
Assessment	Preoperative assessment should include neurological status, device function/settings, volume status, and existing RV function
Risks	Patients are high-risk surgical candidates Risks of bleeding and thromboembolic events are elevated due to alteration in coagulation and platelet function in the perioperative period Hemodynamic instability is commonly caused by loss of VAD preload or vascular tone Do not do chest compression if VAD is in place
Monitoring	Assure adequate VAD function and power supply preoperatively Pulse oximetry may not work in continuous-flow devices Arterial line monitoring is necessary due to the lack of pulsatility VAD monitor provides pump flows/output Echocardiography offers significant value for the assessment of VAD function, intraoperative instability, and management options
Anesthetic Management	Maintain preload and avoid large changes in SVR Reduced pump flows/hypotension often responds to fluid bolus Maintaining RV function is essential—avoid hypoxia, hypercarbia, and acidosis

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106 Perioperative Transtransesophageal Echo

Jacob Evers • Nathaen Weitzel

Transesophageal echocardiography (TEE) is a monitoring tool used extensively in the field of anesthesiology due to its versatility, reliability, and safety. It was initially used as a diagnostic aid primarily by cardiologists but has become a mainstay in intraoperative cardiac anesthesia, and its utility is extending into a growing number of clinical areas. This chapter provides an overview of the indications and contraindications for TEE, the risks and complications associated with its use, as well as the current training and credentialing requirements.

1) **Perioperative management and guidance of cardiac surgery**
   a) TEE has become routine for the intraoperative management of open cardiac surgery. The use of TEE during cardiac surgery helps decide necessary interventions and assess the adequacy of surgical repairs as well as guide intraoperative management and diagnose complications.
   b) **Valvular repair**
      i) TEE is used to assess cardiac valves for abnormalities and diagnose the severity of valvular regurgitation and stenosis.
         (1) The intraoperative TEE exam can help the surgeon determine if a valvular repair is feasible or if replacement is necessary.
            (a) It is also crucial for evaluating the success of the procedure immediately following intervention.
      ii) Complications of valvular repair/replacement can be diagnosed immediately with TEE and direct additional surgical repair if necessary.
         (1) Complications include
            (a) New-onset stenosis or regurgitation
            (b) Perivalvular leak
            (c) Malpositioned valve or stuck valve leaflets
            (d) Obstruction of the left ventricular outflow tract due to systolic anterior motion of the mitral leaflet after mitral repair/replacement

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MV, Chapter 99

   c) **Aortic dissection**
      i) Emergent repair of an ascending aortic dissection requires accurate assessment of the location and extent of the dissection.
      ii) Iatrogenic dissection after aortic instrumentation, such as cannulation for cardiopulmonary bypass, can be promptly identified with TEE.
   d) **Congenital heart lesions**
      i) The anesthetic management of these patients can often be assisted by TEE.
         (1) This is primarily necessary in the neonatal and pediatric population where detailed information about the anomalous cardiac anatomy is required.
         (2) The population of adults with congenital heart disease, repaired or uncorrected, is growing.

e) **Endocarditis repair**
   i) TEE exam of patients with suspected endocarditis can play a major role in the surgical plan.
      (1) The sensitivity for detection of valvular vegetations for TTE is 60%-65% compared to 85%-98% for TEE.[1]
f) **Minimally invasive procedures**
   i) During minimally invasive interventional cardiac procedures such as transcatheter aortic valve replacement, TEE can help guide placement and assess for complications including perivalvular leak.
   ii) TEE is useful in guiding the placement of cardiovascular support devices such as ECMO cannulas and percutaneous LVAD devices.
g) **Pericardial window procedures**
   i) Pericardial effusion and cardiac tamponade are easily identified on TEE and a thorough exam can help guide drainage procedures.
   ii) Posterior or loculated pericardial effusions may be missed by the surgeon but are identified by TEE.
h) **Aortic cross-clamp**
   i) Cross-clamping of the aorta is frequently necessary as part of cardiopulmonary bypass.
      (1) Calcification or atheroma of the ascending aorta can be dislodged by the cross-clamp placing the patient at risk of embolic stroke.
      (2) Accurate assessment of the aorta by TEE prior to cross-clamp helps reduce this risk and allows the surgeon to place the clamp at a safe location.[2]

2) **Other indications**
   a) **Rescue TEE**
      i) During noncardiac surgery, TEE can be emergently placed to aid in the diagnosis and management of unexpected hemodynamic instability in which ventricular function, volume status, or other factor are indeterminate.
      ii) Refractory hypotension is an intraoperative complication that every anesthesiologist encounters on a regular basis, but the cause is not always clear.
         (1) All sources of hypotension must be detected early and treated aggressively.
      iii) Left ventricular end diastolic volume can be determined, providing an accurate assessment of volume status.[3]
      iv) Wall motion abnormalities resulting from coronary ischemia can be detected before EKG changes are evident.[4]
         (1) The transgastric short-axis view at the mid-papillary level (**Fig. 106-1**) allows for simultaneous monitoring of myocardium supplied by the left anterior descending, circumflex and right coronary arteries making it useful for the rapid evaluation of ischemia.[5]

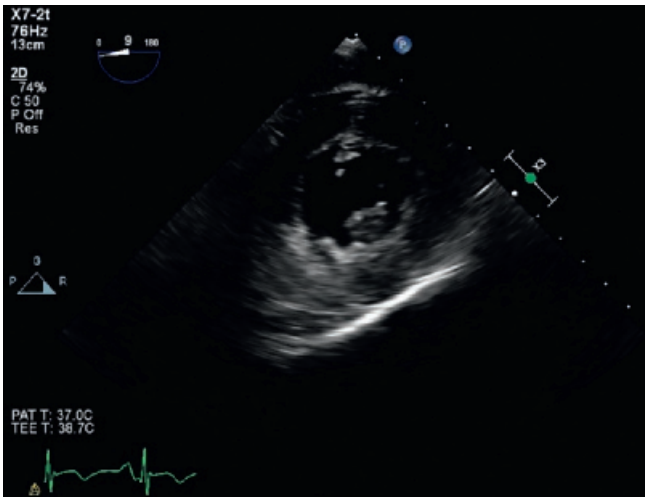


In the setting of refractory hypotension without a clear underlying cause, TEE is an appropriate and effective tool that can help diagnose and guide therapy (**Table 106-1**).

TABLE 106-1 Sources of Hypotension Identified on TEE

1. Hypovolemia
2. Myocardial dysfunction
3. Valvular abnormalities
4. Left ventricular outflow obstruction
5. Cardiac tamponade
6. Pulmonary embolism
7. Ventricular septal defect
8. Aortic dissection

FIGURE 106-1 Transgastric Short-Axis Mid-Papillary View of the Left Ventricle.



b) **Management of the ICU patient**
   i) TEE is commonly used to guide therapy in the critically ill patient, but TEE offers advantages over TTE in a variety of scenarios:
      (1) Mechanical ventilation
      (2) Cardiac tamponade
      (3) Diagnosis of vegetations and complications of endocarditis
      (4) Central pulmonary emboli
      (5) Exclusion of a cardiac source of embolism
      (6) Evaluation of a mediastinal hematoma
      (7) Diagnosis of ascending or descending thoracic aortic dissection
      (8) Structural and functional evaluation of native valves
      (9) Acute hemodynamic instability
   ii) TEE may also be left in place for several hours, allowing the intensivist to assess the patient's response to therapy.
      (1) Hemodynamic TEE (hTEE) comprises a small disposable TEE probe that can be left in place for up 3 days and may be useful in the critical care setting for patients requiring repeated exams.
   iii) TEE offers additional benefit above the capability of a pulmonary artery catheter.
      (1) PA catheters rely on occlusion pressures to estimate LV volume.
         (a) This value can be affected by things such as PEEP, mitral valve abnormalities, and changes to LV compliance leading to over or underestimated data.[6]

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Pulmonary artery catheter, Chapter 13

c) **Assessment of intracardiac masses and embolic sources**
   i) TTE can falsely identify normal intracardiac structures such as a pectinate muscle or Coumadin ridge as a thrombus or tumor.
   ii) TEE provides further information to accurately diagnose intracardiac pathology, which may help to guide therapy.[6]

3) **Contraindications**
   a) There is some debate about the classification of certain scenarios as relative or absolute contraindications.
      i) Ultimately, the clinician must take into consideration the entire clinical situation and weigh the potential risks and benefits of TEE.

Contraindications to TEE Placement	
Absolute	**Relative**
1. Esophageal spasm	1. Unstable neck injuries
2. Stricture	2. Esophageal trauma
3. Laceration	3. Varices
4. Perforation	4. Dysphagia
5. Diverticula	5. Active upper GI bleeding
	6. History chest radiation
	7. Hiatal hernia[7–9]

4) **Risks and complications**
   a) TEE complications are rare, especially in the hands of a trained clinician, but there are risks associated with the modality.[7–10]
      i) Incorrect interpretation
         (1) Can lead to improper surgical or medical interventions just as proper interpretation can correctly guide therapy.
      ii) Probe placement can result in
         (1) Oral trauma
         (2) Pharyngeal, esophageal, or gastric perforation
         (3) Dysphagia
         (4) Esophageal or gastric bleeding
         (5) Thermal injury
         (6) Transient bacteremia
         (7) Displacement of the endotracheal tube
         (8) Hemodynamic changes



Esophageal spasm, stricture, laceration, perforation, and diverticular are all absolute contraindications to TEE.

   b) **Avoid injury**
      i) To avoid oropharyngeal trauma, probe placement must be done by a skilled clinician using the proper technique.
         (1) Generous lubrication with ultrasonic gel is placed in the mouth, and the probe is then guided to the posterior oropharynx.
            (a) A bite block is commonly used to prevent dental trauma and damage to the TEE probe.
         (2) A jaw lift followed by gentle posterior and caudal pressure on the probe usually results in a loss of resistance as it passes through the upper esophageal sphincter.
         (3) When resistance is experienced, it is best to redirect the probe rather than force it in the same direction.
            (a) If resistance is still encountered, aborting the procedure should be considered.
         (4) With adequate depths of anesthesia, a direct laryngoscopy can help displace oropharyngeal structures and assist with probe placement.

5) **Equipment**
   a) **TEE probe**
      i) The TEE probe consists of an ultrasound transducer, which is fixed at one end of a flexible rod.
      ii) The handle consists of a series of controls allowing manipulation of the transducer in a variety of planes.
      iii) The probe can be retro- and anteflexed, flexed to the right or the left, turned right or left, advanced and withdrawn and rotated 180 degrees.

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Performing a TEE examination, Chapter 206.

b) **TEE transducer**
   i) Functions by transmitting ultrasound through the esophagus and into the heart.
      (1) These ultrasound waves are then reflected back to the transducer.
         (a) Because the speed of sound is constant in tissue, the time it takes for the sound waves to return to the transducer from different structures is extrapolated into a visual image in real time.[6]



The Doppler effect states that ultrasound waves directed at a moving object will compress as the object moves toward the source and expand as it moves away.

c) **Color flow Doppler**
   i) It is based on the principle of the Doppler effect, which states that the ultrasound waves, directed at a moving object (in this case, red blood cells), will be compressed as the object moves toward the source and expanded as it moves away.
   ii) The color flow Doppler function typically assigns the color red to ultrasound waves returned at a higher frequency, that is, blood flow toward the transducer and blue to waves returned at a lower frequency, that is, blood flow away from the transducer.
      (1) This allows for analysis of the severity of valvular disease, detection of intracardiac shunts, identification of the true and false lumens of an aortic dissection, and many others.[6]



BART, Blue away, Red toward. An easy way to remember the direction of blood flow of color flow Doppler.

d) **Pulsed and continuous wave Doppler**
   i) Pulsed wave Doppler utilizes a single vibrating crystal to emit an ultrasound wave and focus it on a specific point of interest in the 2D image and calculate blood velocity at that point.
   ii) Continuous wave Doppler differs in that it uses one crystal to continuously emit an ultrasound signal and another to continuously receive signal.
      (1) This modality is used to measure blood flow velocity along the entire path of the ultrasound beam and is useful in identifying the highest velocity such as for a stenotic valve.

e) **Tissue Doppler**
   i) This uses the same Doppler principles and applies it to the myocardium itself.
      (1) By measuring low velocity signals, this modality allows for assessment of myocardial movement and contractility.

f) **3D echocardiography**
   i) 3D imaging has rapidly emerged as a useful tool in TEE imaging.
      (1) Gated reconstruction
         (a) A series of 2D images obtained simultaneously, which are then reconstructed to produce a 3D image.
      (2) Real-time 3D
         (a) Requires a special transducer that emits a pyramidal shaped sector and produces a real-time 3D image.[6]

g) **Strain echocardiography**
   i) Strain imaging is an emerging postprocessing TEE feature that measures the deformation, or change in length, of myocardium during the cardiac cycle.
      (1) Strain imaging can be used to assess regional myocardial function or provide an objective measure of global ventricular function.[11]

6) **Credentialing**
   a) **National Board of Echocardiography (NBE)**
      i) Began offering board certification in TEE in 2004 as a way to recognize those clinicians completing special training in intraoperative TEE.

b) **PTEeXAM**
   i) The Special Competence in Perioperative Transesophageal Echocardiography (PTEeXAM) is offered once a year for eligible physicians.
   ii) Basic and advanced TEE exams are offered with the basic exam focused on intraoperative monitoring and the advanced exam focused on utilizing TEE for diagnostic purposes including the direction of perioperative decision-making.[12]

c) **Board certification**
   i) Previously two pathways for certification. The practice pathway was eliminated and now certification is obtained only after an additional year of advanced cardiac training.
      (1) Training program pathway
         (a) Available for physicians who have completed or are currently enrolled in ACGME accredited cardiothoracic anesthesia fellowship programs.
            (i) Requirements
               1. Medical licensure
               2. Anesthesiology board certification
               3. A passing score on the Advanced PTEeXAM
               4. 300 complete transesophageal echocardiograms under direct supervision
                  a. 150 of these must be performed, interpreted, and reported by the trainee directly.

d) **Testamur**
   i) Physicians who do not qualify for board certification may still take the PTEeXAM and with a passing score, obtain the title of "testamur."

CHAPTER SUMMARY FOR TEE AS A MONITORING TOOL

Indications	Assessment of hemodynamic instability, guidance of cardiac surgical therapy, evaluation of intracardiac masses as well as others
Contraindications	Absolute contraindications include esophageal spasm, stricture, laceration, perforation, and diverticula. All others are relative and the risks and benefits must be weighed by the clinician
Risks and Complications	Rare but include oropharyngeal and esophageal trauma and postoperative dysphagia
Equipment	2D TEE includes a semiflexible probe, ultrasound source, and hand controls. Multiple modalities are available to acquire specific anatomical and physiologic information
Credentialing	Board certification is available through the NBE and requires a passing score on the PTEeXAM as well as advanced training and completion of supervised exams

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107 Obstetrical Anesthesia Overview

Philip Rubin

Obstetrical anesthesia is a rewarding and dynamic specialty requiring **constant communication** among nurses, midwives, obstetricians, and anesthesiologists. Although some patients prefer to avoid medications as part of their birth plan, all patients admitted to the labor ward could require anesthetic intervention. Thus, the anesthesia provider must be cognizant of all patients on the labor ward, clearly explain the risks and benefits of any treatment, all the while respecting and honoring patient's wishes.

1) **Common obstetrical (OB) nomenclature**
   The description of a patient will often include abbreviations, the knowledge of which is important to understanding a patient's obstetric history.
   a) **G refers to Gravida**, which is the number of times the patient has been pregnant.
   b) **P refers to Parity** and is often subclassified with the mnemonic FPAL.
      i) F is the number of full-term births.
      ii) P is the number of preterm births.
      iii) A is the number of abortions, whether spontaneous or induced.
      iv) L is the number of living children.
   c) For example, a G3P1112 patient has been pregnant three times, with one full-term birth, one preterm birth, one abortion, and two living children.



It is important to fully understand the physiologic changes of pregnancy in order to appropriately care for a pregnant woman.

2) **Physiologic changes of pregnancy (Table 107-1)**
   a) **Pregnancy significantly alters physiology, which dramatically affects the perioperative management of these patients.**
3) **Normal stages of labor**[1]
   a) **First stage:** Onset of labor to complete cervical dilation
      i) Pain arises in peripheral nociceptors in the lower uterine segment and cervix and travels in visceral afferent neurons, which enter the neuraxis at T10-L1.
   b) **Second stage:** Pushing and delivery of baby
      i) Pain is predominantly from the perineum and travels in somatic afferent fibers in the pudendal nerve that enter the neuraxis at S2-S4.
   c) **Third stage:** Placental delivery
4) **Analgesia for a laboring patient anticipating a vaginal delivery**
   a) **Nonpharmacologic methods for relieving labor pain**
      i) Success varies with each individual's experience, cultural beliefs, and labor pattern.
      ii) Include Lamaze, transcutaneous electrical stimulation, relaxation training, meditation, massage, water baths, and emotional support.
   b) **Nitrous oxide**
      i) Analgesic and anxiolytic
      ii) Inhibits NMDA, potentiates neurotransmitters such as GABA and glycine.
      iii) Historically used for labor analgesia outside the United States, now becoming increasingly available in the United States as a noninvasive alternative to neuraxial anesthesia.
      iv) Requires patient participation and proper coordination with contractions. Administered as a fixed 50% O_2/N_2O mixture. Mask inhalation with a good seal is

TABLE 107-1 Physiologic Changes of Pregnancy

System	Physiologic Change	Anesthetic Implication
CNS	MAC ↓ by up to 40% ↑ sensitivity to local anesthetics ↓ CSF volume, distention of epidural veins, ↑ pressure in epidural space	↓ anesthetic requirement ↓ doses required, ↑ risk of toxicity ↑ risk of intravascular catheter placement with attempted epidural
Airway	Capillary engorgement and edema	↑ potential for difficult airway ↓ consider endotracheal tube size (6-6.5 mm)
Respiratory	↑ oxygen consumption, ↓ FRC ↑ minute ventilation	Very rapid desaturation with apnea or induction of GA ↓ $PaCO_2$ to ~33 mm Hg by third trimester
Cardiovascular	↑ cardiac output by 40% by end of first trimester and to >80% above prelabor values after delivery (due to autotransfusion of uterine blood) ↓ SVR Aortocaval compression due to uterine enlargement	Potential for decompensation in patients with preexisting cardiac disease, especially immediately following delivery ↓ arterial BP Hypotension with supine position; never position patients >18-20 wk supine without displacement of the uterus (15-30 degrees)
Hematologic	↑ blood volume; ↑ red cell mass (disproportionate to blood volume) ↑ coagulability	Ability to tolerate blood loss at delivery; "physiologic" anemia of pregnancy Hypercoagulability helps reduce blood loss at delivery but may also place patients at risk for DVT/PE and other clotting problems.
Gastrointestinal	↓ gastric emptying ↓ lower esophageal sphincter tone	↑ risk of pulmonary aspiration of gastric contents
Musculoskeletal	↑ lumbar lordosis Widened pelvis	↓ interspinous spaces may make neuraxial procedures more difficult
Genitourinary	↑ glomerular filtration rate	↓ creatinine (0.4-0.6 mg/dL)

BP, blood pressure; CSF, cerebrospinal fluid; DVT, deep venous thrombosis; FRC, functional residual capacity; GA, general anesthesiology; MAC, minimum alveolar concentration; PE, pulmonary embolism; SVR, systemic vascular resistance.

required to open the demand valve, which subsequently closes with exhalation. Gases are removed via a scavenging system.[2]

v) Side effects include drowsiness, nausea, light-headedness.

vi) Efficacy dependent on patient expectations, sometimes influenced by provider or birthing center culture. While N_2O is sufficient for some, its inferiority to neuraxial labor analgesia is well documented.[2]

c) **Systemic pain medications**

i) Commonly administered when neuraxial analgesia is either undesired, unavailable, difficult to place, nonfunctional, or contraindicated.

ii) Opioid medications are the most widely used.

iii) Do not cause lower extremity motor weakness.

iv) Intravenous (IV) route preferred due to the rapid, predictable, onset.

v) Higher doses of IV opioids are usually required as labor progresses.

vi) Maternal side effects of opioids include respiratory depression, postural hypotension, urinary retention, sedation, constipation, nausea, vomiting, and itching.



Opioid medications are a viable option for labor analgesia, but do not completely relieve labor pain in safe doses.

vii) Opioids cross the placenta and may lead to decreased fetal heart rate (FHR) variability.[1]
   (1) Fentanyl
      (a) Rapid onset of analgesia
      (b) Preferred agent for labor and delivery
      (c) IV dose is 50-100 µg
      (d) Peak effect is 3-5 minutes and duration is 30-60 minutes.
      (e) If possible, avoid administration within 2 hours of delivery, due to the risk of neonatal respiration depression.
      (f) Fentanyl patient-controlled analgesia (PCA) is an effective option for patients who cannot receive neuraxial analgesia.
         (i) PCA demand doses range from 10 to 50 µg with a lockout period of 8-20 minutes.
   (2) Meperidine has been widely used in obstetrics.
      (a) The dosage is 25-50 mg IV with a peak effect 5-10 minutes and duration of 3-4 hours.
      (b) Rapidly crosses the placenta.
      (c) Both meperidine and its active metabolite, normeperidine, are present in the neonate for long periods (up to 3-6 days), increasing the risk of respiratory depression and neurobehavioral changes.
   (3) Morphine
      (a) Causes more neonatal depression than other agents and is a less effective analgesic at clinically safe doses.
      (b) Not a recommended agent
   (4) Remifentanil PCA
      (a) Ultra-short acting analgesic with a rapid onset and offset.
      (b) Caution with basal rates when using as a PCA, as this may increase risk of maternal and neonatal respiratory depression
      (c) Administration to be timed with contractions. Demand dose of 0.25 µg/kg with lockout of 2 minutes
      (d) Requires more frequent monitoring for maternal sedation, oxygen desaturation, and respiratory depression.[3]



Every epidural catheter in a laboring patient should be assessed regularly for adequate labor analgesia, which is the best indicator of its successful use for cesarean delivery, if necessary.

d) **Neuraxial analgesia for vaginal delivery**
   i) Provides the best pain relief and highest level of patient satisfaction of all options for pain management during labor (**Table 107-3**).
   ii) The goal is to provide effective analgesia while minimizing lower extremity motor block that may limit expulsive efforts during the second stage of labor.[4]
   iii) If cesarean delivery (CD) becomes necessary, a properly functioning epidural catheter can be bolused for rapid conversion to surgical anesthesia.
   iv) Epidural labor analgesia
      (1) The epidural catheter is advanced into epidural space via Tuohy needle after loss of resistance (LOR) obtained.
      (2) The catheter is aspirated with empty syringe to assess for potential intrathecal (CSF) or intravascular (blood) placement, however not all instances are identified with this practice.
      (3) After negative aspiration, some centers then administer a "test dose" bolus, which is typically local anesthetic + epinephrine, also to assess for potential intrathecal or intravascular placement.
         (a) A lower extremity motor block indicates an inadvertent intrathecal catheter.
         (b) In the absence of a contraction an increase in HR ≥20-30% ± tinnitus/circumoral numbness/metallic taste indicates an intravascular catheter.

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(c) This technique is controversial, as it lacks sensitivity and specificity.[5]
(d) Therefore, some centers simply consider every dose administered as a "test dose," as they assess for these concerns routinely after each epidural bolus.

(4) Epidural analgesia is initiated with local anesthetic ± opioid bolus. An infusion is then started with titratable basal rate ± demand settings (PCEA, patient controlled epidural analgesia), or via PIEB (programmed intermittent epidural boluses).[6]

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v) Combined spinal epidural (CSE) labor analgesia
(1) Spinal needle is inserted through the Tuohy needle after LOR. Local anesthetic and/or opioid injected and needle retracted. Epidural catheter then advanced into epidural space via Tuohy needle.
(2) Advantages
(a) Rapid onset of analgesia, decreased need for boluses.
(b) Decreased likelihood of catheter failure. CSF flow from the spinal needle may confirm appropriate epidural needle position and subsequent catheter placement.[7]
(3) Patients likely to benefit from CSE
(a) Late in labor, especially multiparous women
(b) Those with a history of short labor
(c) Repeat blocks due to nonfunctioning epidural catheters
(4) Disadvantages
(a) When a local anesthetic is injected with a CSE technique, it may not be possible to test the epidural catheter functionality until a later time, when the effect of the spinal dose wears off.
(i) It may be prudent to avoid CSE when the potential for an emergent CD is high (eg, unfavorable fetal heart tone tracings, bradycardia).
(ii) Patients with a known or anticipated difficult airway may not be good candidates for CSE.



A CSE is an excellent option for labor analgesia, especially in multiparous women late in labor or those with a history of short labor.

vi) Dural puncture epidural (DPE)
(1) Same as CSE technique, but without intrathecal injection
(2) Useful as a "confirmatory" test similar to CSE when LOR is questionable. The presence of CSF may provide reassurance that the LOR is truly within the epidural space.
(3) Local anesthetic boluses can cross the dural puncture into the CSF, speeding block onset and improving sacral spread.[8]

vii) The choice of technique depends on many factors, including provider preference and expertise, potential for difficult airway, maternal comorbidities, and progress of the patient's labor (cervical dilation and pain level).

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5) **Instrumented vaginal delivery**
a) **Accomplished with vacuum extraction or forceps**
b) **Requires excellent perineal anesthesia**
c) **Frequently requires concentrated local anesthetic (Table 107-3)**
i) If an epidural catheter is not present, a spinal injection (preferably via the CSE technique in case CD becomes necessary) can be performed and dosed according to **Table 107-3**.
d) The anesthesia provider should **remain present (or immediately available) during delivery** in the event of an emergent CD, additional medication requirements, neonatal resuscitation, or bleeding.

TABLE 107-2 Considerations for Cesarean Delivery

Surgical Phase	Events	Anesthetic Considerations
Phase I	Administration of anesthesia, surgical incision, and dissection to uterus	Choice of RA or GA based on clinical situation (RA preferred) Hemodynamic stability and adequate anesthetic level are primary concerns Communication with obstetrician, patient, and support person essential
Phase II	Uterine incision and delivery of fetus	If RA, notify patient of imminent delivery; observe uterine incision and delivery; monitor placental extraction and the surgical field for adequacy of uterine tone and signs of bleeding
Phase III	Hemostasis and closure	Administer uterotonic agents (eg, oxytocin ± methylergonovine or carboprost tromethamine) if obstetrician requests; vigilantly observe and communicate with surgeon about adequacy of uterine tone and bleeding concerns; patient may spend time bonding with baby; consider postoperative pain management plans

6) **Anesthesia for cesarean delivery (CD) (Table 107-2)**
   a) Regardless of anesthetic technique, CD can be categorized into three distinct phases.
      i) Phase I: Initiation of anesthetic, surgical incision, and uterine exposure
      ii) Phase II: Uterine incision and delivery of the fetus
      iii) Phase III: Hemostasis and closure



If an instrumented vaginal delivery is unsuccessful, the patient may require immediate cesarean delivery. An indwelling epidural catheter can be utilized if this occurs.

7) **Regional anesthesia (RA) for cesarean delivery (CD)**
   a) **RA is the preferred anesthetic for CD for the following reasons:**
      i) Due to the physiologic changes of pregnancy, all parturients are considered a full stomach and are at increased risk for aspiration of gastric contents.
      ii) The incidence of difficult airway is eight times higher during pregnancy than in the general population.
      iii) Anesthetic mortality with general anesthesia (GA) has previously been reported to be 16 times higher than with RA.[9,10]
         (1) Maternal mortality has decreased significantly with the use of RA as well as with improvements in airway management.
         (2) Prevalence of morbidity and mortality with GA still remains higher than with RA, but the overall number of malpractice claims is higher with RA because of its widespread use in obstetrics.[11]
      iv) Maternal satisfaction is higher with RA because the patient is awake for delivery and able to bond with her baby immediately.
      v) Postoperative pain management is improved with the use of long acting neuraxial opioids (eg, preservative-free morphine) administered intrathecally or via an epidural catheter.



The majority of elective cesarean deliveries in the United States are performed with spinal anesthesia.

   b) If RA is used, achieving a T4 sensory level is optimal for effective surgical anesthesia. **Table 107-3 contains suggested medications and dosage.**
   c) Always assess and document adequate surgical anesthesia (bilaterally) prior to incision. A pinprick or cold object (eg, alcohol swab) may be used, testing in

TABLE 107-3 Suggested Analgesic/Anesthetic Regimens for Obstetrics[1,2]

Clinical Scenario	Anesthetic Regimen	Comments/Side Effects
Labor epidural	**Initiation:** 10-15 mL bupivacaine 0.125% + fentanyl 50-100 µg or sufentanil 5-10 µg **Maintenance infusions:** Bupivacaine 0.0625%, 10-15 mL/h Bupivacaine 0.125%, 6-10 mL/h Ropivacaine 0.1%-0.2%, 6-10 mL/h **Added opioid:** Fentanyl 2 µg/mL or sufentanil 0.3-0.4 µg/mL **Avoid** lidocaine due to increased motor blockade **PCEA:** above infusions with boluses of 4-12 mL every 10-20 min, depending on concentration **PIEB:** 5-10 mL every 30-60 min	Lower concentrations and higher volumes of local anesthetics preferred Side effects include motor block, urinary retention, nausea/vomiting, hypotension, pruritus, and respiratory depression (if narcotics are added)
Labor CSE	Intrathecal: Fentanyl 15-25 µg or Sufentanil 2.5-5 µg ±Bupivacaine 1.5-2.5 mg	Side effects are the same as epidural Higher doses associated with fetal bradycardia via unclear mechanism; probably uterine hypertonus secondary to decreased beta-agonism (from sudden drop in catecholamines) Narcotic-only techniques do not cause motor block
Instrumented vaginal delivery	**Epidural**: 5-15 mL lidocaine 1%-2%, or 5-15 mL of 2-chloroprocaine 2%-3% **Spinal**: 2.5-5 mg bupivacaine plus 15-25 µg fentanyl	Cesarean delivery may be necessary if instrumentation fails
Cesarean delivery in a patient with functioning labor epidural	10-20 mL of 2% lidocaine with 1 mEq/10 mL HCO_3 and 1:200 000 epinephrine) 10-20 mL of 3% 2-chloroprocaine	For postoperative analgesia, add epidural preservative-free morphine 3 mg before removing the catheter Chloroprocaine may decrease effectiveness of epidurally administered morphine; may extend block with lidocaine if necessary
Spinal anesthesia for cesarean delivery (no labor epidural)	Bupivacaine 12 mg (use 0.5% isobaric or 0.75% hyperbaric) + Fentanyl 10-15 µg	For postoperative analgesia, add intrathecal preservative-free morphine 100-200 µg
Epidural anesthesia for cesarean delivery (no labor epidural)	20-25 mL of 2% lidocaine with 1 mEq/10 mL HCO_3 and 1:200 000 epinephrine; or 0.5% bupivacaine, 0.5% ropivacaine, 3% 2-chloroprocaine + Fentanyl 75-100 µg or sufentanil 10-25 µg	For postoperative analgesia, add epidural morphine-free morphine 3 mg

a cephalad direction. NOTE: pain may lag temperature loss by up to two sensory levels, so it may not be adequate, for example, to proceed with only a T5 level to cold sensation.

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d) **Spinal anesthesia**
   i) Advantages
      (1) Rapid onset of dense surgical anesthesia
      (2) Usually quick to perform and can be used for urgent delivery
      (3) Low doses of medication are required, which have minimal fetal effects.

ii) Disadvantages
(1) Hypotension
(a) Can be minimized by rapid administration of crystalloid during (coloading) or immediately following block placement and with vasopressors (see below).[12]
(2) Inability to supplement neuraxial medications if anesthesia is inadequate, or if the duration of surgery outlasts spinal duration.

e) **Epidural anesthesia**
i) Advantages
(1) Catheter is in place for the duration of surgery.
(2) May be used as a patient controlled epidural analgesia (PCEA) for postoperative pain management.
(3) If anesthetic level is inadequate, the epidural catheter can be redosed and/or manipulated to improve anesthesia or extend duration.
(4) Hemodynamic changes may be slower to take effect, which may be preferred in patients who may not tolerate acute hypotension (eg, patients with severe aortic stenosis).

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ii) Disadvantages
(1) Slower onset
(2) Less dense block possible
(3) Placement can be more time consuming.

f) **CSE Anesthesia**
i) Combines the advantages and the disadvantages of both spinal and epidural techniques.
ii) Examples of situations where CSE may be indicated include:
(1) Patients who would benefit from the rapid onset of spinal medication but in whom the best dose is unclear (ie, extreme short stature, morbid obesity).
(2) Patients at risk for longer surgical duration (eg, a patient undergoing her third-fourth CD, or with a history of gynecological procedures), all of which lead to scaring/adhesions.

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g) **Hypotension during RA** for CD is common and should be treated aggressively, as there is no autoregulation in the uterus.
i) Typically manifests as nausea and vomiting
ii) Treatment
(1) Ensure adequate left uterine displacement to lessen aortocaval compression.
(2) Ensure adequate intravascular volume.
(3) Pharmacologic treatment includes phenylephrine (50-100 µg IV) as a first-line medication if the heart rate is adequate, otherwise ephedrine (5-10 mg IV).
(a) Due to concerns about neonatal metabolic acidosis with large doses of ephedrine, phenylephrine is the preferred agent.[4]
(b) Initiation of phenylephrine infusions postspinal (eg, 0.5 µg/kg/min) may help maintain hemodynamic stability.
(c) Reflex bradycardia may occur with phenylephrine administration and can be successfully treated with glycopyrrolate (0.2-0.4 mg IV).



In a parturient who has a spinal anesthetic for cesarean delivery, a complaint of nausea is a manifestation of hypotension until proven otherwise.

iii) Rarely, severe bradycardia and even cardiac arrest can occur after spinal anesthesia.
(1) Treat rapidly with atropine starting at 0.2-0.3 mg for severe bradycardia, up to 1 mg IV for cardiac arrest and/or epinephrine 0.2-0.3 mg, escalating to full resuscitation doses if necessary.[13]

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FIGURE 107-1 Suggested Procedure for General Anesthesia for Cesarean Delivery.

Ensure well functioning **IV line**

Consider administration of non-particulate antacid and/or H_2-receptor antagonist

Place patient in the supine position with **left uterine displacement**

Apply standard ASA **monitors**

Have surgeons prepped, draped and ready for surgery

Preoxygenate, ideally for 3-5 minutes

Have an assistant apply **cricoid pressure (when appropriate)**

Administer induction agent (propofol 2 mg/kg, etomidate 0.2-0.3 mg/kg) followed by succinylcholine (1-1.5 mg/kg) with **rapid sequence induction**

Intubate the trachea with a cuffed endotracheal tube

Confirm placement of the endotracheal tube with $ETCO_2$ and/or bilateral breath sounds

Inform the surgeons that the airway is secure and that they may begin surgery

Administer 100% oxygen plus 0.5-1 MAC of halogenated agent

Once baby is delivered, administer 50% nitrous oxide and 50% oxygen, **decrease volatile agent to <0.5 MAC, and consider adding opioids and/or muscle relaxants as necessary**

Administer **uterine contractile agent** (usually oxytocin infusion)

Closely monitor uterine tone and bleeding. Additional uterotonic agents may be necessary

Administer antibiotics as requested by the surgeon (may be done prior to incision)

Extubate the patient fully awake

8) **General anesthesia for cesarean delivery (Fig. 107-1)**
   a) GA in obstetrics can be anxiety provoking but may be necessary when time does not permit spinal placement or extension of epidural anesthesia due to emergent conditions. Occasionally GA may be required due to failure of regional technique or when it is contraindicated.

FIGURE 107-2 Considerations for Aspiration Precautions

- **Nonparticulate antacid (such as 30 mL sodium bicitrate PO)**
- **Histamine$_2$ (H_2) receptor antagonist (ranitidine 50 mg or famotidine 20 mg IV) preferably given at least 30 minutes prior to procedure**
- **Gastric motility agent (metoclopramide 10 mg IV)**
- **Rapid sequence induction**
- **Cricoid pressure**
- **Endotracheal intubation**
- **Awake extubation with airway reflexes intact**

Appropriate for all patients after 12 weeks of gestation and immediately postpartum.[1,2]

b) Constant communication among members of the care team is imperative, especially when deciding on the choice of anesthetic.
c) Aspiration precautions (**Fig. 107-2**) should be taken.
d) It is important to remember that all volatile anesthetics decrease uterine tone and can increase bleeding, so uterine tone must be carefully observed. Minimizing uterine atony postdelivery can be accomplished by decreasing the concentration of volatile anesthetics and supplementing with nitrous oxide to maintain an adequate depth of anesthesia.
e) MAC is decreased in pregnancy, so volatile anesthetic requirements may be reduced for this reason.
f) Postoperative pain can be managed with an IV PCA if neuraxial opioids (eg, preservative-free morphine) are not administered.
   i) Consider multimodal therapy with NSAIDs and acetaminophen.
   ii) Bilateral transversus abdominis plane blocks (TAP) can be placed postoperatively to provide analgesia for incisional pain.



For optimal airway management in the parturient, consider building a "ramp" to elevate the head and shoulders so that the external auditory meatus is at the level of the sternal notch.

9) **Other pregnancy-related procedures**
   a) **Cervical cerclage is performed for cervical incompetence, especially in patients with a history of previous pregnancy loss.**
      i) Due to the physiologic changes of pregnancy, RA is preferred.
      ii) A patient with a dilated cervix and bulging membranes who cannot assume the upright or the lateral position may require GA (with aspiration precautions).
      iii) It is important to speak with the patient's obstetrician ahead of time in order to prepare for the safest anesthetic technique based on the clinical scenario.
      iv) If RA is used, the sensory level should be T8-T10.
         (1) For spinal anesthesia, 7.5 mg of bupivacaine or 60 mg 5% lidocaine with 10-25 µg fentanyl is recommended.[1]
         (2) If an epidural is used, ~12-15 mL of 2% lidocaine with 50-75 µg fentanyl is typically adequate.[1]



When performing GA for an urgent cesarean delivery, clear communication with the obstetricians is essential, so they do not incise before the patient's airway is secure.

   b) External cephalic version (ECV): Procedure to rotate breech presentation fetus to vertex position in an attempt to avoid cesarean delivery
      i) Typically offered at 36-37 weeks of gestation
      ii) Maternal pain is a common reason to abort ECV

iii) ECV success is superior with neuraxial anesthesia than without, perhaps by relaxing abdominal wall muscles, and reducing patient guarding.
(1) In most recent meta-analysis, ECV success was 58% and CD rate was 46% with neuraxial anesthesia compared to without, 43% and 55%, respectively.[14]
(2) Spinal, epidural, or CSE techniques may be used.
(3) ECV success does not improve with spinal bupivacaine doses >2.5 mg.[15]
(4) A CSE technique allows for extension to a surgical level if CD becomes necessary.



Large doses of oxytocin postdelivery should never be bolused IV, as hemodynamically significant hypotension, tachycardia, arrhythmias, and flushing may occur.

c) **Emergent hysterectomy**: In the event of postpartum hemorrhage, hysterectomy may become necessary to control bleeding. Rarely, a hysterectomy may be done electively at CD for medical reasons.
i) RA is still preferred over GA in the absence of extreme hemodynamic instability and/or coagulation abnormalities.[1]
ii) If massive hemorrhage occurs and resuscitation is required, airway difficulty could arise due to increased edema. Early intubation in the setting of an ongoing resuscitation is preferable.[16]

d) **Postpartum tubal ligation**: Tubal ligation may be easily performed in the first few days postpartum.
i) It is unknown when the risk of gastric aspiration reaches prepartum levels.
(1) These patients remain at risk for aspiration and difficult airway.
(a) If GA is performed, appropriate aspiration precautions should be taken (**Fig. 107-2**).
(2) Volatile agents can cause uterine atony and precipitate postpartum hemorrhage; high doses should be avoided.[1]
ii) A spinal or an epidural anesthetic is the preferred method for this surgery.
(1) Spinal dosing is slightly lower than cesarean dosing (ie, approximately 11mg bupivacaine).
(2) If an epidural is in place from delivery, it can be used. The failure rate for catheters left in place >24 hours is up to 20%, so it may be faster to remove the catheter and administer a spinal anesthetic.[17]

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10) **Nonobstetric surgery during pregnancy**
a) **Incidence:** Approximately 0.75%-2% of pregnant patients require nonobstetric surgery.[18]
b) **Timing**
i) Elective surgery should not be performed during pregnancy.
ii) Surgical procedures during the first trimester are associated with increased risk of spontaneous abortion.
iii) Surgery in the third trimester is associated with preterm labor.
iv) If surgery must be done, the second trimester is preferred as these risks are minimized.
v) A procedure should not be postponed until the second trimester if a mother's life is at risk.



In determining whether an *in situ* epidural catheter may function for postpartum tubal ligation, check to see if the catheter has dislodged and ask the patient if it functioned well for labor.

c) **Fetal Monitoring:** Intraoperative fetal monitoring should be discussed with the patient's obstetrician.
i) Prior to 24 weeks' gestation, FHR should be documented pre- and postoperatively.
ii) Although a topic of controversy and variable across different centers, 24 weeks of gestation is often considered the age of fetal viability. For this reason:

(1) Continuous monitoring should be considered, especially for patients undergoing high-risk procedures (eg, intra-abdominal, intracranial, or intrathoracic operations).[19]

(2) When fetal monitoring is performed, a clear plan for treatment of FHR abnormalities should be determined prior to starting the procedure and appropriate equipment/personnel should be available (eg, STAT cesarean delivery).[19]

iii) Anesthetic technique

(1) No specific technique has been associated with any improved fetal outcome.[1]

(2) Fetal oxygenation depends directly on maternal oxygenation and uterine perfusion (which lacks autoregulation).

(a) Blood pressure should be vigilantly monitored (by invasive means if necessary) and hypotension promptly treated.

(b) Hypoxia, and associated fetal acidosis, should be avoided, and PCO_2 maintained within the normal range for pregnancy (~33 mm Hg).

(3) RA is preferred if appropriate for the surgical procedure.



When a patient is under GA and continuous FHR monitoring is performed, the FHR tracing will show no variability.

iv) Aspiration precautions should be taken and the airway should be protected with endotracheal intubation in most patients >12 of weeks gestation, or those with a history significant for gastroesophageal reflux (**Fig. 107-2**).

v) After 18-20 weeks of gestation, the patient should always be positioned with left uterine displacement to minimize aortocaval compression from the gravid uterus.

vi) Some evidence supports the avoidance of N_2O very early in the first trimester (6 weeks of gestation or less) or during long cases.

(1) Complete avoidance of N_2O may necessitate large doses of volatile agent, which may lead to maternal hypotension.

(2) Concentrations of N_2O of 50% or less appear safe, especially when used to avoid maternal hypotension.[1]

(3) Propofol infusions are also safe and may cause less hypotension.

vii) Midazolam and opioids are considered safe in pregnancy.

(1) Teratogenicity not associated with use in clinical doses[1]

(2) No adverse neonatal outcomes reported with midazolam use for anxiolysis during Cesarean delivery.[20]

viii) All volatile agents and opiates cause decreased FHR variability.

(1) Likely due to fetal anesthesia

(2) Despite this, every effort should be made intraoperatively to ensure adequate fetal oxygenation and uterine perfusion.[1]

d) **Postoperative recovery**

i) It is optimal to recover pregnant patients on a unit with FHR and uterine tocodynamic monitoring capability.

ii) Postoperative disposition should be a collaborative decision involving the patient's obstetrician and the physician performing the nonobstetric procedure.

CHAPTER SUMMARY FOR OBSTETRICAL ANESTHESIA

Procedure	Overview of Anesthetic Considerations
Labor Analgesia for Vaginal Delivery	Epidural infusion/PIEB, or CSE most commonly used; aim for maximal pain relief with minimal motor block
RA for Cesarean Delivery	Preferred anesthetic technique; patient should be positioned with left uterine displacement; hypotension should be aggressively treated; block must be tested and adequate anesthetic level confirmed prior to surgical incision; constant communication with the patient and obstetrician is essential
GA for Cesarean Delivery	May be necessary due to contraindications to RA or when emergent; aspiration precautions should always be taken along with preparation for potential difficult airway (eg, video laryngoscopy); vigilantly observe blood loss and decrease volatile agents if necessary; PCA analgesia may be necessary for postoperative pain control
Cervical Cerclage	Performed for cervical incompetence; RA preferred; avoid coughing or straining; outpatient procedure for many patients
Postpartum Tubal Ligation	Performed 24-48 h postpartum while uterine size enables smaller surgical incision; RA preferred
Anesthesia for Pregnant Patients Requiring Nonobstetric Surgery	Done for variety of indications; elective procedures should be delayed until postpartum; second trimester is preferred if surgery is necessary; strive to optimize maternal hemodynamics; ensure left uterine displacement; monitor FHR at beginning/end of procedure, and intraoperatively only after consultation with obstetrician regarding plan of action

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108 Amniotic Fluid Embolus

Edward T. Riley

Amniotic fluid embolism (AFE) is a rare obstetrical event that presents with cardiopulmonary dysfunction or collapse and altered mental status, followed by disseminated intravascular coagulation. This is a poorly understood phenomenon and hence is a diagnosis of exclusion. Treatment is supportive and requires access to major hospital resources such intensive care, operating rooms, massive transfusion, and possibly extracorporeal life support.[1]

1) **Incidence**
   a) The incidence and case fatality of AFE are inconsistently reported since it is a diagnosis of exclusion. The incidence varies depending on diagnostic criteria and what is reported through case series, governmental agencies, and registries.
   b) A pooled estimate of various population studies from North America and Europe revealed an incidence of 1 in 53 800 in Europe and 1 in 15 200 in North America.[2]
   c) **Case fatality rate**
      i) Early estimates of case fatality were extremely high (86% and 61%), but later estimates are much lower (13%-44%).
      ii) Diagnostic criteria, reporting bias, or improvements in care may explain these changes.
   d) **Morbidity is high with many patients developing permanent neurologic sequelae.**

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Crisis management: amniotic fluid embolism, Chapter 142



AFE is a serious pregnancy-related condition and is a diagnosis of exclusion.

2) **Signs and symptoms: a triad of hypotension, hypoxemia, and coagulopathy**
   a) **Initial phase**
      i) AFE usually presents with sudden dyspnea and decreased oxygen saturation occurring during labor or a cesarean delivery.
      ii) Hypotension is common and may lead to cardiac arrest.
         (1) The initial symptoms will be related to right heart failure, with hypoxemia and hypotension being the first symptoms.
         (2) In less severe cases, the hypotension is mild and the hypoxemia is transient.
         (3) In severe cases, complete cardiac arrest may occur due to acute right heart failure.
         (4) If the right heart failure is survived by the patient, there is danger of left heart failure in the second phase of the disease process.
      iii) AFE has been associated with tumultuous deliveries during the second stage. Rather than the rapid delivery being causative of the AFE, one expert theory is that norepinephrine release from the body in response to the insult of the AFE may increase uterine activity and cause the rapid delivery.[1]
      iv) Patients may appear to be having a seizure.
      v) AFE is often accompanied by evidence of acute fetal asphyxia (changes in fetal heart rate pattern).
      vi) May be accompanied by hypertonic contractions
   b) **Secondary phase**
      i) Includes a severe consumptive coagulopathy characterized by:

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Coagulopathies, Chapter 66

(1) Low or undetectable fibrinogen
(2) Low platelets
(3) High D-dimer
(4) Prolonged PT and PTT
(5) The clinical picture is usually very clear. There is diffuse bleeding, and clotting studies are highly abnormal.



The triad of hypotension, hypoxia, and coagulopathy with no other cause should raise suspicion of an AFE and trigger empiric therapy.

c) **The differential diagnosis of AFE includes**[3]
i) Other obstetrical emergencies such as placental abruption, uterine rupture, uterine atony, or eclampsia
ii) Anesthetic causes such as high spinal anesthesia or local anesthetic toxicity
iii) Nonobstetric causes such as air or pulmonary embolism, anaphylaxis, or sepsis

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Obstetrical emergencies, Chapter 111

3) **Pathophysiology**
a) As there is not a definitive understanding of the mechanism that leads to the AFE syndrome, it remains a diagnosis of exclusion.
b) **However, common features that aid in the diagnosis include:**
i) An episode of respiratory distress or oxygen desaturation
ii) Cardiovascular compromise ranging from hypotension to cardiac arrest
iii) Coagulopathy
iv) The presence of fetal squamous cells in the lungs of the patient in a postmortem exam or on the maternal side of the placental circulation suggests, but does not confirm, an amniotic fluid embolus.



Patients with suspected AFE may require immediate airway management.

c) Although the pathophysiology is not known, it is commonly agreed upon that there is some type of systemic response to mediators released in response to an unknown trigger. Whether this is related directly to amniotic fluid is unknown.
d) **Clinical responses to the syndrome include the following:**
i) An initial period of pulmonary hypertension resulting from intense vasoconstriction in the pulmonary vasculature in response to the mediators.
(1) The pulmonary hypertension component of the syndrome is not a physical plugging of the vessels by amniotic fluid. Suggested, but not proven, mechanisms include
(a) Anaphylaxis to fetal material in the maternal circulation
(b) Complement activation initiated by the amniotic fluid
ii) Relatively short-lived right heart failure and hypoxemia due to ventilation/perfusion mismatching
iii) The right heart failure and hypoxemia lead to left heart failure.
e) Some experts propose renaming the phenomenon of AFE as "anaphylactoid syndrome of pregnancy," because it appears to be a response to an abnormal release of endogenous mediators.[1] In this theory, the pathophysiology is more akin to a septic state rather than an embolic event. However, this change has not been adopted by the scientific community at large, and the condition is still known by the arguably less appropriate name of AFE.

4) **Treatment**
a) Treatment is supportive and includes:
i) Delivery of the fetus
ii) Oxygen supplementation and ventilation if needed
(1) May require immediate intubation if the patient's level of consciousness or oxygenation is jeopardized

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Crisis management: amniotic fluid embolism, Chapter 142

iii) Invasive monitoring
  (1) An arterial line is indicated for blood pressure monitoring and frequent blood sampling.
  (2) Central monitoring (CVP or pulmonary artery catheter) may be of limited used in assessing volume status and cardiac function, while transesophageal echocardiography will likely provide this information in a more expedient and accurate manner.
iv) Appropriate fluid resuscitation to maintain preload
v) Vasopressor medications as needed to maintain blood pressure
vi) Packed red blood cell replacement
vii) Blood product replacement for coagulopathy
viii) Newer modalities that may be useful, but are unproven
  (1) Cardiopulmonary bypass or extracorporeal membrane oxygenation should be considered if there is cardiovascular collapse, and pharmacologic treatment alone is inadequate to provide adequate blood pressure.[4,5]
  (2) The right heart failure is transient, but anything that can be done to support right sided ventricular function and decrease pulmonary hypertension during the evolution of the disease may help survival. Treatment modalities to consider may include
    (a) Inhaled aerosolized prostacyclin
    (b) Inhaled nitric oxide
    (c) A right ventricular assist device[6]

b) **Other treatments that theoretically make sense but are of no proven benefit and may be harmful include the following:**
  i) Steroids
    (1) Some experts view AFE as an allergic reaction and theorize that steroids may help resolve the reaction. Currently, this is of unproven value.
  ii) Recombinant factor VIIa (RFVIIa)
    (1) Even though RFVIIa is a useful treatment adjunct during a major obstetric hemorrhage, and its use has been reported in patients with presumed AFE,[3,6] its use in AFE should be avoided. The hemorrhage associated with AFE is a consumptive coagulopathy that may include massive fibrinolysis with high levels of circulating tissue factor. RFVIIa may have the effect of triggering thromboses and fibrosis, leading to further complications.[7]

CHAPTER SUMMARY FOR AMNIOTIC FLUID EMBOLUS

Diagnosis	AFE is a diagnosis of exclusion. Nothing is pathognomonic, but sudden dyspnea, decreased oxygen saturation, and hypotension, followed by heart failure and coagulopathy are highly suggestive
Clinical Pathophysiology	Right heart failure secondary to pulmonary hypertension that is short lived Left heart failure Profound coagulopathy
Treatment	Delivery of the fetus Supplemental oxygen with intubation and mechanical ventilation if needed Supportive care with fluid resuscitation, transfusion of packed cells and other blood products, vasopressors, as needed Cardiopulmonary bypass in case of cardiovascular collapse

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109 Pregnancy and Hypertension

John J. Kowalczyk

Hypertension (HTN) during pregnancy is one of the leading causes of maternal morbidity and mortality in the United States and the world.[1] Preeclampsia affects 2%-5% of pregnancies in the United States and accounts for 9.5% of maternal deaths.[2] Up to an estimated 60% of these deaths are preventable.[3] The diagnosis, management, and anesthetic concerns of HTN in pregnancy are outlined in this chapter.

1) **Classifications of HTN during pregnancy**[4]
   a) **Chronic HTN**
      i) Diagnosis predates pregnancy or <20 weeks' of gestational age.
      ii) Patients typically are on antihypertensive medications prior to pregnancy.
   b) **Gestational HTN**
      i) Defined as HTN after 20 weeks' gestation without proteinuria or end organ dysfunction
      ii) Many go on to develop preeclampsia
   c) **Preeclampsia (Table 109-1)**
      i) Preeclampsia must be viewed as a multisystem disease.
      ii) HTN is associated with end organ dysfunction.
      iii) Diagnosis occurs after 20 weeks' gestation.
      iv) SBP ≥ 140 or DBP ≥ 90 mm Hg (on 2 occasions > 4 hours apart) or SBP ≥ 160 or DBP ≥ 110 mm Hg (confirmed within minutes)[4,5]
      v) Preeclampsia without severe features
         (1) Elevated BP as described above
         (2) Proteinuria ≥ 0.3 g over 24 hours or a protein/creatinine ratio of 0.3 mg/dL
      vi) Preeclampsia with severe features (**Table 109-2**)
         (1) New criteria for diagnosis removed the requirement of proteinuria. The degree of proteinuria does not correlate with severity of symptoms or outcomes.[4]
         (2) Diagnosis is based on elevated BP and one of the following:
            (a) New-onset visual disturbances or headache resistant to analgesics
            (b) Renal insufficiency (creatinine > 1.1 mg/dL or > 2× baseline)
            (c) Impaired liver function (liver enzymes > 2× baseline), severe right upper quadrant or epigastric pain

TABLE 109-1 Risks Associated With Preeclampsia[4]

	Increased Risk of…	Management Suggestions
Preeclampsia without severe features	Labor induction Cesarean delivery Small for gestational age infants	Consider early epidural in case of urgent delivery. May also aid in BP management.
Preeclampsia with severe features	Placental abruption Preterm delivery Maternal mortality Maternal morbidities	Prepare for the possibility of urgent delivery and postpartum hemorrhage. Consult neonatal team to counsel mother regarding neonatal resuscitation and critical care.

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TABLE 109-2 Complications Associated With Severe Preeclampsia

Complication	Clinical Considerations
Cerebral vascular accident	Most common cause of death in patients with preeclampsia. BP management critical.
Pulmonary edema	Carefully monitor volume replacement and urine output.
Renal dysfunction	Check electrolytes. Monitor volume in and out. May require invasive monitoring or point of care ultrasound for assessment of intravascular volume.
Liver dysfunction or rupture	Monitor coagulation, LFTs, and hemoglobin.
Hemorrhage	Type and crossmatch; carefully monitor blood loss.
Disseminated intravascular coagulopathy	Consider early preparation of clotting factors (eg, FFP); monitor surgical field and other sites for microvascular bleeding.
HELLP (Hemolysis, Elevated Liver enzymes, Low Platelets) syndrome	Assess platelet count in severe preeclampsia prior to placing or removing epidural catheter. Prepare for possibility of general anesthesia for cesarean delivery.
Seizures (eclampsia)	Supportive care (see treatment plan below).

(d) Thrombocytopenia (<100 000/mL)
(e) Pulmonary edema

vii) Complications of untreated or undertreated preeclampsia
(1) Eclampsia
(2) Stroke
(a) Preeclampsia leads to dysfunctional cerebral autoregulation, which causes hypoperfusion, vasogenic edema, and ultimately vessel rupture.[6]
(b) >90% of preeclampsia-associated strokes are hemorrhagic.
(c) SBP control is more important than DBP in stroke prevention.
(d) Mortality >50%
(3) Pulmonary edema

d) **Eclampsia**
i) Preeclampsia with new-onset seizures

e) **Chronic HTN with superimposed preeclampsia**
i) Defined as chronic HTN and new-onset proteinuria, worsening proteinuria or BP, thrombocytopenia, or abnormal liver function tests (LFTs)

2) **Treatment**
a) **Delivery of fetus** is the only cure for preeclampsia or eclampsia.[7]
b) **Treatment goals** are to prevent maternal complications and delivery of a mature neonate.
i) BP control
(1) Maintain SBP < 160 mm Hg.[3,8]
(2) Cerebral autoregulation may be altered, and normal BP may decrease blood flow to the brain, uterus, or other vital organs.



Cerebrovascular accident due to uncontrolled HTN is the leading cause of death in patients with preeclampsia.

3) **Antihypertensives**[9]
c) **Vasodilators**
i) The first-line medications include labetalol, nifedipine, and hydralazine.[9,10] These are given every 15-30 minutes until SBP < 160 mm Hg.
ii) β-Adrenergic antagonists
(1) Labetalol
(a) Widely used by obstetricians

(b) 10-20 mg IV boluses are given with onset in ~2 minutes, lasting 2-3 hours and a maximum dose of 300 mg.
(c) Faster onset than IV hydralazine
(d) Use caution in asthmatics since it is not cardioselective.
(2) Esmolol
(a) Generally avoided due to the potential for fetal bradycardia

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Adrenergic agents, Chapter 32.

iii) Hydralazine
(1) One of the most commonly used agents by obstetricians
(2) 5-10 mg IV is given and has an effect in 20 minutes; lasts 2-3 hours.
(3) May cause a reflex tachycardia

iv) Calcium channel blockers
(1) Decrease SVR
(2) Can cause uterine relaxation
(3) May potentiate magnesium toxicity[10]
(4) Nifedipine
(a) PO or sublingual administration, 10 mg, every 15-30 minutes, up to 30 mg
(b) Oral dosing to reach target BP faster and with fewer doses than IV labetalol[10,11]
(c) May also be used for tocolysis
(5) Nicardipine
(a) May be useful in severe, intractable HTN
(b) Only available by IV administration



Nifedipine is given PO and may have the advantages of faster time and fewer doses to achieve SBP < 160 mm Hg. This can allow prompt initiation of BP control in patients without IV access.

v) α-Adrenergic agonists
(1) Methyldopa
(a) May be used for outpatient BP control but typically avoided due to its side effect profile including dry mouth, sedation, bradycardia, and postpartum depression[2,12]

vi) Nitroglycerin
(1) May be useful for severe, intractable HTN
(2) Administered by IV infusion
(3) Very rapid onset

vii) Sodium nitroprusside
(1) May be useful for severe, intractable HTN
(2) Administered by IV infusion
(3) Very rapid onset
(4) Be aware of possible cyanide toxicity in the mother or the neonate.

4) **Maintain adequate intravascular volume**
a) Preeclamptic patients are often fluid restricted and, despite being hypertensive, often have low intravascular volume and require judicious fluid resuscitation intraoperatively.[13]
b) Patients are at risk for pulmonary edema; be careful not to fluid overload.



While magnesium is the anticonvulsant of choice and a peripheral dilator, it should not be used as a substitute for antihypertensives in the treatment for preeclampsia.

5) **Decrease central nervous system hyperactivity with magnesium sulfate.**[4,14]
a) Magnesium functions as an anticonvulsant, tocolytic, and mild vasodilator.
b) **All patients with preeclampsia with severe features and eclampsia should be treated with magnesium**.
i) Mg treatment reduces the risk of preeclampsia with severe features progressing to eclampsia by >50%.

c) Dosing
   i) Regimens may vary, but typically is given as a 4-6 g bolus over 30 minutes, followed by a continuous infusion of 1-2 g/h.
   ii) Continue the infusion for 12-24 hours postpartum.[14]
      (1) Use caution in patients with renal dysfunction as magnesium is excreted by the kidneys.

d) **Anesthetic concerns in patients on magnesium therapy**
   i) The intubating dose of succinylcholine is the same when a patient is receiving magnesium.
   ii) Nondepolarizing neuromuscular blockade will be potentiated by magnesium.
      (1) Doses should be dramatically reduced, and neuromuscular blockade should be monitored.
   iii) As a tocolytic, it can increase the risk of uterine atony and PPH.
   iv) Can delay emergence and augment opioid analgesia.

e) **Toxicity (Table 109-3)**
   i) Deep tendon reflexes are followed clinically, and toxicity is suspected when they are decreased.
   ii) Treat overdoses with 1 g of calcium gluconate.



Even in the therapeutic range, EKG changes with magnesium (prolonged PR and widened QRS) can occur. This progresses to muscle weakness and decreased deep tendon reflexes, cardiac nodal block, and respiratory paralysis and cardiac arrest.



Be aware that babies born to mothers on magnesium may also show signs of magnesium toxicity such as muscle weakness and poor respiration and require intervention.



Calcium gluconate acts intracellularly to reverse the effects of magnesium toxicity.

6) Anesthetic management
   a) **Preoperative**[15]
      i) History and physical examination
         (1) A thorough history and physical should be performed.
         (2) Patients with preeclampsia are at high risk for difficult airway due generalized edema.
         (3) If patient has been admitted on antepartum, confirm time of last dose of venous thromboembolism prophylaxis.
      ii) Patients are at high risk for cesarean delivery due to decreased uteroplacental perfusion. The provision of anesthesia for cesarean delivery should be discussed with the patient.
         (1) Early epidural catheter placement will help with pain control and BP management and should be in place should urgent or emergent cesarean delivery be necessary.[15]
      iii) Laboratories
         (1) Hematocrit
         (2) Platelet count[15]
            (a) There is no recommended threshold for platelet values prior to placement of neuraxial blockade.
            (b) Literature suggests that patients with HELLP syndrome are at high risk for epidural hematoma.[16]

TABLE 109-3 Clinical Effects of Magnesium Therapy

Magnesium Level (mg/dL)	Effect
4-8	Therapeutic range
5-10	EKG changes (prolonged PR interval, widened QRS, prolonged QT)
>10	↓ Deep tendon reflexes and muscle weakness
15-18	SA and AV nodal block
25-30	Cardiac arrest

(c) In the presence of preeclampsia, it is advisable to monitor serial platelet counts and have a recent (within 6 hours) value prior to placement of a neuraxial block and/or removal of an epidural catheter.[17]
(d) Most practitioners would perform neuraxial blockade with a platelet count >100 000/dL.[18]
(e) Most practitioners would not place a neuraxial block in a preeclamptic patient with a platelet count <70 000/dL, although it may be appropriate in very select cases.[19]
(f) Those with a low but stable platelet count may be at less risk for complications from a neuraxial block than patients with rapidly decreasing platelet counts.[19]
(g) The decision to perform neuraxial blockade in patients who fall into the "gray area" should be individualized based on the patient's airway examination, coexisting disease, and rate of platelet drop.
(h) If neuraxial blockade is performed, consider the following:
(i) Small needles such as those used for spinal anesthesia are probably less traumatic than larger epidural needles.[16]
(ii) A lower threshold platelet count >75-80 000/dL may be considered safe for spinal anesthesia.[20]
(iii) Strongly consider having the most experienced practitioner place the block. The risk of hematoma increases with the number of passes of the needle.

(3) Liver function tests (LFTs)
(a) Patients with elevated LFTs are at high risk for coagulation abnormalities.
(4) Blood type and screen
(a) Type and crossmatch should be done in the presence of liver dysfunction or thrombocytopenia or if estimated blood loss is expected to be high.
(5) Urine protein

iv) Treatment of eclampsia (**Table 109-4**)
(1) Seizures are usually self-limited, lasting a few minutes.
(2) Magnesium: 6 g load, if not on Mg or 2 g bolus, if already on infusion
(3) Small dose of propofol or midazolam
(a) Short-acting medications allow neuro examinations to assess for signs of stroke.
(4) Seizures accompanied by apnea (~1 minute) and may need assisted ventilation
(a) Intubate for intractable seizures, signs of aspiration, or continued unconsciousness.
(5) Fetal bradycardia is common but usually resolves.

TABLE 109-4 Treatment of Eclampsia

- Stop the seizure with:
  - Magnesium (bolus additional 2 or 6 g loading dose if infusion not running)
  - Benzodiazepines or propofol as appropriate
- Maintain adequate oxygenation/ventilation
  - Monitor SpO_2
  - Administer 100% O_2
  - Monitor ventilation and assist if necessary
  - Intubate only to protect against hypercarbia or aspiration
- Ensure adequate left uterine displacement
- Control BP
- Consult with obstetrician regarding delivery plan
- Always rule out other causes (eg, hypoglycemia, seizure disorder)

(a) If the mother is stable, allow the placenta to resuscitate the baby prior to delivery.[21]

(6) Deliver the baby after the seizure and BP are controlled.

(7) Seizures can occur antepartum (38%-53%), intrapartum (18%-36%), or postpartum (11%-44%).[22]

Onset of maternal seizures is associated with fetal bradycardia. Treatment of the seizure usually results in resolution of the fetal bradycardia and can allow time to prepare for more gentle induction of general anesthesia.

b) **Intraoperative considerations**

i) Monitoring

(1) Blood pressure

(a) Noninvasive monitoring is usually adequate.

(b) Consider an arterial line if the patient has pulmonary edema or requires an infusion of antihypertensives or frequent blood draws.

(2) Central venous pressure (CVP) monitor/pulmonary artery catheter

(a) There are no data to support routine use of invasive monitors.[15]

c) **Postoperative considerations**

i) Patients are at risk for seizures particularly in the first 48 hours postpartum; 32%-44% of all eclampsia occurs postpartum.[7,23]

ii) Continue magnesium for 12-24 hours.

iii) Repeat platelet counts prior to removal of epidural catheter and use the same criteria for removing the catheter as for placement.

Although delivery is considered the "definitive treatment" of preeclampsia, all complications of preeclampsia can occur pre-, intra- and postpartum.

7) **Management of labor analgesia in patients with preeclampsia**

a) **Neuraxial analgesia**

i) Preferred for patients with preeclampsia to blunt the sympathetic response to labor pain

ii) Contraindicated in the presence of thrombocytopenia or abnormal coagulation[15]

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Obstetrical anesthesia, Chapter 107.

b) **IV analgesia**

i) Appropriate in patients who are unable or unwilling to receive neuraxial analgesia[18]

8) **Management of anesthesia for cesarean delivery**

a) **Neuraxial anesthesia**

i) Epidural anesthesia

(1) If a well-functioning epidural catheter is in place (eg, from labor), it can be used if time permits.

(2) Preferred by some to prevent rapid drop in BP that may occur with spinal anesthesia

ii) Spinal anesthesia

(1) More recent studies indicate spinal anesthesia is safe.[24]

(2) Spinal anesthesia is safe to use and does not precipitate severe hypotension, in fact they may require less fluid and vasopressors than a healthy parturient.[25,26]

iii) Combined spinal-epidural technique has also been shown to have minimal hemodynamic effects.[27]

Spinal anesthesia may be safer than epidural anesthesia for cesarean delivery in patients with severe preeclampsia due to the use of smaller needles and lack of indwelling catheter should the platelet count drop.

b) **General anesthesia**

i) Appropriate when regional anesthesia is not performed due to time constraints or contraindications

ii) Patients are at risk for severe HTN on airway instrumentation.

(1) Remifentanil, esmolol, labetalol, or nitroglycerin may be used during induction to blunt the BP response to intubation.[18]

iii) If the airway is not reassuring secondary to airway edema or other causes, awake fiberoptic intubation may be necessary.

c) Methylergonovine contraindicated as a uterotonic agent as profound HTN can occur, possibly leading to a hypertensive crisis

CHAPTER SUMMARY FOR HTN IN PREGNANCY

Classification	Diagnosis	Clinical Considerations
Chronic HTN	HTN present prior to pregnancy or earlier than 20 wks	Monitor for the development of preeclampsia
Gestational HTN	HTN during pregnancy after 20 wks without proteinuria	Monitor for the development of preeclampsia
Preeclampsia without severe features	New-onset SBP > 140, DBP > 90 on 2 separate readings or SBP > 160, DBP > 110 Proteinuria > 0.3 g/24 h or a protein/creatinine ratio of 0.3 mg/dL	Monitor platelet count and LFTs regularly Treat to SBP < 160
Preeclampsia with severe features	Associated symptoms such as headache, visual changes, liver dysfunction, thrombocytopenia, renal insufficiency, pulmonary edema	Severe, multisystem disease typically treated with magnesium sulfate Regional analgesia and anesthesia preferred in absence of contraindications Delivery based on maternal and fetal status Prepare for possible urgent or emergent delivery
Eclampsia	Preeclampsia with seizures	Supportive care Difficult airway possible Consult OB team about delivery plan
All patients with HTN in pregnancy need to have adequate BP control. Treatment and clinical concerns vary depending on the type of HTN.		

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110 Pregnancy and Coexisting Disease§

Kelly A. Fedoruk • Glennie A. Lane • Andrea J. Traynor

Increasing numbers of women are becoming pregnant in the presence of preexisting illnesses. Coexisting disease states have significant implications for pregnancy outcome and the care these patients require. In addition to the normal physiologic changes of pregnancy, the pathophysiology of individual conditions must be considered during assessment and treatment of pregnant women. Fetal well-being is dependent upon maternal hemodynamic stability and perfusion, and the acutely ill mother must be treated aggressively in order to maintain maternal health and fetal viability.

1) **Introduction**
   a) **Definition**
      i) Coexisting disease in pregnancy is any medical condition that occurs in the pregnant patient. The disease may be unique to pregnancy or present prior to pregnancy. Often, pregnancy exacerbates preexisting disease.
   b) **Epidemiology**
      i) The most common diseases associated with pregnancy are hypertension (HTN) and diabetes mellitus (DM).
      ii) The incidence and complexity of coexisting disease in pregnancy is increasing.
         (1) Women are delaying childbearing to an older age.
         (2) Improved treatment of pediatric diseases, especially congenital heart disease, allows these patients to reach childbearing age and achieve pregnancy.
         (3) For this reason, maternal-fetal medicine and obstetric anesthesiology specialists are realizing an increasing role, as pregnant women with complex maternal or fetal conditions are often referred to their care.
      iii) The most common cause of death of women in the peripartum period currently is cardiac disease, which is most often present prior to pregnancy.[1]
   c) **The approach to the pregnant patient with coexisting illness should include the following:**
      i) A thorough understanding of the underlying disease process and the physiologic consequences of pregnancy, as more specialized care may be necessary in the peripartum period.
      ii) Discussion with an obstetrician about timing and mode of delivery.
      iii) Multidisciplinary conferences with the physicians and providers involved in the care of the patient should be organized to discuss possible peripartum complications and develop a clear plan to manage potential emergencies.
2) **Hypertensive disorders of pregnancy**
   a) **Preexisting (chronic) hypertension**



Pregnancy can be considered a stress test that may reveal a woman's underlying predisposition to develop a variety of chronic diseases.



The most common cause of death of women in the peripartum period is cardiac disease.



Early, comprehensive, and ongoing discussion between the anesthesiologist and obstetrician regarding the goals of care, timing, and mode of delivery is of utmost importance when caring for parturients with complicated medical and obstetrical conditions.

i) Definition
(1) Hypertension that predates pregnancy or <20 weeks of gestational age
(2) Essential hypertension is the cause of 90% of preexisting chronic hypertension.[2]

Hypertensive disorders of pregnancy affect 15% of all

b) **Gestational hypertension**
i) Definition
(1) Hypertension that presents after 20 weeks of gestational age

c) **Adverse pregnancy outcomes associated with HTN**
i) Maternal risks include preeclampsia, HELLP syndrome, coagulopathy and DIC, pulmonary edema, eclampsia, stroke, PRES (posterior reversible encephalopathy syndrome), renal failure, placental abruption, and cesarean delivery.
ii) Fetal and neonatal risks include preterm birth, growth restriction, and fetal demise.

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Pregnancy and hypertension, Chapter 109

3) **Endocrine disease**
a) **Gestational diabetes mellitus (GDM)**[3,4]
i) Epidemiology
(1) Affects 2%-12% of pregnancies
(2) Due to the rising incidence of obesity, the rise in mean maternal age, and changing diagnostic criteria, the incidence of GDM has been steadily increasing.
ii) Definition (**Table 110-2**)
(1) Glucose intolerance of variable severity with onset or first recognition during pregnancy

Insulin has typically been prescribed for GDM, but recently oral hypoglycemic agents have become accepted for use during pregnancy.[5]

iii) Pathophysiology
(1) GDM is caused by a combination of metabolic abnormalities including increased insulin resistance, impaired insulin secretion, and increased hepatic glucose production.
(2) Under normal circumstances, human placental lactogen and human placental growth hormone increase maternal glucose levels, creating a glucose gradient from mother to fetus. In GDM, insulin resistance increases due to excess human placental lactogen and an inability to suppress endogenous glucose production.[6] This results in maternal hyperglycemia.
(3) GDM confers a 50% risk of developing type 2 DM within 5-10 years and a long-term risk of 70%.[4]

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Obstetrical emergencies, Chapter 111

iv) Adverse pregnancy outcomes associated with GDM (**Table 110-1**)
(1) Fetal hyperglycemia occurs due to maternal hyperglycemia, resulting in fetal hyperinsulinemia, which places the infant at risk for life-threatening hypoglycemia in the neonatal period.

TABLE 110-1 Adverse Pregnancy Outcomes Associated with GDM

Preeclampsia	Shoulder dystocia
Intrauterine fetal demise	Birth trauma
Polyhydramnios	Operative delivery
Fetal macrosomia	Perinatal mortality
Neonatal metabolic complications (hypoglycemia, hyperbilirubinemia, erythrocytosis, hypocalcemia)	Preterm labor
Polyhydramnios	

TABLE 110-2 White Classification of DM in Pregnancy

Gestational diabetes	
A1	GDM; diet controlled
A2	GDM; medication (usually insulin) controlled
Diabetes present prior to pregnancy	
B	Onset at age 20 y or older and duration <10 y
C	Onset at age 10-19 y or duration of 10-19 y
D	Onset before 10 y of age, duration over 20 y, benign retinopathy or HTN (not preeclampsia)

Other subtypes of D and classification types are based on associated end-organ disease.
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(2) Maternal hyperglycemia increases the risk of fetal macrosomia and polyhydramnios, with subsequent increased risks of operative vaginal delivery, cesarean delivery, shoulder dystocia, and postpartum hemorrhage.



Mothers with GDM have a 25%-40% risk of cesarean delivery.

v) Intrapartum management
(1) Maintenance of maternal glucose between 70 and 120 mg/dL with the use of IV insulin and dextrose infusions as necessary

b) **Diabetes mellitus (DM) (Table 110-2)**
i) Preexisting DM complicates as many as 2% of all pregnancies, and the proportion of type 2 DM is increasing with the prevalence of obesity.
ii) DM in pregnancy is described by the White classification system, which is based upon duration of diabetes and the presence of end-organ damage.
iii) Hyperglycemia and hyperinsulinemia carry the same risks to mother and fetus as GDM.
iv) Preexisting DM is associated with an increased risk of early-onset preeclampsia, fetal growth restriction, and birth defects.

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Diabetes mellitus, Chapter 59

v) Type I DM
(1) Associated with increased incidence of other autoimmune conditions
(2) Increased risk for hypoglycemia in the first trimester due to an early increase in insulin sensitivity, especially in the fasting state
(3) Diabetic ketoacidosis (DKA) is more commonly associated with type 1 DM but can occur in type 2 and rarely with GDM.
(4) In the first trimester, there are decreased insulin requirements in type 1 DM.[7] Insulin requirements gradually increase throughout pregnancy and peak in the third trimester.



During pregnancy, DKA can occur with less provocation or at lower levels of hyperglycemia compared to the nonpregnant state.

(5) Immediately after delivery of the placenta, insulin requirements fall; insulin should be reduced to near prepregnancy values and glucose carefully monitored to avoid hypoglycemia.[2]
vi) Type II DM
(1) Incidence has increased dramatically among women of younger age groups due to the epidemic of obesity.
(2) Associated with HTN, hyperlipidemia, obesity, and polycystic ovarian disease

c) **Thyroid disease**[8,9]

i) Physiologic changes in thyroid function result in abnormal thyroid investigations during normal pregnancy.
   (1) Decrease in thyroid-stimulating hormone (TSH) is mediated by β-hCG.
   (2) Increase in thyroid-binding globulin causes increased total T3 and T4.
   (3) Free T3 and T4 remain the same as nonpregnant values.

ii) Hypothyroidism
   (1) Causes
      (a) Iodine deficiency is the leading cause of hypothyroidism globally.
      (b) Hashimoto thyroiditis is most common in the United States.
   (2) Associated with infertility and pregnancy loss
   (3) Children exposed to hypothyroidism during gestation are at risk for a range of neurodevelopmental delays.
   (4) Pregnant women with subclinical hypothyroidism may be at risk of stillbirth, preterm birth, preeclampsia, breech presentation, intrauterine growth restriction (IUGR), and placental abruption.
   (5) Treatment
      (a) Thyroxine replacement doses are increased beginning early in pregnancy.
      (b) After delivery, prepregnancy dose is resumed and the TSH followed frequently for the first year, due to risk of postpartum thyroiditis.

iii) Hyperthyroidism
   (1) Causes
      (a) Graves disease
         (i) Most common cause of hyperthyroidism
         (ii) Thyroid-stimulating autoantibodies cross the placenta and place the fetus at risk of fetal or neonatal Graves disease.
         (iii) Fetal Graves disease manifests as fetal tachycardia, high-output failure and hydrops, IUGR, and fetal goiter.
   (2) Treatment
      (a) Subclinical hyperthyroidism
         (i) Defined by low TSH with normal free T4
         (ii) Not associated with adverse pregnancy outcomes
         (iii) Should not be treated, as this risks suppression of the developing fetal thyroid gland
      (b) Overt hyperthyroidism
         (i) Diagnosed by low TSH and elevated free T4 and/or T3
         (ii) Treated with a thionamide, preferably propylthiouracil
         (iii) The goal of treatment is a free T4 in the high-normal range.
         (iv) β-Adrenergic antagonists may be used to control symptoms (ie, tachycardia, tremor).
   (3) Pregnancy complicated by poorly controlled hyperthyroidism is associated with increased rates of spontaneous abortion, preterm labor, low birth weight, stillbirth, preeclampsia, and heart failure.
   (4) Thyroid storm may occur in hyperthyroid pregnant patients in response to labor, infection, preeclampsia, trauma, and cesarean delivery.

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Thyroid disease, Chapter 60



Thyroid storm in hyperthyroid pregnant patients can be precipitated by labor, infection, preeclampsia, trauma, and cesarean delivery.

d) **Pituitary disease**

i) Pituitary adenomas
   (1) Treated with dopamine agonists, which may be required for conception and discontinued once pregnancy is achieved
   (2) Tumor enlargement may occur due to hormonal stimulation of the tumor.

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Pituitary disorders, Chapter 62

(a) Macroadenomas have a higher chance of enlarging enough to cause neurologic symptoms versus microadenomas.
(b) Patients should be followed for the development of headache or visual symptoms.

ii) Hypopituitarism
(1) Caused by various conditions whose mechanisms fall into the categories of tumor, infiltrative disease, congenital insufficiency, or surgical destruction

iii) Sheehan syndrome
(1) Pituitary necrosis in the setting of shock or massive peripartum hemorrhage resulting in panhypopituitarism
(2) Patients often present with infertility or difficulty lactating. Rarely, patients may experience headache, visual field changes, diabetes insipidus, or prolonged hypotension.
(3) Treatment
(a) Appropriate hormone replacement (cortisol, thyroid hormone, DDAVP), including stress dose glucocorticoids when indicated
(b) Periodic measurements of BP and electrolytes and monitoring fetal growth by ultrasound
(4) If acute hypopituitarism is suspected
(a) Management includes fluid resuscitation, IV dextrose, stress dose steroids (hydrocortisone: 100 mg IV q8h)
(b) Diagnostic workup includes measuring prolactin, ACTH, cortisol, TSH, free T4, electrolytes, glucose, and MRI when stable



Sheehan syndrome is a cause of panhypopituitarism unique to pregnancy. The risk is increased in patients with shock due to peripartum hemorrhage.

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Adrenal disorder, Chapter 61.

e) **Adrenal conditions**
i) Pheochromocytoma[9]
(1) Associated with high maternal and fetal mortality rates
(2) It should always be considered in the differential diagnosis of labile, severe HTN.
(3) Proteinuria occurs in 20% of cases, making it difficult to distinguish from preeclampsia.
(4) The most sensitive diagnostic test is urinary or plasma metanephrines.
(5) Once diagnosed, α-adrenergic blockade should be instituted immediately, followed by β-adrenergic blockade titrated with a goal of maternal heart rate between 80 and 100 beats per minute.[9]
(6) If diagnosed in the first two trimesters, the tumor should be surgically removed.
(7) If the patient is in the third trimester and medical therapy is adequate, surgery may be delayed until postpartum.
(8) There is no consensus regarding the preferred route of delivery.[5] A vaginal delivery may be considered in consultation with the obstetrician.



When managing a patient with pheochromocytoma, alpha-blockade must always be initiated before beta-blockade, as the reverse leaves alpha receptors susceptible to unopposed agonism and could result in a hypertensive/tachycardic crisis. Short-acting alpha- and beta-blockers and vasodilators should be readily available.

4) **Hematologic diseases**
a) **Thrombocytopenia in pregnancy**
i) Thrombocytopenia in pregnancy is a frequent concern for anesthesiologists.
ii) The first step in evaluating any thrombocytopenic parturient is to determine the cause of thrombocytopenia.
iii) Other important factors to consider are the rate of platelet drop and whether the patient is on any medications that may affect platelet function (eg, aspirin).



The approach to the parturient with thrombocytopenia includes consideration of the cause of thrombocytopenia, rate of platelet drop, and medication use.

TABLE 110-3 Diagnostic Criteria for Gestational Thrombocytopenia

- Thrombocytopenia is mild (platelets are usually >70 000/μL and average 130-150 000/μL) and the patient is asymptomatic
- No past history of thrombocytopenia outside of pregnancy
- Occurs late in gestation—in the midthird trimester or beyond
- Resolves postpartum
- No associated fetal thrombocytopenia

iv) Thrombocytopenia occurs in ~5% of pregnancies and is usually without consequence when the diagnosis is gestational thrombocytopenia.
v) Gestational thrombocytopenia has five diagnostic criteria (**Table 110-3**).[10,11]
(1) Anesthesia considerations for gestational thrombocytopenia
(a) Usually no change in routine obstetric or anesthetic care is necessary.
(b) The decision to perform neuraxial analgesia/anesthesia is individualized but is generally considered safe in patients with uncomplicated gestational thrombocytopenia.[12]

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Pregnancy and hypertension, Chapter 109

b) **HELLP syndrome and/or severe preeclampsia**[13]
i) Thrombocytopenia complicates ~15% of preeclampsia cases.
ii) Thrombocytopenia in preeclampsia may be a result of HELLP syndrome and/or DIC in the case of severe preeclampsia.
iii) Anesthesia considerations
(1) Assess platelet count and coagulation studies before placement of neuraxial block.
(2) The decision to perform neuraxial procedures is based on the patient's airway exam and how difficult other routes of anesthesia (ie, general anesthesia) are expected to be. The rate of platelet drop and coexisting illnesses are also considered.
(3) Assess patient's airway, renal function, and IV access to formulate the anesthetic plan for delivery.

c) **Idiopathic thrombocytopenia (ITP)**[14,15]
i) ITP may be difficult to differentiate from gestational thrombocytopenia.
(1) Usually presents earlier in pregnancy
(2) Platelet counts are more commonly <50 000 cells/mm^3.
ii) Neonatal thrombocytopenia is an uncommon risk secondary to antiplatelet antibodies that cross the placenta. It is not correlated with the degree of maternal thrombocytopenia.
iii) Anesthesia considerations
(1) Maintenance of platelet counts >50 000 cells/mm^3 is considered safe for vaginal or cesarean delivery.
(2) Hemorrhage does not appear related to the degree of thrombocytopenia.
(a) Blood and blood products should be available for cesarean delivery.
(3) NSAIDs should be avoided in the postpartum period due to risk of worsening already jeopardized platelet function.
(4) In a study of 118 deliveries in women with ITP, 42 received neuraxial anesthesia with no complications reported in those with platelet counts <100 000 cells/mm^3 (62%), <75 000 cells/mm^3 (17%), or <50 000 cells/mm^3 (2%).[14]
(a) Based on these data and the rarity of neuraxial bleeding complications, no absolute platelet number may be determined safe or unsafe. The decision to place a neuraxial block must be individualized.

d) **Thrombotic thrombocytopenic purpura-hemolytic uremic syndrome (TTP-HUS)**[16]
   i) Rare condition in pregnancy (1/25 000), characterized by central nervous system abnormalities, fever, renal dysfunction, hemolysis, and thrombocytopenia
   ii) Treatment includes plasma exchange, hemodialysis, and transfusion.
   iii) If disease is severe, fetus viable, and preeclampsia cannot be excluded, delivery is appropriate.
   iv) Unlike preeclampsia/HELLP, hematologic abnormalities may progress beyond 3 days postpartum.

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Hemorrhage in the parturient, Chapter 112

e) **Disseminated intravascular coagulation**
   i) May occur in the setting of severe preeclampsia/HELLP syndrome or as a sequelae of hemorrhage
   ii) Anesthesia considerations
      (1) May require blood product replacement, supportive care, general endotracheal anesthesia if necessary
      (2) Due to risk of epidural hematoma, avoid removal of epidural catheters until coagulopathy is corrected.



Pregnancy is a hypercoagulable state mediated by an increase in many procoagulant factors, a decrease in fibrinolytic activity, and the presence of venous stasis and increased endothelial damage (Virchow triad).

f) **Acute thromboembolism**[4,12]
   i) Teleologically, the hypercoagulable state of pregnancy can be seen as preparation for blood loss of delivery.
   ii) The risk of venous thromboembolism is increased throughout pregnancy, with the highest risk occurring in the third trimester and peripartum. The increased risk of VTE extends to 6-8 weeks following delivery.
   iii) Diagnosis[4]
      (1) Symptoms and physical findings of deep venous thrombosis (DVT) and pulmonary embolus (PE) are relatively nonspecific and common during normal pregnancy, making the diagnosis challenging.
      (2) No findings on physical exam, ECG, or arterial blood gas have sufficient sensitivity to rule in or out the diagnosis of VTE.
      (3) Radiologic studies required for the diagnosis and treatment of thromboembolic disease should be used during pregnancy.
      (4) Venous ultrasound is the procedure of choice to evaluate suspected DVT.
      (5) *V/Q* scans, starting with perfusion studies, are used to diagnose a PE.
      (6) CT scans may be used to evaluate for PE, but carry higher radiation exposure than V/Q scans to maternal breast tissue, and should be used only when necessary. There is equal radiation risk to the fetus of V/Q scans and CT Angiography.[17]
      (7) D-Dimer, although normally elevated in late pregnancy and postpartum, may also be useful in assigning pretest probability.
   iv) Treatment of thromboembolic disease in pregnancy[4]
      (1) Immediate therapeutic anticoagulation should be initiated with either unfractionated heparin (UFH) or low molecular weight heparin (LMWH).
      (2) Therapeutic anticoagulation is continued for 3-6 months. If the diagnosis of VTE was made early in pregnancy and the patient completes 3-6 months of therapeutic dosing prior to delivery, the patient may be transitioned to prophylactic doses of UFH or LMWH for the duration of the pregnancy and postpartum period.
      (3) There are no randomized controlled trials to guide dosing or monitoring of anticoagulation in pregnant patients.



VTE is one of the most common causes of maternal death.[4]



Pregnancy increases the volume of distribution and renal clearance of UFH and LMWH, necessitating increased dose and frequency when used for therapeutic anticoagulation.

TABLE 110-4 Causes of Thrombophilia in Pregnancy

- Factor V Leiden
- Prothrombin gene mutation
- Deficiency of protein S, C, and/or antithrombin
- Systemic lupus erythematosus
- Antiphospholipid antibody syndrome
- Methyltetrahydrofolate reductase mutations

(a) Compared to heparin, LMWH has a longer half-life and therefore longer dosing interval.
(b) During pregnancy, the pharmacokinetics are altered such that twice daily dosing may be required.
(c) Warfarin is controversial in pregnancy because of its association with congenital malformations and fetal and neonatal hemorrhage, but it may be used while breast-feeding.
(d) Prophylaxis using either unfractionated heparin or LMWH should be recommended for women at high risk of VTE.



Patients with thrombophilias are often anticoagulated during pregnancy, labor, and delivery and require communication between care providers regarding the best plan for anesthesia and analgesia.

g) **Inherited and acquired thrombophilias (Table 110-4)**
i) May predispose women to adverse pregnancy outcomes and thromboembolic disease
ii) Complications are related to altered uteroplacental blood flow and include IUGR, preeclampsia, stillbirth, placental abruption.
iii) Women with a history of VTE should be evaluated for both inherited and acquired thrombophilias, while those with recurrent miscarriages and/or placental-mediated pregnancy complications should be evaluated for the presence of antiphospholipid antibody syndrome.

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Coagulopathies, Chapter 66

h) **Sickle cell anemia**
i) Higher risk of vasoocclusive complications because of the pregnancy-related increase in RBC-adhesive proteins
ii) This manifests in the placental circulation, causing areas of infarction, fibrosis, and villous necrosis, which may result in IUGR or stillbirth.
iii) Additional fetoplacental complications include preterm labor, low birth weight, and placental abruption.
iv) Maternal complications include recurrent urinary tract infections (UTI), painful crises, anemia, acute chest syndrome, splenic infarction, and preeclampsia.
v) Management during pregnancy
(1) Careful prevention of dehydration and frequent screening for UTI
(2) Screening for preexisting renal dysfunction and pulmonary hypertension, if not carried out prior to conception
(3) Prompt treatment of painful crises with hydration, O_2, analgesia, and investigations for an underlying cause, such as infection.
(4) Transfusion should be reserved for severe anemia or acute indications such as preeclampsia, sepsis, renal failure, acute chest syndrome, or anticipated surgery.

READ MORE
Sickle cell disease, hemoglobinopathies, and polycythemia vera, Chapter 65.

i) **von Willebrand disease**[18]
i) Genetic disease affecting von Willebrand factor (vWF) and factor VIII quality and quantity

ii) Several different types exist, with type I being the most common.
iii) Many patients have normal vWF and factor VIII levels during pregnancy, but levels fall postpartum, and bleeding may occur days to weeks later.
iv) Treatment will depend on the type of deficiency:
(1) DDAVP increases vWF and factor VIII levels in patients with types I and IIa. May be given to patients prior to surgery or delivery to decrease bleeding risk.
(a) DDAVP is not recommended in types IIb and III, as it will worsen platelet aggregation, thrombocytopenia, and contribute to a greater risk of bleeding.
(2) Commercial preparations of vWF and factor VIII replace deficient complexes of vWF and factor VIII in those unresponsive to DDAVP. An example currently available is Humate-P.[19]
(3) In the event of an emergency, both fresh frozen plasma (FFP) and cryoprecipitate contain vWF and factor VIII and can be used along with usual resuscitative efforts.[20]
v) Anesthetic concerns
(1) Every effort must be made to determine the type of vWD, as this will determine the method of treatment and optimization prior to delivery.
(2) Laboratory studies should include bleeding time, factor VIII level, vWF level, platelet count, prothrombin time (PT), activated prothrombin time (aPTT), and vWF function studies.
(3) Neuraxial analgesia is not necessarily contraindicated, especially in type I patients, but therapy should be individualized in consultation with a hematologist.



Patients with vWD may have a postpartum hemorrhage days to weeks after delivery, when factor VIII levels begin to fall to prepregnancy levels.



Along with several other procoagulant factors, vWF and factor VIII both increase to near-normal levels during pregnancy, making hemorrhage in women with type 1 vWD less common than in other subtypes.[20]

5) **Neurologic diseases**
a) **Multiple sclerosis (MS)**
i) MS is a chronic demyelinating disease affecting the central nervous system.
ii) The prevalence is 1/1000 in the United States. The peak age at diagnosis is 30 years. Women are more commonly affected than men.[2]
iii) Pregnancy may have a beneficial effect.
(1) Recent studies suggest that pregnancy reduces the likelihood of acquiring MS.[21]
(2) Nulliparous women have more disability due to MS than women who become pregnant, regardless of when MS presents.[22]
iv) Relapse rate is higher in the first 3 months postpartum and is not associated with breast-feeding or epidural analgesia.
v) Treatment of MS during pregnancy includes the use of corticosteroids, azathioprine, and interferon in select cases.
vi) Anesthetic considerations
(1) Increased temperature is associated with relapse in MS.[23] Every effort should be made to monitor temperature, especially in patients with epidural analgesia.
(2) There is a theoretical risk of damage to demyelinated nerves with local anesthesia delivered via spinal administration. Although there is not sufficient evidence to support this speculation, many practitioners prefer epidural over spinal analgesia and anesthesia for these theoretical reasons.

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Anesthesia and diseases of the nervous system, Chapter 55



Patients with MS do not have higher rates of pregnancy complications.[24]

vii) In patients with MS, document preexisting neurologic defects and use clinical judgment to determine if regional anesthesia is appropriate.

b) **Paraplegia**[12]

i) Women with spinal cord injury remain fertile and have specialized issues during pregnancy.

ii) Risk of complication depends on the level of injury.

(1) Pulmonary function may be affected and should be serially assessed with pulmonary function tests.

(2) Thermoregulation may be impaired, resulting in either hypothermia or hyperthermia.

(3) May require operative assistance to achieve vaginal delivery in the absence of maternal expulsive effort

(4) Ability to perceive labor pain is impaired in women with cord injuries below T5-T10.[25]

(5) Autonomic dysreflexia

(a) The principal concern in labor in paraplegic or quadriplegic women is autonomic dysreflexia or hyperreflexia.

(b) Occurs in 85% of women with spinal cord injuries in labor

(c) Similarly, 85% of patients with spinal cord injuries above T6 experience autonomic dysreflexia (AD). Although never completely absolved of the risk, those with lesions below T10 are less likely to experience the phenomenon.[26]

(d) Results in sympathetic hyperactivity in response to noxious stimuli below the level of the lesion

(e) Signs and symptoms

(i) Labile, severe HTN, arrhythmia, headache, loss of consciousness, facial erythema, diaphoresis above the level of the spinal cord injury, with dry, pale skin below the level of the injury

(ii) May wax and wane with contractions

(f) Anesthetic considerations[12]

(i) Epidural analgesia to the T10 level is recommended to prevent this syndrome.

(ii) Antihypertensive medications may be required.

1. In refractory cases, nitroprusside or IV magnesium may be considered.

c) **Seizure disorders**[28]

i) Most patients with epilepsy have uncomplicated pregnancies.

(1) The rate of obstetric complications, including spontaneous abortion, hyperemesis gravidarum, anemia, eclampsia, placental abruption, IUGR, stillbirth, and preterm delivery, is twofold higher.

ii) Medication therapy

(1) Preconception counseling is necessary to determine the best medication regimen due to the significant increase in fetal malformations seen with some antiepileptic drugs (AEDs).

(2) Fetal effects

(a) Infants of mothers with epilepsy are at higher risk for seizure disorder. The risk of birth defects is associated with the number and type of AEDs used during pregnancy.

(b) All AEDs can cause similar fetal dysmorphic features.

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Spine surgery, Chapter 121



Hemodynamic instability associated with autonomic dysreflexia may threaten uteroplacental perfusion and necessitate emergent delivery if not treated.[27]



Epidural analgesia is recommended for laboring parturients with spinal cord injuries, but assessment of the neuraxial blockade can be difficult. Placement of neuraxial analgesia may seem futile because they may not sense contractions or labor pain, but it helps prevent noxious stimuli that can trigger dangerous episodes of AD.



Seizure after 20 weeks of pregnancy in the absence of a known seizure disorder is eclampsia until proven otherwise. Treat with supportive measures and IV magnesium.[29]

(3) The physiologic changes of pregnancy lead to subtherapeutic levels of AEDs during pregnancy.
(a) Levels and therapeutic effect should be monitored as doses may need to be increased to prevent breakthrough seizures.
(b) Folate metabolism may be altered by AEDs, necessitating increased supplementation for 3 months prior to pregnancy and during the first trimester.
(c) Many AEDs inhibit vitamin K transport across the placenta, placing exposed neonates at risk for vitamin K deficiency and neonatal hemorrhage.

iii) Management of acute seizure during pregnancy
(1) If patient has a known seizure disorder, provide supportive care, use a first-line AED, and investigate precipitating cause.
(2) The use of IV benzodiazepines is first-line therapy in status epilepticus.
(3) Always consider the possibility of hypoglycemia causing the seizure and check blood glucose when appropriate.

iv) Intrapartum concerns[20]
(1) Labor and delivery may precipitate a seizure due to sleep deprivation, dehydration, and meperidine administration. AEDs should be continued during labor, and if not tolerated, a parenteral equivalent should be administered.
(2) Treat seizures with IV benzodiazepines during active labor.
(3) Administer O_2 and place the patient in left lateral position.
(4) Generalized seizures during labor may cause maternal hypoxia and prolonged fetal bradycardia.
(5) Cesarean delivery should be performed if frequent uncontrolled seizures occur or if the mother is unable to cooperate with labor.

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Asthma and reactive airway disease, Chapter 52

d) Postpartum considerations include ensuring safety in caring for a new infant.

6) **Pulmonary disease in pregnancy**

a) **Asthma**[12,30]

i) Most common pulmonary disease in pregnancy with 20%-36% of pregnant asthmatics experiencing at least one exacerbation during pregnancy.
(1) Managed with β-adrenergic agonists, inhaled corticosteroids, and mast cell inhibitors
(a) Systemic steroids are used in severe exacerbations.

ii) Exacerbations leading to acid-base changes yielding $pCO_2 > 35$ mm Hg or $pO_2 < 70$ mm Hg represent more severe compromise versus non-pregnant individuals due to the normal physiologic changes of pregnancy that enable O_2 shunting to the fetus.

iii) Respiratory support, including intubation, must be considered to maintain maternal and fetal oxygenation.



Respiratory compromise in the mother results in fetal heart tracing changes consistent with fetal hypoxia and acidosis.

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Obstructive sleep apnea, Chapter 54

b) **Obstructive sleep apnea (OSA)**[31,32]

i) OSA may be precipitated or exacerbated during pregnancy and impaired fetal growth may result from chronic hypoxia.
ii) OSA is associated with hypertensive disorders during pregnancy.
iii) Pregnant patients with known OSA should undergo a cardiac evaluation, including an echocardiogram, to rule out pulmonary hypertension.
iv) CPAP should be prescribed.



OSA patients who receive opioids after cesarean delivery are at risk for respiratory depression postoperatively and must be monitored closely.

7) **Obesity and pregnancy after bariatric surgery**[33,34]
   a) **Bariatric surgery in women of childbearing age is becoming more common.**
   b) **Rates of GDM and preeclampsia are lower following bariatric surgery.**
   c) **Cesarean delivery rates are lower, and neonatal outcomes are similar or better in women who undergo bariatric surgery vs obese women.**
   d) **Surgical complications such as bowel obstruction and intestinal hernias are a concern in pregnancies following bariatric surgery.**
   e) **Anesthetic considerations**
      i) Compared to lean women, obese women have a higher rate of anesthetic complications such as epidural failure (42% vs 6% of nonobese patients), inadvertent dural puncture, and more neuraxial placement attempts.[35]
      ii) As obese women are also at risk for difficult intubation, early placement of an epidural or an intrathecal catheter may obviate the need for emergent intubation.



In obese patients, it is helpful to place the patient in the lateral position prior to securing an epidural catheter in order to prevent catheter migration.[35]

8) **Pregnancy after transplant**[36,37]
   a) **Posttransplant patients are at risk for hypertensive disease and preeclampsia.**
   b) **Patients may require steroid therapy.**
      i) Patients receiving steroids are at risk of GDM and wound dehiscence and infection following cesarean delivery.
      ii) Stress dose steroids are recommended at the time of cesarean delivery but may also be considered with vaginal delivery.
   c) **Organ rejection does not appear to be exacerbated by pregnancy.**
   d) **Surgeons must be mindful of an abdominal kidney at the time of cesarean delivery if the patient has received a renal transplant.**
   e) **Fetal concerns include early pregnancy loss, congenital malformation due to teratogenic antirejection drugs, stillbirth, immunosuppression, prematurity, and low birth weight.**
   f) **Epidural labor analgesia is recommended to control pain and minimize a pain-induced sympathetic responses.**
9) **Dermatologic disorders (dermatoses of pregnancy)**[12]
   a) **Pruritic urticarial papules and plaques of pregnancy**
      i) Occurs first in abdominal striae but may spread to extremities and cause hives. The face, palms, and soles are usually spared.
      ii) There are no risks to the pregnancy with this condition, and it typically does not alter anesthetic management. Consideration should be given to avoid neuraxial techniques in affected areas to decrease the potential for bacterial contamination.
   b) **Intrahepatic cholestasis of pregnancy**[12,38]
      i) A disorder of intense pruritus, most commonly of the palms and soles, occurring in second and third trimesters of pregnancy
      ii) Results in an elevation in serum bile acid concentrations and occasionally elevated transaminases
      iii) Coagulation abnormalities are not typically observed and, if present, may represent vitamin K deficiency due to cholestasis or bile acid sequestrants.
      iv) Ursodiol (ursodeoxycholic acid) is the most commonly prescribed medication to reduce symptoms and bile salts.
      v) Spontaneous *in utero* death is a fetal complication of this disease.
         (1) Risk correlates with the level of bile acid elevation.
         (2) Induction of labor between 36 and 37 weeks is recommended to reduce the risk of term fetal demise.[38]
10) **Cardiac disease in pregnancy**[4,12]
   a) **General principles**

i) In caring for gravid patients with heart disease, one must determine the New York Heart Association (NYHA) functional class.
   (1) In patients with NYHA class I or II lesions, maternal and fetal mortality is ~0.4% and 2%, respectively.
   (2) The risk rises up to 7% in women and 30% in fetuses with class III or IV lesion.
   (3) Generally, regional anesthesia is appropriate for most patients with cardiac disease. Adequate pain control can decrease cardiac work, help avoid maternal tachycardia, and allow operative vaginal delivery.
   (4) Each cardiac lesion has unique requirements, and a multidisciplinary team, including obstetricians, anesthesiologists, and cardiologists, should formulate a detailed delivery plan for each patient with heart disease.

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Aortic valvular disease, Chapter 100



Passive second stage of labor (ie, no "pushing," also known as assisted second stage of labor) with operative vaginal delivery can assist in decreasing the cardiovascular stress associated with the delivery process.

ii) Labor increases cardiac output by ~45% above prelabor values, which can be extremely problematic in patients with poorly compensated cardiac disease.[12]
iii) After delivery, increases in preload due to the autotransfusion from the contracting uterus can add further stress on the heart, making the immediate postpartum period an extremely high-risk time for patients with cardiac disease.[12]
iv) The majority of patients do not require antibiotic prophylaxis.[39]
   (1) Patients with complex congenital heart disease may require antimicrobial prophylaxis for endocarditis for vaginal or cesarean delivery.
   (2) Consultation with the patient's cardiologist and/or the American College of Cardiology (ACC)/ American Heart Association (AHA) guidelines is recommended.[39]

b) **Valvular heart disease**
i) Due to the physiologic 30%-50% increase in plasma volume with gestation, regurgitant lesions are better tolerated than stenotic lesions.
ii) Patients with severe stenotic heart defects will not tolerate sudden drops in systemic vascular resistance.
   (1) If used, epidural medications must be administered very slowly.
   (2) Intrathecal local anesthetics are typically avoided due to the possibility of rapid sympathectomy and tendency to result in rapid loss of preload. Careful consideration should be given to the use of a neuraxial opioid–only technique.
   (3) For cesarean delivery, most patients receive carefully controlled epidural anesthesia or general anesthesia.

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Mitral valve disease, Chapter 99

iii) Mitral stenosis[12,40]
   (1) Pregnancy considerations
      (a) Risks associated with pregnancy are greatest in the third trimester or the puerperal period, correlating with the increased plasma volume seen in these periods. Risks include atrial arrhythmias, thromboembolic events, and pulmonary edema.
   (2) Maternal risks directly correlate with the severity of stenosis, based upon the area of the mitral valve.
   (3) One-third of patients with NYHA class I or II heart failure experience adverse cardiac complications (pulmonary edema, arrhythmias), while two-thirds experience such complications if their functional class is III or IV.
   (4) Rate of maternal and fetal mortality in patients with NYHA class III-IV is up to 20% and 30%, respectively.



The immediate postpartum period is an extremely high-risk time for patients with cardiac disease. Patients should have close monitoring with consideration for admission to the intensive care unit.

(5) Treatment of mitral stenosis
(a) β-Adrenergic blockade, gentle diuresis, and restriction of physical activity can help control heart rate and symptoms in moderate to severe mitral stenosis.
(b) Atrial fibrillation should be aggressively treated with digoxin or β-adrenergic antagonists to control heart rate, as well as anticoagulation to prevent embolization.
(c) In patients refractory to medical treatment, percutaneous balloon valvuloplasty with pelvic shielding can be considered.
(6) Intrapartum management goals
a) Avoid tachycardia and maintain preload by avoiding hypotension.
(7) Anesthetic considerations
(a) Adequate pain control can help prevent tachycardia.
(b) Intrathecal and epidural anesthesia carry a risk of rapid decrease in SVR, leading to hypotension. These risks can be minimized by careful titration and use of very dilute local anesthetics or opioid-only solutions.
(c) If hypotension develops, phenylephrine is preferred to avoid tachycardia.

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Aortic valvular disease, Chapter 100

iv) Aortic stenosis (AS)
(1) In the pregnant population, most often secondary to congenital bicuspid aortic valves.
(2) Bicuspid aortic valves can be associated with coarctation of the aorta, which can predispose patients to aortic aneurysms and dissection.
(3) Pregnancy considerations
(a) Patients have a fixed stroke volume, so variation in cardiac output is determined by heart rate only.
(i) Bradycardia leads to hypotension, and tachycardia decreases ventricular filling time and coronary perfusion, leading to hypotension, syncope, and risk of myocardial ischemia.



Atrial fibrillation decreases the amount of blood volume attributed to the SV by the left atrial kick and is very poorly tolerated by patients with AS.

(4) Severity of AS is defined by valve area and/or the peak gradient across the valve.
(5) Treatment of AS
(a) Severe AS is associated with increased maternal and fetal morbidity and mortality.
(b) Ideally, the aortic valve should be replaced or repaired prior to conception, but intervention can be considered midgestation if the patient is symptomatic and resistant to medical therapy.
(6) Intrapartum management
(a) Passive second stage with operative vaginal delivery is preferred.
(b) Hemorrhage prevention and treatment should be aggressive in order to avoid the detrimental effects of tachycardia and hypotension.
(7) Anesthetic considerations
(a) Regional anesthesia is generally avoided in severe cases. The decision to use neuraxial techniques should be individualized to the patient. If regional anesthesia is used, hypotension should be strictly avoided.
(b) Avoid vasodisslatory agents.

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Mitral valve disease, Chapter 99

v) Mitral regurgitation
(1) Mitral valve prolapse, affecting ~5%-15% of women of childbearing age, is the most common etiology.

(2) Pregnancy considerations
   (a) Increased blood volume and decreased systemic vascular resistance during pregnancy promotsses forward flow across the regurgitant valve.
   (b) Pregnancy is generally well tolerated, and most patients require no medications.
   (c) Severe cases can be associated with markedly dilated left atrium, atrial fibrillation, and pulmonary edema, which can be treated with diuretics.

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Aortic valve disease, Chapter 100

vi) Aortic regurgitation
   (1) Pregnancy considerations
      (a) Regurgitant lesions are often well tolerated in pregnancy. However, a sudden increase in afterload, such as that seen in preeclampsia with severe HTN, can lead to acute cardiac decompensation.
   (2) Treatment
      (a) Hydralazine is the antihypertensive medication of choice.
      (b) Treatment of heart failure involves digoxin, diuretics, and hydralazine. Angiotensin-converting enzyme (ACE) inhibitors are contraindicated in pregnancy but can be used postpartum.

vii) Mechanical heart valves
   (1) Mechanical valves are often used in younger patients (as opposed to bioprosthetic valves) during valve replacement because of their prolonged durability.
   (2) Pregnancy considerations
      (a) Mechanical heart valves carry a very high risk of thrombotic disease, especially in association with the hypercoagulable state of pregnancy.
   (3) Treatment[6]
      (a) Warfarin is a known teratogen, and controversy surrounds its use in pregnancy, even in these very high-risk patients.
      (b) Patients with first-generation mechanical heart valves are at significantly increased risk of clots and may be offered warfarin (to maintain an INR of 2.5-3.5) for 35 weeks of gestation, followed by IV unfractionated heparin. An alternative option may include subcutaneous heparin (UFH or LMWH) for the first 12 weeks of pregnancy to avoid potential teratogenicity, followed by warfarin until 35 weeks at which time they are transitioned to intravenous heparin
         (i) Cesarean delivery should be performed if delivery is indicated while on warfarin.
      (c) A bioprosthetic valve presents a lower risk of thrombosis, and low-dose aspirin (75-100 mg) is recommended throughout pregnancy.[41]
   (4) Intrapartum management
      (a) Most authorities recommend discontinuing anticoagulation during labor, with reinitiation of IV heparin 4-6 hours following an uncomplicated delivery.
      (b) In rare cases, reversal of anticoagulation may be required in patients who require emergent cesarean delivery. Consultation with a hematologist and potentially a cardiac surgeon is suggested in this situation.
   (5) Anesthetic considerations
      (a) The decision to perform neuraxial procedures on patients who require anticoagulation should be individualized based on laboratory data and the risks and benefits to a particular patient.

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Regional anesthesia in the anticoagulated patient, Chapter 171

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Congenital heart disease, Chapter 85

(b) Many patients on chronic anticoagulation will not be candidates for regional anesthesia/analgesia. For labor, these patients should be offered IV opioids, and preparations should be made for general anesthesia should cesarean delivery be necessary.

c) **Congenital heart disease**

i) Coarctation of the aorta

(1) Pregnancy considerations

(a) Severity of aortic coarctation is based upon the difference in blood pressure (BP) between the upper and the lower extremities, with a gradient >20 mm Hg indicating a severe lesion.

(b) Risks of pregnancy include worsening HTN, heart failure, aortic dissection and rupture, as well as rupture of any associated cerebral aneurysms.

(c) Patients with unrepaired aortic coarctation have a mortality rate up to 9%.

(2) Intrapartum management

(a) Severe fluctuations in BP should be prevented by providing adequate pain control.

(b) Consider passive second stage and operative vaginal delivery if cesarean delivery is not performed.

ii) Left-to-right shunting lesions

(1) Pregnancy considerations

(a) Due to volume expansion associated with pregnancy, left-to-right heart shunts (ventricular septal defects [VSDs], atrial septal defects, and patent ductus arteriosus) are generally well tolerated during pregnancy.

(b) Risks of pregnancy are related to shunt size and degree of pulmonary hypertensionss.

(c) Paradoxical embolization across the shunt can cause stroke, so these patients are typically prescribed low-dose aspirin.

(d) Postpartum hemorrhage can lead to acute hypotension with transient shunt reversal and cyanosis.

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Pulmonary hypertension, Chapter 48

(2) Eisenmenger syndrome

(a) Long-standing left-to-right shunt can lead to pulmonary HTN and shunt reversal (Eisenmenger syndrome).

(b) Maternal mortality in the setting of Eisenmenger syndrome is up to 50%, with the greatest risk of death within the immediate postpartum period. Pregnancy is considered to be contraindicated in patients with this condition.

(c) Antepartum management of these patients involves bed rest, supplemental O_2, anticoagulation, and prolonged hospitalization.

(d) Intrapartum management

(i) These patients are at extremely high risk for maternal death and require a multidisciplinary approach.

(ii) There is usually no benefit to cesarean delivery, and vaginal delivery is preferred.

(iii) Patients should receive slow epidural anesthesia or intrathecal narcotics and aggressive avoidance of postpartum hemorrhage.

(iv) If cesarean delivery is required, patients typically receive general anesthesia, possibly with inhaled nitric oxide to mitigate increases in pulmonary vascular resistance. An individualized plan should be made for each patient.

iii) Right-to-left shunting lesions

(1) Tetralogy of Fallot (TOF)[6]

(a) Pregnancy considerations
  (i) Patients with corrected TOF and good residual functional status usually tolerate pregnancy well.
  (ii) Cardiac complications in corrected lesions during pregnancy can include symptomatic right-sided heart failure and arrhythmias.

(b) Uncorrected TOF[6]
  (i) May clinically deteriorate during pregnancy with risk of both maternal and fetal morbidity and mortality.
  (ii) Cardiac complications can include increased right-to-left shunting via a residual VSD resulting in worsening cyanosis, biventricular heart failure, arrhythmias, and cerebral vascular accidents from paradoxical emboli.

(c) Prophylaxis for bacterial endocarditis is recommended.[31]



Auxiliary means to pace a patient with complete heart block, including both transcutaneous and transvenous pacing, should be available during labor and delivery.

d) **Complete heart block**[4]
  i) Patients with complete heart block, with or without a pacemaker, may become symptomatic, particularly during labor and delivery.
  ii) Due to the physiologic increase in heart rate during pregnancy, the pacing heart rate is typically increased by 10%-20% in pregnant women.

e) **Ischemic heart disease**[4]
  i) Historically uncommon in women of childbearing age, the incidence is increasing during pregnancy as women choose to delay childbearing and prevalence of metabolic syndrome (or cardiac risk factors) is more common among this older demographic.
  ii) Maternal mortality rate for acute myocardial infarction (MI) is 7%-35%.
  iii) Medical therapy is the same in the pregnant patient and includes O_2, aspirin, β-adrenergic antagonists, heparin, and nitrates.
  iv) Thrombolytic agents have been used successfully in pregnancy but are associated with placental abruption and hemorrhage, especially when used in the peripartum period.
  v) Management of the pregnant patient with previous MI can include limiting physical activity and continuation of most cardiovascular medications.
  vi) Up to 50% of these patients will experience some cardiac complications during pregnancy, ranging from angina to heart failure.
  vii) Vaginal delivery, with adequate pain control and passive/assisted second stage, is the delivery method of choice.

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Ischemic heart disease, Chapter 98.



Delivery within 2 weeks of an acute MI has been associated with a 50% maternal mortality due to heart failure or arrhythmias.

f) **Peripartum cardiomyopathy**
  i) The diagnosis of peripartum cardiomyopathy is made when a patient develops new-onset heart failure (defined as an ejection fraction of <45%) during the last month of pregnancy, or within 5 months postpartum, in the absence of a determinable cause of heart failure.
  ii) The etiology remains elusive, but known risk factors include advanced maternal age, multiple gestation, and African descent.
  iii) Treatment includes medications that will reduce preload (diuretics), reduce afterload (vasodilators such as hydralazine or nitroglycerin), and provide inotropy (digoxin or dobutamine).
    (1) ACE inhibitors are contraindicated in pregnancy but can be initiated postpartum and are safe to use in breast-feeding.

(2) Anticoagulation should also be considered.

iv) Approximately 50% of patients have marked improvement in clinical symptoms and left ventricular function within 6 months of diagnosis.

CHAPTER SUMMARY FOR PREGNANCY AND COEXISTING DISEASE

General Principles	Understand different underlying disease processes and how they are affected by physiologic changes of pregnancy.
	Consultation with an obstetrician and potentially multiple specialists may be required for optimal care.
	Mode of delivery should be discussed as most patients benefit from vaginal delivery but may require a passive second stage.
Chronic (Preexisting) Hypertension	Pose risks to both mother and fetus. Contrasts from gestational hypertension based on its onset and underlying mechanism of disease.
Diabetes Mellitus	One of the most common coexisting diseases.
	Patients are at high risk for pregnancy complications and cesarean delivery.
Other Endocrine Disease	Thyroid disorders carry significant fetal risks if untreated.
	Pituitary disorders may cause hormonal imbalances and require stress dose steroids.
	Adrenal disorders may include pheochromocytoma.
Neurologic Disease	Paraplegia increases the risk of autonomic dysreflexia.
	Patients with multiple sclerosis are sensitive to hyperpyrexia but generally tolerate pregnancy well.
	Epilepsy medications increase the risk of fetal anomalies.
Hematologic Disease	Thrombophilias are common in pregnancy.
	VTE is one of the leading causes of maternal death worldwide.
	Patients may present with long-term anticoagulation.
	In patients with thrombocytopenia, determine cause and individualize management plans.
Pulmonary Disease	"Normal" $PaCO_2$ levels in a pregnant asthmatic patient may represent severe respiratory compromise due to the physiologic changes of pregnancy.
	OSA is common and should be treated with CPAP.
	Patients with OSA should be carefully monitored after neuraxial opioid administration.
Cardiac Disease	Increasing in frequency in the pregnant population.
	Patients may present with valvular disease, congenital heart disease, and/or ischemic disease.
	Management plans should consider the increased cardiac work with labor and the immediate postpartum period.
	Individualized management often includes a multidisciplinary approach.

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111 Obstetrical Emergencies

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Emergencies can arise at any point during labor and delivery and may be life threatening. While certain high-risk conditions may raise the incidence of delivery complications, many obstetrical emergencies can occur at any time. Obstetrical emergencies are a unique challenge in that they may threaten the well-being of the fetus, mother, or both simultaneously. It is the anesthesia provider's responsibility to understand obstetric complications and respond to them in a timely and appropriate manner. This chapter was written as a collaborative effort between an obstetrician and anesthesiologists in order to explain the approach of each specialty to various emergencies.

1) **Umbilical cord prolapse**
   a) **General principles**
      i) Umbilical cord prolapse (UCP) is a rare obstetrical emergency that requires immediate delivery, generally by cesarean section. The incidence is 1.4-6.2 per 1000 pregnancies.[1]
   b) **Pathophysiology**
      i) UCP occurs when the umbilical cord presents prior to the presenting part of the fetus or alongside the fetus.
      ii) Results in compression of the umbilical cord and can lead to fetal hypoxia.
      iii) UCP can be classified as *overt* (where the cord is palpated) or *occult* (where the cord is not palpated but fetal bradycardia is observed).
      iv) The first indication of a UCP is palpation of the cord following a procedure (eg, artificial rupture of membranes), fetal bradycardia, or moderate to severe variable decelerations following a normal fetal tracing.
   c) **Risk factors**
      i) Prematurity, fetal malpresentation, multiple gestation, polyhydramnios, multiparity, and rupture of membranes
   d) **Treatment and management**
      i) The standard treatment of UCP is immediate cesarean delivery (CD).
         (1) Maneuvers such as elevating the fetal head with a hand, change in maternal position, and distention of the bladder using a Foley catheter may be useful to elevate the presenting part off the cord while a CD can be begun.



Sudden fetal bradycardia after rupture of membranes is concerning for umbilical cord prolapse.

   e) **Anesthetic management**
      i) CD must be immediate.
      ii) General anesthesia is often required due to the urgency of the situation.
      iii) If the patient's airway is not favorable, regional anesthesia or awake intubation may be necessary to avoid compromise of the maternal condition.
      iv) It must be emphasized that time is of the essence for a favorable neonatal outcome, and one must carefully weigh the benefit to the mother of taking more time to achieve optimal regional anesthesia.

2) **Uterine inversion**
   a) **General principles**
      i) Puerperal uterine inversion is a rare condition with an incidence of 1/2000-1/50 000 births.[2]
   b) **Pathophysiology**
      i) Uterine inversion is a partial or full inversion of the body of the uterus.
      ii) Diagnosis
         (1) Patients present with postpartum hemorrhage, shock, shock out of proportion to blood loss, or palpation of pelvic mass during bimanual examination.
         (2) When the uterus does not protrude out of the vagina, the diagnosis can be missed, leading to a subacute or chronic inversion.
   c) **Risk factors**
      i) Fundal placenta (present in 10% of pregnancies), adherent placenta, a short umbilical cord, excess traction on the cord, fundal pressure, and use of oxytocin
      ii) The greatest risk appears to be in primiparous women with fundal placentas.[3]
      iii) Active management of the third stage of labor (placental delivery) may decrease the incidence of uterine inversion.[4]
   d) **Treatment and management**
      i) Treatment depends on prompt recognition.
      ii) Postpartum hemorrhage and shock should be managed with IV fluids and blood product replacement.
      iii) The uterus must be replaced into the pelvic cavity.
         (1) Oxytocin should not be given.
         (2) The placenta should be left *in situ* until the uterus is replaced.
         (3) The obstetrician will attempt to grasp the uterus and push it back into the vagina and pelvis (Johnson maneuver).
         (4) If this is not successful, uterine relaxation is necessary.
            (a) Options for uterine relaxation include:
               (i) Terbutaline 0.125-0.25 mg IV, magnesium sulfate 2 g IV, nitroglycerin 25-100 mcg IV, or nitroglycerin 0.4 mg SL[5]
            (b) General anesthesia with a high-dose volatile agent can be used but will likely require intubation and airway protection.[6]
         (5) Once the uterus is repositioned
            (a) The physician's hand should be kept inside until oxytocin is started and the uterus contracts.
            (b) The placenta can be removed following uterine replacement.
            (c) The uterine cavity should be manually inspected for retained placenta and uterine rupture.
         (6) Rarely, the uterus cannot be manually returned to the pelvis, and an operative approach is indicated. This is most commonly through an abdominal laparotomy.
   e) **Anesthetic management considerations**
      i) Uterine inversion can be associated with hemorrhage and shock.
      ii) Attention should be paid to IV access, blood product replacement if necessary, uterine relaxation to facilitate replacement into the pelvis, and patient comfort during the procedure. Administration of broad-spectrum antibiotics should be considered.
      iii) If the uterus is not easily replaced, transport to the operating room should be considered to facilitate monitoring and visualization.

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Hemorrhage in the parturient, Chapter 112.



Nitroglycerin, administered IV or sublingual, is helpful for uterine relaxation for many obstetrical emergencies.

iv) Nitroglycerin 50-100 mcg is the first-line agent to relax the uterus and facilitate replacement.
v) If other measures fail, administration of volatile anesthetic agents (eg, sevoflurane) may assist with replacement of the uterus into the pelvis.
vi) If these measures are necessary, hemodynamic support and potentially blood product replacement is critical.

3) **Shoulder dystocia**
a) **General principles**
i) The incidence of shoulder dystocia is 0.6%-1.4% of all deliveries and increases with increased birth weight.[7] The incidence of shoulder dystocia is 3%-10% when neonatal weight is between 4000 and 4500 g and 8%-24% when >4500 g.[8]
b) **Pathophysiology**
i) Following the delivery of the neonatal head, the shoulders become impacted against the symphysis pubis and umbilical cord compression occurs. Increased shoulder-to-head and chest-to-head proportions are associated with shoulder dystocia.[9]
ii) Fetal complications
(1) Asphyxia, meconium aspiration, plexus injuries (Erb, Duchenne, Klumpke), and injuries to clavicle or humerus
iii) Maternal complications
(1) Postpartum hemorrhage, vaginal lacerations, and ruptured uterus
c) **Risk factors**
i) Many patients have no identifiable risk factors.
ii) Risk factors
(1) Large gestational weight and gestational diabetes mellitus, advanced maternal age, maternal obesity, postterm pregnancy, history of prior dystocia, multiparity, postterm gestation, history of macrosomic infant, labor induction, and prolonged second stage of labor.
(2) Forceps and vacuum assistance may also increase the risk of shoulder dystocia.
d) **Treatment and management**
i) Prompt recognition followed by initiation of the following steps by the obstetrician to relieve the impacted fetal shoulder
(1) Call for help, perform episiotomy (controversial), drain maternal bladder.
(2) Perform suprapubic pressure and McRoberts maneuver.
(a) McRoberts maneuver is hyperflexion and abduction of hips causing cephalad rotation of symphysis pubis and flattening of the lumbar lordosis that frees the impacted shoulder.
(3) Maneuvers attempted at disimpacting the anterior shoulder. One example is the corkscrew maneuver that involves rotating the anterior shoulder posteriorly to release the impacted shoulder.
(4) Perform Zavanelli maneuver, which is an attempt to replace the fetal head into the uterus followed by CD.
e) **Anesthetic considerations**
i) Attention should be paid to minimizing second-stage motor block and optimal pain management.
ii) A high index of suspicion should exist in patients who have a high estimated fetal weight (EFW) or other risk factors for shoulder dystocia. The anesthesia provider should be prepared for the possibility of CD and perform a preoperative evaluation of the patient while in labor before imminent delivery if possible.
f) **Prevention**
i) Prevention may not always be possible.

ii) A multidisciplinary management plan is helpful to efficiently treat dystocia.
iii) Elective CD is considered for EFW 4500 g in diabetic women,[10,11] EFW >5000 g, and women with a prior history of shoulder dystocia.

4) **Trial of labor after cesarean section (TOLAC)/vaginal birth after cesarean section (VBAC)/uterine rupture**

a) **General principles**
i) Incidence of TOLAC
(1) Has varied overtime with an increase from 3% in 1980 to 31% in 1998 and back down to 9.2% in 2004.[12-14]
(2) These changes in incidence reflect political, medical, legal, and patient/practitioner preferences.
ii) The incidence of uterine rupture due to a prior CD varies depending on uterine scar type.
(1) Rate of rupture with a prior classical or T-shaped uterine incision is 4%-9%.
(2) Rate of rupture with a prior low vertical incision is 1%-7%, and a low transverse incision is 0.2%-1.5%.[15-17]



Patients with a prior midline or T-shaped uterine incision have a significantly higher rate of uterine rupture with labor versus those with a prior low transverse incision.

b) **Pathophysiology**
i) Uterine rupture may occur at any time during the pregnancy but most commonly during labor.
ii) With rupture of the scar and the uterus, the placenta may separate causing fetal hypoxia.
(1) Generally, the first recognizable sign of uterine rupture is fetal heart rate abnormalities (70%) but may also include maternal pain, maternal hypotension, loss of fetal station, and bleeding.
iii) Candidates for a TOLAC include those with a history of one or two prior CD, clinically adequate pelvis, and no other uterine scars or previous rupture. Similarly, there must be physicians immediately available to perform CD and immediate availability of an anesthesiologist.[15]
iv) The American College of Obstetricians and Gynecologists (ACOG) encourage attempts at TOLAC to reduce the CD rate. Calculators exist to estimate chance of successful VBAC.[18]



Since a uterine rupture is a life-threatening emergency for mother and baby, constant communication with obstetricians and nurses regarding progress of labor and adequacy of the epidural catheter is essential for any patient attempting a TOLAC.

c) **Risk factors for uterine rupture during a TOLAC**
i) Age >30, more than one prior CD, history of postpartum fever and/or chorioamnionitis with prior CD, interdelivery interval <18-24 months from prior CD, and dysfunctional labor

d) **Treatment and management**
i) Treatment involves prompt recognition followed by immediate CD.
ii) If the rupture is large and associated with hemorrhage, cesarean hysterectomy may be required.

e) **Prevention**
i) Prevention may not always be possible.
ii) Patients should be appropriately counseled prior to undergoing a TOLAC.
iii) Prostaglandins should not be used in induction of labor during TOLAC as this increases the rate of uterine rupture.
iv) TOLAC should only be performed in hospitals where CD is immediately available.



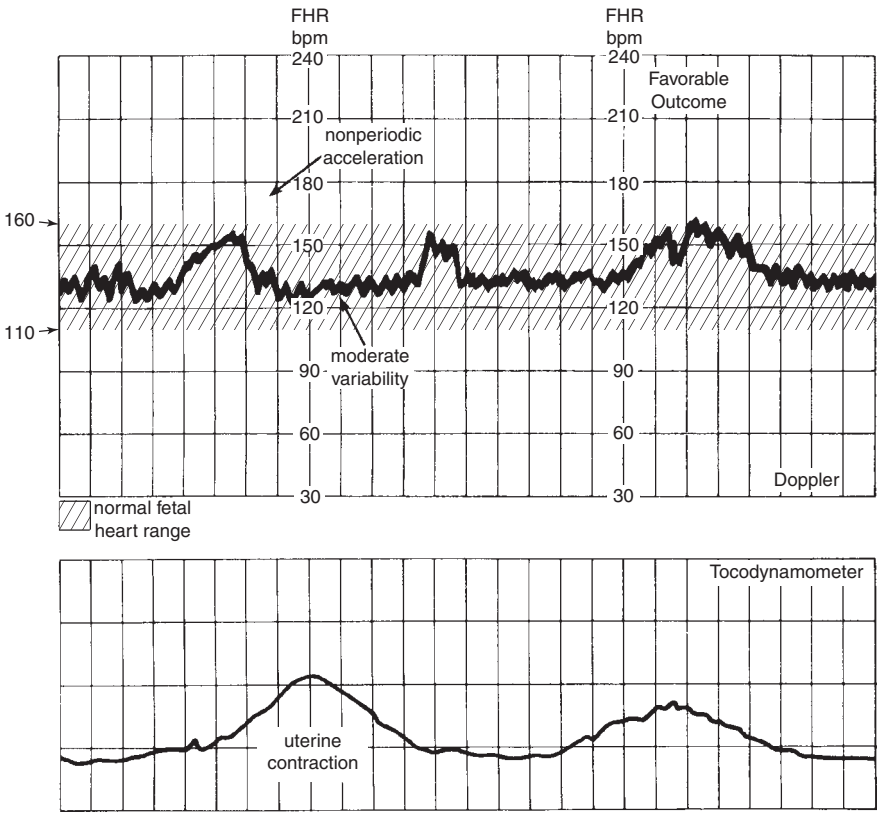
In any TOLAC patient with an epidural having severe breakthrough pain, a uterine rupture should be suspected.

f) **Anesthetic considerations**
i) Early anesthetic evaluation is recommended with special attention paid to the airway examination and counseling regarding early epidural placement.

ii) Although a well-functioning epidural catheter is optimal, there may not be time to dose the catheter for CD and emergent general anesthesia may be necessary.
iii) If a cesarean hysterectomy is required, bleeding could be profuse and mandate invasive monitoring, large-bore IV access, and blood transfusion.

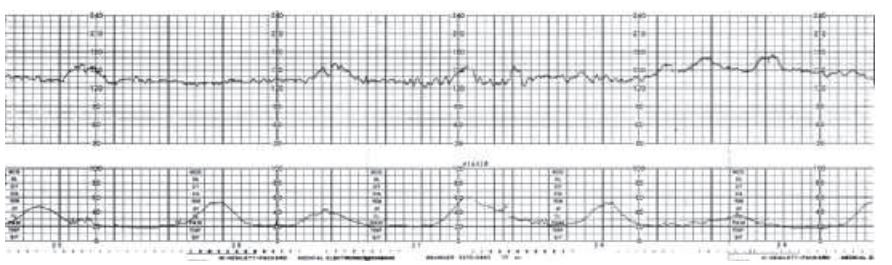
5) **Nonreassuring fetal heart rate (FHR) tracing (fetal distress)**
   a) **Fetal well-being is often determined by FHR monitoring.**
   b) **Criteria to determine fetal status**
      i) Historically, providers have not used the same criteria or definitions to describe fetal distress and FHR.
      ii) There has been dispute over the use of the term *fetal distress*, resulting in ACOG recommending using the term *nonreassuring FHR tracing*.
      iii) The National Institute of Child Health and Human Development (NICHD) Research Planning Workshop on electronic fetal monitoring has developed guidelines for the interpretation of fetal monitoring.[19,20]
   c) **Pathophysiology**
      i) The purpose of fetal monitoring is to distinguish FHR patterns associated with abnormal fetal acid-base status and that are associated with a physiologically compensated fetus.
      ii) The fetus responds to acute hypoxia by redistributing its blood flow to maintain cardiac output while protecting vital organs, decreasing its consumption of O_2, and undergoing anaerobic glycolysis.
      iii) This condition is readily reversible and may sustain a fetus for 30 minutes.
      iv) After 30 minutes or during more severe hypoxia, cardiac output will decrease, resulting in decreased blood flow to the brain and the heart, and hypoxic tissue damage may occur.
   d) **Categories of FHR tracings (Figs. 111-1 to 111-3)**
      i) The NICHD has broken down FHR patterns into three categories: I, II, and III.[21,22]
      ii) Category I FHR tracings
         (1) Normal
         (2) Predictive of normal fetal acid-base status
         (3) Characteristics include:
            (a) Baseline rate of 110-160 bpm
            (b) Moderate baseline FHR variability
               (i) Variability is defined as fluctuations in baseline of two cycles per minute or greater and can be determined visually as the amplitude of the peak to trough in beats per minute.
            (c) Absence of late or variable decelerations
            (d) Early decelerations may or may not be present.
            (e) Accelerations may or may not be present.
      iii) Category II FHR tracings (Fig. 111-4)
         (1) Indeterminant
         (2) Not always predictive of fetal acid-base status
         (3) Encompasses all FHR tracings not category I or III, including:
            (a) Bradycardia not accompanied by absent baseline variability
            (b) Tachycardia
            (c) Minimal or marked baseline variability; absent baseline variability without recurrent decelerations
            (d) Absence of accelerations following fetal scalp stimulation
            (e) Recurrent variable decelerations with minimal or moderate baseline variability
            (f) Prolonged decelerations (>2 minutes but <10 minutes), recurrent late decelerations with moderate baseline variability, variable decelerations with a slow return to baseline, overshoots, or "shoulders"

FIGURE 111-1 Characteristics of a Category I (Reassuring) FHR Tracing



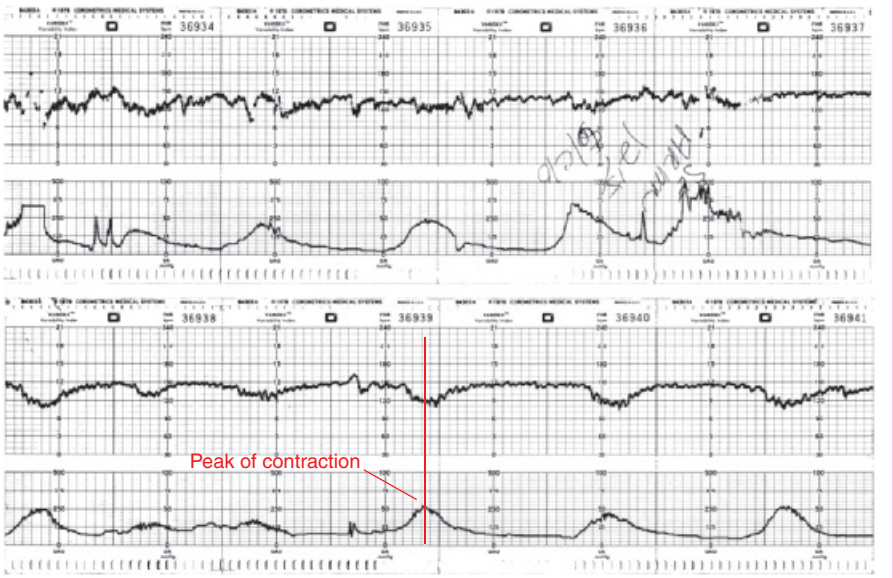
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FIGURE 111-2 Category I (Reassuring) FHR Tracing



FHR is at the **top** and uterine contractions are recorded at the **bottom**.
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FIGURE 111-3 Category I: Early Decelerations



The nadir of the deceleration is at the peak of the uterine contraction (*line drawn*).
Adapted with permission from Cabaniss ML, Ross MG, eds. *Fetal Monitoring Interpretation*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.

iv) Category III FHR tracings (**Fig. 111-5**)
  (1) Abnormal
  (2) Predictive of abnormal fetal acid-base status
  (3) Characteristics include:
    (a) Absent baseline FHR variability and any of the following:
      (i) Recurrent late decelerations
      (ii) Recurrent variable decelerations
      (iii) Bradycardia
    (b) Sinusoidal pattern

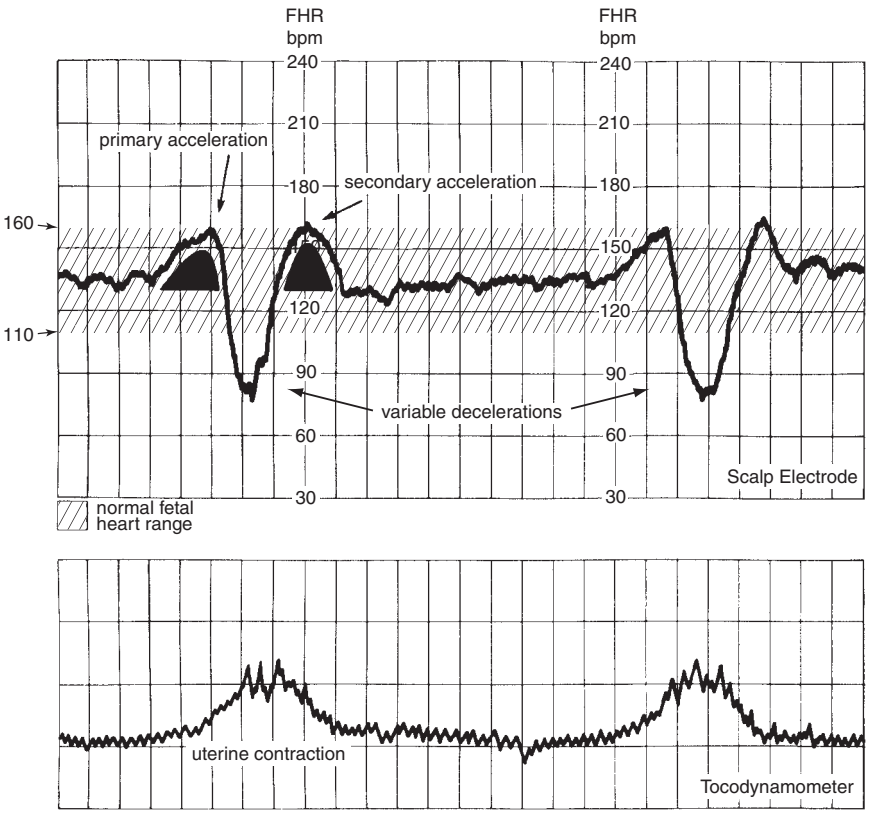
e) **Risk factors for fetal distress**
  i) Preterm infants, multiple pregnancies, induction of labor, oligohydramnios, and pregnancies complicated by intrauterine growth restriction (IUGR), HTN, or type I DM

f) **Treatment and management**
  i) Most practitioners advise using either continuous or intermittent fetal monitoring throughout labor.
  ii) Resuscitative measures aimed at increasing oxygen delivery can be performed.
    (1) Repositioning the patient, increasing IV fluids, treating hypotension (especially after epidural or spinal analgesia), administering tocolytic agents to stop contractions, and administering supplemental oxygen
  iii) If vaginal delivery is remote, CD is performed when a nonreassuring FHR is diagnosed.



It is in the anesthesia provider's best interest to become familiar with normal and nonreassuring FHR tracings. This allows for better communication on the labor ward and decision-making in urgent situations.

FIGURE 111-4 Category II: Characteristics of Variable Decelerations



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iv) If possible, delivery may be expedited with forceps or vacuum the setting of nonreassuring FHR.

g) **Prevention**

i) Optimizing the maternal condition is the best way to prevent fetal distress. This includes avoiding aortocaval compression, hypotension, and acidosis.

ii) Consider early epidural placement in high-risk pregnancies.

iii) Continuous fetal monitoring is generally recommended when labor induction medications are administered (oxytocin and/or prostaglandins) in order to avoid iatrogenic causes.

6) **Retained placenta**

a) **Pathophysiology**

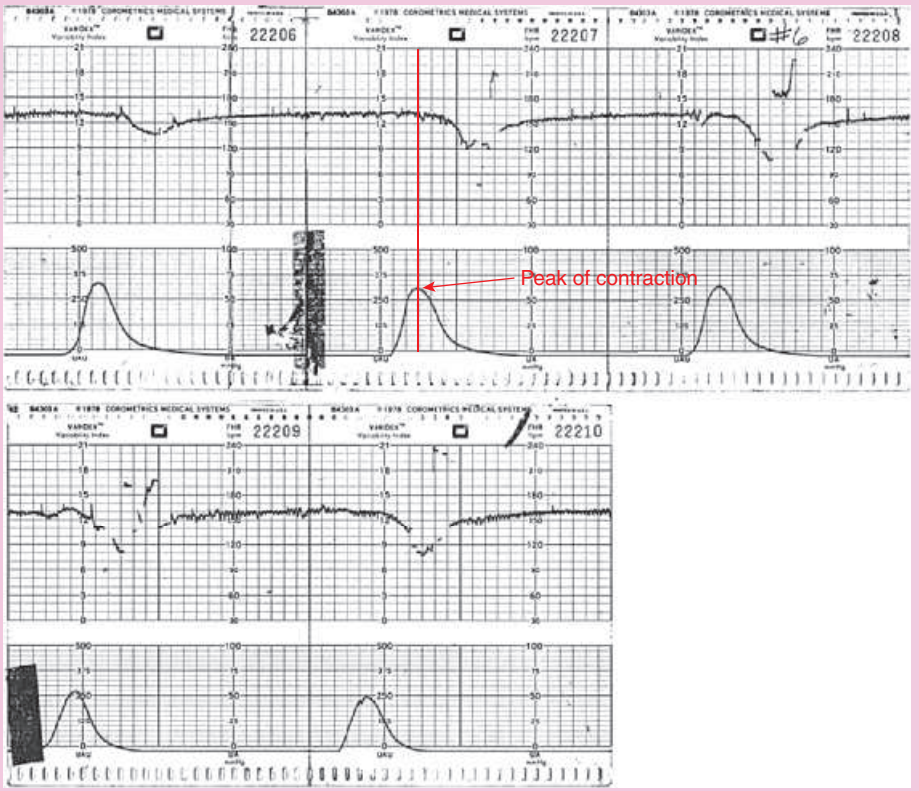
i) The third stage of labor is defined as the time from delivery of the infant to the delivery of the placenta.

(1) A normal third stage of labor is <30 minutes.

(2) After 30 minutes, manual removal of the placenta may be attempted.

(3) Removal should be attempted prior to 30 minutes in the presence of postpartum hemorrhage.

FIGURE 111-5 Category III: Late Decelerations



The nadir of the deceleration is *after* the peak of the uterine contraction (*line drawn*).
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ii) If the majority of the placenta has delivered, but uterine bleeding continues, the patient must be evaluated for retained placental fragments.

b) **Risk factors for retained placenta**
   i) Advanced maternal age, previous uterine curettage, premature rupture of membranes, preterm delivery (especially in the second trimester), history of retained placenta[23,24]

c) **Treatment/management**
   i) Initial treatment is gentle umbilical cord traction and uterine massage.
   ii) Oxytocin and/or prostaglandins may also be administered IM, IV, or vaginally.
   iii) If initial treatment is unsuccessful, the placenta may be removed manually.
      (1) A tocolytic drug or uterine relaxant such as nitroglycerin may be required for uterine relaxation.
   iv) Ultrasound may be performed to look for placental fragments.
      (1) If suspicious for placental fragments, a uterine curettage may be performed using a large curette or a manual sweep may be attempted.



In cases of retained placenta, special consideration must be given to the patient's hemodynamic status.

d) **Anesthetic considerations**

i) The patient may require additional pain control for removal of retained placenta.
ii) It is advisable to transport the patient to the operating room for monitoring and optimal visualization by the obstetrician.
iii) Use of pharmacologic agents for uterine relaxation may be necessary.
iv) If an epidural catheter is in place, a bolus of local anesthetic with or without narcotic should be given.
v) It is difficult to adequately titrate sedative medications in these patients due to the risk of aspiration and possible hemodynamic instability.
(1) If a regional anesthetic is not possible, one should strongly consider general anesthesia.

7) **Abnormal placentation**

a) **General principles**

i) The incidence of placenta accreta spectrum (abnormal adherence of the placental trophoblast to the uterine myometrium) has been estimated to be 1/500-1/300 pregnancies. The increasing rate of CD is related to the increasing incidence of placenta accreta spectrum.[25,26]

b) **Pathophysiology**

i) Placenta accreta
(1) A form of abnormal placental implantation where the placenta is adherent to but does not penetrate the myometrium
ii) Placenta increta
(1) Placental invasion into the myometrium
iii) Placenta percreta
(1) Invasion beyond the uterine serosa and into the abdominal cavity. This form of accreta may in fact invade other abdominal viscera, including the bladder and large vessels.



It is prudent for the entire medical team to review imaging in a patient who has had several uterine surgeries and is at risk for placenta accreta. Regardless of a prior diagnosis, a high index of suspicion, preoperative planning, and quick action to treat postpartum hemorrhage results in fewer complications in patients with placenta accreta.

c) **Diagnosis**

i) Often made at the time of delivery in the face of a retained placenta or placental fragments, sonographic evidence of retained placental fragments, or adherent placenta at CD
ii) Doppler ultrasound and MRI may allow for antepartum diagnosis. However, a recent study showed that MRI may not be reliable in improving predelivery diagnosis.[27]
iii) Placenta accreta is associated with life-threatening maternal hemorrhage, premature delivery, IUGR, and small for gestational age babies.[28]

d) **Risk factors**

i) Advanced maternal age, multiparity, prior uterine surgery including CD, previous uterine curettage, uterine scarring, and placenta previa
(1) 10%-20% of women with placenta previa and one prior CD have a placenta accreta.
(2) 50% of women with placenta previa and two or more prior CD will have a placenta accreta.[29]

e) **Treatment/management**

i) Placenta accreta should be suspected based on risk factors.
ii) A focal placenta accreta may be managed conservatively by oversewing or excising the implantation site at the time of delivery.
iii) Hysterectomy is usually required to remove the placenta *in situ* and control bleeding.
iv) In rare cases, the placenta is left *in situ* with the administration of methotrexate postpartum.

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Hemorrhage in the parturient, Chapter 112
Massive transfusion and resuscitation, Chapter 40

v) Preparation of large amounts of blood products and consultations with other services including interventional radiology, general surgery, urology, hematology, and oncology may be indicated.

f) **Anesthetic considerations**

i) Patients are at high risk for massive blood loss.

ii) One should have a high index of suspicion in patients with placenta previa and multiple previous cesarean sections.

iii) The anesthetic plan should include large-bore or central IV access, blood product availability, and provisions for long surgical time. Invasive arterial monitoring is helpful for blood pressure monitoring and frequent blood draws. Transesophageal echocardiography may be helpful in guiding the fluid management in severe cases.

iv) The anesthetic method is controversial. While an epidural catheter is useful for repeated dosing, maternal satisfaction, and possibly controlling blood loss,[30] some argue that the sympathectomy associated with regional anesthesia may not be desirable in the face of potential massive hemorrhage.[31]

v) While general anesthesia is not mandatory, it should be considered if multiple transfusions are required.[30]

vi) Interventional radiology procedures can preserve fertility and may decrease blood loss, but the patient must be hemodynamically stable for safe transport. Consideration should be taken to placement of iliac or other catheters preoperatively.

vii) Disposition of the patient must also be a consideration. Intensive care and/or step down units will be necessary in cases of massive hemorrhage or other associated complications.

8) **Operative vaginal delivery**

a) **Operative vaginal delivery is the delivery of the fetus with the aid of forceps or a vacuum.**

b) **Pathophysiology**

i) Operative delivery may be performed when fetal station is outlet, low, or mid.

(1) High forceps should not be attempted due to the increased risk of neonatal morbidity and mortality.

ii) In vacuum-assisted deliveries, pressure to the fetal scalp is used to assist in delivery.

iii) In forceps-assisted deliveries, pressure is applied to the fetal skull to assist in delivery.

iv) Selection of vacuum vs forceps is generally provider driven.[32]

c) **Indications for assisted vaginal delivery**

i) Prolonged second stage, fetal compromise or nonreassuring FHR, and shortening the second stage for maternal benefit

d) **Complications**

i) Both vacuum and forceps have been associated with maternal complications including hematomas, lacerations, and pelvic floor injury.

ii) Fetal complications include scalp laceration, cephalohematomas, subgaleal hematomas, intracranial hemorrhage, hyperbilirubinemia, and retinal hemorrhages.

iii) The incidence of shoulder dystocia is increased with operative vaginal delivery.

e) **Contraindications to operative vaginal delivery**

i) Vacuum prior to 34 weeks secondary to the increased risk of cerebral hemorrhage, known fetal bone demineralization or bleeding disorder, unengaged fetal head, unknown position of the fetal head

f) **Treatment/management**
   i) Patients should meet ACOG criteria for operative vaginal delivery.[33]
   ii) Immediate CD should be available.
   iii) Operator experience should determine which instrument is used in each situation.
   iv) Neonatal care providers should be made aware of operative deliveries to assess for neonatal complications.

READ MORE
Obstetrical anesthesia, Chapter 107

g) **Anesthetic considerations**
   i) Pain control is necessary for operative vaginal delivery.
   ii) A well-functioning epidural catheter is optimal.
   iii) A bolus (~5 mL) of higher concentration local anesthetic (0.25% bupivacaine or 1% to 1.5% lidocaine) with or without opioid is recommended.
   iv) One must be prepared for emergent CD, ideally with epidural anesthesia, but general anesthesia may be required if an epidural is not in place or the situation is too emergent.

CHAPTER SUMMARY FOR OBSTETRICAL EMERGENCIES

Clinical Problem	Considerations	Recommended Action
Umbilical Cord Prolapse	Life-threatening fetal emergency.	Emergent CD required, usually with GA.
Uterine Inversion	Potential for hemorrhage and shock.	Treat with uterine relaxant and hemodynamic support.
Uterine Rupture	Life-threatening maternal and fetal emergency. Potential for hemorrhage.	Urgent CD usually required. T&C blood and prepare for potential hemorrhage.
Placenta Accreta Spectrum (Accreta/Increta/Percreta)	Massive blood loss and hysterectomy possible.	T&C blood and products, consider invasive monitoring. Plan for postoperative care.
Shoulder Dystocia	More common with maternal diabetes and large babies. May require CD.	Minimize motor block with epidural analgesia. Prepare for CD.
Nonreassuring FHR Tracing	Category III tracings may be associated with fetal hypoxemia and acidosis.	Prepare for urgent delivery. Consult with obstetrician. May use regional or general anesthesia depending on the situation.
Operative Vaginal Delivery	Forceps or vacuum assistance required. If unsuccessful, may require emergent CD.	Ensure patient comfort, preferably by dosing epidural catheter. Prepare for CD.

GA, general anesthesia; CD, cesarean delivery; T&C, type and cross.

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112 Hemorrhage in the Parturient

Mary Yurashevich • Andrea J. Traynor

Postpartum hemorrhage (PPH) is the leading cause of ICU admission in the United States and one of the leading causes of maternal death worldwide. Morbidity from hemorrhage includes loss of fertility, shock, coagulopathy, adult respiratory distress syndrome, and pituitary necrosis (Sheehan syndrome).

1) **Definition of PPH**
   a) Cumulative blood loss (including intrapartum loss) ≥1000 mL or blood loss accompanied by signs and symptoms of hypovolemia within 24 hours of delivery (irrespective of route of delivery).
   b) Exact amount of blood loss is difficult to determine and often underestimated due to the presence of amniotic fluid and lack of containment of the blood.
   c) Signs of poor perfusion, including tachycardia, decreased pulse pressure, decreased BP, tachypnea, decreased urine output, and altered mental status may not present until blood loss is significant (ie, ~25% of the patient's blood volume or ~1500 mL of blood loss).
   d) **Primary PPH**
      i) Hemorrhage occurring within 24 hours postdelivery
      ii) More likely to result in maternal morbidity or mortality[1]
   e) **Secondary PPH**
      i) Hemorrhage occurring between 24 hours and 12 weeks postdelivery[1,2]
2) **Risk factors for PPH**[1,2]
   a) **Prolonged, augmented, and/or rapid labor**
   b) **Overdistention of the uterus**
   c) **Episiotomy**
   d) **Previous history of PPH**
   e) **Preeclampsia**
   f) **Chorioamnionitis**
   g) **Operative delivery (forceps or vacuum)**
   h) **Asian or Hispanic descent**
3) **Causes of hemorrhage**
   a) **Uterine atony**
      i) Uterine atony causes 70%-80% of PPH.[2]
      ii) Characterized by a soft, poorly contracted uterus
      iii) Risk factors for uterine atony
         (1) Prolonged, augmented, or precipitous labor
         (2) Prolonged use of oxytocin
         (3) Infection (such as chorioamnionitis)
         (4) Overdistention of the uterus from macrosomia, multiple gestations, or polyhydramnios
         (5) Use of tocolytic drugs



A mnemonic for remembering the risk factors for uterine atony is to think of a "big tired or infected" uterus.

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Obstetrical emergencies, Chapter 111

(6) General anesthesia can contribute to atony as volatile agents decrease uterine contractility.
(a) Consider propofol infusion or total intravenous anesthesia if time permits to alleviate the problem of decreased uterine contractility with volatile agents.

b) **Placental abnormalities (previa, accreta, increta, percreta)**
i) Placental abnormalities include placenta previa, placenta accreta, placenta increta, and placenta percreta.
ii) Placenta previa occurs when the placenta covers the cervical os.
iii) The incidence of placenta previa is 4 per 1000 pregnancies.[1]
(1) The other placental abnormalities depend on the depth of invasion into the uterine myometrium, with placenta percreta extending through the myometrium and into the peritoneal cavity.



Patients with abnormal placentation are at risk for massive blood loss at delivery.

iv) Risk factors
(1) Previous uterine surgery (especially cesarean delivery)
(a) The risk of placenta accreta in the presence of placenta previa is 3% with first cesarean and goes up to 67% with five or more caesarean deliveries.[1–3]
(2) Advanced maternal age
(3) Assisted reproductive technology[4]

c) **Coagulation defects**
i) Inherited
(1) Von Willebrand Disease (VWD)
(a) Most common inherited coagulation defect
(b) Patients with vWD are at risk for PPH and commonly have delayed hemorrhage as their coagulation factors reach prepregnancy levels in the weeks following delivery.[5]
(c) May require treatment with desmopressin acetate, recombinant factor VII, humate-P, cryoprecipitate, or fresh frozen plasma during and after delivery, depending on their subtype of disease.[5]
(d) Because there are several different forms of vWD, it is best to consult a hematologist when caring for these patients.

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Coagulopathies, Chapter 66

ii) Acquired coagulation defects
(1) Causes
(a) Syndrome of hemolysis, elevated liver enzymes, and low platelets (HELLP)
(b) Disseminated intravascular coagulation (DIC)
(i) DIC can occur as a result of placental abruption, prolonged intrauterine fetal demise, and amniotic fluid embolism.
(c) Sepsis
(i) Due to chorioamnionitis, retained products of conception, surgical infection, or systemic infection from nonobstetric causes

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Obstetrical emergencies, Chapter 111

d) **Uterine inversion**
i) Characterized by rapid onset of hemorrhage and shock during the third stage of delivery
ii) Incidence of 1 in 3700-20 000 vaginal deliveries and 1 in 1860 cesarean deliveries[2]
iii) Uterus must be restored to its normal position, which may require nitroglycerin, β-agonist agents, magnesium sulfate, or general anesthesia to relax the uterus.

iv) Once in its normal position, uterotonic agents should be administered to facilitate uterine contraction.[1]

e) **Uterine rupture**
   i) Can occur at the site of previous caesarean delivery scar or other surgical procedure that involves the uterine wall (ie, myomectomy site).[2]
   ii) May occur due to intrauterine manipulation or trauma, spontaneously due to abnormal labor or in the setting of congenital malformation.[2]
   iii) Surgical repair is required and supportive care with fluid resuscitation, blood transfusion, and uterotonics may be needed depending on the patient's hemodynamic status.



Nitroglycerin may be useful during a uterine inversion to facilitate replacement of the uterus into the pelvis.

f) **Retained products of conception**
   i) Can cause primary or secondary PPH
   ii) Occurs in approximated 3% of vaginal deliveries and is defined as failure to deliver the placenta within 30 minutes of delivery of the infant.[1]
   iii) Treatment
      (1) Manual removal of the placenta, which can be performed under regional or general anesthesia depending on the hemodynamic stability of the patient[1]
      (2) Nitroglycerin is effective in providing uterine relaxation for removal of retained placenta.[6]



Methylergonovine can cause severe hypertension and should not be given to patients with hypertension.

Prostaglandin $F_2\alpha$ can increase pulmonary vascular resistance and should be avoided in patients with asthma or pulmonary hypertension.

g) **Genital tract or uterine lacerations/hematomas**
   i) Lacerations and/or hematomas are the most common cause of injury during childbirth and should be suspected as a cause of hemorrhage in a patient with persistent vaginal bleeding despite adequate uterine tone.[1]
   ii) Patients who have vWD or who have had a vacuum or forceps delivery are at highest risk.[1,5]
   iii) Retroperitoneal hematomas are the most dangerous hematomas to occur during childbirth.
      (1) Occurs after laceration of one of the branches of the hypogastric artery.
      (2) Typically occurs from injury during caesarean delivery or with rupture of low transverse uterine scar during labor. Hematomas may extend as far as the kidneys.[1]

4) **Treatment of hemorrhage**

a) **Pharmacologic treatment of uterine atony**
   i) The anesthesia provider is commonly asked to administer uterotonic agents.
   ii) The route of administration, dosage, and side effects of the various agents are outlined in **Table 112-1**.
   iii) It is important to be aware of the side effects and contraindications of uterotonic medications (see **Table 112-1**).
   iv) Methylergonovine and prostaglandin $F_2\alpha$ should always be administered intramuscularly.[2]
   v) Surgical techniques to control uterine atony include uterine massage, balloon tamponade (Bakri balloon), B-Lynch sutures, and bilateral uterine or hypogastric artery ligation.[2]
   vi) In cases of placental abnormality and/or extreme uterine atony, hysterectomy may be required.

TABLE 112-1 Medical Management of Postpartum Hemorrhage

Drug[a]	Dose/Route	Frequency	Comment
Oxytocin (Pitocin)	IV: 10-40 units in 1 L normal saline or lactated Ringer solution IM: 10 units	Continuous	Avoid undiluted rapid IV Infusion, which causes hypotension.
Methylergonovine (Methergine)	IM: 0.2 mg	Every 2-4 h	Avoid if patient is hypertensive.
15-Methyl $PGF_2\alpha$ (Carboprost) (Hemabate)	IM: 0.25 mg	Every 15-90 min, 8 doses maximum	Avoid in asthmatic patients; relative contraindication if hepatic, renal, and cardiac disease. Diarrhea, fever, tachycardia can occur.
Dinoprostone (Prostin E_2)	Suppository: vaginal or rectal 20 mg	Every 2 h	Avoid if patient is hypotensive. Fever is common. Stored frozen, it must be thawed to room temperature.
Misoprostol (Cytotec, PGE_1)	800-1000 µg rectally		

[a]All agents can cause nausea and vomiting.
IM, intramuscularly; IV, intravenously; PG, prostaglandin.
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b) In cases of hemorrhage where the patient is relatively hemodynamically stable, the uterine arteries can be embolized.[7]
   i) Advantages of this technique
      (1) Potential avoidance of surgery, decreased blood transfusion, and preservation of fertility
      (2) In patients at risk for hemorrhage, uterine artery catheters and balloons can be placed preoperatively.[8]
   ii) Disadvantages
      (1) Include possibility of infection, vascular injury, or thrombosis.

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Blood component therapy, Chapter 38
Alternatives to blood product replacement, Chapter 39

c) **Blood component therapy**
   i) Normal pregnancy is a hypercoagulable state.
      (1) Increased tissue factor concentration and enhancement of tissue factor–dependent coagulation pathway.
   ii) Many patients with obstetric hemorrhage will require treatment with blood components.
      (1) Red blood cell transfusion should be considered in patients with ongoing bleeding, clinical evidence of decreased oxygen-carrying capacity and/or hemoglobin concentration <7 g/dL.
   iii) Evaluation of clotting is important, and the patient may require treatment with platelets, clotting factors, and synthetic clotting agents in addition to red blood cells.



In the bleeding obstetric patient, constant communication with the surgeon about ongoing blood loss is imperative.

d) **Pharmacologic treatment of OB hemorrhage**
   i) Tranexamic acid
      (1) Mechanism of action is inhibition of breakdown of fibrinogen and fibrin by plasmin.
      (2) Most effective if given within 3 hours of birth[9]
      (3) Early administration significantly reduced death due to bleeding as observed in WOMAN trial.[9]

ii) Activated recombinant factor VII (rFVIIa) can be used to treat intractable obstetric hemorrhage.
   (1) Mechanism of action is characterized by augmentation of the intrinsic clotting pathway by binding with tissue factor and directly activating factors IX and X.[10]
   (2) The most effective dose is 90-100 μg/kg IV.[1,10]
   (3) Side effects in the obstetrical population are rare but include thrombosis of vessels or indwelling devices.
   (4) Use should be reserved for extenuating circumstances when standard therapies have failed and in consultation with an expert in massive hemorrhage.[2]



In an OB patient with PPH, regional anesthesia is an option, but one must carefully consider general anesthesia with endotracheal intubation in patients who are hemodynamically unstable or requiring large amounts of fluids and blood products.

CHAPTER SUMMARY FOR HEMORRHAGE IN THE PARTURIENT

Implications	OB hemorrhage is common. It is the leading cause of ICU admission and one of the leading causes of maternal death worldwide
Definition	>1000 mL EBL (including intrapartum loss) irrespective of mode of delivery
Causes	Uterine atony is most common. Other causes include coagulation defects, lacerations, retained products of conception, placental abnormalities
Treatment	Treat uterine atony with uterotonics or surgical techniques Avoid large boluses of oxytocin IV Avoid methylergonovine in patients with hypertension Avoid prostaglandin $F_2\alpha$ in patients with asthma or pulmonary hypertension General treatment includes volume resuscitation, airway management, and blood product replacement
Postoperative Care	Consider the possibility for ongoing blood loss and/or coagulopathy. Patients may require postoperative intubation and care in an ICU

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113 Neonatal Resuscitation

Echo Rowe

As anesthesiologists, most often our clinical focus resides in the mother during labor or Cesarean delivery, while other medical providers provide neonatal care. According to the American Academy of Pediatrics, ~10% of newborns require some assistance to begin breathing after birth, but 1% will require extensive resuscitation to survive.[1] It is the responsibility of the anesthesiologist to understand the basic principles of neonatal resuscitation and be prepared to provide this life-saving care when necessary.

1) **Introduction**
   a) **Neonatal resuscitation** primarily consists of basic life support with a focus on airway, breathing, circulation, and tone (ABCs).[2]
   b) The ABCs are the heart of our everyday practice and such skills may be called upon when other medical personnel are not available or during certain neonatal emergencies, whether anticipated or not.
2) **Risk factors:** Antepartum and intrapartum factors may increase the likelihood of positive-pressure ventilation or endotracheal intubation at the time of delivery, including gestational age, multiple gestation pregnancy, and meconium stained amniotic fluid.[3]
   a) Important information to obtain when responding to a resuscitation:
      i) Estimated gestational age
      ii) Presence of meconium
      iii) Maternal diseases and/or pregnancy complications
      iv) Any fetal anomalies such as congenital heart disease, congenital diaphragmatic hernia, gastroschisis, or craniofacial syndromes associated with difficult intubation such as Pierre Robin or Treacher Collins
3) **Treatment**
   a) Equipment: Availability of appropriate supplies and equipment saves invaluable time.
   b) The anesthesiologist is most often caring for the mother and thus has a primary responsibility to the mother. If called upon to assist with resuscitation of the infant, the benefit to the child must be compared to the risk to the mother.[4]
   c) Restoring oxygenation and ventilation is the utmost priority as the infant transitions from intrauterine to extrauterine life. Equipment used for this purpose is listed in **Table 113-1**.
4) **Assessment**
   a) The initial assessment should take <30 seconds and includes checking for **respiratory effort, color, heart rate (HR), and muscle tone**.
      i) These are the clinical factors that guide further resuscitation and reassessment.
   b) **Apgar scores (Table 113-2)**
      i) Assigned at 1, 5, and 10 minutes of life
      ii) Apgar scores are a retrospective measure of an infant's need for and response to resuscitative measure.
      iii) Not particularly useful as an assessment tool to guide resuscitative efforts



Anesthesiologists are most likely to provide neonatal resuscitation when other providers are unavailable or unexpected events occur, such as emergent delivery or difficult neonatal airway.

It is helpful to familiarize yourself with the equipment for neonatal resuscitation before an emergency occurs since a variety of bag/mask devices are used that differ from those commonly used by anesthesiologists.

TABLE 113-1 Neonatal Resuscitation Equipment

	Equipment/Supplies	Sizes
General	• Sterile gown and gloves (if CD) • Warm blankets, plastic sheeting • Infant warmer with timer • Neonatal pulse oximeter • ECG electrodes	
Suction	• Bulb syringe • Suction catheter with meconium aspirator	6, 8, 10, 12 Fr
Bag-mask ventilation	• Neonatal resuscitation bag and/or T-piece • O_2 source • Pressure manometer • Cushioned face mask	Newborn and premature
Intubation	• Laryngoscope blades and handle • ETT—uncuffed • Thin stylets • CO_2 detector	• Miller 0 (premature) • Miller 1 (term) • 2.5 mm < 1000 g/28 wk • 3.0 mm 1000-2000 g/28-34 wk • 3.5 mm 2000-3000 g/34-38 wk • 4.0 mm > 3000 g/>38 wk
Medications	• Epinephrine • Naloxone • Normal saline	

CD, cesarean delivery; ETT, endotracheal tube; Fr, French; mm, millimeter; wk, weeks.

5) **Resuscitation: The Neonatal Resuscitation Program (NRP) algorithm is widely available with key points as follows (Fig. 113-1):**
   a) After initial assessment, provide warmth, drying, and stimulation with an emphasis on maintaining normothermia during resuscitation.
   b) Position and clear the airway if necessary. Routine suctioning is no longer recommended as this may cause vagal response.
   c) If apnea, poor respiratory effort, or HR < 100 bpm → begin effective ventilation either by bag-mask or CPAP device such as flow-driven T-piece with manometer.
   d) Consider early placement of preductal pulse oximetry and ECG monitoring to guide resuscitation (**Fig. 113-2**).
   e) For PPV in newborn >35 weeks, begin with 21% FiO_2, for infants <35 weeks begin with 21%-30% oxygen with an FiO_2 blender.
   f) Higher pressures may be required when initiating ventilation to open fluid-filled alveoli. Watch level of chest rise to assess adequate ventilation.



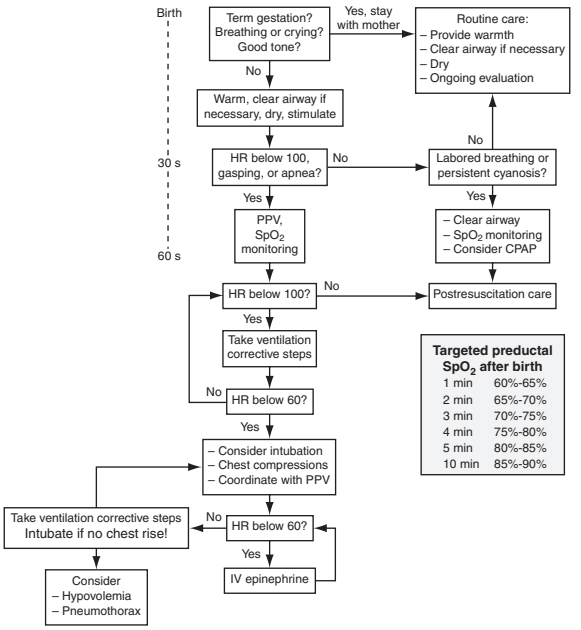
Endotracheal intubation is indicated when:

- Bag-mask ventilation ineffective or prolonged
- Chest compressions indicated
- Special circumstances such as congenital diaphragmatic hernia

TABLE 113-2 Apgar Scoring

	0	1	2
Color	Blue or pale	Body pink, extremities blue	Completely pink
Respiratory effort	Absent	Slow, irregular	Good, crying
Heart rate	Absent	<100 bpm	>100 bpm
Reflex irritability	No response	Grimace	Cough, sneeze, cry
Muscle tone	Limp	Some flexion	Good, active motion

FIGURE 113-1 Neonatal Resuscitation Algorithm.



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FIGURE 113-2 Pre-Ductal SpO_2 Target During Newborn Resuscitation

Targeted Pre-ductal Spo_2 After Birth	
1 min	60%-65%
2 min	65%-70%
3 min	70%-75%
4 min	75%-80%
5 min	80%-95%
10 min	85%-95%

g) If HR still <60 bpm after 30-60 seconds of effective ventilation, intubation is recommended prior to starting chest compressions if possible.
h) Chest compressions should occur at a rate of 100 bpm with a breath given after every third compression.
i) If chest compressions are necessary, FiO_2 should be increased to 100%.
j) If HR remains <60 bpm, administer epinephrine.

6) **Resuscitation tips**
   a) Place infant in sniffing position (shoulder roll may be helpful) and apply gentle chin lift into mask to obtain a proper seal. Avoid downward force onto the mask.
   b) Watch for chest rise as the primary end point of adequate ventilation.
      i) Keep in mind that higher peak inspiratory pressures up to 40 mm Hg may be initially required to open closed alveoli. A pressure manometer is helpful along with clinical indicators such as chest rise.
   c) Avoid insufflation of the stomach as it may further impair ventilation. Decompress if needed.
   d) Chest compressions can be done by encircling the infant with both hands and placing both thumbs on lower sternum or by placing two fingers perpendicular to the chest.
   e) Chest compressions should depress the sternum one-third the depth of the chest while the fingers remain in place at all times.
   f) Compressions to ventilation ratio should be 3:1 at a rate of 90 compressions and 30 breaths per minute.
   g) It can be helpful to keep cadence out loud with "One-and-Two-and-Three-and-Breathe."
   h) When the HR is >100 bpm and ventilation is adequate, resuscitation may be stopped.
7) **Special considerations**
   a) **Meconium**
      i) Occurs in ~7%-20% of live births with up to 10% of these infants developing meconium aspiration syndrome (MAS)[5]
         (1) MAS can lead to hypoxic respiratory failure, persistent pulmonary hypertension, pneumonia, and sepsis all with significant potential for long-term morbidity and mortality.
      ii) Current NRP guidelines no longer recommend routine intubation and suctioning of the trachea of a nonvigorous, meconium-stained infant as it has not been shown to decrease the incidence of MAS and may potentially be harmful.[6,7] The emphasis is now on initiation of ventilation for any nonbreathing or poorly breathing newborn.[6]
   b) **Maternal opioid administration** within 4 hours of delivery may contribute to respiratory depression in the neonate.
      i) If respiratory depression persists after PPV and normal HR, consider administering naloxone; however, PPV and supportive care should continue.
      ii) Keep in mind that the half-life of naloxone may be shorter than that of the circulating opioid and therefore may need to be redosed.
8) **Medications**
   a) **Epinephrine** should be given when the HR remains <60 bpm after 30 seconds of assisted ventilation and an additional 30 seconds of chest compressions.
      i) Can be administered via the endotracheal tube, umbilical vein, IV, or IO
         (1) Epinephrine recommended dose is 0.01-0.03 mg/kg of 1:10 000 intravenously.[6] This is equivalent to 10-30 µg/kg.



Consider the possibility of opioid-induced respiratory depression in any neonate whose mother has received IV narcotics close to delivery.



Do not give naloxone to infants of mothers on methadone maintenance or with chronic addiction.

(2) Epinephrine given via endotracheal route has variable absorption and requires larger doses since it can remain in the tube.[8]

(3) Early epinephrine via endotracheal tube may promote aortic diastolic pressure, coronary perfusion, and return of spontaneous circulation and its use via this route remains an option when intravenous or umbilical vein access is delayed.[9]

b) **Naloxone**
   i) Administration can also be via endotracheal or IV routes.
   ii) Recommended dose is 0.1 mg/kg.

c) **Volume expansion** may be considered when blood loss is known or suspected, evidence of shock, and the newborn's HR has not responded to other resuscitative measures.[6]
   i) Isotonic crystalloid or uncrossmatched O negative blood may be given.
   ii) Recommended dose is 10 mL/kg.
   iii) Rapid infusion of volume expanders has been associated with increased risk of intraventricular hemorrhage in premature infants.



Quick preparation of epinephrine solution: Dilute 1 mg (1 ampule) into 100-mL bag for final concentration of 10 μg/mL. The resuscitation dose is then 1 to 3 mL/kg.



Avoid giving rapid infusion of volume expanders to premature infants, as it may increase risk of IVH.

CHAPTER SUMMARY FOR NEONATAL RESUSCITATION

General Considerations	A large number of infants will require assistance with breathing and/or resuscitation at birth. Neonatal resuscitation should ideally be provided by a provider whose primary responsibility is the infant.
Preparation	Equipment should be prepared prior to an emergency. Doses of medications should be posted and easily accessible.
Primary Goals	Restoring oxygenation and ventilation is the first priority. Position and clear airway of copious secretions. Provide supplemental oxygen and PPV if necessary, starting with FiO_2 0.21 in term infants. Maintain normothermia. Use pulse oximetry and ECG to help guide resuscitation efforts. Follow NRP algorithm in cases of depressed infants.
Special Considerations	Consider naloxone administration in infants who had acute in utero exposure to opioid medications during maternal labor.

PPV, positive pressure ventilation.

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114 Emergency Aids for Obstetric Crises in Anesthesiology

Gillian Abir • Katherine M. Seligman • Larry F. Chu

Multidisciplinary team involvement in obstetric emergencies is fundamental to outcomes, which requires an efficient and organized response. Evidence-based practice involving peer-reviewed protocols and guidelines have become standard throughout health care, in particular in anesthesiology and the subspecialties such as obstetric anesthesiology.[1-6] Cognitive aids are tools intended to refresh memory, enhance recognition of emergencies, as well as structure or standardize actions and communication.[7] Several simulation studies have shown that cognitive aids have improved team management during emergency situations, and the aim is to implement cognitive aids into actual clinical teams to enhance patient care during emergency and nonemergency situations.[8–11]

During emergency situations (eg, postpartum hemorrhage and eclampsia), critical management and treatment steps should be accomplished in a specific order to optimize outcomes, and the use of cognitive aids may help to enable this process, rather than relying on memory alone.[12,13]

When a new process or system is introduced, users (eg, health care providers) need to understand its intended purpose. As health care providers become familiar with tools such as cognitive aids, it will become evident that they can be beneficial for them, their teams, and their patients; however, institutions and individuals vary in regard to how they adopt change in their routine practice.[7] Effective implementation, which involves multiple variables (such as effective design concepts, relevant and specific content, and informed implementation in the clinical setting), is vital for cognitive aids to be routinely and successfully used.[7,14,15]



OBSTETRIC ANESTHESIA

MANUAL



Maternal Cardiac Arrest 967

EMERGENCIES

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Please note, additional cognitive aids are listed in the downloadable version available at:

http://cogaids.stanford.edu

MATERNAL CARDIAC ARREST

DIAGNOSIS

Pregnant + NO Pulse → **START**

CALL FOR CODE TEAM + CART  **BEGIN CPR**

IMMEDIATE

- **Position** → Supine with back board under patient + perform manual left uterine displacement during CPR
- **AED** → Place + assess rhythm
- **Airway** → Place ETT + ventilate (confirm correct placement) 10 breaths/min with 100% oxygen
- **IV access** → 2 large bore IVs, 16G (above level of diaphragm)
- **IO access** → Humeral IO line (if no IV access)

Perimortem cesarean delivery (PMCD)

- → Confirm fundus is at or higher than the level of the umbilicus before proceeding
- → Prepare immediately at site of arrest
- → Alert NICU
- → Perform if no ROSC at 4 min from time of arrest

CPR → **100 compressions per minute**; rotate compressors q2min

Continued on next page

MATERNAL CARDIAC ARREST

BACK

TREATMENT

Defibrillate ⇒ **200 joules** (biphasic energy) if pulseless VT or VF

Drugs ⇒ **Epinephrine** 1 mg IV/IO q3-5min
⇒ **Amiodarone** 1st dose 300 mg bolus IV/IO, 2nd dose 150 mg bolus IV/IO

Repeat cycle until resuscitated
CPR + defibrillate (if pulseless VT/VF)
q2min + drugs

OTHER

Magnesium sulfate ⇒ If running Mg infusion, **STOP**
⇒ Administer 10% $CaCl_2$ 10 mL IV

Anesthetics ⇒ If running neuraxial/inhalation/IV medications, **STOP**

Continued on next page

MATERNAL CARDIAC ARREST

BACK

POTENTIAL FACTORS

Cross-check diagnosis with team

Anesthetic complications
- → High neuraxial block
- → Loss of airway
- → Aspiration
- → Respiratory depression
- → Hypotension
- → LAST

Bleeding
- → Coagulopathy
- → Uterine atony
- → Placenta accreta
- → Placental abruption
- → Placenta previa
- → Uterine rupture
- → Trauma
- → Surgical
- → Transfusion reaction

Cardiovascular causes
- → Cardiomyopathy
- → Myocardial infarction
- → Aortic dissection
- → Arrhythmia

Drugs
- → Anaphylaxis
- → Illicit drug use
- → Overdose (magnesium, opioid, insulin, or oxytocin)

Embolic
- → Pulmonary embolus
- → AFE
- → Air

Fever
- → Infection
- → Sepsis

General nonobstetric causes of cardiac arrest

Hs and Ts:
- → Hypoxia
- → Hypovolemia
- → Hypo/hyperkalemia
- → Hypo/hyperthermia
- → Hydrogen ions (acidosis)
- → Hypoglycemia
- → Tension pneumothorax
- → Tamponade
- → Toxins
- → Thromboembolism
- → Thrombosis (MI)
- → Trauma

Hypertension
- → Preeclampsia/eclampsia/HELLP
- → Intracranial bleed

Continued on next page

MATERNAL CARDIAC ARREST

BACK

FIND TREATABLE CAUSES

Cross-check diagnosis with team

Cardiac tamponade
Diagnosis:
→ Increased CVP, equalization of right and left-sided pressures
If suspected, consider:
→ TEE/TTE to rule out pericardial effusion
→ If present, perform peri cardiocentesis

Coronary thrombosis
If suspected, consider:
→ TEE
→ Emergent revascularization: pharmacological or percutaneous (cath lab)
→ Intra-aortic balloon pump

Electrolyte abnormality
Rule out:
→ Hyperkalemia
→ Hypokalemia
→ Hypocalcemia
→ Acidosis
→ Hypoglycemia

Hypo/Hyperthermia
If <30°C:
→ Rapid rewarming with warm IV fluids, peritoneal lavage, ECMO or CPB
If >40°C:
→ Rule out malignant hyperthermia and treat if suspected

Hypovolemia
IV fluids → Rapid bolus
Hb/Hct → Evaluate
Blood → Consider transfusion

Hypoxia
If suspected, consider:
→ 100% oxygen, in OR rule out switched gas lines, use separate oxygen tank
→ Check connections, re-confirm ETT placement
→ Confirm bilateral breath sounds
→ Suction ETT
→ Rule out other causes with TEE/TTE

Pneumothorax
Diagnosis:
→ Unilateral breath sounds, neck veins, trachea deviated from affected side (consider POCUS)
Needle decompression:
→ Midclavicular line 2nd intercostal space, then place chest tube

Pulmonary thrombosis
If suspected, consider:
→ TEE/TTE to rule out right ventricular failure
→ Thrombolytic therapy, discuss risk/benefits with team

Toxins
Rule out:
→ Existing infusions (for neuraxial analgesia consider LAST
→ Prescriptions
→ Illicit drug use, syringe swaps or drug errors, poisoning
If suspected:
→ Contact poison control
→ Administer appropriate therapy or antidote



For a poison emergency in the US call 1-800-222-1222

Continued on next page

MATERNAL CARDIAC ARREST

BACK

VT/VF CONSIDERATIONS

Consider antiarrythmics ⇒
- Amiodarone → 300 mg IV bolus or
- Lidocaine →100 mg IV bolus (do not administer if concern for LAST)

Hyperkalemia? ⇒ ABG → Evaluate potassium
- → Consider 10% calcium chloride 10 mL IV (over 5-10 min, repeat if necessary)
- → Consider Insulin 10 units IV bolus + glucose 40-60 g IV bolus
- → Consider Albuterol inhaler (6-8 puffs/ETT)

pH <7.20 → Consider sodium-bicarbonate 8.4% 1-2 amps IV bolus

Hypomagnesemia or torsades? ⇒ Consider → magnesium sulfate 2 g IV (over 20 min)

TEAMWORK

Leader

Teamwork → Assign checklist reader + other roles
Open Exchange → Solicit information, ideas and input
Diagnosis/Plan → Cross check for other etiologies. Could this be anything else?
Plan & Thoughts → Share/restate aloud the treatment plan and priorities

Follower

Checklist → Obtain and read aloud
Concerns → Vocalize issues
Task → Focus on assigned task; inform leader of success/failure/issues
Communication → Confirm specific request, question for clarity and indicate completion

END

AMNIOTIC FLUID EMBOLISM

START

DIAGNOSIS

Triad
→ Hypoxia
→ Hypotension
→ Consumptive coagulopathy

Premonitory symptoms
→ Restlessness
→ Agitation

CALL FOR HELP  **OB RAPID RESPONSE**

IMMEDIATE

Team leader ➔ Identify

Airway ➔ Clear?

Breathing ➔ SpO_2 + RR
➔ Auscultation

Circulation ➔ HR + BP
➔ Urine output

Conscious level ➔ Adequate?

Position ➔ Left uterine displacement

Fetal heart rate ➔ Monitor

IV access ➔ 2 large bore IVs, 16G

Invasive monitoring ➔ Arterial line
➔ Central line

Labs ➔ CBC
➔ Coag screen
➔ Fibrinogen
➔ BMP
➔ ABG
➔ TEG/ROTEM

Continued on next page

AMNIOTIC FLUID EMBOLISM

TREATMENT

Oxygen ⇨ 100% (10 L/min) via non-rebreather facemask or ETT

Hypotension ⇨ Cautious IV fluid bolus
⇨ Administer vasopressor boluses prn
→ Phenylephrine 100-200 µg IV
→ Ephedrine 5-10 mg IV
→ Epinephrine 10-100 µg IV
⇨ Consider vasopressor infusion
→ Epinephrine 0.01-0.1 µg/kg/min IV
→ Norepinephrine 0.01-0.1 µg/kg/min IV
→ Vasopressin 0.01-0.04 units/min IV

Coagulopathy ⇨ At risk for massive hemorrhage/DIC
→ Initiate MTP if symptoms of DIC or ongoing hemorrhage and/or atony
→ Early administration of PRBCs, FFPv, Plts, cryoprecipitate, or fibrinogen concentrate (if indicated)
→ Consider tranexamic acid 1 g IV (over 10 min)

Emergent delivery ⇨ Consider

Additional treatments ⇨ Consider hydrocortisone 100 mg IV bolus
⇨ Iloprost 2.5 µg NEB for pulmonary vasoconstriction

OTHER

1. Definitive airway: Intubate if developing hypoxia/pulmonary edema
2. Invasive monitoring: Place arterial line, consider central line
3. EKG, CXR, TEE/TTE
4. ICU consult
5. Consider ECMO or balloon pump in patients with severe left ventricular failure

DDX

1. Anaphylaxis
2. Sepsis
3. Hemorrhage
4. Embolism (PE, air)
5. Eclampsia
6. Medication reaction (LAST)
7. MI

END

DIFFICULT AIRWAY

START

DIAGNOSIS

Failure to intubate → Unable to see cords or pass the ETT into trachea



CALL FOR HELP, DIFFICULT AIRWAY CART

CP

Consider releasing cricoid pressure (CP)

→ Try to improve view with external laryngeal manipulation

OPTIONS

Sniffing position	Optimize
Airway	Mask ventilate with 100% oxygen ± CP
Laryngoscope blade	Consider changing type/ size of blade for 2nd attempt
ETT size	Consider using smaller size (6.0 mm) + use bougie/ stylet
Advanced airway device	Consider using LMA (Pro-seal or classic) or supra-glottic airway (SGA) device
Video laryngo-scope device	Consider using
Prepare	Prepare for surgical airway (tracheostomy)

Continued on next page

DIFFICULT AIRWAY

If second intubation attempt fails

Insert oropharyngeal airway and perform 2-handed facemask ventilation

If experienced provider, attempt 3rd intubation

PLACE SGA

If you cannot oxygenate the patient

→ Release CP

→ Place SGA device, such as LMA (ProSeal or classic)

→ Consider: intubating LMA

→ Awaken patient

OTHER

If you cannot ventilate the patient

Surgical help ⇒ Call for

Alternative airway ⇒ Attempt:
- ⇒ Transtracheal jet ventilation
- ⇒ Percutaneous cricothyrotomy
- ⇒ Surgical cricothyrotomy
- ⇒ Tracheostomy

Patient ⇒ Awaken

SUCCESS

If ventilation is successful

→ Assess maternal/fetal status and consider continuing surgery with SGA device or facemask ventilation

→ Consider other options: awaken patient, perform awake fiberoptic intubation, perform neuraxial anesthesia, delay surgery, awaken patient

→ If ventilation becomes inadequate, resume from top of page

END

DKA IN PREGNANCY

START

DIAGNOSIS

Type I Diabetes

Symptoms
- → Polyuria
- → Polydipsia
- → Nausea
- → Vomiting
- → Abdominal pain

Signs
- → Hyperventilation
- → Tachycardia
- → Hypotension
- → Altered mental status

Diagnostic Criteria

- ⟶ Glucose **>250 mg/dL**
- ⟶ pH **< 7.3**
- ⟶ Bicarbonate **<18 mEq/L**
- ⟶ Ketonuria

CALL FOR HELP

IMMEDIATE

Airway	Adequate?
Breathing	SpO_2 + RR
Circulation	HR + BP
Oxygen	100% (10 L/min) via non-rebreather facemask
IV access	2 IVs, 18G
Labs	Glucose BMP ABG Serum ketones Lactate CBC Urinalysis
Position	Left uterine displacement
Fetal heart rate	Monitor
If arrhythmia	12-lead EKG

Continued on next page

DKA IN PREGNANCY

TREATMENT

1. **Fluid replacement: 1-2 L saline in first hour**
   → Initiate 0.45% saline at 250-500 mL/h
   → When serum glucose <250 mg/dL → 0.45% saline with 5% dextrose 250 mL/h
2. **Initiate regular insulin infusion if potassium is >3.3 mEq/L**
   → 0.1 U/kg IV bolus regular insulin over 5 min
   → Regular insulin infusion 0.1 U/kg/h IV → if serum glucose does not decrease by 50 mg/dL in first hour, double IV insulin infusion rate
   → When serum glucose <200 mg/dL, decrease insulin rate to 0.05 U/kg/h IV
3. **Consider bicarbonate replacement in severe maternal acidosis pH < 7.0**
   → 50 mEq sodium bicarbonate in 500 mL 0.45% saline with 20 mEq of potassium IV over 2 hours
4. **Aggressive electrolyte replacement**
   → Follow potassium, magnesium, phosphorous
5. **Workup for precipitating factors**
   → Infection
   → Hyperemesis
   → Non-compliance, etc.

END

MATERNAL HEMORRHAGE

START

DIAGNOSIS

Antepartum

Intrapartum

Postpartum
→ EBL ≥ 1000 mL post-vaginal delivery
→ EBL ≥ 1000 mL post-cesarean delivery

CALL FOR HELP  **OB RAPID RESPONSE**

IMMEDIATE

- **Airway** → Adequate?
- **Breathing** → SpO_2 + RR
- **Circulation** → HR + BP
  - → Capillary refill
  - → Urine output
- **Conscious level** → Adequate?
- **Monitor** → Vital signs q1-2min (until stable)
- **IV access** → 2 large bore IVs, 16G
- **IO access** → Consider humeral IO line (if no IV access)
- **Labs** → Stat CBC
  - → Coag screen
  - → Fibrinogen
  - → ABO/T+C
  - → ABG
  - → Calcium
  - → Lactate
  - → TEG/ROTEM

  Monitor (if indicated)

Continued on next page

MATERNAL HEMORRHAGE

TREATMENT

Immediate (0-5 min):

→ Oxygen: 100% 10 L/min via non-rebreather facemask

→ Fluids: Bolus 1-2 L IV crystalloid (see Maternal Hypotension)

→ Activate MTP (see Massive Transfusion Protocol)

→ Transfuse blood products (if indicated MTP or type-specific units [time-dependent])

→ Identify cause
- → Atony
- → Laceration
- → Retained placenta
- → Coagulopathy
- → Uterine inversion (see Uterine Inversion)

→ Treat cause
- → Fundal massage
- → Administer uterotonic(s) (see Uterine Atony)
- → Tamponade: Packing, Bakri balloon
- → Surgery: B-Lynch suture, vessel ligation/ hysterectomy

Continued on next page

MATERNAL HEMORRHAGE

TREATMENT

Phase 1 (5-10 min):

→ Recap
→ Assess EBL/QBL
→ Use a rapid-infuser device
→ Consider early administration of cryoprecipitate or fibrinogen concentrate

Phase 2 (10-20 min):

→ Consider antifibrinolytic therapy

→ Tranexamic acid 1 g IV bolus (over 10 min, repeat dose if no response) + 1 mg/kg/h infusion

→ Early transfer to OR (if bleeding is ongoing) or IR (if bleeding ongoing and patient stable)

→ Maintain normothermia

→ Treat hypocalcemia

Phase 3 (severe refractory):

→ Consider prothrombotic therapy (if life threatening and no evidence of AFE or circulating tissue factors)

→ Factor VIIa 40 μg/kg IV bolus (repeat dose after 15 min if indicated)

CONSIDER

1. Invasive monitoring: Place arterial line, consider central line (large bore)
2. TEE/TTE
3. General anesthesia
4. Hematology consult
5. ICU consult
6. Check Plts/coag screen prior to removal of epidural catheter (if indicated)

END

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MATERNAL HYPERTENSION

START

DIAGNOSIS

Acute-onset, severe hypertension:

SBP ≥ **160 mm Hg** or DBP ≥ **110 mm Hg**
(elevated for ≥15 min)

Preeclampsia: SBP ≥ 140 mm Hg or DBP ≥ 90 mm Hg

ACOG Committee Opinion No. 767: Emergent Therapy for Acute-Onset, Severe Hypertension During Pregnancy and the Postpartum Period. Obstet Gynecol. 2019;133:e174-e180

CALL FOR HELP



IMMEDIATE

Team leader	Identify
Airway	Adequate?
Breathing	SpO_2 + RR Auscultation
Circulation	HR + BP Consider arterial line if obese or very high BP for accuracy Urine output
Conscious level	Adequate?
Position	Left uterine displacement
IV access	2 IVs, 18G
Fetal heart rate	Monitor
	CBC BMP LFTs Coag screen (if abnormal LFTs or thrombocytopenia) 24 hours urine protein Urine protein:creatinine

Continued on next page

MATERNAL HYPERTENSION

TREATMENT

Pain related? Yes

→ Administer PO, IV or neuraxial (if indicated) analgesia

Pain related? No

→ Treat for preeclampsia + administer 1st-line antihypertensive medication within 30-60 min; **select 1 chain below**:

1) Labetalol 20 mg IV bolus
→ Repeat BP in **10 min**, if elevated administer
labetalol 40 mg IV bolus
→ Repeat BP in **10** min, if elevated administer
labetalol 80 mg IV bolus
→ Repeat BP in **10** min, if elevated administer
hydralazine 10 mg IV bolus
→ Repeat BP in **20** min, if elevated obtain anesthesia consult

2) Hydralazine 5-10 mg IV bolus
→ Repeat BP in **20** min, if elevated administer
hydralazine 10 mg IV bolus
→ Repeat BP in **20** min, if elevated administer
labetalol 20 mg IV bolus
→ Repeat BP in **10** min, if elevated administer
labetalol 40 mg IV bolus and obtain anesthesia consult

3) Nifedipine 10 mg PO
→ Repeat BP in **20** min, if elevated administer
nifedipine 20 mg PO
→ Repeat BP in **20** min, if elevated administer
nifedipine 20 mg PO
→ Repeat BP in **20** min, if elevated administer
labetalol 20 mg IV bolus + obtain anesthesia consult

2nd-line antihypertensive medication

→ Esmolol infusion 0.05-0.3 mg/kg/min IV

→ Nicardipine infusion 5-15 mg/h IV

Extreme emergency

→ Sodium nitroprusside infusion 0.1-1 mcg/kg/min IV

Committee Opinion No. 767. American College of Obstetricians and Gynecologists. Obstet Gynecol 2019;133:e174-80

Continued on next page

MATERNAL HYPERTENSION

PROPHYLAXIS

Magnesium sulfate
→ Administer for seizure prophylaxis: 4-6 g IV (over 20 min) + 1-2 g/h IV infusion (monitor serum levels)

DDX

1. Preeclampsia (with or without severe features*)
2. Preeclampsia superimposed on chronic HTN
3. Gestational HTN
4. Chronic (essential) HTN
5. HTN crisis (≥180/120 mm Hg)
6. Acute pain
7. CVA → Refer to neurology if suspected

*Severe features

1. SBP ≥ 160 mm Hg or DBP ≥ 110 mm Hg on two occasions, 4 h apart
2. Thrombocytopenia (<100 000/μL)
3. Elevated liver enzymes (2x normal range), RUQ or epigastric pain
4. Creatinine >1.1 mg/dL or 2x normal value
5. Pulmonary edema
6. New-onset cerebral or visual disturbances

END

American College of Obstetricians and Gynecologists. Executive summary: hypertension in pregnancy. Obstet Gynecol. 2013;122:1122-1131

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MATERNAL HYPOGLYCEMIA

START

DIAGNOSIS

Blood sugar **<60 mg/dL**

OR

Symptoms of hypoglycemia (regardless of blood sugar level)
and suspect if patient taking insulin/ hypoglycemic medication(s)

→ Tremor
→ Anxiety
→ Irritability
→ Tachycardia
→ Diaphoresis

CALL FOR HELP  **OB RAPID RESPONSE**

IMMEDIATE

Airway	Clear
Breathing	SpO_2 + RR
Circulation	HR + BP
Temperature	Assess
Conscious level	Assess
IV access	18G
Stat finger stick	Obtain
Serum blood sugar	Obtain

Continued on next page

MATERNAL HYPOGLYCEMIA

TREATMENT

If blood sugar <60 mg/dL (conscious + eating)
→ Treat with 8 oz milk, 4 oz juice, or 3 glucose tablets
→ Recheck blood sugar within 15 min, if blood sugar <60 mg/dL, repeat previous step
→ Recheck blood sugar every 15 min, until blood sugar >60 mg/dL (x2)

If blood sugar <60 mg/dL (conscious + NPO)
→ If on insulin drip, stop infusion
→ Maintain dextrose 5% in Lactated Ringer's solution at 125 mL/h
→ Recheck blood sugar every 15 min until blood sugar >60 mg/dL (x2)

If blood sugar <50 mg/dL (conscious)
→ Give 3 glucose tablets
→ Recheck blood sugar every 15 min until blood sugar >60 mg/dL (x2)

If the patient is unconscious
→ If on insulin drip, stop infusion
→ Obtain stat finger stick and serum blood glucose
→ Administer 50% dextrose 50 mL IV
→ If no IV access, administer glucagon 1 mg IM

DDX

1. Diabetes mellitus, incorrect insulin dosage
2. Acute fatty liver of pregnancy (AFLP)
3. Insulinoma

END

MATERNAL HYPOTENSION

START

DIAGNOSIS

Systolic Blood Pressure

<100 mm Hg OR **20%-30% decrease below baseline**

CALL FOR HELP  **OB RAPID RESPONSE**

IMMEDIATE

Team leader	Identify
Airway	Clear?
Breathing	SpO_2 + RR Auscultation
Circulation	HR + BP Capillary refill Urine output
Conscious level	Adequate?
IV access	2 large bore IVs, 16G (above level of diaphragm)
IO access	Humeral IO, if no IV access
Labs	CBC, ABO/T+C, coag screen, fibrinogen, lactate
Position	Left uterine displacement Consider passive leg raise, Trendelenburg, left lateral pos
Fetal heart rate	Monitor
Medication(s)	STOP or decrease (eg, epidural infusion, Mg, oxytocin infusion as indicated)

Continued on next page

MATERNAL HYPOTENSION

TREATMENT

Oxygen ⇒ 100% (10 L/min) via non-rebreather facemask

IV fluid bolus ⇒ 500-1000 mL IV crystalloid (pressurized) **Repeat, consider albumin/colloid if no immediate response**

Vasopressors
- ⇒ **HR > 40 bpm**
  - → **Phenylephrine** 100-200 μg IV bolus
- ⇒ **HR < 40 bpm**
  - → **Ephedrine** 5-10 mg IV bolus or **Glycopyrrolate** 100-200 μg IV bolus
- ⇒ **HR < 40 bpm, and hypotensive after spinal anesthetic**
  - → Consider Bezold-Jarisch reflex
  - → Fluids, left uterine displacement, passive leg raise, Trendelenburg, left lateral position
  - → **Phenylephrine** 100-200 μg IV bolus
  - → **Atropine** 0.1-0.2 mg IV bolus
  - → **Ephedrine** 5-10 mg IV bolus
  - → **Epinephrine** 10-100 μg IV bolus

Epinephrine ⇒ **10-100 μg IV bolus prn** if no immediate response or severe refractory hypotension

Vasopressor infusion
- ⇒ **Phenylephrine** 0.5-1 μg/kg/min IV
- ⇒ **Norepinephrine** 0.01-0.1 μg/kg/min IV
- ⇒ **Epinephrine** 0.01-0.1 μg/kg/min IV

Blood transfusion ⇒ If hemorrhage suspected

CONSIDER

1. Inadequate left uterine displacement
2. Vasodilation (sympathectomy) from epidural or spinal
3. Hemorrhage
4. Dehydration
5. LAST
6. Anaphylaxis
7. AFE
8. Cardiac event: MI, cardiac failure

END

MATERNAL HYPOXIA

START

DIAGNOSIS

PaO_2 < 80 mm Hg (on room air) **or** **$SaO_2 \leq 94\%$** (on room air)

IMMEDIATE

Airway	⇒	Clear?
Breathing	⇒	SpO_2 + RR
	⇒	Auscultation
Circulation	⇒	HR + BP
IV access	⇒	2 IVs, 18G
Fetal heart rate	⇒	Monitor
Labs	⇒	ABG, CBC
Other	⇒	POCUS, TTE

TREATMENT

Oxygen	⇒	100% (10 L/min) via non-rebreather facemask
Respiratory therapy	⇒	If indicated
Non-invasive ventilation	⇒	Consider (CPAP or BiPAP)
Intubation + mechanical ventilation	⇒	Consider (with PEEP)
Administer	⇒	Bronchodilators
	⇒	Antibiotics (if indicated)
	⇒	Steroids
	⇒	Diuretics (if indicated)
Reversal agent	⇒	Administer if secondary to drug effect, eg, opioids

Continued on next page

MATERNAL HYPOXIA

OTHER

Patient's torso ➔ Elevate (avoid supine position)

DDX

Hypoventilation
→ Drugs: narcotics, benzodiazepines, residual muscle blockade

→ Other: obesity, high neuraxial block

VQ mismatch
→ Atelectasis, aspiration, bronchospasm, pulm. edema, pleural effusion, mucus plug, emb. (air/blood/amniotic fluid), pneumothorax, pulm HTN

Right-to-left shunt
→ Anatomic (intracardiac, AVMs), physiologic shunts (pneumonia, ARDS)

Impaired diffusion
→ Interstitial lung disease

Reduced inspired oxygen tension
→ Altitude

END

LOCAL ANESTHETIC SYSTEMIC TOXICITY

START

DIAGNOSIS

Central Nervous System
- → Tinnitus
- → Metallic taste
- → Dizziness
- → Confusion
- → Seizures

Cardiovascular System
- → Bradycardia
- → Tachycardia
- → Hypotension
- → Hypertension
- → Arrthymias

CALL FOR HELP  **OB RAPID RESPONSE**

IMMEDIATE

Epidural injection and/or infusion	Stop
Team leader	Identify
Airway	Clear?
Breathing	SpO_2 + RR
Circulation	HR + BP
IV access	2 large bore IVs, 16G
Position	Left uterine displacement

Continued on next page

LOCAL ANESTHETIC SYSTEMIC TOXICITY

TREATMENT

1. Oxygen
→ 100% (10 L/min) via non-rebreather facemask

2. 20% Fat emulsion (immediate)
→ 1.5 mL/kg IV bolus + 0.25 mL/kg/min IV infusion
→ Repeat bolus x 1-2 if persistent CVS collapse
→ Increase infusion rate to 0.5 mL/kg/min IV if BP remains low
→ Continue infusion for at least 10 min after CVS stable

3. Seizing?
→ Midazolam 1-2 mg IV bolus or Lorazepam 4 mg IV bolus

4. Cardiac arrest
→ Start CPR and immediately give 20% fat emulsion IV (if not already)
→ May require prolonged CPR (>1 h)
→ Use reduced epinephrine doses (10-100 µg IV bolus) initially
→ Avoid lidocaine and vasopressin
→ Consider CPB/ECMO

OTHER

Definitive airway ⇒	Intubate if altered mental status or hemodynamic instability
Invasive monitoring ⇒	Consider arterial line, central line
Perimortem cesarean delivery (PMCD) ⇒	Consider in cardiac arrest

END

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MASSIVE TRANSFUSION PROTOCOL

DIAGNOSIS

Maternal hemorrhage
→ Antepartum
→ Intrapartum
→ Postpartum

Stanford MTP contains
→ 6 RBC units
→ 4 FFP units
→ 1 plts (6 pooled units)

START

CALL FOR HELP  **OB RAPID RESPONSE**

IMMEDIATE

1. Activate MTP
→ Closed loop verbal communication to nurse
→ Or, activate in electronic medical record
→ Nurse to call Blood Bank (no. on back cover) to activate MTP
→ Send runner to blood bank with a patient sticker

2. Send blood sample for ABO typing (prior to administering MTP)

3. Refer to Maternal Hemorrhage for other on-going management

TREATMENT

1. Transfuse (MTP or type-specific units) in the ratio of
→ 6 PRBCs
→ 4 FFP
→ 1 Plts (6 pooled units)

2. Replace fibrinogen early if coagulopathy suspected or fibrinogen <200 mg/dL
→ Cryoprecipitate (takes 45 min to thaw) or
→ Administer RiaSTAP (fibrinogen concentrate 1-2 g IV over 10 min), (swirl, do not shake) reconstitute in 50 mL sterile water

3. Labs
→ CBC
→ Coag screen
→ Fibrinogen
→ TEG/ROTEM
→ ABG
→ Calcium
→ Lactate

OTHER

→ Always stay ahead with availability of at least one full MTP

→ Once patient is stable and no further need for transfusion, return un-used units to blood bank

END

PLACENTAL ABRUPTION

START

DIAGNOSIS

→ Vaginal bleeding (not in all cases)
→ Abdominal pain +/– back pain
→ Preterm labor
→ Fetal heart rate abnormalities
→ Maternal hypotension

CALL FOR HELP  **OB RAPID RESPONSE**

IMMEDIATE

Team leader	Identify
Airway	Clear?
Breathing	SpO_2 + RR Auscultation
Circulation	HR + BP
Conscious level	Adequate?
IV access	2 large bore IVs, 16G
Labs	CBC Coag screen Fibrinogen T+C TEG/ROTEM
Position	Left uterine displacement
Fetal heart rate	Monitor
Invasive monitoring	Consider arterial line

Continued on next page

PLACENTAL ABRUPTION

TREATMENT

→ See Maternal Hypotension

→ May need to proceed with cesarean delivery (use neuraxial anesthesia after confirming no DIC)

OTHER

→ Vital signs can be inconsistent with the amount of blood that is visible as it may be concealed (retro-placental hematoma)

→ Delivery management or expectant management depending on maternal + fetal status

→ Risk of DIC + fetal demise

→ Can be acute or chronic

DDX

1. Labor
2. Placenta previa
3. Uterine rupture
4. Subchorionic hematoma

END

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PLACENTA ACCRETA

START

DIAGNOSIS

→ Placenta accreta
→ Placenta increta
→ Placenta percreta

IMMEDIATE

Multidisciplinary team meeting
→ OB/MFM/GYN-ONC/ANES/NICU/Blood bank

Discuss anesthetic plan with patient
→ Neuraxial (CSE), GA, or combination

TREATMENT

→ 2 large bore IVs, 14-16G
→ Awake pre-induction arterial line
→ RIC or central line (large bore)
→ 1-2 MTP, fibrinogen concentrate, tranexamic acid, calcium chloride (in OR prior to start)
→ Rapid infuser connected to large bore PIV or central line
→ Uterotonics (in OR prior to start): oxytocin, methylergonovine, carboprost, misoprostol
→ Forced-air warming
→ See General Anesthesia if indicated
→ See Maternal Hemorrhage if indicated

OTHER

Discuss need for IR pre-op
→ Uterine artery balloons

Consider cell saver

Have vasopressors prepared in OR
→ Phenylephrine
→ Norepinephrine
→ Epinephrine
→ Consider TEE/TTE to guide resuscitation

Turn off oxytocin after uterine arteries are clamped/tied
→ Hysterectomy

Consider ICU admission post-op

Check Plts/coag screen prior to epidural catheter removal
→ If applicable

END

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PROLAPSED UMBILICAL CORD

DIAGNOSIS

Prolapsed umbilical cord (visual or palpable) following
→ SROM
→ AROM
→ Delivery of Twin A

START

CALL FOR HELP  **OB RAPID RESPONSE**

IMMEDIATE

OB team ⇒ Continuous fetal monitoring
⇒ Relieve compression on the umbilical cord by manually elevating the presenting part of the fetus until delivery + continuously monitor fetal heart rate

IV access ⇒ 18G

Immediate surgery ⇒ Prepare for stat cesarean delivery (see Stat Cesarean Delivery)

TREATMENT

If fetal compromise
→ Administer GA (see General Anesthesia)

If no fetal compromise (discuss with OB)
→ Administer neuraxial anesthesia with continuous decompression of the umbilical cord + continuous monitoring of the fetal heart rate
→ Single-shot spinal in lateral position, or
→ CSE in lateral position, or
→ Dose *in situ* epidural catheter in supine position with left uterine displacement
→ Abandon neuraxial technique and proceed with general anesthesia if fetal heart rate changes indicate

END

MATERNAL SEIZURE

START

DIAGNOSIS

Pregnant or Postpartum + **Seizure**

CALL FOR HELP  **OB RAPID RESPONSE**

IMMEDIATE

- **Team leader** ⇒ Identify
- **Airway** ⇒ Clear?
- **Breathing** ⇒ SpO_2 + RR
- **Circulation** ⇒ HR + BP
- **Position** ⇒ Left uterine displacement
- **IV access** ⇒ 18G

TREATMENT

→ 100% (10 L/min) oxygen via non-rebreather facemask

→ Open airway maneuvers

→ Trendelenburg with lateral tilt

→ Protect limbs

→ Midazolam 2 mg IV bolus or lorazepam 4 mg IV bolus if seizure not self-terminating

→ Consider propofol 20-40 mg IV bolus if seizure not self-terminating (anesthesiologists only)

→ If Ddx Eclampsia: Administer magnesium sulfate 4-6 g (loading dose over 20 min) + 1-2 g/hr IV infusion (if already receiving magnesium, administer 2nd loading dose of 2 g IV [over 3-5 min])

→ Monitor fetal heart rate

Continued on next page

MATERNAL SEIZURE

OTHER

Induction of GA + intubation ⇒ Consider if:
- → Non-terminating seizure
- → Risk of aspiration
- → Hypoxic
- → Remains unconscious postseizure

Antihypertensive medications ⇒ Consider (see Maternal Hypertension)

Labs ⇒
- CBC
- LFTs
- BMP
- Glucose
- Magnesium (if on infusion)
- Toxicology screen

Delivery of fetus ⇒ Consider if prolonged fetal bradycardia after termination of seizure

DDX

1. Eclampsia
2. Epilepsy
3. Pseudoseizure
4. Hypoxia
5. Hypoglycemia
6. CVA
7. AFE
8. Medication cause (LAST, drug error, substance abuse)

END

MATERNAL SEPSIS

START

DIAGNOSIS

- → Hypothermia
- → Tachycardia
- → Hypotension
- → Hypoxia
- → Tachypnea
- → Diaphoresis
- → Oliguria
- → Altered mental status

CALL FOR HELP

IMMEDIATE

Team leader	Identify
Airway	Clear?
Breathing	SpO_2 + RR Auscultation
Circulation	HR + BP Capillary refill Urine output
IV access	2 large bore IVs, 16G
Position	Left uterine displacement
Labs	Lactate CBC BMP Glucose Coag screen ABG CRP
Obtain cultures (as appropriate)	Blood Urine Sputum CSF Wound Stool

Continued on next page

MATERNAL SEPSIS

TREATMENT

Respiratory support
→ Oxygen: 100% (10 L/min) via non-rebreather facemask
→ Consider non-invasive ventilation (CPAP or BiPAP)
→ Consider invasive ventilation if respiratory failure

Hemodynamic support (*Crit Care Med*. 2017;45:486-552)
→ Volume resuscitation: 30 mL/kg crystalloid in first 3 hours (if hypotensive or lactate ≥4 mmol/L)
→ Consider albumin infusion or blood transfusion
→ Vasopressor infusion if MAP < 65 mm Hg despite fluid resuscitation:
→ Norepinephrine
0.01-0.1 mcg/kg/min IV
→ Epinephrine
0.01-0.1 mcg/kg/min IV

Administer broad spectrum antibiotics within *1 hour* of diagnosis of sepsis
→ Obtain blood cultures (x2) prior to administration of antibiotics (plus other cultures as indicated)
→ Consult local protocols and consider the most likely source, examples include:
→ Piperacillin/tazobactam + vancomycin
→ If patient has a penicillin allergy, substitute piperacillin/tazobactam with gentamicin + clindamycin

OTHER

→ Monitor fetal heart rate

→ Consider arterial line + central line for monitoring or if inotropic support required

→ Consider corticosteroids for refractory septic shock: hydrocortisone 50 mg IV bolus q6 h

DDX

1. Sepsis
2. Septic shock
3. AFE
4. Hemorrhagic shock
5. Cardiogenic shock
6. Anaphylactic shock

END

HYPOTENSION AFTER NEURAXIAL ANESTHESIA

START

DIAGNOSIS

After neuraxial anesthesia block or bolus
→ Rapid rise in sensory blockade
→ Numbness and/or motor weakness in upper limbs
→ Hypotension: 20%-30% decrease in SBP below baseline or SBP < 100 mm Hg
→ Bradycardia (may be preceded by tachycardia)
→ Vocal changes
→ Dyspnea → apnea
→ Altered mental status → unconsciousness
→ Cardiac arrest

CALL FOR HELP  **OB RAPID RESPONSE**

IMMEDIATE

Team leader	Identify
Airway	Clear?
Breathing	SpO_2 + RR + auscultation
Circulation	HR + BP
Conscious level	Adequate?
IV access	2 IVs, 18G
Position	Left uterine displacement, passive leg raise, Trendelenburg, left lateral position
Fetal heart rate	Monitor

TREATMENT

Oxygen ⇒ 100% (10 L/min) via non-rebreather facemask

IV fluid bolus ⇒ 500-1000 mL IV crystalloid (rapidly infuse)
Repeat, consider albumin/colloid if no immediate response

Vasopressors ⇒ **HR > 40 bpm**
→ **Phenylephrine** 100-200 µg IV bolus
⇒ **HR <40 bpm**
→ **Ephedrine** 5-10 mg IV bolus or
Glycopyrrolate 100-200 µg IV bolus

Continued on next page

HYPOTENSION AFTER NEURAXIAL ANESTHESIA

TREATMENT

Vasopressors ⇒ **HR < 40 bpm, and hypotensive after spinal anesthetic**
- → Consider Bezold-Jarisch reflex
- → Fluids, left uterine displacement, passive leg raise, Trendelenburg, left lateral position
- → **Phenylephrine** 100-200 µg IV bolus
- → **Atropine** 0.1-0.2 mg IV bolus
- → **Ephedrine** 5-10 mg IV bolus
- → **Epinephrine** 10-100 µg IV bolus

Vasopressor infusion ⇒ Phenylephrine 0.5-1 µg/kg/min IV
⇒ Norepinephrine 0.01-0.1 µg/kg/min IV
⇒ Epinephrine 0.01-0.1 µg/kg/min IV

Fetal assessment ⇒ Monitor fetal heart rate continuously
⇒ If fetal distress, prepare for emergency cesarean delivery

Cardiac arrest ⇒ Start CPR (see Maternal Cardiac Arrest), administer epinephrine, consider PMCD (aim for delivery of fetus by 5 min)

END

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STAT CESAREAN DELIVERY

DIAGNOSIS

Immediate surgical delivery for maternal and/or fetal indications

START

CALL FOR HELP  **OB RAPID RESPONSE**

IMMEDIATE

→ Immediately transfer patient to the OR

→ Discuss with OB if time permits for neuraxial anesthesia

TREATMENT

→ Monitors: EKG, BP, SpO_2, RR

→ IV access (18G) + fluids

→ Left uterine displacement (also consider ramped position for airway)

→ Administer nonparticulate antacid (if GA)

→ Spinal/CSE/epidural (if not contraindicated) or GA (see General Anesthesia, #27)—TIME DEPENDENT

→ If functional epidural catheter *in situ*: bolus with 2% lidocaine/bicarbonate/epinephrine (1:200 000) in 5 mL increments (or 3% chloroprocaine in 5 mL increments)

→ Inform Peds team

OTHER

If no neuraxial opioids, consider

- Multimodal analgesia regimen + opiate-based PCA
- Consider bilateral TAP block
- Consider local anesthesia infiltration at incision site

END

TRANSFUSION REACTION

START

DIAGNOSIS

→ Recent or ongoing blood product transfusion

→ 20%-30% ↓ in SBP below baseline, or SBP <100 mm Hg

→ Fever, chills, pruritus, urticaria, wheezing or respiratory distress

CALL FOR HELP  **OB RAPID RESPONSE**

IMMEDIATE

Transfusion	**Stop** + disconnect blood product tubing
Team leader	Identify
Airway	Clear?
Breathing	SpO_2 + RR + auscultation
Circulation	HR + BP
Conscious level	Adequate?
IV access	2 large bore IVs, 16G
Position	Left uterine displacement
Fetal heart rate	Monitor

TREATMENT

→ Oxygen: 100% (10 L/min) via non-rebreather facemask

→ Give IV fluid bolus → 500-1000 mL crystalloid, repeat as indicated

→ If hypotensive, see Maternal Hypotension

→ If developing severe reaction, consider administering epinephrine + antihistamine, see Anaphylaxis.

Continued on next page

TRANSFUSION REACTION

DDX

1. TRALI, TACO, hemolytic reaction
2. Anaphylaxis
3. Sepsis

OTHER

→ Inform blood bank

→ Keep blood product bag/tubing + return to blood bank for testing

→ Other considerations:
May progress to DIC, respiratory failure, cardiovascular collapse

END

TWIN VAGINAL DELIVERY

START

DIAGNOSIS

→ **Vertex-vertex presentation**

→ **Vertex-nonvertex presentation**

→ **Twin B—Vaginal delivery or cesarean delivery**

IMMEDIATE

Review Physiology ⇒ Multiple gestation increases physiologic and anatomic changes of pregnancy
- ↑ Aortocaval compression
- ↑ Hypotension
- ↓ FRC
- ↑ Oxygen consumption

Logistics ⇒ Transfer patient to the OR

Airway + breathing ⇒ Monitor HR/BP/SpO_2

IV access ⇒ Ensure adequate IV access (2 IVs, 16-18G)

Analgesia ⇒ Ensure adequate neuraxial analgesia (epidural catheter *in situ*, or place CSE)

Anticipate + plan ⇒ Prepare for possible stat cesarean delivery under GA
- → Position patient (ramp?)
- → Airway equipment
- → Drugs

Continued on next page

TWIN VAGINAL DELIVERY

TREATMENT

Following delivery of Twin A, monitor Twin B and assess immediate need for:

- → Uterine/cervical relaxation to deliver Twin B vaginally (may need breech extraction or internal/external version)
  - → Nitroglycerine 400-800 µg (1-2 sprays) SL
  - → Nitroglycerine 100-250 µg IV bolus
  - → Terbutaline 250 µg SC or IV bolus
  - → RSI with high concentration of volatile anesthetic
- → Conversion to general anesthesia for stat cesarean delivery of Twin B
  - → See General Anesthesia

OTHER

Risks:

- → After delivery of Twin A, Twin B may have
  - → Non-reassuring fetal heart rate
  - → Non-vertex presentation
  - → Umbilical cord prolapse
  - → Head entrapment
- → PPH + uterine atony (see Maternal Hemorrhage + atony + review contraindications of uterotonic medications)
- → Retained placenta
- → Infection, if prolonged intrauterine manipulation

END

UTERINE ATONY

START

DIAGNOSIS

→ **Surgeon notes atony by palpation**

→ **Uterus appears boggy + non-contractile**



CALL FOR HELP



IMMEDIATE

Team leader ➔	Identify
Airway + breathing ➔	SpO_2 + RR
Circulation ➔	HR + BP
IV access ➔	2 IVs, 16-18G
2nd-line uterotonics ➔	Obtain medications (PPH kit) if not available in OR

Continued on next page

UTERINE ATONY

TREATMENT

1. IV fluids
→ Bolus 1000 mL crystalloid + repeat if indicated
→ Consider colloid, blood transfusion

2. Oxytocin bolus
→ 2 U IV bolus

3. Oxytocin infusion
→ 30-40 U/500 mL fluid at 125-500 mL/h (7.5-40 U/h)

4. Reassess uterine tone + EBL (2 min)
→ If adequate: Decrease oxytocin infusion rate to 125 mL/h (7.5 U/h)

→ If inadequate: Repeat oxytocin 2 U IV bolus

5. Reassess uterine tone + EBL (2 min)
→ Consult with OB and administer 2nd-line uterotonic agents, if required:
→ Methylergonovine 0.2 mg IM q2-4h (contraindicated in HTN/PreE), or
→ Carboprost 0.25 mg IM q15 min (contraindicated in asthma), or
→ Misoprostol 600-800 µg SL or 1000 µg PR

6. Reassess uterine tone + EBL (2 min)
→ Consult with OB + give a 2nd-line uterotonic agent, if required

OTHER

If uterine tone remains inadequate, discuss with OB team about other/surgical interventions:

1. Bakri balloon
2. B-Lynch suture
3. IR embolization
4. Hysterectomy

→ If EBL > 1000 mL, see Maternal Hemorrhage

END

UTERINE INVERSION

START

DIAGNOSIS

→ Uterine fundus palpable at cervical os or uterine tissue visualized at introitus

→ Fundus not palpable on abdominal exam

→ High suspicion in setting of manual extraction of placenta

→ Hypotension, profuse vaginal bleeding postpartum

→ Unexpected shock state out of proportion to bleeding

CALL FOR HELP  **OB RAPID RESPONSE**

IMMEDIATE

Airway → Clear?
Breathing → Sats + RR
Circulation → HR + BP

Oxygen → 100% (10 L/min) via non-rebreather facemask

IV access → 2 large bore IVs, 16G

Hemodynamics → IV fluid + vasopressors + anticholingeric medication as indicated

Move to OR → If no **immediate** uterine replacement possible by OB

Continued on next page

UTERINE INVERSION

TREATMENT

Stop oxytocin infusion

Relax uterus to facilitate uterine replacement

→ Nitroglycerin 1-2 sprays SL (0.4 mg/spray) or 50-100 µg IV bolus prn (AND administer 50-100 µg phenylephrine IV bolus prn)

→ Terbutaline 250 µg SC

→ If replacement of the uterus is unsuccessful, aid relaxation with intubation and general anesthesia using a volatile agent

If hemodynamically unstable

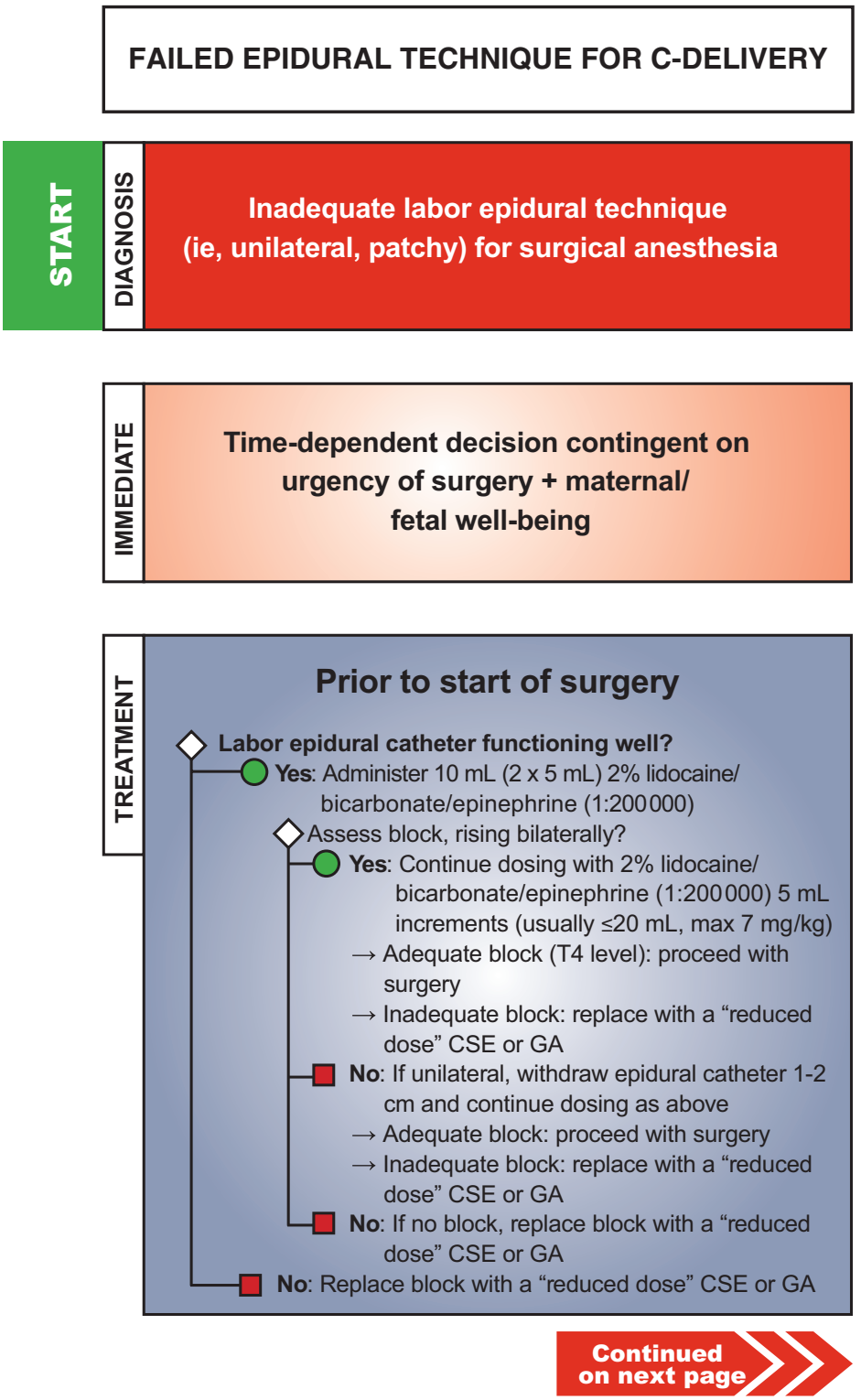
→ IV fluid bolus 1-2 L crystalloid (rapidly infuse)

→ Hypotension + HR < 40 bpm (can have profound vagal response): administer atropine 0.1-0.2 mg IV bolus

→ Hypotension + HR > 40 bpm: phenylephrine 100-200 µg IV bolus or ephedrine 5-10 mg IV bolus

→ Prepare for massive transfusion, high risk of blood loss with inversion (see Maternal Hemorrhage, MTP and Uterine Atony)

END



Continued on next page

FAILED EPIDURAL TECHNIQUE FOR C-DELIVERY

TREATMENT

Intraoperative

Surgery started with an adequate block, but now inadequate

Prior to uterine incision (depending on urgency)

→ Dose epidural catheter with 2% lidocaine/bicarbonate/epinephrine (1:200 000) 5 mL increments (max 7 mg/kg) + epidural fentanyl 100 µg (if not already administered)
- → If adequate: proceed with surgery
- → If inadequate: convert to GA

→ Convert to GA

After uterine incision

→ Dose epidural catheter with 2% lidocaine/bicarbonate/epinephrine (1:200 000) 5 mL increments (max 7 mg/kg) + epidural fentanyl 100 µg (if not already administered)

→ Consider 50%/50% N_2O/O_2
- → IV narcotics or adjuvants
  - → Fentanyl 50-100 µg IV bolus
  - → Morphine 10-15 mg IV or hydromorphone 1-2 mg IV in divided boluses
  - → Midazolam 1-2 mg IV bolus
  - → Ketamine 10-20 mg IV bolus

→ Convert to GA

OTHER

Alternative local anesthetic solution

→ 3% chloroprocaine 5 mL increments administered via the epidural catheter

Administer preservative-free morphine 3 mg

→ Via epidural catheter (may still have a postoperative analgesia effect, despite inadequate surgical anesthesia efficacy)

→ After delivery if preservative-free morphine not administered intrathecally

Reduced dose CSE

→ Intrathecal component
- → 0.75% Hyperbaric bupivacaine 0.8-1.4 mL
- → Fentanyl 15 µg
- → Preservative-free morphine 100-150 µg

END

GENERAL ANESTHESIA FOR C-DELIVERY

START

DIAGNOSIS

Indications for general anesthesia for cesarean delivery

→ Failure to extend *in situ* labor analgesia epidural technique for surgical anesthesia, or insufficient time for epidural technique to be functional prior to surgery

→ Inadequate time to place neuraxial anesthesia

→ Neuraxial anesthesia contraindicated

- → Coagulopathy
- → Critical aortic stenosis
- → Hemodynamic instability
- → Patient preference
- → Infection
- → Patient refusal
- → Other

IMMEDIATE

H+P ⇒ Perform

Equipment check ⇒ Anesthesia machine
⇒ Airway equipment
⇒ Video laryngoscope
⇒ Difficult intubation cart
⇒ Fiberoptic bronchoscope

Induction medications ⇒ Prepare

Monitors ⇒ EKG
⇒ BP
⇒ SpO_2
⇒ $ETCO_2$
⇒ Fetal monitor

IV access ⇒ 18G
⇒ IV fluid bag attached + infusing

Position ⇒ Good head/neck position (build ramp if indicated, tragus aligned with sternum)
⇒ Left uterine displacement

Other ⇒ Available staff member to escort partner out of the OR

Continued on next page

GENERAL ANESTHESIA FOR C-DELIVERY

TREATMENT

1. Premedications (if time permits)
- → Sodium bicitrate 30 mL po
- → Ranitidine 50 mg IV bolus + metoclopramide 10 mg IV bolus

2. Preoxygenate
- → 100% oxygen via anesthesia circuit for 3 min, or 4 maximal capacity breaths
- → CPAP/semi-upright position, if indicated

3. Patient prepped, surgeon scrubbed and ready to start?

4. Perform RSI with cricoid pressure
- → Propofol 2.0-2.5 mg/kg IV bolus (or etomidate 0.2 mg/kg IV bolus, or ketamine 2 mg/kg IV bolus in severely hypotensive patients)
- → Succinylcholine 1.5 mg/kg IV bolus (unless contraindicated)

5. After confirming correct placement of ETT, inform the surgeon to start

6. Maintenance anesthesia
- → Before delivery 50%/50% N_2O/O_2 + 0.75-1.0 MAC volatile (sevo or iso)
- → After delivery 70%/30% N_2O/O_2 + 0.5 MAC volatile (sevo or iso)

7. Administer prophylactic antibiotic(s)
- → If time permits prior to skin incision (but do not delay induction of anesthesia), otherwise administer when able

8. Midazolam 2 mg IV bolus (if indicated for amnesia)

9. Additional monitors
- → Temperature probe
- → Consider awareness monitor

10. Administer uterotonic(s) after delivery

11. Narcotics (after delivery)
- → Fentanyl 200-300 µg IV bolus
- → Morphine 10-15 mg IV in divided boluses, or
- → Hydromorphone 1-2 mg IV in divided boluses, or
- → Preservative-free morphine 3 mg via epidural catheter (if functional)

12. Administer antiemetics
- → Ondansetron 4 mg IV bolus
- → Dexamethasone 4 mg IV bolus

13. Local anesthesia (if no neuraxial block)
- → Consider bilateral TAP block, or
- → Infiltration at incision site

Continued on next page

GENERAL ANESTHESIA FOR C-DELIVERY

TREATMENT

14. Ensure patient fully awake prior to extubation
→ Semi-upright
→ Or left lateral position with Trendelenburg

15. Oxygen
→ 100% (10 L/min) via non-rebreather facemask

16. Postoperative analgesia (in addition to step13 above)
→ Multimodal (acetaminophen + NSAIDs)
→ IV PCA (if no neuraxial opioid)

OTHER

→ Be prepared for a difficult intubation
→ Modify RSI technique in patients with preeclampsia/eclampsia/raised intracranial pressure
→ Need to reduce a hypertensive response to laryngoscopy, therefore supplement induction drugs with
→ Nitroglycerine 1-2 μg/kg IV bolus + esmolol 1-2 mg/kg IV bolus and/or
→ Remifentanil 1.5 μg/kg IV bolus (if time permits) or
→ Fentanyl 100-200 μg IV bolus

END

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MATERNAL INTUBATION

START

DIAGNOSIS

Elective or emergency requirement for a definitive airway

IMMEDIATE

- **H+P** → Perform
- **Equipment check** →
  - Anesthesia machine
  - Airway equipment
  - Video laryngoscope
  - Difficult intubation cart
  - Fiberoptic bronchoscope
- **Induction medications** → Prepare
- **Monitors** →
  - EKG
  - BP
  - SpO_2
  - $ETCO_2$
  - Fetal monitor
- **IV access** →
  - 18G
  - IV fluid bag attached + infusing
- **Position** →
  - Good head/neck position (build ramp if indicated, tragus aligned with sternum)
  - Left uterine displacement

Continued on next page

MATERNAL INTUBATION

TREATMENT

Preoxygenate
→ 100% oxygen (10 L/min) via anesthesia circuit for 3 min, or 4 maximal capacity breaths

Rapid sequence induction with cricoid pressure

Medications
→ See General Anesthesia

Direct or indirect laryngoscopy
→ Short handled laryngoscopy blade with Miller 2 or Mac 3 blade
→ Consider video laryngoscope if anticipate difficult direct laryngoscopy
→ Anticipate difficult/failed intubation (see Difficult Airway)
→ Have LMA (ProSeal + classic) size 3 + 4, bougie, stylet, oral airways available

If not NPO, place OG tube + suction stomach contents

OTHER

→ Size 6.0-7.0 endotracheal tube with stylet

→ Extubation (on OR table)
→ Criteria: Following commands, adequate tidal volumes on minimal pressure support, maintaining saturations on low FiO_2
→ Position patient semi-upright or left lateral position with slight Trendelenburg
→ Oxygen 10 L/min via non-rebreather facemask

END

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ANESTHESIA FOR ECV

ECV

→ External cephalic version (ECV)

→ Usually attempted ≥36 weeks of gestation

→ Terbutaline 250 mcg SC may be administered for uterine relaxation (discuss with OB)

START

RISKS

→ **Fetal heart rate abnormalities**

→ **Placental abruption**

→ **Uterine rupture**

→ **Stat cesarean delivery**

ANESTHETIC OPTIONS

→ CSE: Intrathecal dose 0.75% hyperbaric bupivacaine 1 mL (dose range 2.5-10 mg) + fentanyl 10-20 μg (or sufentanil 5 mcg) to achieve T6 level

→ If proceeding to cesarean delivery bolus epidural catheter to appropriate T4 Level

→ Bolus epidural catheter with 2% lidocaine/bicarbonate/epinephrine (1:200 000) 5 mL increments (max 7 mg/kg) or 3% chloroprocaine 5 mL increments

Epidural catheter left *in situ* for labor analgesia or cesarean section depending on success of version

END

TROUBLESHOOTING LABOR EPIDURAL TECHNIQUES

TOPPING OFF

Patient complains of pain with labor epidural catheter *in situ*

→ Interview patient to determine location, intensity + quality of pain

→ Assess dermatome level of neuraxial blockade, examine epidural catheter insertion site + catheter/tubing connector

Unilateral block

→ Position patient with painful side downward + administer a bolus of 0.125%-0.25% bupivacaine 5-10 mL via the epidural catheter

→ Or position as above + withdraw the epidural catheter 1-2 cm + administer bolus of 0.125%-0.25% bupivacaine 5-10 mL via the epidural catheter

Insufficient dermatomal spread

→ Administer bolus of 0.125% bupivacaine 5-10 mL via the epidural catheter

Insufficient dermatomal density

→ Administer bolus of 0.25% bupivacaine 5-10 mL via the epidural catheter

Sacral sparing or assisted delivery

→ Sit patient upright + administer bolus of 0.25% bupivacaine 5-10 mL via the epidural catheter

→ Consider a bolus of fentanyl 100 mcg via the epidural catheter

Always check fetal heart rate after position change

Alternative local anesthetic: 0.2% ropivacaine 5-10 mL bolus

Continued on next page

TROUBLESHOOTING LABOR EPIDURAL TECHNIQUES

If you have bolused an epidural catheter x2 with insufficient pain relief, consider replacing with a CSE

TROUBLESHOOTING

CSF aspirated via epidural catheter

↳ Intrathecal catheter

→ Dose as an intrathecal catheter, or

→ Remove catheter and replace

Blood aspirated via epidural catheter

↳ Intravascular catheter

→ Withdraw epidural catheter 1-2 cm, flush 2 mL of normal saline, aspirate, if negative administer a test dose (1.5% lidocaine/epinephrine (1:200 000) 3 mL) + if negative proceed with use, or

→ Remove epidural catheter + replace

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115 Outpatient Anesthesia§

Stephen M. Klein

The scope of ambulatory anesthesia and surgery is continually expanding. This chapter discusses issues important to providing successful ambulatory anesthesia, including surgery and patient risk factor evaluation, postanesthetic recovery and discharge, unanticipated inpatient admission, perioperative emergencies and transfer, and office-based anesthesia.

1) **Overview**
   a) Outpatient anesthesia, or ambulatory anesthesia, is an anesthesiology subspecialty encompassing the preoperative, intraoperative, and postoperative anesthetic care of patients undergoing elective, same-day surgical procedures.
   b) By definition, patients undergoing ambulatory surgery do not require admission to the hospital and are well enough to be discharged from the facility after the procedure.
   c) In the United States in 2010, 48.3 million ambulatory surgeries were performed, 25.7 million in hospitals and 25.7 million in ambulatory surgery centers (ASCs).[1]
   d) Advancements in surgical technique and technology resulting in less invasive surgery, and advancements in anesthesia care and postoperative pain management, are allowing patients and surgeries once requiring inpatient care and resources to be scheduled in the outpatient setting. These advancements have resulted in reduced expenses in a competitive, cost-conscious health care environment with improved patient satisfaction.[2]
   e) The ASA Guidelines for Ambulatory surgery encompass recommendations pertaining to physicians, staff, facility, equipment, and clinical care that apply to anesthesiology personnel administering ambulatory anesthesia in all settings.[3]

READ MORE
Preanesthesia assessment, Chapter 4

2) **Risk evaluation for outpatient surgery**
   a) **Risk identification is the foundation for perioperative risk reduction.**
   b) **Conditions associated with increased risk in the outpatient population include:**[2,4–8]
      i) Congestive heart failure (CHF)
      ii) Coronary artery disease (CAD)
      iii) Hypertension
      iv) Cerebrovascular accident (CVA), transient ischemic attack (TIA)
      v) Asthma
      vi) COPD
      vii) Pulmonary hypertension
      viii) Obesity
      ix) Smoking
      x) Sleep apnea
      xi) Gastroesophageal reflux (GERD)



Risk identification is the foundation for perioperative risk reduction.

   c) **Signs and/or symptoms of instability or insufficient management in any of these areas require further investigation, optimization, and/or inpatient setting.**
   d) **Based on risk identification, the anesthesiologist should be able to mitigate adverse events and provide optimal care for the case.**

e) While an individual risk factor is important, it is the combination of different factors and their management that determines the final outcome, increasing or decreasing the risk of adverse events.[9]

f) When deciding whether a particular surgery and/or patient is suitable for the outpatient setting, it is necessary to identify whether the risk would be minimized in an inpatient environment and what hospital-based resources might be needed.



When deciding whether a particular surgery and/or patient is suitable for the outpatient setting, it is necessary to identify whether the risk would be minimized in an inpatient environment and what hospital-based resources might be needed.

g) A multidisciplinary approach to handling concerning issues will optimize perioperative management and lead to system improvements.[10]

h) While current evidence-based medicine can provide recommendations on some high-risk ambulatory issues, for others the evidence is still lacking and/or requires larger, well-designed studies (**Table 115-1**).

3) **Postanesthetic recovery and discharge**

a) **Reasons for delays after ambulatory surgery are multifactorial (Table 115-2).**[10]

b) **Postoperative pain and postoperative nausea and vomiting (PONV)**

i) Commonly prolongs postanesthesia care unit (PACU) stay after ambulatory surgery.[15,16]

ii) An aggressive plan for each of these issues must be addressed prior to surgery.

c) **Pain**

i) Pain control should be managed using a multimodal coordinated approach.

(1) Agents should be used in combination to provide analgesia with a focus on minimizing side effects.

TABLE 115-1 Concerns With Ambulatory Surgery

Cardiac	• Elective noncardiac surgery is not recommended within 14 days of coronary revascularization with balloon angioplasty.[11] • Following percutaneous coronary stent implantation, elective noncardiac surgery should be postponed 30 days after bare-metal stents (BMS)s and 6 mo after drug-eluting stents (DES).[11] • Elective noncardiac surgery after DES implantation in patients for whom P2Y12 inhibitor therapy (eg, clopidogrel) will need to be discontinued may be considered after 3 months if the risk of further delay of surgery is greater than the expected risks of stent thrombosis.[11] • When noncardiac surgery is required in patients currently taking a P2Y12 inhibitor, a consensus decision among treating clinicians as to the relative risks of surgery and discontinuation or continuation of antiplatelet therapy can be useful.[11] • Elective noncardiac surgery should not be performed within 30 days after BMS implantation or within 3 months after DES implantation in patients in whom DAPT will need to be discontinued perioperatively. • If possible, aspirin should be continued perioperatively.[11]
Respiratory	• Patients without acute respiratory symptoms do not need preoperative spirometry.[12] • Postoperative management of obstructive sleep apnea in the outpatient setting should include minimization of narcotics, regional anesthesia when appropriate, PACU protocols, and perioperative continuous positive airway pressure.[12]
Pediatric	• Ex-premature infants at a postconceptual age of 52-60 wk are considered amenable for ambulatory surgery due to a low incidence of postoperative apnea and bradycardia; anemia should be excluded.[13]
Geriatric	• While overall risk for elderly patients undergoing day surgery is low, in a study of patients >65 years of age, increased risk of inpatient hospital admission or death within 7 days was associated with age >85, prior inpatient hospitalization within 6 mo, surgical procedure performed in an office or outpatient setting, and invasiveness of surgery[14]

TABLE 115-2 Factors Associated With Delayed Discharge After Ambulatory Surgery

Preoperative	Intraoperative	Postoperative
Female gender	Long-duration surgery	PONV
Increasing age	General anesthesia	Pain
History of congestive heart failure (CHF)	Spinal anesthesia	Drowsiness
		No escort

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(2) Frequently used medications may include some or all of the following to maximize analgesia and minimize side effects: nonsteroidal anti-inflammatory, acetaminophen, NMDA antagonists, and opioids.

ii) Peripheral nerve blocks (PNBs) provide site-specific surgical anesthesia and postoperative analgesia, reducing opioid requirements and opioid-related side effects.[17]

iii) As part of a multimodal approach to postoperative pain management, PNBs with long-acting local anesthetic (LA) can provide prolonged analgesia.

(1) The duration can be extended further with the placement of a perineural catheter and subsequent continuous LA infusion at home.[17]

(2) Discharging patients home prior to regression of motor and sensory block can be done safely.[18]

(3) Patients receiving lower-extremity PNBs should receive coaching on crutch walking.

(4) Alternative care should be arranged for those identified as unsafe or without adequate home resources.

(5) Patients receiving upper-extremity PNBs should have the affected extremity placed in a sling.

(6) Instructions specific to protection and care of the blocked extremity should be provided.

(7) If being discharged with a perineural catheter, additional instructions on catheter care and removal, and signs and symptoms of LA toxicity should be included.

(8) Timing of PNB resolution and initiation of pain medications should be reviewed to minimize and anticipate pain when the PNB resolves.

(9) Intravenous lipid emulsion 20% must be available as part of the treatment strategy for LA-induced cardiotoxicity if PNBs are utilized.

d) **PONV**

i) PONV associated with ambulatory surgery increases health care costs due to delayed discharge (pharmaceutical costs and nursing care) and hospital admission.

READ MORE
Postoperative nausea and vomiting, Chapter 129



Postoperative pain control should be managed using a multimodal approach.

READ MORE
Peripheral nerve blocks, Chapter 44



PONV associated with ambulatory surgery increases health care costs due to delayed discharge and hospital admission.

READ MORE
Postanesthesia care unit, Chapter 130.

TABLE 115-3 Oral Fluid Intake and Voiding After Ambulatory Surgery

Oral fluid intake prior to d/c not mandatory, required only for select patients on case-by-case basis[10]
Low-risk patients can be d/c home without voiding. These patients should be instructed to return to the hospital if unable to void within 6-8 h. This includes patients undergoing short-acting spinal anesthetics for procedures at low risk for urinary retention.[21]
Patients at high risk for urinary retention should be required to void prior to discharge with a residual volume <300 mL as measured by ultrasound. If the bladder volume ≥500-600 mL, patients should undergo straight catheterization prior to discharge.[21]

ii) It accounts for 0.1%-0.2% of unanticipated admissions, which is significant in the United States where more than 31 million patients undergo ambulatory anesthesia each year.[19]
iii) An aggressive PONV prophylaxis and treatment algorithm is prudent.[19]

e) **Discharge criteria**
i) The major accreditation bodies in the United States require that policies and procedures be implemented to ensure the safe recovery of patients after ambulatory anesthesia.
ii) Various scoring systems have been devised to facilitate timely and safe patient PACU discharge and home readiness after ambulatory surgery.[10,20]
iii) Discharge criteria of an outcome-based system.
(1) Patient alert and oriented to time and place
(2) Stable vital signs
(3) Pain controlled by oral analgesics or PNB
(4) Nausea or emesis controlled
(5) Able to walk without dizziness
(6) No unexpected bleeding from the operative site
(7) Oral fluid intake and voiding on case-by-case basis[10,21] **(Table 115-3)**
(8) Discharge instructions and prescriptions received from surgeon and anesthesiologist
(9) Patient accepts readiness for discharge
iv) These systems maximize patient safety and avoid inappropriate or premature discharge.
v) Responsible adult escorts are mandatory after ambulatory surgery.[22]

4) **Unanticipated inpatient admission after ambulatory surgery**
a) **The reported incidence of unanticipated inpatient admission after ambulatory surgery is 1%-2%.[10]**
b) **Reasons for unanticipated inpatient admissions are multifactorial including long duration of surgery, postoperative bleeding, pain, and PONV[10] (Table 115-4).**

TABLE 115-4 Most Common Factors Associated With Unanticipated Inpatient Admission

- Long duration of surgery
- Postoperative bleeding
- Pain
- PONV

Modified by permission from Springer: Awad IT, Chung F. Factors affecting recovery and discharge following ambulatory surgery. *Can J Anaesth.* 2006;53:858-872.

c) **The low incidence reflects that preoperative identification and management of identified factors is successful.**
   i) Risk identification, including case selection
   ii) Multidisciplinary approach to handling concerning problems
   iii) Multimodal analgesia including regional anesthesia with PNBs
   iv) Aggressive PONV prophylaxis and treatment algorithm

5) **Perioperative emergencies and transfer**
   a) Anesthesiologist should be physically present until all patients have been discharged from anesthesia care.
   b) Personnel with training in advanced resuscitative techniques (eg, ACLS, PALS) should be immediately available.
   c) Availability of these providers allows for acute stabilization of perioperative emergencies.
   d) Perioperative emergencies extending beyond the resources or available patient care of the ambulatory or office-based setting must be identified immediately.
   e) Emergency care and transfer protocols must be in place and activated without hesitation.
   f) Options for transfer to the inpatient setting include:
      i) Activation of the 911 system
      ii) Hospital emergency medical services
      iii) Physically transporting the patient across a parking lot, hospital tunnel, etc. in a stretcher with oxygen, monitors, manual resuscitator, and any other resuscitative equipment
   g) Direct communication between the anesthesiologist, the admitting physician, and the emergency room or intensive care unit physician receiving the patient must occur, providing a thorough transfer of care.
   h) Training for such emergencies should occur at frequent intervals to ensure that in the event of an emergency, all personnel, both medical and administrative, can execute their roles, and that supplies are in order.[23]
   i) All outpatient centers should have medications (eg, dantrolene), equipment, and written protocols available to treat malignant hyperthermia when triggering agents are used.
   j) All outpatient centers should have intravenous lipid emulsion 20% available as part of the treatment strategy for LA-induced cardiotoxicity if PNBs are utilized.
   k) Surgeons should have admitting privileges at nearby hospitals, a transfer agreement with another physician who has admitting privileges, or an emergency transfer agreement with a nearby hospital.[23]



Perioperative emergencies extending beyond the resources or available patient care of the ambulatory or office-based setting must be identified immediately.

6) **Office-based anesthesia**
   a) Like surgery visits to freestanding outpatient surgery centers, office-based surgeries are increasing.
   b) There are special problems that anesthesiologists must recognize when administering anesthesia in the office setting.[24]
   c) In a comparative outcome analysis of procedures performed in physicians' offices and ASCs, the death rate per 100 000 procedures was 9.2 in offices and 0.78 in day surgery centers.[9]
   d) ASA guidelines for office-based anesthesia encompass recommendations pertaining to physicians, staff, facility, equipment, and clinical care that apply to anesthesiology personnel administering ambulatory anesthesia in the office-based setting:[24]
      i) Compared with acute care hospitals and licensed ambulatory surgical facilities, office operatories currently have little or no regulation, oversight or control by federal, state, or local laws.

ii) Anesthesiologists must satisfactorily investigate the office-based setting and structure, including:
   (1) Governance
   (2) Organization
   (3) Construction
   (4) Equipment
   (5) Policies and procedures, including fire, safety, drugs, emergencies, staffing, training, and unanticipated patient transfers.

iii) All office-based facilities should have a reliable source of oxygen, suction, resuscitation equipment, and emergency drugs (including dantrolene if triggering agents are used) and intravenous lipid emulsion 20% if PNBs are utilized.

iv) The anesthesiologist should adhere to the standards for pre- and postanesthesia care, anesthetic monitoring, and guidelines for ambulatory anesthesia and surgery.[3]



All office-based facilities should have oxygen, suction, resuscitation equipment, and emergency drugs (including Dantrolene if triggering agents are used) and intravenous lipid emulsion 20% if PNBs are used.

CHAPTER SUMMARY FOR OUTPATIENT SURGERY

Risk Identification	Critical for risk reduction and for assessing appropriateness of ambulatory surgery.
Postoperative Pain and Postoperative Nausea and Vomiting (PONV)	Pain and nausea/vomiting commonly prolong stay after ambulatory surgery.
Ambulatory Anesthesia	Regional anesthesia with PNBs should be used when possible PONV prophylaxis is essential A multidisciplinary perioperative approach will optimize perioperative management and lead to system improvements The anesthesiologist should be physically present until the patient has been discharged from anesthesia care
Emergency Care, Medications, and Transfer Protocols	Must be in place in the ambulatory as well as office-based settings. Personnel with training in advanced resuscitative techniques (eg, ACLS, PALS) should be immediately available until all patients are discharged home, allowing for acute stabilization of perioperative emergencies.

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116 Anesthesia for Gastrointestinal, Pancreatic, and Hepatobiliary Surgery

Barbara J. Wilkey • Kellie Hancock • Matthew J. Fiegel

Gastrointestinal (GI) surgeries include procedures involving the esophagus, stomach, small and large intestines. Other important organs involved in abdominal surgery are the gallbladder, extrahepatic biliary system, liver, and pancreas. Impairment of the GI tract and associated organs can affect the balance of water, electrolytes, and nutrients in the body as well as the functions of other body systems. Preoperative assessment of volume status, serum electrolytes, acid-base status, nutrition, and associated organ dysfunction becomes critical in these cases.[1]

1) **Gastrointestinal (GI) surgery**
   a) **Exploratory laparotomy** is performed to diagnose and treat various conditions including inflammation, infection, perforation, and abnormal growths. Patients presenting for exploratory laparotomy range from those with new-onset acute abdomen to those with abdominal sepsis and multiorgan failure, so careful preoperative evaluation with physical exam ination and review of laboratory data is important.[2]
      i) Preoperative evaluation:
         (1) Surgeries are often performed emergently.
         (2) It is important to balance the need for optimization of the patient's preexisting medical problems and correction of fluid deficits with the risk of a deteriorating condition if surgery is delayed.
         (3) Consider blood product or approved synthetic component therapy to correct underlying coagulopathy and pre- or intraoperative blood loss. If patients do not accept blood products, discussion and alternative therapy options should be agreed upon and documented in detail prior to operative intervention.
         (4) Additional intraoperative monitoring including arterial and central lines may be appropriate in patients with cardiovascular disease or hemodynamic instability.
         (5) Consider other monitors of volume status and cardiac output such as transesophageal echocardiography and pulse pressure variation monitoring devices.[3]
         (6) For elective colorectal surgery, steps for enhanced recovery can improve patient outcomes. Preoperative interventions include decreased fasting time (2 hours for clear liquids, 6 hours for a full meal), carbohydrate loading, multimodal pain plan, and administration of μ opioid antagonists.[4]
         (7) Consider neuraxial or regional anesthesia in eligible patients. For patients with open abdominal surgeries, thoracic epidural analgesia provides better postoperative pain control, decreased cardiopulmonary morbidity, and earlier return of GI function compared to opioids. Single-shot spinal analgesia and transversus abdominal plane blocks are other opioid-sparing options (**Table 116-1**).[4]



Check for autoanticoagulation, anticoagulant medication, and antiplatelet medication before neuraxial anesthesia!

TABLE 116-1 Appropriate Epidural Levels for Surgical Procedures

Level	Procedures
T6-7	Gastrectomy, esophagectomy, cholecystectomy, Whipple, hepatic resection
T8-9	Ovarian cancer debulking
T9-10	Colectomy, bowel resection, laparoscopic cholecystectomy, A-P resection
T10-12	Pelvic exenteration

ii) Intraoperative management: Intraoperative management will vary depending upon the indication for and type of surgery. Here are a few key points.
   (1) Consider placement of an arterial line preinduction if patients have baseline hypotension or there is clinical suspicion for hemodynamic deterioration immediately after induction.
   (2) For patients with full stomach, bowel obstruction, delayed gastric emptying, or pregnancy, a rapid sequence induction is indicated.[5]
   (3) For intraoperative hypotension in the setting of septic shock, norepinephrine may be the best first-line vasopressor.[6]
   (4) For colorectal surgery, steps for enhanced surgical recovery include prophylactic antibiotics, judicious red blood cell transfusion based on clinical situation for patients with hemoglobin between 6 and 10 g/dL, goal-directed fluid therapy, use of standardized intraoperative anesthesia pathways, and normothermia.[4]
   (5) Though a heavily contested topic, a recent study suggests that use of colloid over crystalloid for goal-directed fluid therapy in major abdominal surger may result in improved outcomes.[7]

iii) Postoperative considerations:
   (1) Maintenance of temperature, use of short-acting anesthetic agents, and adequate reversal of muscle relaxation can improve extubation success.
   (2) If rapid sequence intubation is deemed necessary, care is required during emergence from anesthesia because the patient is at high risk of aspiration as they return to consciousness with impaired airway reflexes.[8]
      (a) Waiting until the patient is fully awake and following commands, elevation of the head of the bed prior to extubation, and placement in left lateral position after extubation may reduce the risk of aspiration.
   (3) For patients with continuing massive blood transfusion, or requiring high levels of hemodynamic support during surgery, it may be prudent to postpone extubation and plan for postoperative ventilation.
   (4) Respiratory complications are common following abdominal surgery. 13%-33% of patients have postoperative respiratory problems, including splinting, abdominal distension, diaphragmatic dysfunction, and acute lung injury.[9]
   (5) ICU care may be necessary for patients with large fluid shifts, cardiovascular instability, poor respiratory function, or signs of acute kidney injury.



Check to see if your facility has Enhanced Recovery After Surgery guidelines for major abdominal surgery

b) Cancer debulking procedures can increase the efficacy of chemotherapy and radiation and help relieve symptoms caused by tumor bulk. Cytoreduction surgery is often long and complicated. For certain tumors, hyperthermic intraperitoneal chemotherapy

(HIPEC) is an option. HIPEC involves the delivery of cytotoxic drugs in a hyperthermic environment. HIPEC is often done in conjunction with tumor debulking.[10]

i) Preoperative evaluation highlights:
   (1) Chemotherapy drugs may have significant side effects, and it is important to review the toxicity and possible side effects of the patient's chemotherapeutics.
      (a) *Bleomycin* and *Busulfan* have pulmonary effects.
      (b) *Doxorubicin* and *Adriamycin* cause dose-dependent cardiomyopathy.
      (c) *Cisplatin*, *Paclitaxel*, and *Vincristine* are associated with peripheral and/or autonomic neuropathies.[10]
   (2) Patients often have baseline anemia, thrombocytopenia, electrolyte abnormalities, and malnutrition and may suffer from the effects of metastatic lesions in liver, lung, bone, or brain.[10]
   (3) HIPEC causes increased myocardial oxygen consumption. Cytoreduction with HIPEC often requires aggressive fluid resuscitation. Patients with underlying coronary artery disease, ventricular dysfunction, and/or valvular disease may not tolerate this procedure.[11]
   (4) During HIPEC, patients develop an increased cardiac output, a decreased systemic vascular resistance, tachycardia, and elevated end-tidal CO_2.[11] Given these physiologic changes and potential for blood loss, invasive arterial blood pressure monitoring and cardiac output monitoring are felt essential. Some centers prefer peripheral to central access; however, patients may not have peripheral venous architecture amenable to large-bore peripheral access. In this case, a central venous access is necessary.
   (5) If the patient is to have a laparotomy, epidural placement for postoperative analgesia should be considered in patients without contraindications. Epidurals have proven safe during HIPEC procedures.[11]

ii) Intraoperative management highlights[11]:
   (1) These patients generally have significant abdominal distention and intra-abdominal fluid accumulation. Rapid sequence induction is generally indicated.
   (2) Goal-directed fluid therapy is generally considered appropriate.
   (3) During the HIPEC phase, all fluid and bed warmers should be turned off. Significant electrolyte and pH abnormalities can occur during this phase. Arterial blood gas and electrolytes should be monitored and treated. Do not forget to check magnesium levels!
   (4) If HIPEC is done with a closed abdomen, there is risk of impaired preload and abdominal compartment syndrome.
   (5) During closed abdomen HIPEC, hepatic blood flow and liver function decrease. This is a temporary phenomenon that occurs when the abdomen is full of chemotherapeutic agent.[12]
   (6) Cardiac output generally returns to baseline soon after restoration of normothermia.
   (7) The patient is at risk for acute kidney injury due to nephrotoxicity of chemotherapeutic agents.
   (8) Consider nasopharyngeal temperature monitoring.

iii) Postoperative considerations:
   (1) Most patients can be extubated at the end of the operation.
   (2) See postoperative considerations for exploratory laparotomy for further information.



HIPEC may be done with the assistance of a perfusion team, though no vessel cannulation is involved.

2) **Pancreatic surgery**
   a) **Whipple procedure**
      i) Most common operation performed for pancreatic cancer
      ii) Classic Whipple is a pancreaticoduodenectomy followed by pancreaticojejunostomy, a hepaticojejunostomy, and a gastrojejunostomy. Cholecystectomy is also a part of the procedure.
      iii) May be performed by laparotomy or laparoscopy
      iv) Preoperative evaluation highlights:
         (1) Pancreatic cancer and smoking tobacco are strongly associated.
            (a) Evaluate for underlying pulmonary disease.
            (b) Smoking cessation should occur at least 8 weeks prior to surgery.[13]
         (2) Most patients with pancreatic cancer have diabetes mellitus or glucose intolerance. An intraoperative insulin infusion will likely be required.[13]
         (3) The presence of jaundice is associated with increased incidence of postoperative complications.[13]
         (4) Blood loss depends on metastatic involvement of hepatic and portal vessels, preexisting coagulation abnormalities, and the presence of portal hypertension.[14]
         (5) All patients require large-bore IV access, type and crossmatch (T&C), and fluid warmers.
         (6) Invasive monitoring, including arterial line and central line, should be considered but is not mandatory. Decision should be made upon patient's comorbidities, anticipated blood loss, and institutional practice.
         (7) For an open procedure, thoracic epidural should be considered for postoperative pain management.
      v) Specific intraoperative considerations:
         (1) Carefully titrate muscle relaxants in the beginning of the operation as unresectable disease may be found after opening and the operation may be cancelled.
         (2) Though there are no specific studies evaluating the effectiveness of goal-directed fluid therapy in pancreatic surgery, patients undergoing pancreatic surgery have been parts of larger studies looking at goal-directed fluid therapy in major abdominal surgery. Relative intraoperative fluid therapy restriction has been shown to decrease postoperative mortality and complications in pancreatectomy.[15]
         (3) Intraoperative complications include possible hemorrhage from portal vein or vena caval injury.[14]
      vi) Postoperative considerations:
         (1) Most patients can be extubated at the end of the operation.
         (2) Anastomotic leak, intra-abdominal abscess, and fistula formation are the more common surgical complications.[14]
         (3) See postoperative considerations for exploratory laparotomy for further considerations.



For cancer surgeries, careful titration of muscle relaxants early in the case is important due to the possibility of an "open and close" procedure if advanced disease is found.

3) **Hepatobiliary surgery**
   a) Disorders of the liver and biliary tract can be categorized as parenchymal disease (hepatitis and cirrhosis) and cholestasis (involving the extrahepatic biliary pathway).
   b) Liver biopsy is an important tool for diagnosis and staging of liver disease.
      i) Preoperative evaluation[16]:
         (1) Methods for obtaining liver tissue include percutaneous, transjugular, laparoscopic, and ultrasound or CT-guided fine needle aspiration.

(2) Antiplatelet medications and anticoagulants should be discontinued prior to the procedure. Additionally, coagulation status should also be evaluated.
(3) The American Association for the Study of Liver Diseases (AASLD) recommends platelet transfusion prior to biopsy for a platelet count <50 000. The AASLD also recommends a transjugular approach for patients with a prothrombin time prolonged by more than 4 seconds.
(4) DDAVP may be considered in patients with chronic renal failure as the interaction between platelets and factor VIII and vWF may be the primary hemostatic defect in uremia.
(5) Patients should have at least a type and screen performed preoperatively.

READ MORE
See Chapter 56 for further discussion of coagulopathy management in end-stage liver disease.

ii) Specific intraoperative considerations[16]:
(1) In general, these procedures should be performed under light sedation.
(2) If the patient is uncooperative, the procedure may need to be done under heavy sedation or general anesthesia.
(a) If general anesthesia is required, rapid sequence induction may be necessary.

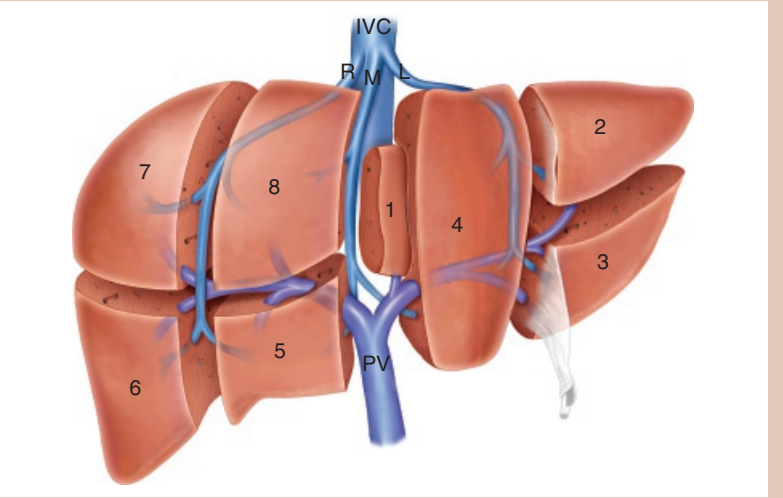
iii) Postoperative considerations:
(1) Complications include intraperitoneal hemorrhage, hematomas, hemobilia, bile peritonitis, and transient bacteremia.

c) **Liver resection**

i) Up to 80% to 85% of the liver can be safely resected assuming good function of the remaining liver (17). The liver is divided into eight segments, with the left lobe being made of segments 2, 3, an 4 and the right lobe of segments 5-8. The caudate lobe is segment 1[18] (See **Fig. 116-1**).
(1) Preoperative evaluation highlights (**Table 116-2**):
(a) Etiology and sequdelae of liver disease should be assessed.
(b) Specifics of the surgical plan
(c) Large bore vascular access is necessary. Invasive arterial monitoring should be considered.
(d) Type specific and crossmatched blood should be available. Consider cell salvage.
(e) Vasopressors should be available.
(f) Enhanced recovery protocols are beneficial after hepatic resection. These generally involve opioid sparing/multimodal pain control regimens including thoracic epidural, early ambulation, diet advancement, and avoidance of hypervolemia.[17]

ii) Specific intraoperative considerations:
(1) If ascites or other cause of delayed gastric emptying is present, rapid sequence induction should be performed.
(2) The anesthesiologist should use techniques to lower the central venous pressure prior to resection to decrease bleeding during resection. Techniques include fluid restriction, reverse Trendelenburg position, epidural infusion, and nitroglycerin infusion.[17]
(3) The surgeon may use a vascular occlusion strategy to limit bleeding. One strategy is called the Pringle maneuver. This involves clamping the hepatic artery, portal vein, and common bile duct.

FIGURE 116-1 Segments 2 and 3: Left Lateral Segment; Segment 4: Left Medial Segment; Segments 6 and 7: Right Posterior; Segments 5 and 8: Right Anterior; Segment 1: The Caudate Lobe.



Reprinted with permission from Cardinal JS. Left Hepatic Lobectomy. In: Hughes SJ, Mullholland MW, eds. *Operative Techniques in Hepato-Pancreato-Biliary Surgery*. Philadelphia, PA: Wolters Kluwer;2016:183.

(a) Hemodynamic changes during Pringle include hypertension, increased systemic vascular resistance, and decreased cardiac output.[17]

(4) If the resection is due to a tumor near the IVC or portal vein, total vascular occlusion may be necessary. Total vascular occlusion is a Pringle maneuver plus clamping of the supra and infrahepatic inferior vena cava.[17] This type of vascular exclusion is likely to result in significant hypotension requiring vasopressor administration.

(5) Use of the thromboelastogram helps guide coagulopathy correction and may reduce transfusion requirements during hepatobiliary operations.[18]

(6) Patients often have postoperative hepatic dysfunction due to decreased liver volume and transient dysfunction of the remnant liver. The anesthesiologist should take this into consideration when administering intraoperative medications as well as when prescribing opioids in the recovery area and postoperative unit.

TABLE 116-2 Approach to the Patient Undergoing Liver Resection

- The etiology of the liver disease should be noted so associated pathology can be addressed.
- The patient should be assessed for sequelae of liver disease such as portal hypertension portopulmonary syndrome, pulmonary hypertension, and renal insufficiency.
- The anesthesiologist should have a clear understanding of the exact sections to be resected, as well as the vascular occlusion techniques the surgeon plans to use.
- Patients should have vascular access that allows rapid, large volume transfusion. Rapid and significant blood loss is possible. Arterial monitoring should be considered.
- Patients should be type and crossed. Additionally, use of cell salvage should be considered as part of a blood conservation strategy.
- Vasopressor should be immediately available.
- Consider use of an enhanced recovery protocol.

iii) Postoperative considerations:
(1) Most patients can be extubated at the end of the operation.
(2) Complications include liver failure, renal impairment, bleeding, and biliary leak.
(3) See postoperative considerations for exploratory laparotomy for further information.



There is decreased metabolism of opioids after liver resection. If a significant amount of liver is removed, consider decreasing opioid doses and placing patient on continuous pulse oximetry monitoring postoperatively.

d) **Cholecystectomy**
i) Gall stones are the most common cause of diseases of the gall bladder.
ii) The gall bladder can be removed via an open technique or laparoscopy, occasionally requiring cholangiogram to delineate anatomy.
iii) Opioid-induced sphincter of Oddi spasm is an often-discussed phenomenon. However, incidence is very low and probably not a factor in the selection of pain management drugs. If spasm occurs, it can be treated with IV glucagon, naloxone, or nitroglycerin.[19]
iv) Bile leakage, hemorrhage from the gall bladder bed, liver injury, and iatrogenic bile duct injury can occur.
v) Anesthetic considerations for laparoscopic cholecystectomy are like those for other laparoscopic procedures.

READ MORE
Anesthesia for laparoscopic and robotic surgery, Chapter 117

CHAPTER SUMMARY FOR GI AND HEPATOBILIARY SURGERY

Overview	GI surgeries include procedures involving the esophagus, stomach, small and large intestines, pancreas, and gallbladder. Patients may have abnormalities with electrolyte and acid-base balance, and nutritional deficits.
Preoperative Considerations	Assess volume status, electrolytes, acid-base status, and coagulation. Balance optimization of the patient's preexisting medical problems and correction of fluid deficits and laboratory abnormalities with the risk of a deteriorating surgical condition.
Intraoperative Considerations	The degree of IV access and invasive monitoring is determined by the extent of surgery and the patient's underlying medical conditions. Vigilant monitoring of hemodynamic status, fluid and blood losses, coagulation, electrolytes, and acid-base status is critical.
Postoperative Considerations	Thoracic epidural analgesia for postoperative pain has many advantages in elective gastrointestinal and hepatobiliary surgery, but hypovolemia, bacteremia, and coagulopathy may preclude its use.

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117 Anesthesia for Laparoscopic and Robotic Surgery§

Pedro P. Tanaka • John H. Nguyen

Laparoscopic surgery is one of the principal techniques for minimally invasive surgery of the abdomen and is used for procedures that range across multiple surgical subspecialties. It describes a group of operations performed with the aid of a camera placed in the abdomen or the pelvis. During recent years, such techniques have been enhanced by robotic manipulation of laparoscopic surgical instruments. With growing market pressures for minimally invasive procedures, the role of robotic-assisted surgery and its advantages of improved surgical precision over standard open and laparoscopic procedures will likely continue to grow. Anesthesiologists should have knowledge of these systems to formulate an anesthetic plan, recognize potential complications, and provide safe patient care.

1) **Overview of laparoscopic surgery**
   a) Indications include gynecologic, abdominal, urology and, thoracic procedures.
   b) **Advantages**
      i) When compared to open abdominal surgery, advantages include reduced surgical trauma, less pain, fewer postoperative pulmonary complications, reduced time to initiation of oral diet, faster return of gastrointestinal function, reduced health care costs, and shorter recovery times.[1]
   c) **Disadvantages**
      i) Technical difficulty of the procedure
      ii) Operative position of the patient
   d) Prevalence of technique
      Because expertise and equipment have improved, laparoscopy has become one of the most common surgical procedures performed on an outpatient basis.
   e) Establishment of pneumoperitoneum
      i) Veress needle insertion
         (1) Inserted blindly, often into an intraumbilical incision through fascia into the peritoneum
         (2) CO_2 gas is insufflated through the needle to establish a pneumoperitoneum.
      ii) Trochar insertion
         (1) Trochars are ports through which laparoscopic instruments are inserted into the abdomen.
         (2) Trochars are inserted once a pneumoperitoneum has been established.
      iii) Minilaparotomy
         (1) Pneumoperitoneum can also be established by inserting an insufflation cannula surgically through an open mini-laparotomy procedure.



A Veress needle is a spring-loaded needle used to create a pneumoperitoneum for laparoscopic surgery. It contains an inner spring-loaded stylet with a dull tip to protect any viscera from injury when penetrating through tissue.

2) **Pathophysiologic effects and complications**
   The pneumoperitoneum is achieved by insufflating the abdomen with CO_2. These maneuvers induce a number of clinically relevant pathophysiologic effects during surgery.

a) **Cardiovascular system**
   i) Hemodynamic effects
      (1) Pneumoperitoneum *increases*
         (a) Systemic vascular resistance (SVR)
         (b) Mean arterial pressure (MAP)
         (c) Cardiac filling pressures
      (2) Pneumoperitoneum *decreases*
         (a) Venous return
         (b) Cardiac output (CO)
      (3) These effects are proportional to the increase in intra-abdominal pressure (IAP) and are significant with insufflation pressures >10 mm Hg.
      (4) Mechanism
         (a) Increased IAP causes compression of the abdominal organs and vessels.
         (b) Resistance to flow through arterial beds is increased due to both mechanical and neurohumoral factors.
         (c) Caval compression and increased venous resistance lead to decreased venous return.
         (d) Intrathoracic pressure transmitted to the heart causes increased cardiac filling pressures.
         (e) Decreased preload and increased afterload result in decreased CO.
         (f) MAP is increased overall because increases in SVR exceed decreases in CO_2.
      (5) Management
         (a) The lowest insufflation pressure required to achieve adequate surgical exposure (<15 mm Hg) should be used.
         (b) Increases in SVR may be treated with vasodilating agents, centrally acting α_2-agonists, or beta-blockers.
         (c) Decreases in venous return and CO may be attenuated by appropriate preloading prior to the induction of pneumoperitoneum.
   ii) Complications
      (1) Cardiac arrhythmias
         (a) Bradyarrhythmias, dysrhythmias, and asystole can occur during insertion of laparoscopic ports or during insufflation of the abdomen.
         (b) Mechanism
            (i) Sudden stretching of the peritoneum can precipitate a sudden and reflexive increase in vagal tone.
         (c) Management
            (i) Slow insufflation of CO_2 can decrease the risk of arrhythmias.
            (ii) Administration of anticholinergic medications may be appropriate for bradyarrhythmias.
            (iii) If the arrhythmia persists or results in hemodynamic compromise, prompt interruption of the surgery and release of pneumoperitoneum is indicated.



Sudden stretching of the peritoneum can precipitate a sudden and reflexive increase in vagal tone leading to bradycardia.

Slow insufflation of CO_2 can decrease the risk of arrhythmias.

b) **Pulmonary and respiratory system**
   i) Ventilatory effects
      (1) Mechanism
         (a) Pneumoperitoneum transmits pressure to the thorax and decreases pulmonary compliance by 30%-50%.
         (b) Elevation of the diaphragm results in decreased functional residual capacity (FRC), leading to atelectasis, *V/Q* mismatching, and hypoxemia.

(c) Hypercapnia results from systemic absorption of the CO_2 pneumoperitoneum.[2]

(2) Management

(a) Institution of positive end-expiratory pressure (PEEP) can mitigate the decreases in FRC and atelectasis.

(b) Hypercapnia can be managed by increasing respiratory rate instead of tidal volume.

(c) Protective lung ventilation with low tidal volume (6-8 mL/kg ideal body weight) and PEEP (5-10 cm H_2O) during pneumoperitoneum is associated with fewer pulmonary complications.[3]

ii) Complications

(1) CO_2 subcutaneous emphysema

(a) This is the most common respiratory complication during laparoscopy.

(b) It is suggested by an increase in end-tidal CO_2 ($ETCO_2$) >25% or an increase that occurs >30 minutes after abdominal CO_2 insufflation.

(c) The cause is extraperitoneal insufflation of CO_2. Some cases are accidental. Others are intentional (eg, in nephrectomies), where retroperitoneal exposure is required.

(d) Management

(i) Hypercapnia can be treated with an increase in mechanical ventilation.

(ii) In cases where the degree of hypercapnia becomes unmanageable with hyperventilation alone, the pneumoperitoneum can be temporarily released to allow for CO_2 elimination.

(2) Pneumothorax

(a) Mechanism

(i) Movement of gas from the peritoneum into the thorax can occur under pressure through weak areas and defects in the diaphragm.

(ii) Pneumothorax may be asymptomatic.

(iii) Pneumothorax may manifest as increased peak airway pressures, decreased O_2 saturation, and hypotension.

(iv) In severe cases, profound hypotension and cardiac arrest can occur.[3]

(b) Management

(i) Early diagnosis and treatment can be lifesaving.

(ii) Surgery should be stopped and the pneumoperitoneum released.

(iii) Supportive measures should be continued while confirming the diagnosis, either clinically or with chest radiograph.

(iv) Depending on the degree of cardiopulmonary compromise, the pneumothorax may be observed or treated with an intercostal cannula or a thoracostomy tube.

(v) After stabilization of the patient, conversion to an open procedure may be indicated.[4]



Early diagnosis and treatment of intraoperative pneumothorax can be lifesaving.

Surgery should be stopped and the pneumoperitoneum released.

(3) Endobronchial intubation

(a) Mechanism

(i) Elevation of the diaphragm during pneumoperitoneum can lead to endobronchial intubation.

(ii) Manifests as a decrease in O_2 saturation as a result of intrapulmonary shunt and increased airway pressure[3]

(b) Management

(i) The lungs should be auscultated, and the endotracheal tube should be slightly withdrawn as needed.

(4) Gas (CO_2) embolism
  (a) Mechanism
    (i) Gas embolism, although rare, has a mortality rate of nearly 30%.
    (ii) Profound hypotension, arrhythmias, or asystole can occur as a result of a "gas lock" in the vena cava or right ventricle (RV) that interrupts circulation.
    (iii) An increase in $ETCO_2$ is observed. This may be followed by an acute decrease in $ETCO_2$ if there is severe hypotension.
    (iv) The major cause is intravascular insufflation of gas from misplacement of the Veress needle or trocar either directly into a vessel or into a parenchymal organ.
    (v) Risk factors include hysteroscopy, hypovolemia, and a history of prior abdominal surgeries.
  (b) Management
    (i) Initial steps include immediate deflation of the pneumoperitoneum, institution of 100% FiO_2.
    (ii) Place patient in the left lateral head-down position to remove air from the RV outflow track.
    (iii) Hyperventilate to eliminate the increased $PaCO_2$ caused by the sudden increase in pulmonary dead space.
    (iv) A central line may be required to aspirate gas from the RV.
    (v) CPR may be required.
    (vi) Hyperbaric O_2 treatment should be considered if there is suspicion of cerebral gas embolism.[4]

(5) Well-leg compartment syndrome[4]
  (a) Definition
    Refers to the occurrence of compartment syndrome in an uninjured limb in the absence of an underlying systemic predisposing cause.
  (b) Mechanism
    (i) Impaired arterial perfusion to raised lower limbs
    (ii) Compression of venous vessels by lower limbs supports
    (iii) Reduced femoral venous drainage due to the pneumoperitoneum
    (iv) Risk factors include surgery more than 4-hour duration, muscular lower limbs, obesity, peripheral vascular disease, hypotension, and steep Trendelenburg positioning.
    (v) Presents after operation with disproportionate lower limb pain, rhabdomyolysis, and potentially myoglobin-associated acute renal failure
    (vi) Avoid intermittent compression stockings, moving the patient's legs at regular intervals during surgery, and using heel/ankle supports instead of calf/knee supports.[5]
  (c) Effects of positioning
    (i) Hemodynamic effects
      1. The head-down position generally increases venous return and CO, whereas the head-up position has the opposite effect. Steep Trendelenburg position causes a further increase on these parameters.
    (ii) Ventilatory effects
      1. The head-down position generally decreases FRC, overall lung volumes, and lung compliance.

3) **Patient monitoring**
   a) Routine ASA monitors are indicated.
   b) Neuromuscular monitors should be used to ensure adequate paralysis.
   c) In elderly ASA III and IV patients with cardiopulmonary comorbidities, consider invasive monitoring.[6]
4) **Anesthetic technique**
   a) A wide variety of anesthetic drugs have been used for laparoscopic procedures, and most are satisfactory.
   b) **Induction of anesthesia**
      i) IV induction agents, such as propofol and etomidate, and a variety of muscle relaxants, including succinylcholine, rocuronium, cisatracurium, and vecuronium, have been reported.
      ii) Although the benefit of rapid sequence induction has been questioned, it is still standard of care in patients with risk of pulmonary aspiration.
      iii) Special attention should be given during pneumoperitoneum creation, due to occurrence of embolic episodes and dysrhythmias.


Special attention should be given during pneumoperitoneum creation due to occurrence of embolic episodes and dysrhythmias.

   c) **Maintenance of anesthesia**
      i) As more procedures are conducted on an outpatient basis, the choice of maintenance agent is likely to be reduced to short-acting drugs such as sevoflurane, desflurane, and infusions of propofol.
      ii) There is no clinical advantage to omitting nitrous oxide.[6]
      iii) Because laparoscopy surgery has generally been performed with tracheal intubation and controlled ventilation, short- or intermediate-acting neuromuscular blockers should be administered based on surgical length.
      iv) General anesthesia has been performed without intubation safely and effectively with ProSeal laryngeal mask airway in nonobese patients.[6]
         (1) Careful attention is required for patients with high airway pressures (>30 cm H_2O) or where extreme head-down tilt will be applied.
      v) Ventilation: The use of pressure-controlled modalities affords higher instantaneous flow peaks, minimizing peak pressures, and have been shown to provide improved alveolar recruitment and oxygenation in laparoscopic surgery.[6] The addition of titrated levels of PEEP can be used to minimize alveolar derecruitment.
      vi) Emergence from general anesthesia
         (1) Reversal of neuromuscular blockade
            (a) Consider administering neuromuscular blocking reversal agents to patients who receive neuromuscular blocking drugs.
            (b) The association between drugs such as neostigmine and glycopyrrolate and postoperative nausea and vomiting (PONV) is controversial.
            (c) Even minor degrees of residual neuromuscular block can produce distressing symptoms and potentially increased risk of respiratory complications.
      vii) Regional anesthesia (spinal, epidural) can be used in laparoscopic procedures (diagnostic, infertility, and tubal ligation).[7] This is not widely done because the high thoracic sensory level required for patient comfort during pneumoperitoneum may result in respiratory impairment.


There is no clinical advantage to omitting nitrous oxide for laparoscopic surgery.[6]

5) **Postoperative recovery**
   a) **Nausea and vomiting**
      i) PONV is common after laparoscopic surgery, although its etiology is not clear.
      ii) Ondansetron or other 5-HT3 antagonists given at the end of surgery result in a significantly greater antiemetic effect compared with preinduction dosing.

iii) Prophylactic dexamethasone (4 mg) decreases the incidence for nausea and vomiting after laparoscopic cholecystectomy and hysterectomy relative to placebo and may decrease the severity of pain with no adverse effects noted from this single steroid dose.[8]
   (1) The antiemetic effect of dexamethasone begins ~2 hours after IV injection, so administration should be at the beginning of the case.
   (2) Single use of a low-dose dexamethasone is effective in preventing PONV, and its use can slightly increase blood glucose levels within 24 hours. This should be considered in diabetic patients.

READ MORE
Postoperative nausea and vomiting , Chapter 129

b) **Pain**
   i) Postoperative pain in laparoscopic surgery is less severe compared to an open procedure but still considerable.
   ii) The most effective pain relief can be obtained by combining opioids, local anesthetics, and NSAIDs into balanced analgesia.
   iii) This approach at least allows reduced opioid dose by the use of other modalities, thereby limiting side effects, decreasing postoperative pain and analgesics, and facilitating an earlier return to normal activities.
   iv) Transversus abdominis plane (TAP) block, local infiltration of the laparoscopy portals, and intraperitoneal local anesthesia can be used as regional techniques for pain relief.[7]

6) **Robotic systems**
   a) The da Vinci surgical system is currently the only commercially available system in the United States.
   b) This system has three components: a console, an optical three-dimensional vision tower, and a surgical cart.
   c) **Advantages of computer-assisted robotic surgery over standard laparoscopy include:**
      i) Improved operative field visibility with three-dimensional imaging systems with adjustable magnification up to 10-fold
      ii) Computer-assisted scaling improves control of fine movements and reduces the "fulcrum effect" that amplifies unwanted motions such as hand tremor.
      iii) More ergonomic, anatomic control of instruments that closely mimics the movement of the human wrist. This results in reduced fatigue from instrument manipulation.
      iv) There is evidence of shorter learning curves with robotic-assisted surgery compared to laparoscopic surgery.


Some surgical procedures are easier to perform and result in less blood loss with a robot than standard techniques.

   d) **Pitfalls/limitations include:**
      i) Several pieces of large, bulky equipment require large amounts of OR space. The new generation of robots are much easier to handle and unlikely to be a problem in the near future.
      ii) The robot itself is quite large and care must be taken in positioning robotic arms.
      iii) Impaired access to patient once robotic arms are positioned and engaged.
      iv) The increased direct cost of surgical equipment and maintenance is often prohibitive.
      v) Operating times are increased compared to traditional approaches across a range of procedures.


The operating team must be trained in rapid undocking of the robot in case of emergencies

7) **Indications**: Although the da Vinci robotic systems were initially designed for cardiothoracic surgeries, there are an increasing number of other surgical procedures utilizing robots. These include:

a) **General surgery:**
   i) Virtually all gastrointestinal procedures have been performed safely by surgeons using this technology.
   ii) Procedures most commonly performed include cholecystectomy, fundoplication, Heller myotomy, bariatric surgery, and colectomy.[9]
b) **Cardiac surgery:** Mitral valve surgery, atrial septal defect repair, internal mammary artery harvest, PDA ligation, coronary artery bypass grafting, atrial fibrillation surgery, and left ventricular pacemaker lead placement
c) **Thoracic surgery:** Esophageal procedures, resection of mediastinal masses, and thymectomy
d) **Urologic surgery:** Radical prostatectomy, radical cystectomy, radical and simple nephrectomy, live donor nephrectomy, pyeloplasty, and adrenalectomy
e) **Gynecologic surgery:** Sacral colpopexy, myomectomy, treatment of endometriosis, hysterectomy, salpingo-oophorectomy, ovarian cystectomy, and repair of vesicovaginal fistula
f) **Pediatric surgery:** Useful features include improved dexterity, motion scaling, tremor filtration, greater optical magnification, and the elimination of the fulcrum effect when compared to conventional laparoscopy. However, its technical requirements can make it cumbersome or not feasible for smaller patients.
g) **Orthopedic surgery:** Total hip and knee arthroplasty
h) **Ear, nose, and throat surgery:** Transoral robotic surgery (TORS) is a technique used to treat oral, throat, and skull base cancers using a minimally invasive robotic approach through the mouth and throat.

8) **Contraindications**
   a) Poor pulmonary function test results may be a contraindication to robotically assisted cardiac surgery because single-lung ventilation may be poorly tolerated.
   b) The prolonged Trendelenburg position required for robotically assisted radical prostatectomy is relatively contraindicated in patients with a history of stroke or cerebral aneurysm.[9]

9) **Anesthetic considerations: Patient safety during robot-assisted surgery requires advance planning and preparation.**
   a) **Patient positioning**: Robotic surgery with the da Vinci system does not allow for changes in patient position on the OR table once the robot has been docked. A well-secured endotracheal tube with adequate breathing circuit extensions is essential.
      i) Pelvic procedures such as prostatectomy are usually done in lithotomy and steep Trendelenburg position, while upper abdominal surgeries are best done in the supine position with reverse Trendelenburg position.
      ii) Intrathoracic procedures often require the lateral position with variations of Trendelenburg and reverse Trendelenburg depending on the surgical site.
      iii) Mediastinal surgeries often require lateral positioning with lateral table tilt.
      iv) Before positioning the robotic cart, pressure points must be carefully padded.
      v) Close attention should be directed to the robot and the patient's head and chest during placement and removal of the robot to avoid injury.
      vi) After robotic instruments are engaged, any patient movement can be disastrous.
   b) **Limited intraoperative access to patient**
      i) Preparation and open communication between the anesthesiologist and the surgical team are essential.

ii) Because of the proximity of the side cart to the patient's head, there is limited access to the patient's airway and neck, and the head must be protected against inadvertent collision with the robotic arms when moving.



Any patient movement after the robot is docked can be disastrous. The OR team should be trained for rapid removal of the robot in case of airway emergency or cardiac arrest.

c) **Patient preparation and monitoring**
   i) Bilateral peripheral IV access is valuable because the upper extremities may not be immediately available during surgery.
   ii) Neuromuscular blockade is very important in avoiding any patient movement while the robot is engaged.
   iii) Arterial and central venous catheterization, if necessary, should be placed prior to robotic docking.
   iv) Core temperature should be maintained with warm IV fluids and forced air warming.
   v) No differences in respiratory mechanics and hemodynamic disturbances have been demonstrated with the use of volume-controlled ventilation or pressure-controlled ventilation during robotic surgery. To minimize the adverse effects of pneumoperitoneum and steep Trendelenburg, a tidal volume of 6-8 mL/kg and a PEEP of 4-7 cm H_2O are recommended to prevent atelectasis.[10]
d) Special considerations for intrathoracic robot-assisted surgery
   i) All robotic procedures that demand entrance into the thoracic cavity require single-lung ventilation.
   ii) Most commonly, the left-sided double-lumen endotracheal tube is used for lung isolation.
   iii) CO_2 insufflation into the thoracic cavity to a pressure of 10-15 mm Hg maintains the lung away from the operative area.

READ MORE
Thoracic surgery and placement of double-lumen tube, Chapters 124 and 165

e) Special considerations for pediatric robot-assisted surgery
   i) Confirming proper endotracheal tube depth with fluoroscopy after patient positioning may help prevent an airway emergency.[11]
   ii) A left-sided precordial stethoscope monitors for inadvertent right main stem intubation.

10) **Potential complications:** The physiologic perturbations during robotic surgery are similar for both laparoscopic and thoracoscopic procedures.
   a) The presence of a pneumoperitoneum affects many organ systems. See previous discussion in the chapter.
   b) Increased risk for nerve injury caused by:
      i) Patient positioning required for optimal surgical conditions
      ii) Possible prolonged operative times as surgeons gain experience with robotic-assisted surgery
      iii) Ocular injuries range from corneal abrasions to serious ischemic optic neuropathy
   c) Close attention to fluid management is required when patients are placed in a steep Trendelenburg position. Liberal fluid therapy increases the risk of head and neck edema. There is also risk of retinal injuries and peripheral neuropathies.
   d) Anesthesia-related challenges in thoracic robotic surgery
      i) Prolonged one-lung ventilation
      ii) Hemodynamic instability due to obstruction of venous return and profound hypotension, associated with CO_2 insufflation of the hemithorax.[11]
      iii) CO_2 insufflation in the chest cavity will lead to an increase in peak airway pressure, particularly during one-lung ventilation.[12]



Pay careful attention to patient positioning prior to robot docking in order to minimize the potential for nerve and other injuries.

CHAPTER SUMMARY FOR ANESTHESIA FOR LAPAROSCOPIC SURGERY

Laparoscopy	Minimally invasive surgery with important physiologic effects.
Insertion of Veress Needle	Watch for inadvertent intravascular placement and CO_2 embolism.
Insertion of Trocars	Watch for inadvertent intravascular placement, especially if minilaparotomy is not performed.
Pneumoperitoneum	Can cause hypertension (↑ SVR due to ↑ CO_2), hypotension (↓ venous return), arrhythmias, ↓ SpO_2 (due to ↓ FRC, ↑ peak pressure).
Trendelenburg Positioning	Ensure patient strapped to bed, mainstem intubation, ↓ FRC.
Postoperative Concerns	PONV common and should be treated, decreased pain compared to similar procedures done with open approach.

CHAPTER SUMMARY FOR ROBOTIC SURGERY

Robotic Surgery	Several surgical advantages over standard laparoscopy. Large, bulky equipment presents anesthetic challenges.
Indications	Multiple surgical procedures are completed with the use of robots. Robot-assisted radical prostatectomy is currently the most common.
Contraindications	Poor pulmonary function may not allow for single-lung ventilation during thoracic surgery. History of stroke or aneurysm may prevent the use of prolonged Trendelenburg positioning.
Anesthetic Considerations	Patient safety requires advance planning, preparation, and open communication. All vascular access and positioning must be complete before robotic docking. Any patient movement after docking may be disastrous. The OR team must be trained for rapid removal of robot in case of airway emergency or cardiac arrest.
Potential Complications	Pneumoperitoneum affects multiple organ systems. Increased risk for nerve injury and edema exists.

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118 Anesthesia for Urologic Surgery[§]

Cosmin Guta

Urologic surgery encompasses a variety of procedures to treat prostatic masses or tumors, urinary tract conditions, kidney stones, or testicular tumors. Many chronic or malignant diseases can benefit from urologic resection or surgery. Providing anesthesia for these surgical procedures can pose unique challenges for the anesthesiologist, including potential for large blood loss, potential for fluid absorption, and patient positioning challenges.

1) **Overview**
   a) Urologic surgery integrates operations of the pelvis that include the colon and urogenital and gynecologic organs.
      i) Common reasons for urologic surgery include malignancy, organ dysfunction or obstruction, pain, and inflammatory diseases.
      ii) Common operations include nephrectomy, bladder surgery, prostate surgery, testicular surgery, and surgery involving the urethra and ureters.
   b) **Anesthesia for urologic surgery presents many challenges, including:**
      i) Potential for large blood loss during surgery in the highly vascular pelvic region
      ii) Potential for fluid absorption during cystoscopic surgeries
      iii) Patient positioning in lateral or flexed positions
2) **Anesthetic considerations for urologic procedures**
   a) **Radical prostatectomy**
      i) Indications
         (1) Prostate cancer is the most common type of cancer in men, affecting 10% of all men at some point in their life.
         (2) Involves resection of large prostatic masses or, more commonly, tumors.
         (3) The procedure can be performed by open laparotomy, but laparoscopic and robotic surgery are being used frequently in many centers. Current data show similar functional outcomes between open and laparoscopic/robotic prostatectomies.[1]
         (4) Radical retropubic prostatectomy is often associated with significant operative blood loss and longer recovery times compared with robotic prostatectomies, which have a lower rate of transfusion and shorter length of hospital stay.[1]
         (5) Laparoscopic/robotic prostatectomy with pelvic lymph node dissection requires a steep (>30 degrees) Trendelenburg position for surgical exposure and has the potential for increased carbon dioxide absorption from the retroperitoneum.



Radical retropubic prostatectomy is often associated with significant surgical blood loss.

      ii) Preoperative considerations
         (1) Patients presenting for prostatectomy are usually elderly males. Preoperative evaluation should focus on comorbid disease processes that are prevalent in this patient population.[2]
      iii) Positioning
         (1) Hyperextended supine position to facilitate exposure of the pelvis.

(2) The operating room table is tilted head-down to make the operative field horizontal.
(3) A steep Trendelenburg position is required for laparoscopic/robotic prostatectomies.

iv) Monitoring
(1) ASA standard monitors and temperature monitoring are required. An arterial line might be indicated based on patient's comorbidities.
(2) Central venous pressure monitoring might be required to assess the volume status.[2]

v) Access
(1) Consider large-bore IVs (significant blood loss may occur) and an arterial line ± central venous access (depending on comorbidities) as significant blood loss and large volume shifts occur.

vi) Anesthetic technique
(1) Regional, general, or a combined regional/general technique can adequately provide anesthesia for radical retropubic prostatectomy.
(2) General anesthesia with muscle relaxation is preferred for radical perineal prostatectomy because the patient is placed in the exaggerated lithotomy position, which interferes with respiratory function.

vii) Complications
(1) Include blood loss, hypothermia, anemia, coagulopathy, venous air embolism, and peroneal nerve injury due to positioning

b) **Transurethral resection of the prostate**

i) Indications
(1) Transurethral resection of the prostate (TURP) is the second most common surgical procedure in men over age 65.
(2) The most common indication for TURP is symptomatic benign prostatic hypertrophy. This patient population has a greater anesthetic risk because of a greater prevalence of coexisting cardiovascular and pulmonary problems.
(3) This procedure is performed using a modified cystoscope (resectoscope) with a wire loop connected to an electrocautery unit (monopolar, M-TURP or bipolar, B-TURP) or by using laser vaporization energy (L-TURP), for resection of tissue and coagulation of bleeding vessels.
(4) During the procedure, large amounts of irrigation solution are absorbed into the circulation and the periprostatic and retroperitoneal spaces.
(a) Normally, about 10-30 mL/min of irrigation fluid is absorbed. In clinical practice, it is almost impossible to accurately assess the volume absorbed.
(b) Absorption of irrigating fluid depends on
(i) Pressure, volume, and type of irrigating fluid
(ii) Intravesical pressure
(iii) Duration, amount, and quality of resection
(iv) Degree of bleeding
(c) Absorption of irrigating fluid will cause hypervolemia, dilutional hyponatremia, hemolysis, and toxicity of solutes in the irrigating fluid.
(d) The ideal irrigating fluid should be
(i) Isotonic
(ii) Nonelectrolytic
(iii) Transparent



Large amounts of irrigation solution are absorbed during TURP and can cause a syndrome of pulmonary edema, cerebral edema, and hyponatremia.



Nonmetabolized, nontoxic, and rapidly excreted.

e) Irrigation solutions used and their potential side effects during TURP include (see table)

Irrigation Solution	Potential Side Effects
Glycine	Hyperammonemia (coma) and possible visual disturbances (transient postoperative visual syndrome)
Distilled water	Hemolysis and hyponatremia/hypo-osmolality
Mannitol	Osmotic diuresis
Sorbitol	Hyperglycemia and possible lactic acidosis
Normal saline or lactate Ringer's	Fluid overload but does not cause hemolysis upon intravascular absorption (*used for B-TURP and L-TURP*)



The ideal irrigating fluid should be isotonic, nonelectrolytic, transparent, nonmetabolized, nontoxic, and rapidly excreted.

ii) Positioning
  (1) Lithotomy.
  (2) Functional residual capacity decreases, predisposing patients to atelectasis and hypoxia.
  (3) Rapid lowering of the legs at the end of the operation acutely decreases venous return and can result in severe hypotension.
  (4) Common peroneal nerve compression at fibular head can cause loss of dorsiflexion of the foot if pressure points padding is not adequate. Sciatic and femoral nerve injuries have been described in the literature.

iii) Monitoring
  (1) Standard ASA monitors, including temperature monitoring

iv) Anesthetic technique
  (1) The procedure can be performed safely under general or regional anesthesia and the choice of anesthetic should be based on patient comorbidities and preference. There is no evidence of a difference in mortality or morbidity between regional and general anesthesia.
  (2) Regional anesthesia might be preferred, since an awake patient can be helpful for an early diagnosis of:
    (a) TURP syndrome: neurological manifestations
    (b) Bladder perforation: abdominal and/or shoulder pain
  (3) Spinal anesthesia offers some advantages over general anesthesia for patients undergoing TURP.
    (a) Spinal anesthesia may reduce the risk of pulmonary edema and permits early detection of mental status changes because increased absorption of irrigating fluid during the procedure may cause fluid overload associated with hyponatremia and with cardiovascular and neurologic implications.
    (b) Adequate anesthesia with minimal hemodynamic effects can be achieved with a hyperbaric solution of local anesthetic with or without an opioid.
    (c) Regional anesthesia does not abolish the obturator reflex (external rotation and adduction of the thigh secondary to stimulation of the obturator nerve by electrocautery current through the lateral bladder wall). The reflex is *reliably* blocked only by muscle paralysis during general anesthesia or obturator nerve block during spinal anesthesia.



For a satisfactory anesthesia, a sensory block up to T10 is required to prevent the discomfort of bladder distention.



Spinal anesthesia may be preferable in TURP because it may reduce the risk of pulmonary edema and permits early detection of mental status changes associated with TURP syndrome. A sensory block up to T10 is required to prevent the discomfort of bladder distention.

v) Complications
   (1) Complications associated with TURP include absorption of irrigation fluid (proportional to the height of the irrigation container above surgical table and duration of the resection), excessive circulatory volume associated with hyponatremia and hypoosmolality, TURP syndrome (2%), hemorrhage (2.5%), bladder perforation (1.2%), hypothermia, septicemia (6%-7%), disseminated intravascular coagulation (**Tables 118-1 and 118-2**).

vi) TURP syndrome
   (1) Manifestations are primarily of circulatory fluid overload, water intoxication, and, occasionally, toxicity from the solute in the irrigation fluid.
   (2) Clinical signs and symptoms
      (a) Early clinical signs and symptoms of TURP syndrome include restlessness, headache, and tachypnea.
      (b) It may progress to respiratory distress, hypoxia, pulmonary edema, nausea, vomiting, confusion, and coma.
      (c) TURP syndrome consists of pulmonary edema, cerebral edema, and hyponatremia due to absorption of large amounts of hypotonic irrigation fluids.
         (i) The most important physiological derangement of CNS function is not hyponatremia but acute hypo-osmolality because the blood-brain barrier is essentially impermeable to sodium but freely permeable to water.
         (ii) Cerebral edema caused by acute hypo-osmolality can increase intracranial pressure with bradycardia, hypertension, and neurologic symptoms.
         (iii) When serum sodium level falls to <120 mEq/L, signs of cardiovascular depression can occur.
         (iv) Levels <115 mEq/L will cause bradycardia, widening of the QRS complex, ST-segment elevation, ventricular ectopic beats, and T-wave



The most important physiologic derangement of CNS function in TURP syndrome is acute hypo-osmolality.

TABLE 118-1 Signs and Symptoms Attributed to Transurethral Resection of the Prostate (TURP) Syndrome

Neurological manifestations	• Restlessness • Disorientation • Confusion • Depressed consciousness • Blurring of vision transient • Blindness (glycine neurotoxicity) • Convulsions
Cardiovascular manifestations	• Hypertension • Chest pain • Bradycardia • Dysrhythmia • ECG changes (wide QRS, ST changes) • In severe and late cases, hypotension, CHF, and pulmonary edema may occur
Pulmonary manifestations	• Respiratory distress • Hypoxemia • Pulmonary edema
Metabolic changes	• Hyponatremia • Hyperglycemia • Hyperammonemia • Hypo-osmolarity

TABLE 118-2 Other Potential Complications of TURP

Blood loss	• Blood loss is difficult to quantify, particularly in prolonged cases. • Visual estimation of hemorrhage may be difficult due to dilution of blood with irrigant solution. • Usual warning signs (tachycardia and hypotension) may be overshadowed by overhydration due to irrigant absorption.
Coagulopathy	• Disseminated intravascular coagulation (DIC) is responsible for severe postoperative bleeding after TURP in <1% of the cases. • Caused by release of a plasminogen activator from the prostate into the circulation during surgery. • Treatment may include administration of aminocaproic acid.
Bladder perforation	• The incidence of bladder perforation during TURP is about 1%. • Can be difficult to recognize in the presence of a high spinal block (may mask abdominal pain) or general anesthesia. • *Extraperitoneal perforation* presents with pain in the periumbilical, inguinal, or suprapubic regions. • *Intraperitoneal perforations* may present with generalized pain in the upper abdomen or referred pain from the diaphragm to the precordial region or the shoulder associated with abdominal rigidity, nausea, vomiting, and hypotension.[1]
Bacteremia and septicemia	• TURP facilitates the entry of bacteria into the bloodstream via open prostatic venous sinuses. • Bacteremia following TURP is usually asymptomatic and transient. • Septicemia may occur and is treated with antibiotics, and cardiovascular support is warranted.[1]
Hypothermia	• Elderly patients have a reduced thermoregulatory capacity. • Appropriate measures to reduce heat loss are warming blankets (Bair Hugger), heated irrigating solution, and warm intravenous fluids. • Associated with a significantly higher incidence of postoperative myocardial ischemia.

inversion. A serum sodium level <110 mEq/L can cause respiratory and cardiac arrest.[3]

(d) The risk of TURP syndrome can be reduced by limiting the time of surgery to <1 hour and by maintaining the irrigating fluid suspended no more than 30 cm above the operating room table during the resection.



The risk of TURP syndrome can be reduced by limiting surgical time <1 hour and suspending irrigation fluid no more than 30 cm above the OR table.

(3) Treatment

(a) Treatment of TURP syndrome depends on early recognition and should be based on the severity of symptoms. The syndrome has a significant mortality unless it is recognized and treated promptly.

(i) Signs will be detected earlier in the awake patient.

(ii) In the anaesthetized patient, the only clue may be tachycardia and hypertension.

(iii) Diagnosis can be confirmed by finding a low serum sodium. An acute fall to <120 mEq/L is always symptomatic.

(b) Surgery should be terminated as soon as possible and IV fluid should be stopped.

(i) Check arterial blood gas, serum sodium, and hemoglobin.

(ii) Support respiration with oxygen or intubation and ventilation if required.

(iii) Most patients can be managed with fluid restriction and a loop diuretic.
(iv) If pulmonary edema or hypotension develops, invasive hemodynamic monitoring is recommended as a guide for pharmacologic support and fluid management.
(v) Symptomatic hyponatremia resulting in seizures or coma should be treated with hypertonic saline (NaCl 3%) until symptoms resolve.
1. The rate of correction of the serum sodium should not be more than 12 mEq/L in the first 24 hours—rapid administration of hypertonic saline has been associated with an increased risk of central pontine myelinolysis (CPM, a.k.a osmotic demyelination syndrome).
2. CPM has been reported after both rapid and slow correction of serum sodium concentration in TURP patients.[3]
3. Endotracheal intubation is advisable to prevent aspiration until the patient's mental status normalizes.


Central pontine myelinolysis may occur if hyponatremia is corrected too rapidly.

c) **Cystoscopy**
i) The most commonly performed urological procedure
ii) Performed to diagnose and treat lesions of the lower (urethra, prostate, bladder) and upper (ureter, kidney) urinary tracts
iii) Positioning
(1) Lithotomy
iv) Monitors
(1) Standard ASA monitors
v) Access
(1) Generally one peripheral IV is all that is required.
vi) Anesthetic management
(1) Varies with the age and gender of the patient and the purpose of the procedure. Spinal anesthesia has been shown to be associated with a lower reoccurrence and a delayed time to reoccurrence of bladder tumors following TURBT.[4]
(2) Most cystoscopies are performed as an outpatient procedure, and the duration is usually short (15-20 minutes), so a general anesthetic is usually used.
(3) If a regional anesthetic (spinal or epidural) is used, a sensory level to T10 provides anesthesia for nearly all cystoscopic procedures.
(4) The obturator reflex that can occur during the procedure is reliably blocked only by muscle paralysis during general anesthesia or obturator block in addition to spinal anesthesia.
vii) Complications
(1) Complications are related to lithotomy position and bladder perforation (see TURP).

d) Cystectomy
i) Indications
(1) Radical cystectomy is recommended for invasive bladder tumors (usually transitional cell) and simple cystectomy for interstitial/hemorrhagic/radiation cystitis.
(2) A procedure to divert the urinary tract is also needed, usually a piece of small bowel brought out as a stoma (ileal conduit). It can also be a pouch constructed out of the bowel and reattached to the urethra (neobladder).
(3) Extensive lymph node dissection is typical in radical procedures.

ii) Preoperative evaluation
  (1) Smoking is a major risk factor for bladder cancer, so patients have high coincidence of CAD and COPD.
  (2) Patients with interstitial cystitis often have chronic pain, and opioid tolerance is common.
iii) Positioning
  (1) Lithotomy or supine position.
  (2) Good medial padding is needed in lithotomy, as injuries have been described with leg adduction as a result of obturator reflex from use of cautery near obturator nerve during pelvic node dissection.
  (3) Robot-assisted procedures are often done with exaggerated Trendelenburg (ie, 45 degrees or more).[2]
iv) Access
  (1) Consider large-bore IVs (significant blood loss may occur) and an arterial line ± central venous access (depending on comorbidities) as significant blood loss and large volume shifts occur.
v) Monitoring
  (1) ASA standard monitors and temperature monitoring.
  (2) Urine output may not be measurable when bladder is open.
vi) Anesthetic technique
  (1) General endotracheal anesthesia with a muscle relaxant is typically required.
  (2) A low-thoracic epidural (usually T10-T12) should be considered for intraop use and/or post-op pain control.
  (3) An upper body forced-air warming blanket should be used to prevent hypothermia.
vii) Complications
  (1) Significant blood loss and fluid shifts are common.
  (2) Colonic or ileal conduits, performed immediately following radical cystectomy, are associated with hyperchloremic metabolic acidosis.



Significant surgical blood loss can occur during radical cystectomy and large-bore IVs, and arterial pressure monitoring should be considered.

e) **Retroperitoneal lymph node dissection**
i) Indications
  (1) Advanced or recurrent testicular tumors
ii) Preoperative evaluation
  (1) Patients who have had chemotherapy may have had bleomycin. Pulmonary function should be evaluated. These patients may be at greater risk of pulmonary complications, including oxygen toxicity.
iii) Positioning
  (1) Usually supine position is used for this procedure.
iv) Monitoring
  (1) ASA standard monitors and temperature monitoring
v) Access
  (1) Consider large-bore IVs and an arterial line ± central venous access (depending on patient's comorbidities).
vi) Anesthetic technique
  (1) General anesthesia; also consider low-thoracic epidural for intraop use and/or postoperative analgesia.
  (2) If an epidural is used, it should cover the T6-T7 dermatomes, depending on the incision (sometimes thoracoabdominal is used).
vii) Complications
  (1) Plan for the possibility of significant blood loss.

(2) Depending on epidural use, sympathetic discharge might be noticed (eg, tachycardia) during dissection near sympathetic chain.

f) **Nephrectomy**

i) Indication

(1) Radical nephrectomy (includes ureter, often adrenal gland) is performed for neoplasms, usually renal cell carcinoma.

(2) Partial nephrectomy is sometimes done for smaller tumors, which spares nephron mass but often results in greater intraoperative blood loss.

(3) Simple nephrectomy is performed for chronic infection, large stones, or transplant donors.



Smoking is a risk factor for renal cell carcinoma and bladder cancer. Patients may have coincident risk of CAD and COPD.

ii) Preoperative evaluation

(1) Smoking is a risk factor for renal cell carcinoma, so patients have high coincidence of CAD and COPD.

(2) Preoperative anemia and renal insufficiency are common. Renal cell carcinoma patients require preoperative staging.

(3) If the tumor extends into the inferior vena cava (IVC) or right atrium, partially or fully occlude the IVC; poor venous return and hypotension may occur.

iii) Positioning

(1) Lateral decubitus or supine position

iv) Approach

(1) Retroperitoneal or transperitoneal

(2) Open or laparoscopic/robotic assisted

(3) If done laparoscopically with retroperitoneal approach, insufflation of CO_2 into retroperitoneum can track up above the diaphragm.

v) Monitoring and access

(1) ASA standard monitors and temperature monitoring.

(2) Consider large-bore IVs and an arterial line as significant blood loss and large volume shifts occur.

(3) Central venous access may be necessary for central pressure monitoring as well as for rapid transfusion when necessary.

(4) Intraoperative transesophageal echocardiogram can be used for monitoring patients at risk for having tumor fragments embolize to the pulmonary circulation.

vi) Anesthetic technique

(1) General anesthesia with muscle relaxation plus a thoracic epidural (usually T7-T8 level) for intraop use and/or post-op pain control should be used.

(2) In case the tumor extends intracardiac and occupies more than 40% of the right atrium, cardiopulmonary bypass may be used intraoperatively.



There is a risk of pneumothorax from diaphragmatic injury during nephrectomy.

vii) Complications

(1) There is a risk of pneumothorax because of diaphragm injury (near superior pole of the kidney).

(2) In renal cell carcinoma that involves IVC or right atrium, prepare for significant blood loss, impairment in venous return, possible need to clamp the IVC intraop, venous air embolism, pulmonary embolism of tumor or associated thrombus.[2]

g) **Nephroureterectomy**
   i) Indication
      (1) For upper tract transitional cell tumors (eg, ureter).
      (2) Similar considerations as nephrectomy; however, this surgery also involves open component to resect ureteral insertion into bladder with a margin of bladder tissue.
   ii) Monitoring
      (1) ASA standard monitors and temperature monitoring.
      (2) Urine output may not be measurable when the bladder is open.
   iii) Access
      (1) Consider large-bore IVs and an arterial line.
   iv) Anesthetic technique
      (1) General anesthesia with muscle relaxation plus a thoracic epidural (usually T7-T8 level) for intraop use and/or post-op pain control should be used.

h) **Orchiectomy**
   i) Indication
      (1) Testicular tumors most commonly diagnosed in men between the ages of 15 and 34.
   ii) Preoperative evaluation
      (1) Patients who have had neoadjuvant chemotherapy may have had bleomycin. Pulmonary function should be evaluated.
   iii) Positioning
      (1) Supine or lithotomy.
      (2) The incision is usually similar to that for inguinal hernia repair.
   iv) Anesthetic technique
      (1) Spinal/epidural (a T9 sensory level is required) or general anesthesia is an acceptable anesthetic technique.
   v) Complications
      (1) Patients treated with bleomycin may be at greater risk of pulmonary complications, including oxygen toxicity.[5]
      (2) Reflex bradycardia or nausea may occur from traction on the spermatic cord

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Anesthesia for laparoscopic surgery, Chapter 117

i) **Laparoscopic and robot-assisted surgery in urology**
   i) Laparoscopic surgery is used to perform traditionally open procedures like pelvic lymph node dissection, radical prostatectomy, nephrectomy (radical or partial), cystectomy, pyeloplasty, and stone extraction.
   ii) Laparoscopic surgery reduces perioperative morbidity compared with similar open approaches and allows for better visualization of the operative site.
   iii) Anesthetic considerations
      (1) Similar to those for laparoscopic procedures in general surgery or gynecology.
      (2) General anesthesia with endotracheal intubation and muscle relaxation is required to control ventilation in setting of pneumoperitoneum.
   iv) Monitoring and access
      (1) ASA standard monitors and temperature monitoring.
      (2) Consider large-bore IVs and an arterial line as significant blood loss can occur during some of these procedures.
      (3) Central venous access is dependent on patient-related comorbidities.
   v) Complications
      (1) Related to pneumoperitoneum required during the procedure.

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Laparoscopic and robotic surgery, Chapter 117.

(2) Subcutaneous emphysema may extend all the way up to the head and neck, compromising the upper airway in the most severe cases.[6]

(3) Because of the prolonged duration of these procedures, a significant amount of carbon dioxide can be absorbed, causing marked acidosis.



Subcutaneous emphysema can occur during laparoscopic surgery due to insufflation of CO_2 gas to create the pneumoperitoneum.

j) **Extracorporeal shock wave lithotripsy (ESWL)**

i) ESWL is the treatment of choice for disintegration of urinary stones in the kidney and upper part of the ureters.

ii) Shock wave–induced cardiac arrhythmias previously reported in 10%-14% of patients undergoing lithotripsy are now extremely rare.[7]

iii) Patients with a pacemaker or an internal cardiac defibrillator (ICD) are at risk for developing arrhythmias induced by shock waves during ESWL. These patients are considered acceptable candidates if the pacemaker is set to the asynchronous mode (if the patient's normal heart rate is pacemaker dependent) and the defibrillator is turn off during the procedure. Treatment should be gradually increased while observing pacemaker function.

iv) The internal components of pacemaker and ICD devices can be damaged during ESWL.

v) Pregnancy and untreated bleeding disorders are contraindications to ESWL.

vi) Newer lithotripters (dry SWL) use a smaller water-filled coupling device to provide an interface with the patient. This allows delivery of a highly focused sound beam that is causing less pain at the entry site.

vii) Immersion in a heated water bath may produce significant changes in the cardiovascular and respiratory system.

(1) Cardiovascular changes include hypotension (caused by vasodilation) and increase in central venous pressure and central venous volume.

(2) Respiratory changes include a reduction in functional residual capacity, tidal volume, and vital capacity.

viii) Monitoring

(1) Electrocardiograph pads should be attached securely with waterproof dressing prior to immersion.

(2) Synchronization of the shock waves to the R wave from the electrocardiogram (ECG) decreases the incidence of arrhythmias during ESWL. The shock waves are usually timed to be 20 ms after the R wave to correspond to the ventricular refractory period.

ix) Anesthetic technique

(1) General anesthesia and regional anesthesia (spinal, epidural, or flank infiltration with or without intercostal blocks). General anesthesia might be preferred because of its rapid onset and control of patient movement.

x) Complications

(1) Cardiac dysrhythmias, hematuria, ureteral colic, renal (subcapsular) hematoma, and severe pulmonary or intestinal damage

CHAPTER SUMMARY FOR ANESTHESIA FOR UROLOGIC SURGERY

Overview of Anesthetic Considerations

Radical Prostatectomy	Used for resection of large prostatic masses or tumors. Consider large-bore IV and arterial pressure monitoring in anticipation of blood loss for retropubic approach.
Transurethral Resection of the Prostate	Used for symptomatic benign prostatic hypertrophy. During the procedure, large amounts of irrigation solution are absorbed into the circulation and the periprostatic and the retroperitoneal spaces. Beware of TURP syndrome.
Cystoscopy	The most commonly performed urological procedure, used to diagnose and treat lesions of the lower (urethra, prostate, bladder) and upper (ureter, kidney) urinary tracts. Most cystoscopies are performed as an outpatient procedure.
Cystectomy	Radical cystectomy recommended for invasive bladder tumors (usually transitional cell). A procedure to divert the urinary tract is also needed. Consider large-bore IV and arterial pressure monitoring in anticipation of blood loss for retropubic approach.
Retroperitoneal Lymph Node Dissection	Used for advanced or recurrent testicular tumors. Prepare for significant blood loss.
Nephrectomy	Radical nephrectomy (includes ureter, often adrenal gland) is performed for neoplasms, usually renal cell carcinoma. Diaphragmatic injury during surgery may result in pneumothorax.
Nephroureterectomy	For upper tract transitional cell tumors (eg, ureter). Involves open component to resect ureteral insertion into bladder with a margin of bladder tissue.
Orchiectomy	Used for testicular tumors most commonly diagnosed in men between the ages of 15 and 34. Risk of reflex bradycardia from traction on the spermatic cord.
Laparoscopic Surgery in Urology	Used to perform traditionally open procedures like pelvic lymph node dissection radical prostatectomy, nephrectomy (radical or partial), cystectomy, pyeloplasty, and stone extraction. Reduces perioperative morbidity compared with similar open approaches; allows for better visualization of the operative site.
Extracorporeal Shock Wave Lithotripsy	For disintegration of urinary stones in the kidney and upper part of the ureters. Patients with a pacemaker or an internal cardiac defibrillator (ICD) are at risk for developing arrhythmias induced by shock waves during ESWL. Immersion in a heated water bath produces significant changes in the cardiovascular and respiratory systems.

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119 Anesthesia for Orthopedic Surgery

Danielle B. Ludwin

Orthopedic surgical procedures range from short outpatient procedures (eg, carpal tunnel) to long, complex cases with the potential for significant blood loss (eg, revision total hip arthroplasty). Orthopedic procedures are often painful, so it is important to provide both surgical anesthesia and postoperative analgesia. This chapter discusses anesthetic considerations for orthopedic surgical procedures, including potential complications and postoperative rehabilitation.

1) **Overview**
   a) **Orthopedics is a branch of surgery involving conditions affecting the musculoskeletal system.**
      i) A wide range of surgical procedures encompass orthopedic practice, from carpal tunnel release surgeries to complex spinal fusion and laminectomy surgeries.
   b) **The goals of anesthesia for orthopedic surgery include:**
      i) Surgical anesthesia. Anesthetic techniques must be adequate to provide optimal surgical operating conditions (see **Table 119-1**).
         (1) Appropriate depth of anesthesia
         (2) Adequate pain control
         (3) General anesthesia, neuraxial anesthesia, peripheral nerve blocks, or a combination can be used.
      ii) Postoperative analgesia. Many patients will have significant pain after orthopedic surgeries and a plan for postoperative analgesia should be carefully considered.
         (1) Peripheral nerve blockade with or without catheter
         (2) Opioids for breakthrough pain
         (3) NSAIDs and adjuvant analgesic medications
      iii) Optimize surgical exposure. Neuromuscular blockade, neuraxial anesthesia, or a peripheral nerve block is often required to facilitate optimal surgical exposure for orthopedic surgeries because they involve manipulation of the musculoskeletal system.
      iv) Prevent stretch/compression nerve injuries. Orthopedic surgeries can require the lateral, prone, or beach chair position; therefore, careful patient positioning is important to prevent injuries.



The goals of orthopedic surgery are to provide surgical anesthesia, postoperative analgesia, optimize surgical exposure, and prevent stretch or compression nerve injuries.

2) **Local anesthetics in regional anesthesia**
   a) Short- (chloroprocaine, lidocaine), intermediate- (mepivacaine), or long-acting (ropivacaine, bupivacaine, liposomal bupivacaine) local anesthetics can be used for peripheral nerve blockade or neuraxial blockade depending on the duration of the procedure and the anticipated postoperative pain.
   b) Most peripheral nerve blocks are done with 20-40 mL of local anesthetic.
   c) The ideal spinal anesthetic for ambulatory surgery is controversial.
      i) Lidocaine has an increased incidence of transient neurologic syndrome (TNS) especially when used for cases in the lithotomy position and therefore is not commonly used in clinical practice.

READ MORE
Local anesthetics, Chapter 25.
Overview of peripheral nerve blocks, Chapter 44.

TABLE 119-1 Benefits/Risks/Considerations for Regional Anesthesia in Orthopedic Surgery

Benefits	• ↓Risk of deep venous thrombosis; pulmonary embolism; pulmonary complications; surgical site infections, length of stay, ICU admission
Risks	• Nerve damage • Bleeding risk • Infection risk • Partial/failed block
Additional considerations	• Site for nerve blockade should be assessed preoperatively • A thorough history and physical exam ination should be documented • If there are preexisting neurologic deficits, risk-benefit ratio should be carefully considered • Postsurgical physical therapy goals are a factor in determining optimal analgesic regimens (eg, adductor canal blockade may be preferable to a femoral nerve blockade for knee surgery to preserve quadriceps function)

From Smith LM, Cozowicz C, Uda Y, et al. Neuraxial and combined/neuraxial/general anesthesia compared to general anesthesia for major truncal and lower limb surgery: a systematic review and meta-analysis. *Anesth Analg*. 2017;125:1931-1945; Neal JM, Barrington MJ, Brull R, et al. The second ASRA practice advisory on neurologic complications associated with regional anesthesia and pain medicine: executive summary. *Reg Anesth Pain Med*. 2015;40:401-430.

ii) Mepivacaine has approximately a 6% TNS incidence.[1]
iii) Chloroprocaine has been used off-label for spinal anesthesia.

d) Care should be taken for patients receiving a combination of anesthetic techniques in regard to local anesthetic toxicity.

READ MORE
Local anesthetic toxicity, see Chapter 149

3) **Upper extremity surgery**—Shoulder, elbow, hand surgery (see **Table 119-2**)

a) **Anesthetic considerations:**

i) Peripheral nerve block with or without general anesthesia is often used.
ii) Surgery to the proximal arm may limit access to the airway. If the patient has received a regional anesthetic and airway obstruction is a concern, securing the airway with an LMA or an ETT at the beginning of the case may be prudent.
iii) Placement of an IV line and NIBP on the nonoperative arm may interfere with infusions or fluid administration; therefore, consideration should be given to placement of a lower extremity IV.
iv) For patients with comorbidities such as morbid obesity and significant coronary artery disease, invasive BP monitoring may be beneficial.



For upper extremity surgery, if the patient has received a regional anesthetic and airway obstruction is a concern, securing the airway with an LMA or an ETT at the beginning of the case may be prudent.

b) **Shoulder surgery**

i) Positioning—"Beach chair" or lateral position is often used. The patient's neck should be maintained in a neutral position.
(1) Bezold-Jarisch reflex—Profound hypotension and bradycardia can present in the beach chair position after interscalene block or with general anesthesia.
(a) Pretreatment with β-adrenergic antagonists and vagolytics has been shown to prevent this reflex.[2]
(b) Treat early with small doses of epinephrine (10-100 μg).
(2) Maintenance of cerebral perfusion pressure (CPP) in the beach chair position is critical.
(a) CPP is the net perfusion pressure causing blood to flow to the brain (CPP = MAP − ICP or CVP, whichever is greater).

TABLE 119-2 Regional Techniques for Upper Extremity Surgery

Brachial Plexus Technique	Level of Blockade	Peripheral Nerves Blocked	Surgical Applications	Comments
Axillary	Peripheral nerve	Radial, ulnar, median; musculocutaneous can be blocked reliably with ultrasound guidance	Surgery to the forearm, hand, and elbow	Unsuitable for proximal humerus or shoulder surgery; patient must be able to abduct the arm to perform
Infraclavicular	Cords	Radial, ulnar, median, musculocutaneous, axillary	Surgery to the elbow, forearm, and hand	Catheter site (near coracoid process) easy to maintain; no risk of hemo- and pneumothorax
Supraclavicular	Distal trunk-proximal cord	Radial, ulnar, median, musculocutaneous, axillary	Surgery to the shoulder, midhumerus, elbow, forearm, and hand	Risk of pneumothorax, phrenic nerve paresis variable
Interscalene	Upper and middle trunks	Entire brachial plexus, although inferior trunk (ulnar nerve) not blocked in 15%-20% of cases	Surgery to the shoulder, proximal/middle humerus	Phrenic nerve paresis approaches 100% of patients for block duration; patients unable to tolerate a 25% reduction in pulmonary function need careful consideration of risks, benefits, and alternatives

Duration of block performed with long-acting local anesthetic (bupivacaine or ropivacaine) is 12-20 hours; intermediate-acting agents (lidocaine or mepivacaine) will resolve after 4-6 hours.
Modified with permission from Kirksey MA, Haskins SC, Soffin EM, et al. Anesthesia for orthopaedic surgery. In: Barash PG, Cullen BF, Stoelting RK, et al., eds. *Clinical Anesthesia*. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2017:1441-1457.

(b) There have been case reports of cerebral ischemia in this position as the brain is 20-30 cm higher than at the site of BP measurement[3]; thus, the blood pressure is lower in the brain than in the arm. CPP can be maintained with fluids and vasopressors as needed.

c) **Hand and elbow surgery**
   i) Typically, the supine position is used. Occasionally, the patient may be lateral or prone.
   ii) See **Table 119-2** for commonly placed peripheral nerve blocks.
   iii) Digital blocks—For distal extremity surgery, digital blocks that block the terminal branch of a nerve can be done by the surgeon or the anesthesiologist.
   iv) Bier block—This is an intravenous regional technique that provides complete anesthesia to an extremity distal to the tourniquet site. It is used in cases of <1.5-2 hours.[4]
      (1) An IV is inserted into the operative extremity. Another IV on the nonoperative side should also be placed for routine fluid and drug administration.
      (2) A proximal and distal pneumatic tourniquet with alarms is placed on the operative extremity.

READ MORE
Invasive arterial blood pressure monitoring, Chapter 12.
Anesthesia for intracranial procedures, Chapter 123.



Maintenance of cerebral perfusion pressure in the beach chair position is critical.

(3) The operative extremity is raised for at least 1 minute to allow for passive exsanguination.
(4) An Esmarch bandage is then wrapped from the distal part of the extremity until the tourniquet site to further exsanguinate the extremity.
(5) The proximal tourniquet is then inflated typically to 100 mm Hg greater than the systolic blood pressure.
(6) Once the tourniquet is adequately inflated, the Esmarch is released.
(7) Local anesthetic, typically lidocaine, is injected into the IV of the operative extremity.
(8) The local anesthetic will diffuse from the bloodstream to anesthetize the surrounding nerves.
(9) If the patient begins to complain of tourniquet pain, inflate the distal tourniquet and then deflate the proximal tourniquet.
(10) The tourniquets cannot be deflated until at least 30 minutes after injection of the local anesthetic for risk of local anesthetic toxicity.
(11) A two-stage deflation of the cuff can be done by deflating for 10 seconds and then reinflating for 1 minute to slow the washout of the local anesthetic from the extremity.



Maintenance of cerebral perfusion pressure in the beach chair position is critical to avoid cerebral ischemia and risk of stroke.



Bupivacaine is contraindicated in a Bier block due to its cardiotoxic properties if it were inadvertently released systemically.

4) **Lower extremity surgery—hip, knee, ankle, and foot surgery (Table 119-3)**
a) **Hip surgery**

TABLE 119-3 Lumbosacral Techniques for Major Lower Extremity Surgery

Peripheral Technique	Area of Blockade	Duration of Blockade	Perioperative Outcomes
Lumbar plexus femoral	Femoral, partial lateral femoral cutaneous, and obturator	12-18 h	Improved analgesia and joint range of motion, decreased hospital stay compared with PCA; fewer technical problems, less urinary retention, and hypotension than epidural analgesia (TKA)
Fascia iliaca	Femoral, partial lateral femoral cutaneous, obturator, and sciatic (S1)		Improved analgesia and joint range of motion compared with PCA (TKA)
Psoas compartment	Complete lumbar plexus; occasional spread to sacral plexus or neuraxis		Reduced morphine consumption, pain at rest compared with PCA (TKA, THA); reduced blood loss (THA); analgesia equivalent to continuous femoral block (TKA)
Sciatic	Posterior thigh and leg (except saphenous area)	18-30 h	Supplemental sciatic required (TKA); proximal approaches allow block of posterior femoral cutaneous nerve (TKA)

Duration of block performed with long-acting local anesthetic (bupivacaine or ropivacaine) and intermediate-acting agents (lidocaine or mepivacaine) will resolve after 4-6 hours.
Outcomes most marked in patients who receive a continuous lumbar plexus catheter with infusion of 0.1%- 0.2% bupivacaine or ropivacaine at 6-12 mL/h for 48-72 hours.
PCA, patient-controlled analgesia; TKA, total knee arthroplasty; THA, total hip arthroplasty.
Reproduced with permission from Horlocker TT. Anesthesia and pain management. Revision Hip and Knee Arthroplasty. Edited by Berry DJ, Trousdale RT, Dennis D, et al. Philadelphia, PA: Lippincott Williams & Wilkins; 2008.

i) Hip fracture
  (1) Preoperative considerations
    (a) Consider the etiology of the fracture/fall and associated risks and comorbidities, for example, mechanical fall vs cardiac or neurologic cause that may require further workup (ie, carotid duplex ultrasound, echocardiogram).
    (b) Delaying surgery for cardiac evaluation is an independent risk factor of postoperative complication, irrespective of the patient's medical status.[5]
    (c) Patients are often elderly.
      (i) They may have extensive comorbidities such as severe coronary artery disease, pulmonary disease, and renal disease.
      (ii) They can be hypovolemic from prolonged NPO status or diuretic use that may require aggressive fluid resuscitation and slow titration of anesthetics.
      (iii) Fluid administration should be done cautiously in patients with impaired left ventricular function.
  (2) Intraoperative considerations/anesthetic techniques
    (a) Supine or lateral position
    (b) Neuraxial anesthesia, lumbar plexus block, or general anesthesia
      (i) Neuraxial anesthesia is typically preferred in patients with concomitant cardiopulmonary disease (see **Table 119-1**).
      (ii) Compared to general anesthesia, neuraxial anesthesia decreases the odds of pulmonary complications, surgical site infections, thromboembolic events, and ICU admissions.[6]
      (iii) General anesthesia is often preferable for patients unable to cooperate for neuraxial anesthesia and anticoagulated patients who require emergent surgery.
    (c) A preoperative femoral nerve or a fascia iliaca compartment block can be helpful in decreasing perioperative opioid requirements as well as for positioning for a neuraxial anesthetic.[7]
    (d) Invasive blood pressure monitoring will depend on patient comorbidities such as extensive coronary artery disease or need for frequent blood sampling.
  (3) Postoperative considerations
    (a) Patients are at high risk for thromboembolic complications.
    (b) Patients often receive compression stocking and thromboprophylaxis medications (eg, LMWH, Coumadin).
ii) Total hip arthroplasty
  (1) Intraoperative considerations
    (a) Commonly performed in the lateral or supine position depending on surgical approach.
    (b) Anesthetic options include neuraxial or general anesthesia with/without a lumbar plexus block for postoperative analgesia.
    (c) Deliberate hypotension may be requested by the surgeon to decrease blood loss and improve surgical exposure.



Do not use epinephrine in a Bier block because of risk of IV injection; it is contraindicated in distal extremities due to risk of vasoconstriction causing nerve ischemia.



Patients undergoing lower extremity surgery often have functional limitations secondary to pain that can make assessing their exercise tolerance difficult and may require further cardiac workup prior to surgical interventions.



Hip fracture patients are at high risk for perioperative thromboembolic events.



Enhanced recovery after surgery (ERAS) protocols for hip fracture patients include preoperative regional analgesia and multimodal medications, a standard intraoperative anesthesia pathway, PONV prophylaxis, glycemic control, and a postoperative multimodal analgesia approach.[8]

(i) Deliberate hypotension is a technique using neuraxial anesthesia, general anesthesia, or IV agents (antihypertensives such as labetalol, nicardipine, or IV sedatives like propofol) to intentionally decrease BP to an MAP of 50-65 mm Hg.

(ii) The acceptable decrease in BP is controversial due to concerns of hypoperfusion and ischemia to vital organs. Benefits and risks must be weighed for each patient, and patients with chronic hypertension may be at higher risk for ischemia.[9]

(iii) Tranexamic acid is routinely given for hip arthroplasty cases. It is an antifibrinolytic drug that inhibits fibrinolysis and decreases blood loss and need for perioperative transfusions.[10]



The acceptable decrease in BP using deliberate hypotension is controversial due to concerns of hypoperfusion and ischemia to vital organs.



ERAS protocols for hip arthroplasty patients include decreasing fasting duration and carbohydrate loading, multimodal medications, a standard intraoperative anesthesia pathway, goal-directed fluid therapy, normothermia, tranexamic acid, glycemic control, and a postoperative multimodal analgesia approach.[11]

b) **Knee surgery**

i) Total knee arthroplasty

(1) Intraoperative considerations

(a) Supine position

(b) Anesthetic options: Neuraxial anesthesia, femoral or saphenous and sciatic nerve block, general anesthesia, or a combination of the above

(2) Postoperative analgesia

(a) Epidural, spinal opioids, femoral block/catheter, sciatic block/catheter

ii) Knee arthroscopy/ACL reconstruction

(1) Intraoperative considerations

(a) Anesthetic options: Spinal, femoral, or saphenous and sciatic nerve block, general anesthesia, local and MAC

(2) Postoperative analgesia

TABLE 119-4 Anesthetic Techniques for Common Foot and Ankle Operations

	Surgical Procedure	Regional Technique	Comments
Forefoot	Hallux valgus	Metatarsal, ankle, popliteal blockade	Sural nerve block not necessary for surgery
	Amputations	Ankle, popliteal blockade	Popliteal blockade is the technique of choice in the presence of infection or swelling
Midfoot	Transmetatarsal amputations	Popliteal, ankle blockade	
Hindfoot	Ankle arthroscopy	Spinal, epidural, or general anesthesia	Operation typically requires good muscle relaxation for manipulation; thigh tourniquet
	Achilles tendon repair	Spinal, epidural, or popliteal blockade	Spinal or epidural anesthesia whenever thigh tourniquet is required
	Triple arthrodesis	Spinal or epidural	Neuraxial technique preferred for bone graft harvesting; popliteal blockade for postoperative analgesia

Femoral or saphenous block required if the incision extends to the medial aspect of the foot or ankle.
Reproduced from Horlocker TT, Wedel DJ. Anesthesia for orthopaedic surgery. In: Barash PG, Cullen BF, Stoelting RK, et al., eds. *Clinical Anesthesia*. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009:1375-1392, with permission.

(a) Spinal opioids, femoral block/catheter, saphenous block/catheter, intra-articular opioids, local anesthetic infiltration

c) **Foot and ankle surgery (Table 119-4)**
   i) Intraoperative considerations
      (1) Anesthetic options: Neuraxial anesthesia vs general anesthesia
   ii) Postoperative analgesia
      (1) Femoral with/without saphenous nerve block, popliteal nerve block, or ankle block

5) **Complications**
   a) **Fat embolus syndrome**
      i) Definition: The triad of pulmonary distress, mental status changes, and petechial rash 24-48 hours after a pelvic or a long-bone fracture.
      ii) Signs and symptoms fall into major and minor criteria and are summarized in **Table 119-5**.
      iii) Treatment is supportive. Fluids and vasopressors should be used as needed to maintain hemodynamics.
   b) **Bone cement implantation syndrome**
      i) Definition: This syndrome is characterized by hypoxia, hypotension, and/or unexpected loss of consciousness occurring around the time of cementation, prosthesis insertion, reduction of the joint or, occasionally, during limb tourniquet deflation in a patient who has received cement.
      ii) Bone cement is used for arthroplasty (joint replacement surgery).
      iii) This can cause sudden hypotension, hypoxemia.
      iv) Treatment includes hydration, vasopressors, 100% FiO_2 (stop N_2O before methyl methacrylate is inserted).[14]
   c) **Thromboembolic events**—Orthopedic patients are at high risk of thromboembolic events such as deep vein thrombosis and pulmonary embolism.



ERAS protocols for knee arthroplasty patients include decreasing fasting duration and carbohydrate loading, multimodal medications, a standard intraoperative anesthesia pathway, goal-directed fluid therapy, normothermia, tranexamic acid, glycemic control, and a postoperative multimodal analgesia approach.[12]



Fat embolus syndrome is characterized by pulmonary distress, mental status changes, and petechial rash 24-48 hours after a pelvic or a long-bone fracture.

TABLE 119-5 Signs and Symptoms of Fat Embolus Syndrome

Major Criteria	**Hypoxemia**
	CNS depression
	Pulmonary edema
	Subconjunctival/axillary petechiae
Minor Criteria	Tachycardia
	Hypothermia
	Retinal fat emboli
	Urinary fat globules
	Anemia
	Thrombocytopenia
	Increased erythrocyte sedimentation rate
	Sputum fat globules

From Kirksey MA, Haskins SC, Soffin EM, et al. Anesthesia for orthopaedic surgery. In: Barash PG, Cullen BF, Stoelting RK, et al., eds. *Clinical Anesthesia*. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2018:1441-1457. Originally appearing in: Hadzic A, Vloka JD: Anesthesia for ankle and foot surgery. *Techniques in Regional Anesthesia and Pain Management* 1999;3(2):113. Ref.[13]

i) DVT prophylaxis should be initiated in every patient. Preventing DVT and thromboembolic events is balanced against the risk of causing major bleeding from anticoagulation.
   (1) Prophylaxis can range from encouraging ambulation, to compression stockings to pharmacological thromboprophylaxis.
ii) The decision to perform neuraxial anesthesia in patients on anticoagulants should be based on the risks and benefits for each individual patient.

d) Regional anesthesia techniques have been shown to decrease the risk of thromboembolic events.[6]

e) **Tourniquets** are often used in distal extremity surgery.
   i) A tourniquet is a pressure cuff that is inflated on the operative limb, proximal to the surgical site. Inflation of a tourniquet prevents blood flow to the limb and enables the surgeons to work in a bloodless field.
   ii) Prolonged tourniquet times typically are defined as >2 hours for the lower extremity and 90 minutes for the upper extremity and may result in permanent injury to muscles, vessels, and nerves.
   iii) Tourniquet pain is believed to involve type C pain fibers.[15]
   iv) Tourniquet release leads to a transient metabolic acidosis often with an increase in $ETCO_2$, hypotension, and tachycardia.
   v) Tourniquet pain is best treated with release of the tourniquet. If it is not surgically possible to release the tourniquet, the anesthetic can be deepened.

f) **Peripheral nerve injury** can occur due to a peripheral nerve block, the surgical procedure, or be a complication from improper patient positioning.[16]

g) **Comorbid conditions**
   i) Patients with rheumatoid arthritis may have associated musculoskeletal, pulmonary, and cardiac complications in addition to cricoarytenoid arthritis and cervical spine instability requiring extra precautions.

6) **Postoperative considerations**
   a) Early mobilization is advantageous to improve rehabilitation.
   b) Lower extremity peripheral nerve blockade can allow patients to participate more fully in physical therapy.
   c) This benefit must be weighed against the possibility of fall risk in a patient with a dense block.



Bone cement implantation syndrome can present with the sudden onset of hypotension and hypoxemia intraoperatively. Treatment is supportive.

READ MORE
Deep vein thrombosis and pulmonary embolism, Chapter 47.



Tourniquet release leads to a transient metabolic acidosis often with an increase in $ETCO_2$, hypotension, and tachycardia.



The ideal postoperative regional technique would provide prolonged sensory blockade with intact motor function.

CHAPTER SUMMARY FOR ANESTHESIA FOR ORTHOPEDIC SURGERY

Definition	Orthopedics is a branch of surgery involving conditions affecting the musculoskeletal system.
	A wide range of surgical procedures encompass orthopedic practice, from carpal tunnel release surgeries to complex spinal fusion and laminectomy surgeries.
Preoperative Considerations	In patients with preexisting neurologic deficits, the risk-benefit ratio of peripheral or central neuraxial blockade must be considered.
Intraoperative Considerations	Since orthopedic procedures are frequently painful, orthopedic anesthesia should provide surgical anesthesia and postoperative analgesia. Regional anesthesia and multimodal analgesia are often used to attain these goals.
	Patient positioning should optimize surgical exposure and prevent stretch/compression nerve injuries.
Postoperative Considerations	Orthopedic patients are at high risk for thromboembolic events. DVT prophylaxis should be initiated in every patient.

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120 Anesthesia for Eye Surgery

Megha Karkera Kanjia • Brian M. Davidson

Ophthalmic surgery is common and presents unique challenges for the anesthesiologist. The ophthalmologic patient population is unique as these patients are often geriatric and inevitably have more cardiopulmonary comorbidities. A second group of patients are those who come for emergency procedures after trauma and require minimizing increases in intraocular pressure (IOP). Avoiding anesthesia-related increases in IOP, management of systemic side effects of ophthalmic medications, and mitigating the oculocardiac reflex (OCR) are important anesthesia concepts for eye surgery. Patients experience improved surgical outcomes when the anesthesiologist recognizes and properly manages these unique challenges.[1]

1) **Ocular physiology**
   a) Intraocular pressure (IOP) is the pressure within the rigid globe of the eye. Normal pressure is 10-20 mm Hg.
   b) **Increases in IOP**
      i) Increased IOP may result from increases in arterial BP, central or local venous pressure, and local external pressure increases on the eye.[1]
   c) **Anesthesia events/procedures that may increase IOP**
      i) Direct laryngoscopy and intubation
      ii) High ventilation pressures—both endotracheal and mask
      iii) Trendelenburg position (**Table 120-1**)



Increased IOP may result in extrusion of vitreous humor in the situations such as an open globe during a surgical procedure or trauma.

2) **Anticholinergic medications**
   a) Topical anticholinergics cause pupillary dilation (mydriasis) and may precipitate closed-angle glaucoma.
   b) Systemic atropine and glycopyrrolate are not associated with ↑IOP.[1]
3) **Oculocardiac reflex (OCR)**
   a) Results from traction on the eye or extraocular muscles
   b) **Signs and symptoms**
      i) Bradycardia, junctional rhythm, ventricular ectopy, or asystole
      ii) Awake patients may experience nausea or somnolence.
   c) OCR involves the ophthalmic trigeminal (V_1) afferent and vagal (X) efferent pathway.[1]
   d) More common in pediatric patients undergoing strabismus surgery.
   e) **Prevention and treatment (Table 120-2)**
      i) Consider asking surgeon to temporarily remove the stimulus.
      ii) Administer anticholinergic drugs such as atropine (10 µg/kg) or glycopyrrolate (4 µg/kg).
         (1) Use with caution in patients with coronary artery disease.
         (2) Care should also be taken when using atropine in elderly patients due to CNS effects.
         (3) The rapid onset of atropine may make it the medication of choice in dire clinical situations.[1]



The OCR pathway may be easily remembered as the "five and dime" reflex because it involves cranial nerves (CNs) V and X.

TABLE 120-1 Anesthetics Impacting IOP

Anesthetic	Effect on IOP	Mechanism
Volatile agents **Opiates** **Benzodiazepines**	↓ IOP (dose related)	↓Systemic vascular pressures ↓Muscle relaxation (extraocular) Pupillary constriction (↑aqueous outflow)
Ketamine	↑ IOP	Sympathomimetic effects ↑ BP
Succinylcholine	↑ IOP	Depolarization of extraocular muscles • May last up to 10 min • Relative contraindication for open-globe trauma or known elevated IOP • Risk vs benefit assessment for risk of aspiration and hypoxia if rapid sequence induction is anticipated
Nondepolarizing neuromuscular blocking (NMB) agents	No increase in IOP	

4) **Systemic effects of ophthalmic medications**
   a) Topical medications
      i) Used frequently and absorbed into the body through blood vessels in the eye
      ii) Rate of absorption is slower than IV but faster than subcutaneous route of administration.
         (1) Echothiophate: Irreversible cholinesterase inhibitor for treatment of glaucoma
            (a) Side effects may include blurred vision, headache, eye erythema, and local or systemic allergic reaction.
         (2) Epinephrine: Topical drops used to reduce bleeding in topical ophthalmic procedures
            (a) May cause tachycardia, hypertension, and dysrhythmias in patients with and without a previous cardiac history
         (3) Timolol: β-Adrenergic antagonist used to lower IOP by reducing aqueous humor production
            (a) Rare reports of hypotension, bradycardia, and bronchospasm



Nitrous oxide administration during retinal surgery can cause **gas bubble expansion and ↑IOP** secondary to the agent's increased blood solubility.

5) **Intraocular gas expansion**
   a) Gas bubble may be placed by ophthalmologist into posterior chamber to aid repair of a detached retina.
      i) Avoid nitrous oxide for the entire case or within 10 minutes of any gas bubble insertion.
   b) Sulfur hexafluoride (SF6) may be used for the bubble owing to its lower solubility and prolonged therapeutic effect.[1]

TABLE 120-2 Management of the Oculocardiac Reflex (OCR)

Immediate management of cardiac symptoms resulting from OCR
1. **Cease** surgical traction or stimulation.
2. **Evaluate** vital signs and depth of anesthesia.
3. **Give atropine** 10 μg/kg IV if patient is unstable or reflex persists.
4. Recurring reflex symptoms may be managed with further anticholinergic doses or local anesthesia injection into the extraocular muscles by surgeon.

i) SF6 bubble will increase in size naturally and stay in place up to 10 days.

6) **Anesthesia for ophthalmologic surgical procedures**

a) General, regional, and topical anesthesia approaches are available for ophthalmic surgery patients.

i) Preoperative considerations

(1) Patients undergoing eye procedures frequently include an older population with significant comorbidities, including cardiac, respiratory, endocrine, and orthopedic pathology.

(2) Appropriate preoperative testing and evaluation should occur in these patients even though the inherent risk of most ophthalmic procedures is low.

(3) The patients' ability/preparedness to cooperate and remain still in the supine position must be confirmed before sedation is considered for the anesthetic plan.



When considering sedation for eye surgery, carefully assess the patient's ability to lie still while supine.

ii) Intraoperative considerations

(1) Airway access may be limited as the patient's head is usually 90-180 degrees from the anesthesiologist.

(2) Standard ASA monitoring requirements apply. Consider special monitors based on the patient's medical history and condition.

iii) General anesthesia

(1) Indicated for more invasive procedures and uncooperative patients.

(2) Choice of induction technique is based on patient's general medical condition.

(a) Trauma patients may require rapid sequence induction (RSI) without the use of succinylcholine.[2]

(b) Succinylcholine avoidance with open-globe injuries has been suggested as it may increase IOP and result in extravasation of ocular contents. Although clinical evidence supporting such avoidance is very limited, succinylcholine has been used successfully in open-globe injuries in patients requiring RSI.[2]

(c) Hypoxia and hypercarbia also greatly increase IOP, making a failed airway an equally problematic situation.

(d) The potential for rapid reversal with sugammadex allows consideration for an RSI dose nondepolarizing NMB in this situation.



Oral Ring, Adair and Elwyn (RAE) tubes are useful for eye cases requiring general anesthesia by providing greater surgical field access and less likelihood of tube obstruction/kinking.

(3) Patients with traumatic rupture (an open globe) require special care.

(a) IOP must be controlled during induction, maintenance, and extubation to prevent extrusion of vitreous content.

(b) Deep anesthesia should be maintained to prevent increases in IOP.

(c) Paralysis to prevent movement should be strongly considered.

(d) Hemodynamic control is essential.

(e) Smooth extubation plan is necessary to avoid Valsalva from coughing.

(i) Consider extubating the patient under deep anesthesia (contraindicated with difficult airway or full stomach).

(ii) Lidocaine 1.5 mg/kg 2 minutes prior to extubation may reduce laryngeal responsiveness and facilitate extubation.

(iii) Remifentanil (0.025-0.1 µg/kg/min) infusion may also be used to provide a smooth emergence with reduced coughing.

(iv) Dexmedetomidine (0.75 µg/kg) may be another adjuvant for smoothing emergence.[3]



PONV after eye surgery is common and can be dangerous due to potential increases in IOP; aggressive antiemetic and fluid resuscitation is prudent.

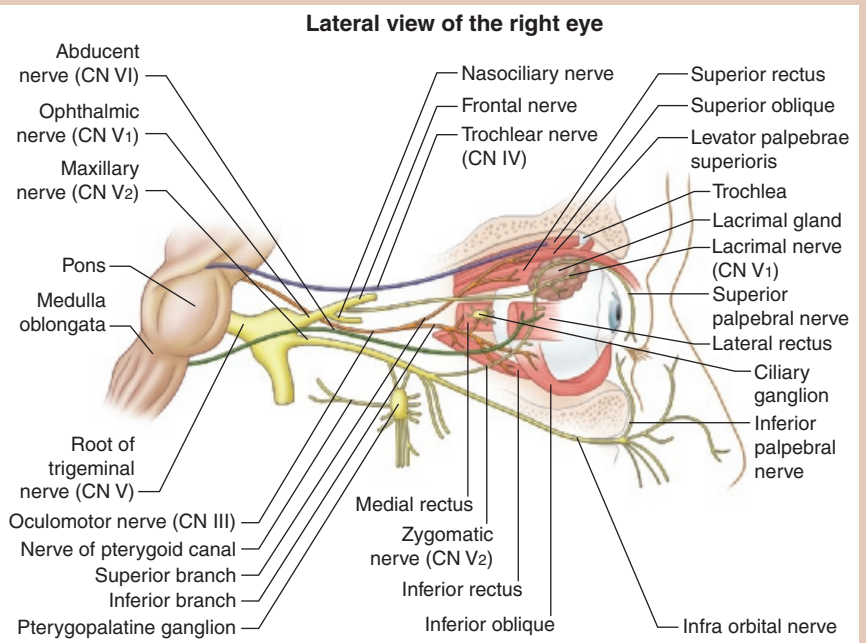
7) Intraoperative anesthetic implications in patients with glaucoma
   a) Topical mydriatic drugs (atropine, scopolamine) are contraindicated; however, systemic use for premedication and reversal with neostigmine is considered to be safe.
   b) Acute angle-closure glaucoma is typically spontaneous but can occur after spinal and general anesthesia.
   c) Acute glaucoma is a true emergency; ophthalmology consult should be obtained to decrease IOP quickly.[1]
      i) Regional and topical anesthesia (see below for techniques)
         (1) Most common technique for nontraumatic eye surgeries
         (2) Sedation must be kept to a minimum for ophthalmic surgery because patient cooperation and stillness are necessary for most of the intraoperative period.[1]
            (a) Short periods of heavier sedation are appropriate for placement of regional blocks.[4]
   d) **Postoperative considerations**
      i) Postoperative nausea and vomiting (PONV)
         (1) Valsalva with vomiting can ↑IOP.
         (2) Because PONV is commonplace after strabismus surgery, aggressive antiemetic medication should be administered.
      ii) Adequate pain control
         (1) Important because increased pain can lead to ↑ BP and ↑IOP.



PONV after eye surgery is common and can be dangerous due to potential increases in IOP.

8) **Regional and local techniques for eye surgery (Fig. 120-1)**
   a) **Retrobulbar block**
      i) Local anesthetic is injected behind the operative eye via the lateral third of the lower eyelid.

FIGURE 120-1 Eye Anatomy and Nerve Innervations



ii) A 3.5-cm, 25-gauge needle is advanced along the floor of the orbit toward the cone formed by the convergence of the extraocular muscles.
iii) 2.5 mL of local anesthetic (lidocaine or bupivacaine common) *without* epinephrine is injected after negative aspiration to avoid intravascular or intracranial injection.
iv) A successful block provides anesthesia, akinesia, and blockage of the OCR.
v) Hyaluronidase is added to improve block spread by increasing connective tissue permeability of local anesthetic.
vi) Complications
(1) Retrobulbar hemorrhage, globe trauma, optic nerve damage, and systemic/CNS injection of local anesthetics
(2) Systemic/CNS injection of local anesthetic may induce seizure, obtundation, apnea, or cardiovascular collapse due to the anatomic continuity between the optic nerve and the CNS.[1]
(a) Prompt and aggressive supportive care is necessary for the duration of the local anesthetic



Inadvertent CNS injection during retrobulbar block may cause seizures, apnea, and cardiovascular collapse.

b) **Peribulbar block (sub-Tenon block)**
i) Less invasive and more common than retrobulbar block
ii) Injection of 0.5 mL of local anesthetic into superior quadrant of subconjunctiva along the sub-Tenon space
iii) Not appropriate for anterior chamber surgery
iv) Complications include localized bleeding, infection, and eyelid droop.

c) **Topical anesthesia**
i) Anesthetic eye drops, such as oxybuprocaine and tetracaine, are applied preoperatively at 5-minute intervals for five applications.
ii) Anesthetic gel is later applied with a swab to upper and lower conjunctival sacs.
iii) Common for cataract and other minor anterior chamber surgery
iv) Complications include corneal abrasion and toxic keratopathy.

d) **Facial nerve block**
i) It prevents eyelid squinting during surgery and allows placement of lid speculums.
ii) Multiple techniques are utilized depending on the location and requirements of the surgical procedure.[5]
(1) Examples include lacrimal, zygomatic, supraorbital, supratrochlear, infratrochlear, and infraorbital nerve blocks.
iii) Complications include local hemorrhage and block failure.

CHAPTER SUMMARY FOR EYE SURGERY

Management Considerations	Control of IOP, Eye medications, OCR, Patient comorbidities
Effects of Anesthesia on IOP	↑ = intubation, mask ventilation, ketamine, succinylcholine, N_2O with gas bubble for retinal surgery ↓ = volatiles/benzos/opiates ↔ = Nondepolarizing NMBAs
OCR	Caused by traction on eye muscles Mediated by cranial nerves V and X Symptoms: ↓HR, bradyarrhythmias, asystole, nausea Treatment: Cease, Evaluate, Atropine!
Operative Considerations	Sedation, regional, vs general Prolonged supine position requires patient cooperation/stillness Minimal airway access, RSI has a risk of increasing IOP
Regional Anesthesia	Local/topical, peribulbar, retrobulbar, facial blocks
Open Globe	General: RSI with succinylcholine may ↑IOP, avoid Valsalva, smooth emergence

IOP, intraocular pressure; RSI, rapid sequence induction; OCR, oculocardiac reflex; NMBAs, nondepolarizing neuromuscular blocking agents; HR, heart rate.

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121 Spine Surgery

Scott Vogel • Barbara J. Wilkey

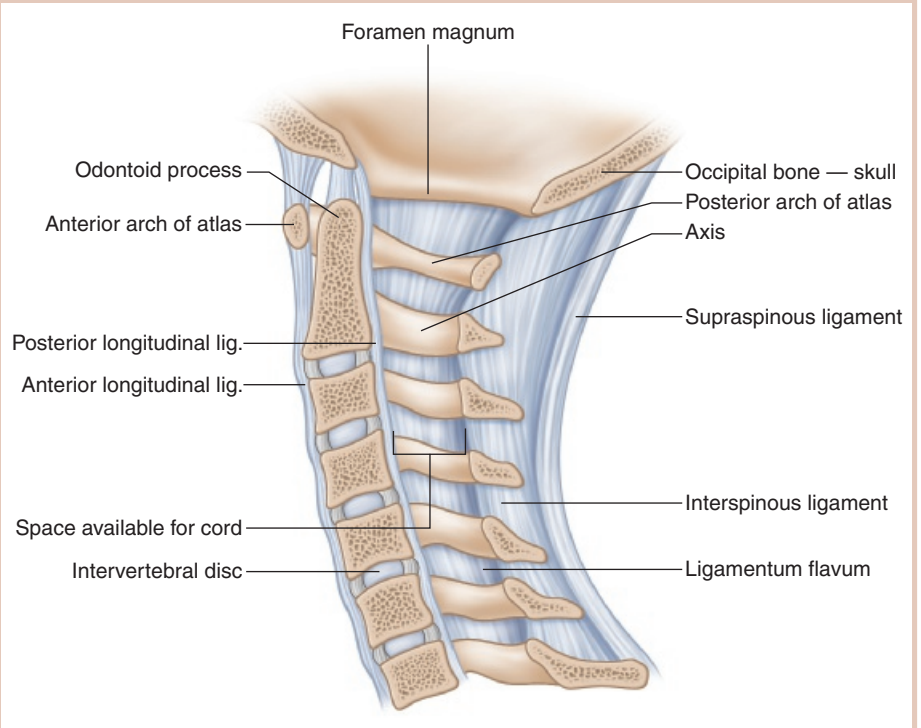
Spine surgery can be very challenging for an anesthesiologist. Perioperative issues potentially include difficult airway management, positioning injury, ventilation challenges, large volume shifts, coagulopathy, and postoperative visual loss. This chapter provides a brief overview of spinal anatomy, pathophysiology, procedures, and fundamental perioperative considerations.

1) **Anatomy of the spine**
   a) Composed of vertebrae, intervertebral discs, the spinal cord, and vascular structures with support by ligaments.
   b) There are 33 vertebrae.
      i) Seven cervical, 12 thoracic, 5 lumbar, 5 sacral, and 4 coccygeal.
      ii) The sacral and the coccygeal vertebrae are fused.
   c) Intervertebral discs
      i) Located between the vertebral bodies of C2-S1
      ii) Function to absorb axial force exerted on the spine and allow movement between vertebrae
   d) Spinal cord
      i) Located in the vertebral canal
   e) Spinal nerves
      i) Exist as pairs, with 8 in the cervical region, 12 in the thoracic region, 5 in the lumbar and sacral regions, and 1 pair in the coccygeal region.
      ii) Most exit through intervertebral foramina.
   f) Cervical spine (**Fig. 121-1**)
      i) Anatomically divided into the upper and lower cervical spines
      ii) Upper cervical spine
         (1) Contains the occipitoatlantal articulation between the atlas (C1) and the axis (C2).
         (2) There is no vertebral body associated with the atlas.
         (3) The axis has a small vertebral body that gives rise to the odontoid process.
         (4) The atlas rotates around the odontoid process.
         (5) The axis and atlas are devoid of intervertebral foramina.
         (6) Flexion and extension are greatest at C1-C2.
      iii) Lower cervical spine (C3-C7)
         (1) More conventional vertebral anatomy.
         (2) Has a lordotic curvature.
         (3) In the lower cervical spine, flexion and extension are greatest at C5-C7.
         (4) The cervical enlargement of the spinal cord starts at C3 and traverses the cervical spinal canal down to T2.
   g) Thoracic spine
      i) Upper thoracic vertebrae resemble cervical vertebrae.
      ii) Lower thoracic vertebrae resemble the lumbar vertebrae.



Spinal cord ends at approximately T12-L2 in most adults and L3 in newborns.

FIGURE 121-1 Anatomy of the Lower Cervical Spine.



Adapted from Soames RW, ed. Skeletal system. In: *Gray's Anatomy*. 38th ed. New York, NY: Churchill Livingston Inc.; 1995:425-736, with permission from Elsevier.

iii) The thoracic spine has a natural kyphotic curvature.
iv) The spinous processes slant downward.
(1) Spinous processes of upper third of thoracic spine end approximately one vertebral level lower.
(2) Spinous processes of middle third of thoracic spine end approximately one half vertebral level lower.
(3) Spinous processes of lower third of thoracic spine end approximately at the same vertebral level.
v) The lumbar enlargement of the spinal cord begins at the T9 vertebral body and ends at T12.

h) Lumbar spine
i) Has a lordotic curvature.
ii) Spinous processes are nearly horizontal.

2) **Spine pathology**

a) **Osteoarthritis (OA)**[1]
i) Can be idiopathic (primary) or secondary to factors such as injury, congenital anomalies, or metabolic disorders
ii) Pathophysiology
(1) Loss of articular cartilage, which may eventually result in pain and decreased range of motion.

(2) Spinal OA can involve the facet joints, the uncovertebral joints in the cervical spine (joints of Luschka), and the intervertebral discs.
(3) Pain can be localized, referred, or radicular.
(a) Radicular pain is due to compression of the spinal nerves and may be accompanied by sensory and/or motor deficits.
(4) Spinal cord compression can result from osteophytes or ligamentous hypertrophy.

b) **Rheumatoid arthritis (RA)**[1]

READ MORE
Arthritis, Chapter 69.

i) Pathophysiology
(1) Inflammatory disease process that, in its natural course, results in proliferation of the synovium.
(a) May result in cervical spine instability and spinal cord compression[4]
(i) Flexion-extension radiographs may be helpful to determine the degree of atlantoaxial instability.
(b) May be associated with myelopathy.
(2) Other comorbidities
(a) Cardiopulmonary sequelae, keratoconjunctivitis sicca, cricoarytenoid arthritis and dislocation, synovitis of the temporomandibular joint.
(b) Patients are often on chronic steroid therapy.



Since flexion-extension radiographs are not 100% reliable in predicting atlantoaxial instability in RA patients, it is prudent to presume an RA patient does have instability and treat the airway as such with minimal cervical extension during intubation.

c) **Ankylosing spondylitis**[2]
i) Pathophysiology
(1) Inflammatory arthropathy with localized or systemic findings.
(2) The entire spine may be involved resulting in the characteristic "bamboo spine."
(3) Cervical mobility may be significantly limited.
(a) Fiberoptic intubation (FOI) may be required.
(4) Other comorbidities may include pulmonary fibrosis, decreased chest wall compliance, aortic insufficiency, and conduction abnormalities.[2]



Patients with ankylosing spondylitis can develop bone fractures with minimal to no trauma. Hyperextension can lead to C-spine fracture. Be gentle with initial positioning and subsequent movement. Support kyphosis, if present.[2]

d) **Spinal cord compression**[3]
i) Due to multiple etiologies
ii) May be acute, subacute, or chronic
iii) Spinal cord compression due to arthritis
(1) Spinal cord compression can occur with OA due to posteriorly directed osteophyte formation.[1]
(2) Spondylosis
(a) OA involving the intervertebral disks, which may result in osteophyte formation on the disc, inflammation of the disc, and subluxation of the facet joints
(b) May be associated with myelopathy if a large posterior osteophyte forms on the disc
iv) Herniated nucleus pulposus[4]
(1) Disks can herniate median, paramedian, or lateral.
(2) Disease is determined by the structure compressed.
(3) Myelopathy occurs due to median (87%) or paramedian (13%) herniation.
(4) Radiculopathy occurs due to median (70%), paramedian (26%), or lateral (4%) herniation.

(5) Cauda equina syndrome may occur with either median or paramedian herniation in the lumbar spine.

v) Spinal stenosis
(1) Narrowing of the spinal canal, which may be congenital or acquired.
(2) Acquired spinal stenosis is generally due to multiple factors, such as disc degeneration, facet joint arthritis, and/or subluxation.

vi) Tumors
(1) Cause spinal cord compression by mass effect or by bone destruction resulting in vertebral collapse.
(2) Pain is usually localized.

vii) Trauma
(1) There is controversy related to the timing of surgery for patients who have suffered trauma to the spine, even when there is cord compression.
(2) In the polytrauma patient, the spine injury may not be the primary injury requiring surgery.
(a) Patients may have spine instability requiring special management.
(i) Consultation with an orthopedic or neurologic spine surgeon for guidance on proper positioning and management is recommended.
(3) Use of succinylcholine in patients with spinal cord injury
(a) Polytrauma patients may have crush injuries, which increases their risk of hyperkalemia.
(b) The amount of time since the spinal cord injury must be considered when choosing a muscle relaxant.
(i) Succinylcholine is likely safe in the first 24 hours following injury.[5]
(c) The risks and benefits of succinylcholine use in the 24-48 hours after spinal cord injury should be considered, and after this time period, it should be avoided.
(4) Hypotension in the trauma patient is often due to hypovolemia.
(a) If persistent hypotension occurs despite adequate volume resuscitation and surgical hemostasis, consider occult bleeding, spinal shock, or adrenal insufficiency in a patient with known inflammatory disease on steroids.
(b) Induction is a very dangerous time for hypotensive trauma patients. Ensure adequate IV access and resuscitation fluids/medications are available.
(c) Although seemingly deleterious in timing, it may be in the patient's best interest to rapidly transfuse RBC unit(s) prior to induction.

e) **Spinal cord transection**

i) Acute spinal cord transection
(1) Associated with flaccid paralysis and sympathetic nervous system dysfunction
(2) Spinal shock[6]
(a) Presents as hypotension, often with associated bradycardia.
(b) Spinal cord injury at the level of T6 or above can result in spinal shock due to a temporary sympathectomy.
(c) Lesions at T4 and above can also result in bradycardia.

READ MORE
Trauma, Chapter 125.



Extrajunctional receptors begin to proliferate and place the patient at risk for life-threatening hyperkalemia after 24-48 hours if succinylcholine is administered.[5]



Sugammadex administration allows for RSI doses of rocuronium to be rapidly and completely reversed following induction if motor evoked potentials are not present at baseline prior to positioning. Do not risk a hyperkalemic arrest with succinylcholine if the patient is close to 24 hours postinjury.



Spinal cord injury at the level of T6 or above can result in spinal shock due to a temporary sympathectomy.

(d) Hemodynamic instability may last for several weeks and will improve when spinal cord reflexes begin to recover.
(e) Risk factors for post-op pulmonary complications include:
(i) Lesion at the midthoracic level or above
(ii) Delayed surgery
(iii) Methylprednisolone administration

(3) Symptomatic treatment of spinal shock[3]
(a) Hypotension
(i) Treat with volume resuscitation, vasopressors, and inotropes as necessary.
(ii) Consider minimizing or avoiding PEEP if hypotension persists.
(iii) Be aware that if the patient is placed in reverse Trendelenburg position, there will be significant dependent pooling of intravascular volume.
(b) Bradycardia should be treated with direct-acting beta-adrenergic agonists.
(c) Consider arterial line placement for close monitoring of BP.
(d) Neurogenic shock can last 1-3 weeks.



Other invasive monitors may be necessary to monitor hemodynamics, especially in patients with injuries above T6.

ii) Chronic spinal cord transection[3]
(1) Characterized by hyperreflexia, paralysis, and possible spasticity
(2) Autonomic hyperreflexia
(a) An autonomic dysregulation syndrome caused by lack of descending sympathetic inhibition due to spinal cord damage.
(b) Stimulation leading to autonomic hyperreflexia can be cutaneous or visceral and must be below the level of the lesion.
(i) Common causes for stimulation include full bladder, pressure from positioning belts, pressure on extremities, or pressure on the abdomen.
(ii) May cause severe hypertension and a baroreceptor-mediated bradycardia.
(iii) Other dysrhythmias may also be observed.
(iv) Above the level of spinal cord injury, vasodilatation occurs.
(c) Autonomic dysreflexia is a serious situation and may progress to seizures, intracranial hemorrhage, or myocardial infarction if untreated.
(d) Treatment
(i) Have quick-onset, relatively short-acting vasodilators and heart rate control agents available.
(ii) Consider regional or neuraxial anesthesia prior to surgical stimulation.
(iii) If signs occur intraoperatively, deepening the anesthetic may help.
(iv) Uncover the patient to find a source of stimulus.
(v) Consider arterial line placement.



Patients with lesions at T10 or above are at risk for autonomic dysreflexia. Highest risk patients have lesions above T6.



Autonomic hyperreflexia leads to severe hypertension and reflex bradycardia

3) **Spinal surgery procedures**
a) **Spinal fusion**[7]
i) Indications
(1) Spinal instability, pain, fracture, stenosis, degenerative disease, tumor removal, disc herniation (post discectomy), infection, scoliosis, and failed previous fusion.

ii) Procedure
(1) Spinal fusion has a wide range of indications; thus, there are many methods used to fuse the spine.
(2) In general, fusion restores stability and/or relieves deformity.
(3) Techniques may involve bone grafting, cage placement, osteotomy, pedicle screws, plates, and rods at one or more levels.

iii) Approaches
(1) Cervical spine
(a) Anterior approach
(i) For upper cervical spine, approach may be transoral or anterior retropharyngeal.
(ii) For middle and lower cervical spine, the approach is usually from the left of midline.
(iii) The patient will be supine and usually in traction.
(b) Posterior approach requires the patient to be prone and in pins with or without traction.
(2) Cervical-thoracic junction
(a) With a trans-sternal or transclavicular approach, the patient will be supine.
(b) If the area is reached via thoracotomy, the patient will be lateral and one-lung ventilation may be required.
(c) Posterior approach is the most common and requires prone positioning.
(i) Whether to pin the head or pad the face depends on the cephalad extent of the fusion.
(3) Thoracic
(a) Anterior approach
(i) Positioning will be lateral decubitus and may require one-lung ventilation.
(b) Transdiaphragmatic approach
(i) Used for T11-L1 surgery.
(ii) Lateral decubitus position.
(iii) One-lung ventilation is generally not required.
(c) Posterior approach requires prone positioning and is the most common.
(i) Face padding in the form of a prone view device is typically used.
(4) Lumbar
(a) Approach may be anterior, posterior, or lateral.
(5) Combined anterior/posterior approaches are used for fusions of three or more vertebral levels and may require a table that rotates.



When rotating a bed for and anterior/posterior spine case, disconnect most if not all lines from the patient and keep your hands free to prevent injury.

b) **Microdiscectomy**[7]
i) Indication is typically acute or chronic pain from a herniated disc.
ii) Procedure
(1) Performed through a small posterior incision with the appropriate level confirmed radiographically.
(2) The ligamentum flavum is exposed and then an operating microscope is used to remove the ligamentum flavum and the disc.
iii) Position may be prone or lateral decubitus.

c) **Laminectomy**[7]
i) Indications
(1) Spinal element decompression and exposure for removal of intraspinal masses.
(2) Decompression of the cord from canal narrowing secondary to other pathology such as OA or spinal stenosis.

ii) Procedure
(1) Performed through a midline posterior incision with the appropriate level confirmed radiographically.
(2) The lamina (and possibly disc) is exposed and removed in pieces.
(3) Be aware that there is risk for epidural and retroperitoneal bleeding.
iii) Position is prone.

d) **Kyphoplasty and vertebroplasty**[7]
i) Indication
(1) Pain from vertebral compression fracture, traumatic, osteoporotic, or osteolytic
ii) Kyphoplasty procedure
(1) The fractured vertebral body is entered percutaneously, and a bone tamp is inflated, which restores vertebral height.
(2) The tamp is then removed, and a filler is placed in the created space to maintain the newly created architecture.
(3) Fluoroscopy is used extensively.
iii) Vertebroplasty procedure
(1) Same as a Kyphoplasty but without the use of a balloon tamp.
iv) Position is prone.
v) General anesthesia or sedation may be used, depending on patient and institution.


The goal of the procedure in kyphoplasty or vertebroplasty is alleviation of neural compression, not height restoration.

e) Laminoplasty
i) Very similar to a laminectomy except the lamina is left in place.
(1) Lamina are cut on one or both sides to relieve pressure in the intrathecal space.
ii) Typically done on the cervical spine with prone positioning and pins or face padding.

4) **Perioperative approach to the patient undergoing spinal surgery**[7]

a) **Preoperative considerations**
i) Preoperative evaluation
(1) Neurologic
(a) Perform a preoperative neurologic exam and document any preexisting sensory or motor deficits or symptomatology.
(b) Discuss seizure history, current pain issues, and pain medication regimen.
(c) Discuss the role and implications of neuromonitoring


Recognize that alternate and unconventional pain therapies are often used by chronic pain patients and may influence your anesthetic plan

ii) Pulmonary/airway
(1) Evaluate for respiratory insufficiency from neuromuscular weakness or diaphragmatic insufficiency. If the patient has scoliosis, determine severity because it could impair pulmonary function.
(2) In the airway exam, skip neck flexion/extension if patient has an unstable cervical spine or neurologic symptoms with movement. Review cervical spine imaging studies if available. If the surgery is being performed for correction of cervical instability or if it is a trauma patient, the surgeon may be an invaluable reference for determination of stability.
(3) Determine need for awake vs asleep intubation.
(4) Determine need for advanced airway techniques, that is, fiberoptic bronchoscopy and/or video laryngoscopy (VL).


Even cleared plain films of the cervical spine in a trauma patient do not rule out ligamentous injury.

iii) Cardiovascular
(1) Evaluate for history of spinal shock or autonomic hyperreflexia.
(2) For trauma patients, evaluate volume status.
(3) Consider invasive monitoring if there is concern about extensive blood loss, history of autonomic hyperreflexia, and/or coexisting cardiovascular disease.

iv) Hematologic
(1) Over-the-counter, prescription, and herbal medications may impair hemostasis.
(a) Determine the need for preoperative or early intraoperative coagulation studies, for example, TEG and/or PT, INR, PTT, fibrinogen.
(2) Discuss any history of coagulopathy, transfusion history, and current willingness to accept blood products.
(3) Discuss cell recovery and normovolemic hemodilution if applicable.
(4) Consider sending blood for type and cross as well as using TXA prior to incision.

v) Positioning
(1) Ask if any position causes significant pain or provokes neurologic symptoms. Some conditions, such as spinal stenosis, may cause dynamic symptoms.
(2) Prior to placing the patient in the prone position for midthoracic and lower spine surgery, it is important to evaluate his or her ability to raise the arms above the head.
(3) Remember to maintain the shoulders and elbows flexed <90 degrees.
(4) Consider padding the anterior shoulder to prevent injury to the brachial plexus.
(a) This type of injury is often seen on somatosensory monitoring.

b) **Intraoperative considerations**

i) Airway management
(1) Cervical spine instability[3]
(a) Spinal movement should be minimized during airway management.
(b) Blade elevation during laryngoscopy can cause superior rotation at the occiput-C1 and inferior rotation at C2-C5. With tracheal intubation, there is superior rotation at the occiput-C1.[3]
(c) Cervical collars
(i) The patient may present to the operating room with a cervical collar in place.
(ii) Cervical collars do not eliminate cervical spine movement during airway manipulation and may make intubation more difficult due to decreased mouth opening.
(iii) Some advocate removing the anterior portion of the collar after manual inline immobilization (MILI), prior to laryngoscopy.



Some cervical collars are placed for stability and some for patient comfort. It is important to know what the indication is, and the only way to know this answer is to review the imaging and speak with the operative team.

(2) Manual inline immobilization (MILI)[3]
(a) May be performed either at the head or side of the bed.
(b) If performed at the head of the bed, one holds the mastoid processes with the fingertips and the occiput rests in the palms.
(c) If performed from the side of the bed, the mastoids are held in the palms and the occiput is grasped with the fingertips.
(d) When laryngoscopy is performed, the individual providing MILI applies pressures equal and opposite to maintain a neutral position.



Polytrauma patients may require a halo for spinal stability, which can complicate airway management and positioning.

(3) Techniques[3]
  (a) Mask ventilation
    (i) May cause more cervical spine movement than nasal or oral tracheal intubation.
  (b) Laryngeal mask airway
    (i) May exert pressure on the upper cervical vertebrae, but the clinical significance of this finding is unclear.
    (ii) LMA-assisted intubation may be another option, which has been demonstrated in a small study to have equal C1-C2 motion compared to FOI.[8]
  (c) Direct laryngoscopy (DL) vs video-assisted technique, awake vs asleep
    (i) There are no clear data as to which technique results in a better outcome.
    (ii) Traditionally, awake FOI was thought to be the safest option.
    (iii) Widespread use of VL has made this a very familiar and safe technique.
    (iv) Comparative studies between awake and asleep various techniques (ie, DL, VL, FOI, and LMA assisted) have not shown clinically significant differences in terms of success or complication rates.[9]
    (v) Backup techniques should always be available.



The best airway management technique is probably one which considers the resources and experience present as well as the individual preference of the provider.

ii) Induction
  (1) Hemodynamically stable patient
    (a) Induction agent of choice is Propofol.
  (2) Hemodynamically unstable patient
    (a) Consider ketamine or etomidate if hypotension is present.
    (b) If spinal shock is present, direct-acting alpha- and beta-adrenergic agonists should be available.
    (c) Blood pressure can be individually titrated based on spinal cord neuromonitoring or maintained at the pre-op level.
    (d) For traumatic spinal cord injury, the optimal perfusion pressure is harder to identify.
      (i) Guidelines are MAP 85-95 mm Hg for 7 days,[10] but this may be possible to individually titrate; studies are pending.
  (3) Choice of muscle relaxant
    (a) Acutely, succinylcholine is likely safe but should be avoided after the first 24 hours.[5]



Patients with acute spinal cord lesions above T4 may not generate a compensatory tachycardia in the face of hypovolemia due to a lack of sympathetic input.

iii) Positioning
  (1) Avoid positions that exacerbate preexisting neuropathies. Consider positioning prior to sedation.

READ MORE
Positioning of the surgical patient, Chapter 7
Neurophysiologic monitoring and anesthetic management, Chapter 16

iv) Maintenance
  (1) Prepare for the possibility of autonomic hyperreflexia or spinal shock.
  (2) In the trauma patient, consider that hypotension could be caused by things other than spinal shock (eg, hypovolemia, myocardial ischemia, tension pneumothorax, venous air embolism [VAE], etc.)

READ MORE
To venous air embolism section of the Neuroanesthesia chapter 157. Relative risk for VAE in spinal fusion and cervical laminectomy is medium risk.



A common stimulus for autonomic hyperreflexia is bladder or gastrointestinal distention. Autonomic hyperreflexia may occur in recovery after the anesthetic has worn off.

(a) Cervical and upper thoracic spine surgery is considered medium risk for VAE due to prone positioning with the surgical site above the heart, and the risk of VAE increases as the degree of surgical exposure increases.

(3) Consider invasive monitoring.

(4) If significant blood loss is anticipated, consider tranexamic acid bolus and infusion.

(5) Intraoperative neuromonitoring[12]

(a) Prior to induction, discuss neuromonitoring with the surgeon.

(b) Place bite blocks between the molars if motor evokes potentials (MEPs) are to be performed to prevent injury to the oral soft tissue.

(c) In general, IV anesthetics will have less effect on SSEPs and MEPs than inhaled agents.

(i) Consider baseline neurologic status and if neurologically impaired start with full TIVA to aid neuromonitoring.

(d) Anesthetic technique does not affect EMG, but paralytics should be avoided for EMG monitoring.

(e) Notify the neuromonitoring team about any changes in hemodynamics or anesthetic technique.

(f) Wake-up test.

(i) Involves waking the patient during the operation and evaluating movement of extremities.

(ii) Requires a cooperative patient.

(iii) Coordinate timing with the surgeon, so muscle relaxation is not inadvertently given close to the wake-up time.

(iv) Generally, anesthetic agents and muscle relaxants are allowed to wear off.

(v) Only limited movement of hands and feet is required.

(vi) Used less often with increased availability of advanced neuromonitoring.

Recognize that even if the surgical area is insensate, stimulation of areas below the level of the lesion can result in autonomic hyperreflexia.

READ MORE
VAE, Chapter 157

Don't forget to place bite blocks prior to turning a patient prone. Having to place bite blocks in a prone position is awkward and dangerous.

Start with maximizing reasonable multimodal therapy and then adding narcotics for pain control as opposed to the classic approach of narcotics first then multimodals.

c) **Postoperative considerations**

i) Neurologic exam

(1) A neurologic exam should be performed in the PACU, with a follow-up exam on your postoperative check.

(a) Abnormalities should be discussed immediately with the surgeon.

ii) Pain control

(1) Patients undergoing spine surgery may be taking a significant amount of narcotics preoperatively, making traditional dosing of narcotics less effective.

(2) Multimodal therapy

(a) Preoperative and scheduled postoperative options

(i) Acetaminophen

(ii) Gabapentin or pregabalin

(iii) Meloxicam or ketorolac depending on individual surgeon preference and surgery type

(iv) Methadone

(v) Oxycodone or partial agonist if other narcotics are not used

(b) Intraoperative and possible postoperative options
  (i) Ketamine bolus plus repeat dosing or infusion
  (ii) Dexmedetomidine or clonidine
  (iii) Lidocaine infusion

(3) Epidural[13]
  (a) Either the surgeon can place an epidural catheter intraoperatively through which opioids and/or local anesthetics can be infused or the anesthesia team can place a single shot epidural injection prior to induction.

(4) Intrathecal morphine[14]
  (a) Preservative-free morphine placed in the intrathecal space by the surgeon has been shown to provide better pain relief than placebo in patients undergoing lumbar spine surgery.

(5) Regional Techniques
  (a) Erector spinae blocks are being used in select patients and have shown promise but further study is needed.

CHAPTER SUMMARY FOR SPINE SURGERY

Preoperative Evaluation	Focus on the indication for surgery, preexisting neurologic deficits, spine stability, hemodynamic stability, need for and type of blood products, and required access. Incorporate neuromonitoring needs into your anesthetic plan.
Intraoperative Management	Consider need for an augmented induction plan if hemodynamics are compromised. Succinylcholine should be avoided in patients with spinal cord injuries after the first 24 hours. Have a multistep airway plan based on spine stability. Consider the need for prepositioning baseline neuromonitoring signals and how this effects muscle relaxant choice and BP management on induction. Prepare for spinal shock or autonomic hyperreflexia if the clinical situation is appropriate. Consider antifibrinolytics and cell salvage if multilevel approach. Blood pressure and anesthetic consistency are needed to minimize false-positive alerts if using neuromonitoring. Perform a cuff leak check prior to extubation following a prolonged prone case or if there are overt signs of facial swelling. Risk facts for postoperative vision loss are largely unmodifiable with the exception of BP management and fluid choice.
Postoperative Pain Control	Chronic pain spine patients are almost always taking a combination of analgesics. Consider multimodal therapies such as acetaminophen, pregabalin or gabapentin, NSAIDs, dexmedetomidine, ketamine, lidocaine infusion, and regional anesthesia as appropriate. Discussion with the patient preoperatively in terms of realistic PACU pain expectations will benefit all involved.
Postoperative Exam	Notify the surgeon if new neurologic abnormalities are found and augment hemodynamics as needed. Postoperative airway compromise is a concern following anterior cervical spine surgery due to hematoma formation or recurrent laryngeal nerve compromise.

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122 Otolaryngologic Surgery[§]

Kevin Malott[†] • Erin Bushell

Otolaryngologic or ear, nose, and throat (ENT) surgery can be challenging to the anesthesiologist because these patients are often at risk for airway compromise. Many patients have coexisting illnesses that can complicate anesthetic management. Lasers can be advantageous for airway surgery but require advanced knowledge and preparation to prevent complications.

1) **Surgical considerations for ENT procedures**
   a) Otolaryngologic procedures constitute a large share of all surgical procedures.
   b) With the population aging, demand will likely increase, especially for resections of head and neck neoplasms and for increasingly sophisticated otological interventions for hearing loss.
   c) Common operations include various ear, nose, sinus, throat, and laryngeal procedures; tracheostomy; definitive oncologic resections; surgery to relieve obstructive sleep apnea (OSA); reconstructive procedures; and panendoscopy to aid in the diagnosis of disorders of the aerodigestive tract.
2) **Preoperative assessment**
   a) **Patients are often elderly and may have significant comorbidities.**
      i) HTN, COPD, CAD, and OSA are the most common.
   b) **Preoperative assessment of the major organ systems and optimization of preexisting disorders is warranted.**
   c) **Oncologic patients:** These patients present at an older age with comorbidities and usually require longer, more invasive, and more complex surgeries.
      i) Tobacco exposure is the most significant risk factor for cancers of the head and neck.
         (1) Patients presenting for oncologic surgery have a high prevalence of COPD.
      ii) Patients may be malnourished secondary to dysphagia and further debilitated from concomitant chemoradiation therapy.
      iii) Patients often present with anemia, coagulopathy, electrolyte imbalances, and occasionally, aspiration pneumonia.
   d) **A thorough airway exam is critical (Table 122-1).**
      i) Close attention to the ease of air movement; noting the presence and timing of stridor, wheezing, or other adventitious breath sounds; as well as noting any limitations in mouth opening and radiation changes in neck tissue are important in assessing the airway in these patients.
      ii) If the patient, while wide awake and with intact airway reflexes, is having difficulty breathing, he or she may be impossible or exceedingly difficult to mask ventilate after the induction of anesthesia.
      iii) Likewise, if supraglottic or glottic anatomy is overly obscured by profuse bleeding, edema, or tumor, direct visualization of the glottis and endotracheal intubation may be impossible, and using a technique to secure the airway with the patient spontaneously ventilating or awake should be considered.
   e) **Patients with chronic airway obstruction and OSA may have chronic hypoventilation with hypoxemia, which results in increased risk of pulmonary hypertension and right-sided heart failure.**



For ENT procedures, remember that the surgeon is also an airway expert. Preoperative discussion with the surgeon about the patient's airway exam may yield essential information.

TABLE 122-1 Predicting Unanticipated Difficult Airway Management (in Decreasing Order of Predictive Value)[1]

- Mallampati 3 or 4
- Neck radiation changes or neck mass
- Male sex
- Limited thyromental distance (<3 cm)
- Presence of teeth
- Body mass index ≥30 kg/m^2
- Age > 46
- Presence of beard
- Thick neck
- Sleep apnea
- Unstable cervical spine or limited neck extension
- Limited or severely limited jaw protrusion

Other Conditions Associated With Likely Difficult Airway Management

- Previous history of difficult mask ventilation or intubation
- Airway obstruction manifested by tachypnea, stridor, or wheezing (stridor at rest suggests severe airway narrowing, and the timing suggests the location of obstruction with an inspiratory stridor, suggesting a supraglottic process, and an expiratory stridor, suggesting a subglottic obstruction)
- Acute or chronic oropharyngeal or laryngeal swelling secondary to infection, epiglottitis, allergic reaction, neoplasm, vascular anomaly, or bleeding

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i) Clinical signs
   (1) Cyanosis, jugular venous distension, a loud P2, clubbing, right axis deviation and RVH on ECG, poor exercise capability, polycythemia.
   (2) If present, further cardiologic evaluation is recommended.

3) **General principles**
   a) **Airway management**
      i) For ENT procedures in general, and airway procedures in particular, close communication with the surgeon is paramount.
      ii) Discussion about preoperative assessments, such as radiology studies, nasal endoscopies, and your joint physical exam findings, are important to create a safe plan for managing the airway.
      iii) Surgeons often have individual preferences regarding type of endotracheal tube (ETT) (nasal vs oral, standard vs reinforced, laser vs plastic, etc.), the feasibility of using a supraglottic airway (SGA), how best to secure the tube, and head positioning.



If a difficult airway is anticipated, the surgeon should be present to assist during anesthesia induction, as their assistance can be invaluable when implementing advanced airway techniques and rescue maneuvers, including establishing a surgical airway.

   b) **Deliberate hypotension**
      i) Except for very superficial procedures, the use of deliberate relative hypotension to limit blood loss and improve surgical visibility may be requested by surgeons for ENT procedures.
      ii) It is important to accurately assess your patient's ability to tolerate induced hypotension without end-organ dysfunction or injury.
         (1) Consideration MUST be given toward maintaining a safe minimum BP to assure adequate renal, coronary, and cerebral perfusion.
      iii) Achieving deliberate hypotension
         (1) In anticipation of a smooth emergence, it is less problematic to achieve deliberate hypotension with antihypertensive agents as opposed to merely deepening anesthetic levels.
         (2) If hypotension is induced with anesthetic agents, use very short-acting drugs.



If deliberate hypotension is requested, ALWAYS consider the patient's coexisting diseases when determining a lower limit for hypotension and clearly communicate with the surgeon. In some cases, it may not be safe.

c) **Teamwork**
   i) A team approach toward airway management is critical.
   ii) The surgeon, recent scans, and nasopharyngoscopy results can provide important information for the assessment of the likelihood of difficulty in managing the airway.
   iii) ENT surgeons are also experts at securing an airway, so their abilities and assistance can be invaluable.
      (1) Surgeons should be present and ready to assist, as necessary, during anesthetic induction and all crucial steps of airway management.
   iv) It is important to discuss the timing and use of muscle relaxants, use of corticosteroids to reduce airway edema, and post-op airway and pain management.

4) **Difficult airway management**
   a) **Awake vs asleep**
      i) When there is no concern about the ability to ventilate by mask, it is reasonable to proceed with induction of general anesthesia.
      ii) If difficulty is anticipated with mask ventilation and intubation, the patient's airway should be secured while awake, either after instituting topical anesthesia to the airway or by tracheostomy.
      iii) Preoperative endoscopic airway exam (PEAE) may provide useful information regarding the need for awake intubation. The nares are treated with a vasoconstrictor and local anesthetic and a small fiberoptic scope is used to assess the airway and determine the need for an awake intubation.[2]



Regardless of the airway management plan, it is important to have back-up plans should the primary plan fail.

   b) **Fiberoptic endotracheal intubation**
      i) May be performed with the patient awake or asleep.

READ MORE
Difficult airway, Chapter 21

   c) **Special airway management options**
      i) In addition to the standard assortment of straight and curved laryngoscope blades, SGAs, bougies, stylets, and nasal and oral airways, many other options exist for the establishment of a secure or at least manageable patent airway.
         (1) If the airway is not expected to be fragile or prone to injury
            (a) A "blind" technique, such as a lighted stylet, intubation through an SGA either with a kit such as LMA Fastrach (Teleflex) or a small ETT inserted blindly through an SGA, or blind nasal approach is reasonable.



If difficult mask ventilation is anticipated, strongly consider securing the patient's airway while awake and/or spontaneously ventilating.

      ii) If a tumor is encroaching on the airway
         (1) Visualization of the passageway to the glottis is preferable. PEAE may provide information to help in formulating plan.
            (a) Techniques, such as direct laryngoscopy, use of a video laryngoscope (VL), or fiberoptic scope, or a combined technique using VL and fiberoptic scope are preferable to blind techniques.

READ MORE
Fiberoptic intubation, Chapter 23, and Cognitive aid, Chapter 164.

      iii) SGA use for ENT procedures (**Table 122-2**)
         (1) The SGA can be an excellent choice for ENT surgeries as its use avoids the stress of tracheal stimulation and offers a smooth emergence.
         (2) SGAs have been used successfully for a variety of ENT procedures, including tonsillectomies.
         (3) Risk/benefit assessment
            (a) It may seem against conventional wisdom not to protect the airway from blood and secretions with an ETT during

READ MORE
Supraglottic airway, Chapter 22

TABLE 122-2 Uses of the Supraglottic Airway for ENT Surgery

- May facilitate oral ETT placement either by intubation through SGA or as a guide for FOI.
- As a rescue airway when mask ventilation not possible.
- As a primary airway management device. Advantage of smoother emergence may be particularly advantageous after sinus, facial, nasal, and other sensitive procedures, where the avoidance of mask ventilation, coughing, or bucking on emergence is desirable.
- An alternative to MAC anesthesia cases requiring heavy levels of sedation to isolate oxygen-enriched airway gases away from the surgical field (see section on fire prevention).
- Provide less stimulation than ETT, making it easier to keep BP within low normal range for procedures where this is desirable such as sinus surgery.

SGA, Supraglottic airway; FOI, fiberoptic intubation; ETT, endotracheal tube; MAC, monitored anesthesia care; BP, blood pressure.

ENT procedures, particularly when the operative table is turned away from the anesthesia machine.

(b) However, blood and secretions will typically pool above the SGA where they can readily be seen and suctioned by the surgeon.

(c) Advantages gained by a smoother emergence can be well worth the mild decrease in control of the airway with the SGA as compared to an ETT.

(4) The LMA Flexible (FLMA, Teleflex) is well suited for ENT cases.

(a) Has a smaller outer diameter per given cuff size

(b) Less prone to kinking

(c) Less likely to become dislodged

(i) More pliable so manipulation of the SGA shaft does not transmit movement to the cuff as easily as the standard SGA.

5) **Considerations for anesthetic emergence in ENT procedures**

a) **A smooth controlled emergence is desirable** to avoid increased bleeding from excessive hypertension or damage to freshly sutured tissues from postextubation airway manipulation.

b) Upon awakening, blood, secretions, and the inherent irritation from operating on this highly sensitive area of the body may predispose to coughing, straining, and laryngospasm.

c) **Finding a balance between suppression of airway irritation with good analgesia while avoiding oversedation is an important challenge.**

i) If the case warrants, use of an SGA may decrease the likelihood of a turbulent emergence, with a reduced incidence of coughing on emergence, SaO_2 desaturations, laryngospasm, sore throat, and laryngeal trauma.

ii) Judicious use of local anesthesia and cranial nerve blocks can enhance patient comfort and decrease postoperative narcotic requirements.

iii) Consider using adjuncts such as dexmedetomidine and ketamine as part of a safe and balanced anesthetic plan.

iv) Measures to prevent postoperative nausea and vomiting should be followed vigorously.

v) Consider administering corticosteroids to help reduce airway edema.

d) **If little post-op pain is expected**

i) Short-acting IV agents such as remifentanil may be advantageous in facilitating a pleasant and cooperative emergence.

6) **Postoperative considerations**

a) **If doubts remain about the ability to maintain an airway**

i) Extubation over an airway exchange catheter should be attempted prior to committing to complete extubation. These catheters are well tolerated by patients and may be left in during the PACU stay.

ii) Conversely, the patient may be left intubated and sedated until extubation is appropriate.

b) **Airway obstruction secondary to edema**
   i) Manifested by stridor, tachypnea, and increased work of breathing
   ii) Must be treated aggressively
      (1) Administer steroids, nebulized racemic epinephrine, and humidified O_2.



If doubts remain about the ability to maintain an airway after extubation, a trial of extubation with an airway exchange catheter should be attempted prior to committing to complete extubation.

c) **Negative pressure pulmonary edema (NPPE)**
   i) Any type of irritated airway increases the risk of this dreaded phenomenon.
   ii) Potentially severe and life threatening
   iii) Most common in young, healthy, strong patients who can generate high negative pressures
   iv) Occurs when patients attempt to inhale against an obstructed airway (eg, closed glottis, obstructed ETT from biting, or any type of upper airway obstruction)
   v) Results in severe pulmonary edema with profound hypoxemia[3]
   vi) Treatment ranges from mild interventions such as supplemental oxygen and observation to ventilatory support with PEEP, high FiO_2, and possibly extracorporeal membrane oxygenation (ECMO) if hypoxemia is profound enough to cause cardiovascular collapse, and pulmonary edema/hypoxemia is not readily reversible with less invasive measures.

7) **Specific situations**
   a) **Otologic and neurotologic surgery**
      i) Preoperative considerations
         (1) General concerns, with patients presenting from all age groups
      ii) Intraoperative considerations
         (1) Avoidance of muscle paralysis during facial nerve monitoring
         (2) May need to avoid nitrous oxide with tympanoplasty
         (3) Resections of acoustic neuromas and skull-based lesions
            (a) Usually performed in collaboration with neurosurgery
            (b) May require relative hypotension, neuromonitoring, and a smooth emergence
         (4) If surgery was intradural, coughing and straining on emergence may increase ICP and predispose to a dural leak.
      iii) Postoperative considerations
         (1) Cranial nerve palsies causing dysphagia and predisposing to pulmonary aspiration are possible.
   b) **Panendoscopy**
      i) Indications
         (1) Assessment of aerodigestive tract, foreign body removal, removal of superficial lesions
         (2) Procedures include esophagoscopy, laryngoscopy, and bronchoscopy.
      ii) Preoperative considerations
         (1) Patients may have head and neck cancers so the aforementioned concerns for oncologic patients apply.
      iii) Intraoperative considerations
         (1) These procedures are profoundly stimulating, but of brief duration and have little post-op pain.
         (2) Often require an open anesthesia circuit to ambient air for surgical access to the airway
         (3) Transnasal humidified rapid-insufflation ventilatory exchange (THRIVE) using the Optiflow (Fisher & Paykel) or some other high-flow nasal cannula (HFNC)

is being used at some centers to provide apneic oxygenation during these procedures.[4,5]

(4) TIVA with short-acting agents is an excellent choice.
(5) Use caution when intubating patient to avoid traumatizing tongue base tumors.
(6) Flexible bronchoscopy may be performed through a Patil-Syracuse mask during mask ventilation or a large diameter ETT.
  (a) See airway management under ventilation strategies below
(7) Muscle relaxation
  (a) Suggested to avoid patient injury from movement when in suspension
  (b) The advent of sugammadex now allows for profound paralysis with rapid reversal.

iv) Postoperative considerations
(1) Complications include esophageal perforation and barotrauma.

c) **Tracheostomy**

i) Indications
(1) Elective
  (a) Useful for intubated patients requiring prolonged respiratory support with the following advantages:
    (i) Increases comfort level
    (ii) Decreases potential for laryngeal injury
    (iii) Enhances airway access for clearance of secretions
    (iv) Improves airway protection from pulmonary aspiration
  (b) May be performed for severe OSA
  (c) Occasionally performed as part of a larger ENT procedure such as total laryngectomy
(2) Emergent
  (a) Patients with impending or progressing airway obstruction in whom other techniques are unsafe

ii) Preoperative considerations
(1) Intubated patients will have significant comorbidities and may be critically ill.
  (a) Patients should be reasonably hemodynamically stable and any significant metabolic or coagulation derangements should be corrected.
  (b) Assess whether the patient can tolerate not only brief periods of apnea inherent to the procedure but also transport to and from the operating room.
(2) Accurate assessment of the airway is important.
  (a) If safety in inducing unconsciousness and managing the airway is not assured, use a technique to secure the airway with the patient awake or place the tracheostomy under local anesthesia. HFNC such as with the Optiflow (Fisher & Paykel) may be used during awake tracheostomy done under local. This provides the patient with symptomatic relief of stridor due to positive airway pressure and a greater margin of safety against desaturation.

iii) Intraoperative concerns
(1) Risk of airway fire if electrosurgical techniques are used (see preventing airway fire algorithm in this chapter)
  (a) The trachea should not be opened with an electrosurgical device.
(2) When the surgeon is ready to open the trachea, increase FiO_2 to 100% and keep the ETT cuff below the level of incision.
  (a) Patients with tracheal edema or fragile tissues are presumably at highest risk.
  (b) If using Optiflow, discontinue it before trachea is entered.

(3) When the surgeon requests removal of the ETT, only withdraw as much as needed to allow placement of the tracheal tube, but keep the original ETT in the trachea. Placing an airway exchange catheter prior to the exchange can provide another layer of safety.
(4) It is possible (even after initial visualization of the ETT/tracheal lumen) for the surgeon to pass the new tracheal tube into a false passage created by a mucosal separation.
(5) Only after the presence of $ETCO_2$ through the new airway should the original ETT be removed.

d) **Acute upper airway obstruction**
i) Indications
(1) Oropharyngeal abscess (OA), tumor, trauma, epiglottitis, or angioedema

READ MORE
Epiglottitis, Chapter 93

ii) Preoperative considerations
(1) Patients may have stridor at rest, cyanosis, or worsening hypercarbia.
(a) These patients are in imminent danger of airway loss.
(b) HFNC with Optiflow (Fisher & Paykel) may be useful in these patients.
(2) OA may be deceivingly large and result in significant airway obstruction during airway management.
(a) Manipulation of the airway can lead to disruption of abscess with the potential for copious drainage and aspiration of its contents.
(3) Epiglottitis
(a) With the widespread use of the *H influenzae* vaccine, epiglottis is rarely seen in children and now is most commonly encountered in adults.
(b) Patients are at risk for respiratory failure and may be intubated under general anesthesia in the operating room.
iii) Intraoperative considerations
(1) Seriously consider, and be prepared for the placement, of a surgical airway under local anesthesia.
(2) Induction and airway management
(a) Anesthesia may be slowly induced with inhalational agents, in 100% O_2, while maintaining spontaneous ventilation.
(b) Once a deep plane of anesthesia is reached, direct visualization of the glottis and endotracheal intubation can be attempted.
(c) Use smaller than normal ETTs (5.0-6.0 mm).
(d) Proceed very cautiously in caring for patients with airway tumors.
(i) Tumors near the base of the tongue are often fragile and not only may physically block the route to the glottis but may bleed or become dislodged and further obstruct the airway.

e) **Sinus/maxillofacial/nasal surgery**
i) Indications
(1) Chronic sinus disease, nasal polyps, control of epistaxis, and aesthetic concerns
(2) Functional endoscopic sinus surgery (FESS) done for the resection of inflamed bone and tissue to restore normal mucociliary clearance
ii) Preoperative considerations
(1) May be associated with triad of nasal polyposis, NSAID hypersensitivity, and reactive airway disease
iii) Intraoperative considerations
(1) SGA may be especially useful, given the importance of a smooth emergence with the avoidance of postoperative mask ventilation. The FLMA protects the

airway from blood and surgical debris by forming a reliable seal across the oropharyngeal inlet, so any bleeding will typically pool above the SGA and can be readily seen and suctioned by the surgeon.
(2) Expect periods of highly stimulating surgical manipulation with little postoperative pain.
(3) Safe deliberate hypotension may be preferred to minimize blood loss as hemorrhage can be copious and will diminish surgical visibility.

iv) Postoperative considerations
(1) Complications include possibility of dural, nasolacrimal, ocular, or carotid injury.

f) **Surgery to relieve OSA**

i) Preoperative considerations
(1) See preoperative assessment for chronic airway obstruction.

READ MORE
OSA, Chapter 54

ii) Intraoperative considerations
(1) Patients frequently are morbidly obese and may be difficult to mask ventilate.
(a) Consider awake FOI if there is sufficient concern for ability to ventilate (see **Table 122-1**).
(b) These patients are at risk for airway obstruction after extubation secondary to glossopharyngeal obstruction or bleeding from uncontrolled HTN.
(c) Patients are susceptible to postoperative HTN, airway edema, and pulmonary edema.
(i) Consider using nonnarcotic adjuncts for pain control to decrease likelihood of respiratory depression.
(ii) Avoid overhydration.

iii) Postoperative considerations
(1) Patients are more prone to over sedation with narcotic analgesics and may warrant more monitoring and longer hospital stays.
(2) Close BP control may decrease risk of airway hemorrhage and edema.

g) **Emergent surgical airway**

i) Indications
(1) Part of difficult airway algorithm when attempts at endotracheal intubation, and SGA or facemask ventilation have been unsuccessful

READ MORE
Difficult Airway, Chapter 21

(2) If an experienced surgeon is not present, should be placed by anesthetist.
(3) There is currently some controversy regarding the best approach to obtain emergency front of neck access (FONA).[6]

READ MORE
Difficult Airway, Chapter 21

(4) Some still advocate the needle cricothyroidotomy and use of the Seldinger technique.
(5) However, both the Difficult Airway Society (DAS) and the Veterans Administration (VA) now advocate the use of the scalpel, bougie, tube method.
(6) Jet ventilation or insufflation of oxygen with permissive hypercapnia may be used initially until the airway can be dilated sufficiently to allow adequate tidal volume. This carries an extremely high risk of barotrauma and is not generally advised.
(7) THRIVE has been successfully used to maintain oxygen saturation during emergency surgical airways for period of up to 30 minutes. It is important to ensure airway patency for THRIVE to be effective. Nonetheless, the degree of patency needed is much less than that for effective mask ventilation, and therefore, THRIVE may be lifesaving in these circumstances.[7,8]

h) **Parotidectomy**

i) Indications
(1) Neoplasms and sialolithasis

ii) Preoperative considerations
(1) Trismus may occur with parotid disease. Although this is not common, it is prudent to carefully assess mouth opening in these patients.
iii) Intraoperative considerations
(1) A nasal intubation, preferably in the contralateral nare, is preferred by some surgeons to reduce the risk of damage to the facial nerve.
(2) Paralysis should be avoided so as not to interfere with facial nerve monitoring.
iv) Postoperative considerations
(1) Facial nerve palsies and paralysis are the most common complications.

8) **Laser surgery**
a) **Indications**
i) Ablation or debulking of various tumors of the head, throat, and airway
ii) Lasers are notably helpful in the precise excision of tumors located on or near critical structures, such as the vocal cords, while causing minimal collateral tissue damage.
b) **Safety precautions**
i) Lasers are high-energy focused beams of light that may easily ignite combustible material, particularly in the presence of supplemental O_2.
ii) Care must be taken to eliminate any readily combustible material from the surgical field.
(1) Surgical drapes around the patient should be nonflammable and wet towels can be placed over the patient's eyes and face for added safety.
iii) Eyes are especially prone to damage. Everyone present in the operating room must wear appropriate, laser-specific eye protection.
iv) Smoke should be properly scavenged.
v) If excised lesions are thought to be from an infectious process, such as human papillomavirus, OR personnel should wear fitted masks to filter aerosolized viral particles.
c) **Airway management**
i) The airway will be shared, so close communication with the surgeon is critical.
d) **Minimizing risk of airway fire (Table 122-3 and 122-4)**
i) FiO_2 should be kept to a minimum, preferably <30%.
ii) Nitrous oxide supports combustion and should not be used.
iii) Avoid introducing any combustible material into the airway, including oil-based lubricants or dry cottonoids.
e) **Ventilation strategies for laser surgery**
i) Endotracheal intubation
(1) Laser resistant ETTs should be used.
(2) Double cuffed ETTs
(a) Provide additional protection because if the proximal cuff is punctured, the distal cuff will prevent airway gases from leaking and supporting combustion.
(b) It is beneficial to fill the cuffs with saline colored with dye (eg, methylene blue) to alert the surgeon to a cuff rupture.
ii) Intermittent apnea
(1) May be considered if ETT is not used due to the nature of the surgery and the patient can tolerate periods of apnea
(2) Intermittently ventilating by mask, SGA, or ETT and then extubating each time the surgeon works is a viable option.
(a) The surgeon will have an unimpeded field of view, and the problem of potentially combustible airway adjuncts will be avoided.

TABLE 122-3 Preventing Fires in ENT During Laser or Electrosurgery

1. Minimize FiO_2; avoid N_2O; use O_2-N_2, O_2-Air, or O_2-helium mixtures; use PEEP.
2. If using MAC, meticulous care must be taken to avoid the accumulation of oxygen within the surgical drapes or bedding. At the most, use low-flow supplemental oxygen. If higher flows are needed for patient safety, then general anesthesia should be used with an SGA or ETT to isolate airway gases from the surgical field.
3. All flammable prep solutions must be allowed time to completely dry.
4. For laser surgery, the surgeon should use lowest laser intensity necessary and short intermittent laser bursts. Use laser safe ETT. Fill ETT cuff with blue dye and keep cuff out of surgical field.
5. Avoid electrosurgery within the airway if patient requires high FiO_2.
6. Saline should be ready and available to extinguish an airway fire.

iii) Venturi technique
   (1) A small, unobtrusive catheter is positioned just above the glottic opening and short bursts of high-pressure ventilation entrain room air to achieve adequate tidal volumes.
   (2) Close observation of chest expansion with confirmation of complete exhalation is critical to avoid severe barotraumas, such as pneumothorax, pneumomediastinum, or severe gastric distention and/or rupture.

iv) Jet insufflation
   (1) Similar to Venturi technique, but catheter is placed at or below the level of the vocal cords either by transglottic or transtracheal route.
   (2) Should be avoided in patients with obstructive lung disease or severe obesity, as their passive lung deflation is impaired.
   (3) Advantages and concerns are similar to the Venturi technique but with higher risk of barotrauma.[6]

v) Manual ventilation through side port of bronchoscope
   (1) Often difficult to achieve effective ventilation secondary to the bronchoscope impeding airflow but may be acceptable if procedure is of short duration.

vi) THRIVE: Is being used at several centers during laser laryngoscopic surgery. Despite the high FiO_2, the risk of fire is considered low due to the lack of fuel as the ETT is absent. However, great care must be taken to ensure that all combustible materials such as gauze are removed from airway prior to using laser.[6,9]

9) **Transoral robotic surgery (TORS)**[7]
   a) Indications
      i) Minimally invasive resection of cancers of the oropharynx such as tonsillar cancers and base of tongue tumors
   b) Preoperative considerations



Every anesthesia provider should know the location of the OR's fire extinguisher.



All unprotected nonmetal tubes are prone to laser-induced ignition.



High-flow nasal cannula is a new method for delivering high FiO_2 without instrumenting the airway. It may be helpful in cases such as tracheostomy, emergent surgical airway, and panendoscopy.

TABLE 122-4 Treating an Airway Fire[6]

1. Stop all gas flows. This is most easily accomplished by disconnecting the inspiratory and expiratory limbs of the circuit from the anesthesia machine.
2. Immediately remove burning material.
3. Flush the airway with saline.
4. Reestablish airway and ventilation.
5. Perform bronchoscopy to assess injury; consider lavage.
6. Supportive care: mechanical ventilation, PEEP, corticosteroids, CXR, ABG, reassessment later if extent of injury unclear. Consider low tracheostomy.

From Baker PA , O'Sullivan EP , Kristensen MS , Lockey D. The great airway debate: is the scalpel mightier than the cannula? *Br J Anaesth*. 2016;117(suppl_1):i17-i19. Copyright © The Author 2016. Reproduced by permission of Oxford University Press.

i) Patients may have associated comorbidities as described at the beginning of this chapter.

c) Intraoperative considerations
   i) A nasal intubation, preferably in the nare contralateral to the tumor, provides the best exposure for the surgeon.
   ii) Profound paralysis provides optimal surgical conditions.
   iii) Neck dissection for assessment and treatment of metastatic spread is often done as part of this procedure.

d) Postoperative considerations
   i) The external carotid artery is often ligated to reduce the risk of postoperative bleeding.
   ii) Postoperative pain is often significant, and a multimodal approach to pain management is recommended.

CHAPTER SUMMARY FOR ENT AND LASER SURGERY

Patient Assessment	Emphasis on thorough airway exam and consideration of coexisting disease.
Airway Management	Coordinate plan with surgeon, maintain spontaneous ventilation, or secure the airway with the patient awake if necessary.
Fire Prevention	Use caution with laser or electrosurgery within the airway, avoid N_2O, minimize FiO_2, and have a plan to quickly extinguish any fire.
Laser Surgery	Pay meticulous attention to protecting the patient and OR personnel from thermal injury.
Emergence	Strive to avoid coughing, mask ventilation, or loss of the airway on patient awakening.
Postoperative Concerns	Avoid PONV, airway edema, oversedation, or hypertension to help minimize bleeding or airway compromise.

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123 Anesthesia for Intracranial Procedures

Leslie C. Andes

Neuroanesthesia is the provision of anesthesia for intracranial and spine operations. A thorough understanding of neurophysiology, including the concepts of intracranial pressure and cerebral blood flow, is required. Neurosurgical cases can be lengthy, and preoperative planning and painstaking attention to detail intraoperatively will aid in a favorable anesthetic and surgical outcome. This section gives a brief overview of neurophysiology and includes common intracranial neurosurgical procedures and perioperative issues to be considered. For a discussion of anesthesia for spinal surgery see Chapter 121.

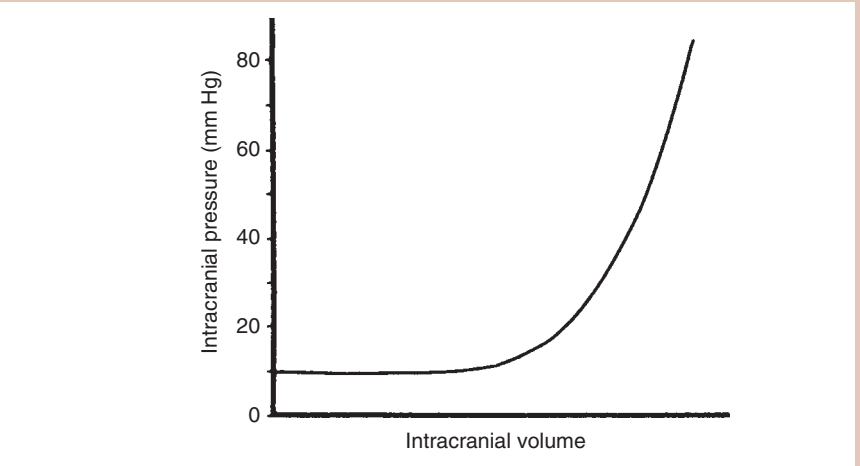
1) **Intracranial physiology and mechanics**
   a) **Intracranial constituents**
      i) Brain parenchyma
      ii) Cerebrospinal fluid (CSF)
         (1) Nourishes and cushions the brain in the cranial vault
         (2) Circulates from the ventricles through the subarachnoid space around the brain and spinal cord and is passively absorbed into the venous circulation primarily via the arachnoid granulations
      iii) Blood
   b) **Intracranial volume**
      i) Considered to be fixed and incompressible (the "Monro–Kellie Doctrine").
      ii) If the volume of one constituent increases, the volume of at least one other must decrease for intracranial pressure (ICP) to remain normal.
   c) **Intracranial pressure (ICP)**
      i) ICP is the pressure exerted throughout the cranium by the tissues and fluids inside the skull.
         (1) Normal ICP is 5-15 mm Hg in healthy supine adults and 3-7 mm Hg in children.
         (2) ICP is normally tightly regulated but can become elevated in many pathological conditions.
         (3) Increased ICP, also called intracranial hypertension (ICH), can require emergency treatment if severe, due to potential compressive effects on tissue and restriction of cerebral blood flow (CBF).



Due to the high turnover of CSF, obstruction of CSF flow may cause ICH within a few hours, necessitating emergency treatment.

      ii) Adjustable components of ICP
         (1) CSF production and drainage, as well as redistribution with the brain and spinal cord
         (2) Blood flow in the brain and spinal cord
      iii) Intracranial hypertension (ICH)
         (1) When growth of an intracranial lesion is slow, ICP will remain stable for a while because of shunting of CSF into spinal reservoirs and blood into the central vascular system.

FIGURE 123-1 Idealized ICP/Volume Curve



Reprinted with permission from Kass IS. Physiology and metabolism of the brain and spinal cord. In: Newfield P, Cottrell JE, eds. *Handbook of Neuroanesthesia*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2012:18, Figure 1-9.

(2) When an increase in volume can no longer be accommodated, ICP will increase rapidly with any small change in volume of any of the three components (**Fig. 123-1**).
(3) ICP above 20 mm Hg will reduce cerebral perfusion pressure (CPP) (CPP = mean arterial pressure [MAP] − ICP), and must be treated.
  (a) When local tissue pressure exceeds perfusion pressure of the arterioles, the resultant ischemia can cause endothelial injury and increased transudation across capillary membranes, leading to cerebral edema.
  (b) If ICH remains untreated, CBF will be low due to maximal arterial constriction, and death will quickly ensue due to limited CBF and/or herniation.
(4) Conditions in which ICH may occur include
  (a) Parenchymal volume increase due to hemorrhage, tumor, traumatic brain injury (TBI), edema following ischemic events, encephalopathies, and idiopathic ICH
  (b) Hydrocephalus due to absolute or relative excess of CSF. Causes include
    (i) Increased CSF production (meningeal disease, arachnoid cyst)
    (ii) Obstruction to CSF flow (Arnold–Chiari malformation, mass effect from tumors, blood in the ventricles from subarachnoid hemorrhage [SAH])
    (iii) Normal pressure hydrocephalus
      1. Characterized by near-normal ICP that slowly increases due to gradually worsening obstruction of flow
      2. Patients are typically older than 60 and develop a triad of dementia, gait problems, and incontinence.
  (c) Increased cerebral blood volume
    (i) Cerebral arterial dilation (hypoxemia, arterial hypertension [HTN], volatile anesthetic administration, hypoventilation with resultant hypercapnia)
    (ii) Increased venous pressure (venous sinus thrombosis, right sided heart failure, or obstruction of jugular or great thoracic veins)
  (d) CSF leak

(i) May occur from several sites, including the nose (rhinorrhea), external auditory canal (otorrhea), or traumatic or operative defects in the skull or spine

ii) Causes

1. CSF rhinorrhea may follow trauma and skull base surgery (eg, transsphenoidal resection of pituitary tumor, translabyrinthine acoustic neuroma resection, mastoid surgery).
2. Otorrhea can be seen in perforated tympanic membrane with fracture of the petrous or temporal bone, and mastoid or translabyrinthine surgery.
3. Spinal CSF leak may be seen in trauma, lumbar puncture (LP), inadvertent "wet tap" during epidural placement, Valsalva or nose-blowing, or postoperatively.
4. Spontaneous CSF leak
   a. Rare and may be difficult to diagnose and treat.
   b. Often the best closure success is with endoscopic repair.



Signs and symptoms of increased ICP are diminished consciousness, nausea/vomiting, visual problems, and headache.

(5) Diagnosis of ICH

(a) Symptoms may be subtle, and include decreased level of consciousness, visual problems, headache, nausea and vomiting, personality changes, memory loss, ataxia, and weakness.

(b) Signs include papilledema, neurologic deficit, and unstable vital signs (hyper- or hypotension, arrhythmias).

(c) If patient is pregnant, preeclampsia should be ruled out.

(d) Diagnostic imaging

(i) May be normal or show enlarged ventricles and/or lateral shift.

(ii) Evaluate scan for cerebral vascular accident (CVA), mass effect from tumor or hemorrhage, as well as signs of impending herniation.



Lumbar puncture, used to diagnose various neurologic conditions, may induce herniation and should not be performed if ICH is suspected.

iv) Monitoring and measurement of ICP

(1) Assists in determining optimal therapy and may prevent herniation or worsening of brain injury

(2) Types of monitoring devices:

(a) Fiberoptic microsensor devices (aka "bolts") that are placed in the parenchyma, ventricle, or subdural space only measure pressure.

(b) Catheter devices inserted into a ventricle (external ventricular drain [EVD] or lumbar drain [LD]) will also allow for drainage of CSF in addition to measuring pressure. These can be placed pre- or intraoperatively when anticipated that brain relaxation could be needed during the case, or if potential for postoperative CSF leak exists (see above).

(c) Devices which combine a microsensor with a catheter

(3) Measurement of ICP

(a) ICP is measured relative to a reference level, above which positive ICPs will result in CSF flow if the system is attached to a drain and open.

(b) Usually the catheter is zeroed at the level of the external auditory meatus, then the top of the reservoir is raised to a height above the ear that equals the acceptable ICP; CSF will flow into the reservoir bag once the ICP exceeds this level.

(i) Care must be used in determining the height at which to hang the reservoir bag as it is possible to over-drain CSF if the bag is too low,



An external ventricular drain should be zeroed at the level of the external auditory meatus.

with potential complications such as subdural hematoma (SDH), intracranial hypotension, rupture or rebleed of an aneurysm, or even herniation.

(ii) Volume-limited (20-30 mL) CSF drainage systems are available and often used for LD.

(iii) Removal of 3-5 mL from an EVD or LD can be quickly accomplished and may be sufficient to treat acute ICH.

iv) The EVD is generally closed during transport.



Dextrose-containing fluids should not be administered to patients with ICH because once the dextrose is metabolized, the free water which remains can worsen brain edema.

v) Medical management of ICH

(1) Elevation of the patient's head up to 30 degrees is rapidly effective.

(2) Ensure there is no obstruction to venous drainage from the head.

(3) Hyperventilate patient to $ETCO_2$ of 25-30.

(a) $ETCO_2$ will return to normal via bicarbonate buffering after 4-8 hours of hyperventilation.

(b) Overventilation to $ETCO_2 < 25$ may cause ischemia secondary to severe vasoconstriction.

(4) Control BP to maintain systolic BP 110 > MAP > 70.

(5) Treat hypoxia.

(6) IV fluids should be isotonic or hypertonic to avoid further increase in brain water.

(7) Osmotic or loop diuretics

(a) Mannitol is currently the first-line agent at a dose of 0.25-1 g/kg. Lasix may be given next if mannitol is not as effective as expected.

(b) Hypertonic saline (HS)

(i) HS expands plasma volume and may increase CPP, unlike mannitol which can decrease intravascular volume.

(ii) Initial dose is 30 mL of 23.4% HS via central venous catheter over 15-20 minutes, after which 3% HS may be infused at 30-50 mL/h to keep serum Na^+ at 140-145 (checked with osmolality at least every 4 hours).



Patients with intracranial hypertension should receive little or no preoperative sedation.

(8) Prevent shivering, muscle movements, pain, and seizures with narcotics, paralytics, antiepileptics, sedatives, or barbiturates as necessary, once appropriate ventilation is assured.

(9) Patients in whom ICP therapy is ongoing may need CVP and arterial line to optimize volume status and CPP.

vi) Surgical treatment of ICH

(1) Preoperative planning

(a) Patients with ICH who are awake and responsive should get little or no preoperative sedation as hypoventilation can further increase ICP, possibly causing herniation.

(b) ABG after induction may be helpful in determining the best ventilatory parameters in patients where significant end-tidal to arterial CO_2 gradient may exist.



Extreme care should be taken when intubating a patient with ICH because increased BP on laryngoscopy can cause herniation.

(2) Intraoperative technique

(a) Voluntary hyperventilation while awake followed by early mask hyperventilation during anesthetic induction may minimize changes in ICP or CPP during induction and intubation.

(b) Assurance of anesthetic depth before intubation is important.

(c) Anesthetic agents

(i) Propofol and etomidate decrease ICP.

(ii) Any effects of volatile anesthetic agents to increase ICP can be overcome by mild hyperventilation.
(iii) The effects of N_2O on ICP vary with concentration.
1. Increase in ICP has been shown in humans but is largely attenuated with prior hyperventilation or administration of induction agents, which cause decreased ICP.
2. N_2O should not be used when there is a chance of intracranial air. Most anesthesiologists do not use it at all for intracranial procedures.
(iv) Narcotics have no direct effect on ICP but may cause hypoventilation leading to ICH.
(v) If the dura looks "tight" upon turning the bone flap, additional doses of propofol may be administered to reduce ICP and prevent the brain from herniating through the dural incision, and other measures listed above may be added (eg, head up, hyperventilation).

(3) Surgical procedures
(a) Shunt procedures
(i) Shunts are silastic catheters used to drain CSF from an area of high pressure (ventricles or cistern) to one of lower pressure.
(ii) Used for urgent but nonemergent increase in ICP due to obstruction of CSF flow (patient symptomatic but herniation not imminent)
(iii) The proximal end is most often placed in the right lateral (intracranial) ventricle.
(iv) The distal end is tunneled under the skin to one of several locations (the peritoneal cavity, right atrium via the internal jugular vein, pleural cavity, gall bladder).
1. Venous placement of the distal end is most often used if the patient is obese, has peritonitis, or previous abdominal surgery.
2. The hypertensive response frequently seen with skin tunneling of the catheter should be forestalled by increasing anesthetic depth (narcotics, propofol, sevoflurane) or β-adrenergic antagonists.



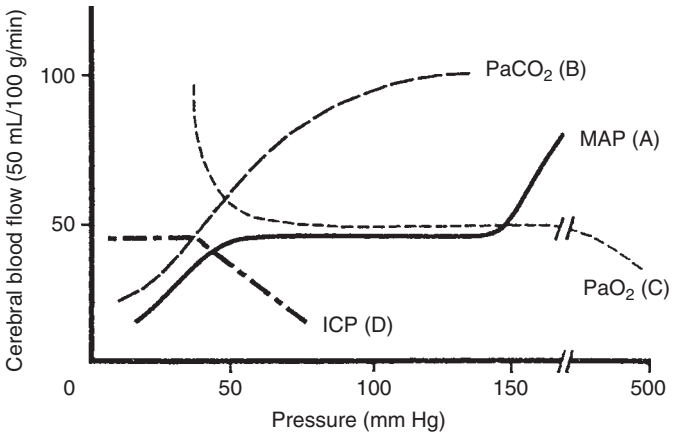
VP shunts have a fairly high failure rate over time and are frequently revised, or may need to be removed and subsequently replaced if infection develops.

(v) If there is free communication between intracranial and spinal CSF, an LP shunt (from lumbar subarachnoid space into peritoneum) may be placed.
(vi) For shunt removal due to infection, hold antibiotics until cultures are taken.
(vii) No special intraoperative monitoring is necessary for a shunt procedure.
(b) Minimally invasive neuroendoscopic surgery for treatment of CSF obstruction
(i) Used effectively to treat many types of obstructive hydrocephalus and can be lower risk than craniotomy or shunt procedures due to very small incision and shorter operative times
(ii) Aqueductoplasty and aqueductal stenting are examples of other therapeutic options afforded with the endoscope.
(c) Emergency craniotomy for refractory ICH
(i) Decompressive craniectomy (DC) solely for ICH is controversial and is more commonly used to treat large volume intracranial hemorrhage.
(ii) Rarely, surgical resection of brain tissue may be necessary.
(iii) If ICP remains elevated at the end of a craniotomy (surgeon unable to put on bone flap without compressing brain tissue), the bone may be left off, frozen, and replaced at a later date.

(4) Postoperative care
  (a) Maintain slight head up position.
  (b) If an ICP monitor or EVD is in place, watch the location and level of the bag and ICP monitor as the patient is transferred to a bed and transported, and take strict care to ensure the catheter is not pulled out.
  (c) Carry sedative agent, vasoconstrictors, and vasodilators during transport in case of urgent need to keep patient sedated or manipulate BP, especially if transporting for a postoperative scan before returning to the ICU.

d) **Cerebral perfusion pressure (CPP) and cerebral blood flow (CBF)**
  i) CPP is the net perfusion pressure causing blood to flow to the brain (CBF). This can be expressed as: CPP = MAP − ICP (or central venous pressure [CVP], whichever is greater)
  ii) Autoregulation of CBF (**Fig. 123-2**)
    (1) Autoregulation is the ability of the cerebral vasculature to maintain relatively constant CBF despite large changes in blood pressure.
    (2) Effected via dilation or constriction of the cerebral blood vessels, with some matching (or "coupling") of flow to metabolism of glucose and cerebral metabolism of oxygen ($CMRO_2$)
    (3) Range for autoregulation
      (a) Historically thought to be between 50 and 150 mm Hg
      (b) Recent expert opinion is that the MAP for the lower limit of autoregulation (LLA) may be ≥70 mm Hg, but much more research needs to be done. In the meantime, the recommendation is to be conservative.[1]
    (4) Once the limits of autoregulation are exceeded, flow is dependent on perfusion pressure.

FIGURE 123-2 Pressure-CBF Relationships. **A.** The traditional view of CBF autoregulation between MAP 50 and 150 mm Hg. The LLA is more likely ≥70 mm Hg. **B.** Linear relationship between partial pressure of arterial carbon dioxide ($PaCO_2$) and CBF for $PaCO_2$ = 20-80 mm Hg. **C.** PaO_2 and CBF. **D.** ICP and CBF.



Adapted with permission from Newfield P, Cottrell JE, eds. *Handbook of Neuroanesthesia*. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2007:113.

(a) Areas of the brain that are chronically underperfused may be maximally vasodilated, and blood flow will be dependent on MAP.
(b) Acceptable intra- and postoperative BP must be individualized for each patient based on their usual BP readings.[1]

(5) Cerebral autoregulation can be impaired by the following:
(a) Vascular disease
(b) Anesthesia
(c) Tumors
(d) Disruption in the blood-brain barrier (BBB)
(e) Medications
(f) Cerebral edema
(g) Trauma

iii) CBF is directly proportional to $PaCO_2$ between 20 and 80 mm Hg, and tightly coupled regionally (**Fig. 123-2B**).
(1) Blood vessels in ischemic regions do not react normally to changes in CO_2 levels.
(2) Hypoventilation with hypercarbia may result in a steal phenomenon with increased CBF to normal areas.
(3) Hyperventilation may result in an inverse steal effect with improved CBF to damaged brain areas.
(4) Changes in CBF due to changes in $PaCO_2$ called "cerebrovascular reactivity" ($CVR\text{-}CO_2$); a recent review of the literature shows that in usual clinical anesthetic doses, $CVR\text{-}CO_2$ is maintained with propofol as well as inhalational agents.[2]



The cerebral autoregulation curve is shifted to the right in patients with chronic HTN, and these patients require higher MAP to maintain CBF.

iv) CBF is inversely affected by PaO_2 (**Fig. 123-2C**).
v) CaO_2 and viscosity changes caused by changes in hematocrit will also affect CBF.
vi) Phenylephrine in normal clinically used doses will not cause cerebral vasoconstriction or changes in CBF.[1]
vii) Cerebral metabolism of oxygen ($CMRO_2$)
(1) The cerebral metabolic rate for oxygen ($CMRO_2$) is the brain's oxygen consumption requirement to maintain normal function and viability.
(2) Normal coupling between $CMRO_2$ and CBF is maintained under anesthesia.
(3) A decrease in $CMRO_2$ is the basis for most current neuroprotection therapies. EEG burst suppression can decrease $CMRO_2$ by up to 60% from baseline, which is the maximum achievable as the remaining 40% is used for cellular maintenance. Propofol is the current preferred agent.

e) **Blood-brain barrier (BBB)**
i) A cellular barrier between the vascular system and brain tissue that protects the CNS.
ii) Tight junctions in capillary endothelial cells prevent passage of large, water-soluble, or charged substances between the blood and brain cells.
iii) May be disrupted by inflammation and oxidative stress in multiple conditions: HTN, drugs, TBI, and disease (eg, tumors, encephalopathies, meningitis, ALS, MS, stroke, epilepsy)[3]



The goal of anesthesia for neurosurgical procedures is maintenance of the normal balance between ICP, CPP, and $CMRO_2$.

(1) Vasogenic cerebral edema
(a) Results from disruption of the BBB which causes protein leakage into extracellular areas
(b) Seen with tumors

(c) Responds to steroids (which may improve BBB integrity) and diuretics

(2) Cytotoxic edema

(a) BBB is not disrupted, so there is no protein leak; intracellular

(b) Not improved with steroid use

(3) Ischemic edema

(a) Blend of vasogenic and cytotoxic

(b) May have a delayed onset

iv) Much exciting research is being done with regard to BBB breakdown and repair.[4]

2) **Preoperative planning: intracranial and neurovascular procedures**

a) **Patient assessment**

i) A discussion with the surgeon is recommended to assess neurosurgical issues impacting planned anesthetic.

(1) Films should be reviewed for signs of increased ICP, compression or invasion of other structures. Examples include

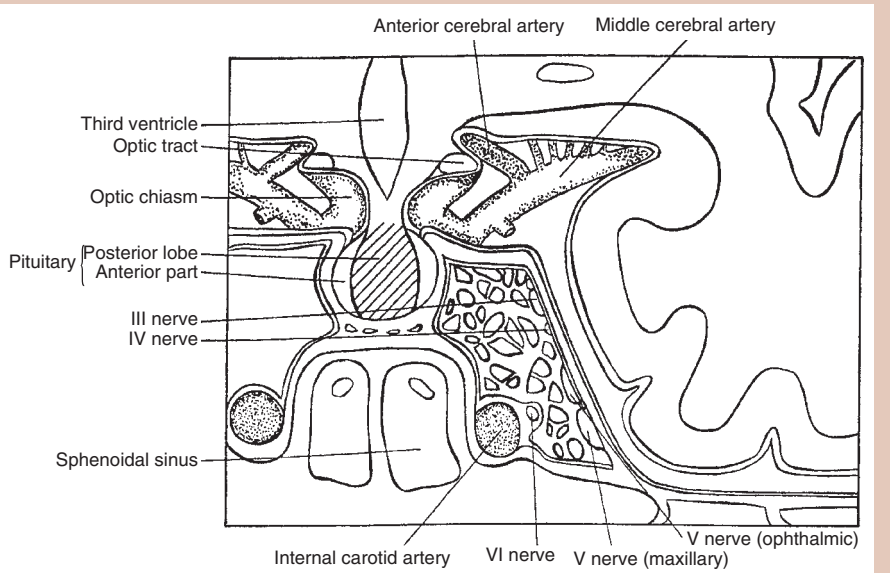
(a) Compression of brain stem by tumor (eg, large acoustic neuroma, clival mass) or vascular malformation

(b) Large pituitary tumor invading the cavernous sinus, with proximity to the carotid artery (**Fig. 123-3**)

(2) Underlying structures that may be entered with the chosen surgical approach, especially venous sinuses.

(3) Positioning for best surgical exposure should be discussed with the surgeon. Optimal positioning will place the plane the surgeon wishes to work in parallel to the floor.

FIGURE 123-3 Structures Within or Near the Cavernous Sinus



Reproduced with permission from Newfield P, Cottrell JE, eds. *Handbook of Neuroanesthesia*. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2007:188.

ii) Blood and components should be readily available for surgeries where rapid and extensive blood loss may be likely, based on type and location of lesion.

iii) Preoperative airway management planning is an important aspect of neuroanesthesia.

(1) Difficult airway is common in neurosurgical patients, due to concerns such as spinal pathology, presence of halo fixation device, facial trauma, congenital deformities.

(2) A wide variety of airway devices should be available.

(3) Proceed with rapid sequence induction and intubation if full stomach is expected.

(4) Minimal or no preoperative sedation is best for a patient with increased ICP due to the possibility of inducing hypoventilation and intracranial HTN.



Location, size, and type of lesion are important determinants of positioning, monitoring, and anesthetic induction and maintenance.

iv) Preoperative discussion with patient

(1) Prepare the patient for frequent postoperative serial neurologic examinations until neurologic stability is assured.

(2) Pituitary surgery patients may be instructed to breathe through the mouth if nasal packing is placed at the conclusion of surgery.

3) **Intraoperative considerations**

a) **In addition to standard ASA monitors, special monitors may be considered for neurosurgical procedures.**

i) Intravascular monitors

(1) Arterial pressure lines are useful for continuous monitoring of BP and to obtain arterial blood for analysis.

(a) When large changes in arterial blood pressure are expected and/or undesirable

(b) May be placed preinduction for patients in whom aggressive BP control is necessary (eg, ruptured aneurysm).

(c) Insert an arterial line for all large craniotomies, or if indicated for medical condition of patient.

(d) The patient's arms will often be wrapped and unreachable during neurosurgical cases; thus, it is critical to ensure the arterial line functions reliably prior to positioning the patient.

(e) As with the ICP monitor, the arterial line should be zeroed at the level of the external auditory meatus for any position in which the patient is placed.[1]

(2) Central venous access is useful for monitoring CVP and infusing medications quickly into the central circulation. A CVP catheter is placed prior to positioning:

(a) For larger craniotomies

(b) If large volume shifts are expected

(c) If the lesion is very vascular or bleeding is expected (eg, renal cell carcinoma metastases, AVM)

(d) To improve ease of care in ICU postoperatively.

(3) Pulmonary artery catheters are currently rarely used in neurosurgery.

READ MORE
Neurophysiologic monitoring, Chapter 16
Positioning of the surgical patient, Chapter 7

ii) Neurologic monitors: Monitors for neurophysiological monitoring and awareness are covered in Chapter 16. Please refer for further reading on the topic.

iii) Neuroimaging navigation system (aka "wand guidance")

(1) Correlates the topographic anatomy of the patient's head to the preoperative MRI or CT scan
(2) Used to optimize surgical approach and help the surgeon avoid critical structures
(3) Once the neuronavigation frame is attached to the head holder and calibrated, the frame and the system should not be moved.



Correct and safe positioning of the neurosurgical patient requires preoperative consultation with the surgeon, and although the ultimate responsibility rests with the surgeon, anesthesiologists are frequently called on to assist.

iv) ICP if monitor is already *in situ*
v) A urinary output catheter is placed for any case expected to be longer than 2 hours, and in all patients with spinal cord injury.

b) **Positioning**
i) General considerations:
(1) Surgical approach will be chosen by the surgeon to maximize view while minimizing retraction of brain tissue.
(2) The patient must be protected during what could unexpectedly become a very lengthy case.
(3) If table rotation is planned, the patient may be taped to the table across the chest and thighs.
(4) The position should be as anatomically correct as possible.
(5) All pressure points should be well padded; ensure there are no restrictive elements on the patient (armbands, rings, and so on).
(6) Patient access may be limited once the case has started.
(a) The table will frequently be turned 90-180 degrees, placing the anesthesiologist at the side or foot of the bed.
(b) Secure all lines and tubes completely, and ensure all monitors are showing parameters, before draping.
(c) The endotracheal tube (ETT) must be well secured to the patient and all connections checked.



For a craniotomy, the head will be positioned so that a plane through the craniotomy will be roughly parallel to the floor.

ii) Head positioning
(1) Application of the head holder ("pins")
(a) Can cause intense adrenergic stimulation, bradycardia, or even asystole (more likely in children or patients with high vagal tone).
(b) Pre-treat the patient with narcotic, propofol, esmolol, and/or an increase in gas concentration.
(c) Pins may also be used in short cases if wand guidance is planned (eg, VP shunts, brain biopsy).
(d) The horseshoe head holder is not used in cases expected to last longer than 2 hours, due to potential effects of pressure on the scalp.



If BP control is critical, the arterial line should be placed prior to pin application.

(2) Limiting rotation of the head to 45 degrees or less is preferred. If more rotation is required, the shoulder opposite from the direction of the head turn can be "bumped up" with a sandbag, and the table can be rotated as necessary after the patient is secured to it.
(3) Disastrous consequences have been reported from overextension or overflexion of the head and/or neck with resultant inadequate arterial flow to the brain stem and spinal cord. Avoid venous outflow obstruction as well.
iii) Prone position
(1) For high posterior cervical and occipital surgeries, the head will often be in pins with arms tucked at the sides. If a Jackson table is preferred, a foam headrest and possibly Gardner-Wells tongs may be utilized.

(2) In cases where the head is quite flexed, a soft gauze bite block will ensure that the patient does not bite the tongue or occlude the ETT.
(a) Chin position should be checked and padded as needed; at least two fingerbreadths thyromental distance should be maintained.
(b) If peak inspiratory pressures increase after positioning, check for ETT obstruction; surgeon to reposition the head as necessary.

iv) Sitting position
(1) Used infrequently. Preoperative echocardiogram ruling out PFO is required due to risk of paradoxical air embolus, and if PFO is found (~25% of general population), the surgeon should decide on another position for the case.
(2) Patients with carotid stenosis may have insufficient CBF in the sitting position.
(3) The arterial pressure transducer should be placed at the level of the Circle of Willis (ear level), as it is possible for CPP to be undesirably low if BP is adjusted based on the pressure transduced at heart level.
(4) Place Doppler monitor over right second intercostal space, to alert in case of venous air embolus (VAE).

c) **Induction and maintenance**
i) Choice of anesthesia
(1) GA is used for most intracranial (IC) cases; no difference in outcome has been shown between use of volatile agents vs total intravenous anesthetic (TIVA) in intracranial cases.
(2) Awake (or intermittently awake) anesthetics may be used for seizure focus resection or tumors where the lesion is in an eloquent area, as well as in thermal rhizotomies to facilitate placement of the probe.

4) **Anesthetic agents and other medications**
a) **Induction agents**
i) Intravenous
(1) Propofol (1-2 mg/kg) will decrease CBF (via vasoconstriction) and ICP and preserve autoregulation.
(2) Etomidate (0.1-0.4 mg/kg) decreases CBF and $CMRO_2$ and may be a reasonable induction agent for patients with unstable cardiovascular status.
(3) Ketamine, long thought to increase ICP, is still generally not used in IC surgery although has been shown to be safe under most conditions.
ii) Inhalation agents are rarely used for induction due to potential increase in CBF and ICP.
iii) Neuromuscular blockade (NMB)
(1) Usually accomplished with an intubating dose of rocuronium (0.5-0.6 mg/kg), vecuronium (0.1-0.2 mg/kg), or cisatracurium (0.15-0.20 mg/kg)
(2) Succinylcholine may transiently increase ICP; it is contraindicated in some chronic neuromuscular and CNS diseases and in trauma with crush injury which has occurred more than 24 hours earlier.
(3) For most neurosurgeries, repeat dosing of NMB is not necessary (even if the patient is in pins) after intubation as long as anesthetic depth is maintained; they especially should not be redosed in cases where electromyography (EMGs) or MEPs are monitored. Sugammadex administration (1-2 mg/kg) is helpful in obtaining EMGs or MEPs before final positioning.

b) **Maintenance**
i) Intravenous agents
(1) TIVA with propofol is popular for cases requiring MEP monitoring; the use of a volatile agent at $\leq$1 MAC with a small amount of narcotic may be as effective.
(2) Narcotics do not have clinically important direct effects on $CMRO_2$, CBF, or ICP.

(a) Fentanyl, sufentanil, remifentanil, morphine, meperidine, and hydromorphone are all acceptable choices in neurosurgery.
(b) Ventilation must be adequate to prevent hypercarbia.

(3) Dexmedetomidine, an α_2 agonist, can be useful in BP control and may decrease the dose of narcotics required.

ii) Inhalation agents

(1) Volatile agents

(a) At >1 MAC may increase CBF due to vasodilation. In patients where CBF is already compromised, even small doses of volatile agents may cause a further decrease in CBF.
(b) Sevoflurane is least problematic in terms of effects on CBF; isoflurane would be the next best choice.
(c) Desflurane may increase CBF and cause loss of autoregulation, and can also increase ICP through increased CSF production.

(2) Nitrous oxide (N_2O)

(a) Use of N_2O in intracranial neurosurgery is controversial as it can increase CPP.
(b) However, studies show no difference in outcomes with use of N_2O in aneurysm surgery in patients with SAH.
(c) The increase in CBF from N_2O can usually be overcome by hyperventilation.
(d) N_2O will interfere with MEPs and should not be used in cases where MEPs are monitored.
(e) N_2O will increase volume of an intracranial air space and if used it may be desirable to turn it off near the end of the case before the dura is closed.
(f) ETT cuff pressures may increase during a long case if N_2O is used.



Chronic therapy with medications that induce hepatic enzyme production (eg, antiepileptics) may necessitate increased dose of anesthetic agents.

c) **Adjuvant agents include the following:**

i) Mannitol 0.25-1 mg/kg
ii) Dexamethasone 4-10 mg if cerebral edema
iii) Vasoactive agents

(1) Phenylephrine is the vasopressor of choice in neurosurgical cases; it increases MAP and may cause reflex decrease in HR. It is often used during neuroradiologic procedures when anesthesia must be deep enough that the patient remains immobile, but the surgical stimulation is not intense.



Phenylephrine is the vasopressor of choice in neuro-surgical cases; it increases MAP and may cause reflex decrease in HR.

(2) Norepinephrine use as a vasopressor has increased recently, especially in longer cases (where tachyphylaxis sometimes develops to phenylephrine) or those where the inotropic effects may be desirable.
(3) β-Adrenergic antagonists or nicardipine are the most frequently used agents to decrease MAP intra- or postoperatively for neurosurgical patients.
(4) Sodium nitroprusside causes vasodilation and impairs autoregulation and currently is rarely used.
(5) Adenosine is occasionally administered IV in aneurysm surgeries to facilitate permanent clip placement if temporary clip placement is not practical in large or difficult-to-isolate aneurysms. By causing short-duration hypotension and possibly brief asystole, adenosine acts as a "pharmacologic temporary clip."

(a) If use is planned, consider application of defibrillator pads and an additional set of ECG pads to the patient before draping.
(b) Dose recommended is 0.2-0.4 mg/kg ideal body weight to achieve 45-60 seconds of profound hypotension. Most common initial dose is 12-18 mg

followed by up to two more doses, but escalating doses may be needed during the procedure if permanent clip placement is difficult. Total doses >700 mg have been reported with no sequelae.[5]

iv) Antibiotics
v) Anticonvulsants (phenytoin or fosphenytoin, levetiracetam)
vi) Desmopressin and vasopressin may be given for their hormonal effects; be aware they can cause changes in blood pressure depending on dose and site of administration.



In neurosurgical procedures, the fluid management goal is euvolemia (normal CVP), while maintaining normal glucose and electrolyte levels.

5) **Fluid and electrolyte management**
   a) **The goal is euvolemia (normal CVP), while maintaining normal glucose and electrolyte levels.**
   b) **Management of intraoperative glucose levels**
      i) Avoid dextrose-containing fluids: hyperglycemia worsens outcome in both global and focal ischemia.
      ii) Current practice is to treat glucose levels >150-200; best intraoperative control may be obtained with use of insulin infusion protocols.
   c) **Large amounts of IV saline can cause hyperchloremic acidosis; use isotonic fluids if available and limit fluids to necessarssy amount**
   d) **Check electrolyte and glucose values as appropriate for the case**



Avoid dextrose containing fluids in neurosurgical procedures because hyperglycemia worsens outcome for global and focal ischemia.

6) **Cerebral protection**
   a) Neuroprotection entails the use of certain pharmacologic agents or hypothermia to protect the CNS from injury during periods of low blood flow, via decrease in $CMRO_2$.
   b) The maximum achievable decrease in $CMRO_2$ has occurred when burst suppression is seen on EEG; however, an isoelectric EEG may not be required for cerebral protection.[6]
   c) During a focal ischemic event, propofol may be used to decrease $CMRO_2$ and afford a measure of neuroprotection, although it has not been proven to be effective in global ischemia at clinically useful doses.
   d) Other agents may also decrease $CMRO_2$ but should not be assumed to be neuroprotective.
   e) Volatile agents also decrease cerebral metabolism until the EEG becomes isoelectric, but the dose required to do so (>1.5-2 MAC) may increase CBF and interfere with SSEPs.
   f) **Moderate hypothermia neuroprotective in global ischemia**
      i) If used during neurovascular cases, the patient may be rewarmed once the lesion has been treated.
      ii) Though hypothermia decreases $CMRO_2$, mild intraoperative hypothermia (33°C) does not improve outcome during hospitalization or at 3 months postoperatively in favorable-grade (WFNS score I, II, or III) SAH patients.
   g) **Intraoperatively, begin EEG burst suppression therapy once the risk of an ischemic event is present.**
      i) During aneurysm surgery, this is usually when the aneurysm is visualized but before the surgeon is closely dissecting. If an aneurysm has ruptured preoperatively, a neuroprotective agent may be started on induction.
      ii) Arteriovenous malformations (AVMs) are often larger and more complicated lesions, requiring higher total dose due to length of case.
      iii) Propofol is most commonly used; dose at 25-100 µg/kg/min. Volatile agents can be decreased accordingly during this time.
      iv) Once the lesion has been resected or treated, burst suppression can be discontinued if ICP is not grossly elevated.

v) The relatively short action of propofol means that most patients can be extubated at the end of the case if indicated.

7) **ICP management**
   a) Can be critical in the perioperative period.
   b) See preceeding section, where this is covered extensively.
8) **High venous pressures**
   a) **May cause excessive intraoperative venous bleeding and obscure surgical field**
   b) **Treatment**
      i) Ensure no venous compression or restriction of venous drainage in the neck.
      ii) Raise head of bed up to 30 degrees.
      iii) Change respiratory parameters (decrease tidal volume, increase respiratory rate).
      iv) Low-dose venodilators (NTG, morphine) can be administered if increased CBF is not a concern, or if deliberate hypotension may be useful.
      v) Redose neuromuscular blocking agents.
9) **Emergence**
   a) Most desirable would be to have the patient awake at end of case; wakeup must be smooth to avoid increase in ICP or CBF.
   b) Extubation under deep anesthesia, when appropriate, may help prevent coughing.
   c) Nausea and vomiting are especially common after posterior fossa surgeries and can be mitigated with a judicious dose of an antiemetic prior to emergence.
   d) **If the ability to maintain an airway could be compromised, it may be prudent to keep the patient intubated until awake. Examples include**
      i) Patient has not regained consciousness after larger doses of neuroprotective agent.
      ii) Posterior fossa surgeries where CNs IX, X, and XII might be injured or edematous.
      iii) Cases in which brain stem swelling might be expected (tumor, hemorrhage, long delicate surgery).
      iv) Lengthy prone cases where dependent edema of the airway is possible.
   e) Watch for BP spikes during emergence and extubation. Treat promptly or expectantly.
   f) If a postoperative MRI is planned, remove all metal from the patient (SaO_2 probe, esophageal stethoscope, wires from neurophysiologic monitoring), and replace NIM-EMG ETT with plain ETT.
10) **Postoperative care**
   a) **Frequent serial neurologic examinations should be performed; any postoperative decrement in the examination should prompt immediate investigation as to cause.**
      i) Assess level of consciousness (LOC) and mental status.
      ii) Check pupillary response.
      iii) Perform basic cranial nerve function.
         (1) Eyes follow finger
         (2) Smile
         (3) Gag
         (4) Tongue function
      iv) Flexion/extension of all four extremities
   b) Continue meticulous BP control (systolic <160) so that recently coagulated blood vessels on and within the brain do not bleed. Keep BP even lower (systolic <100-120 mm Hg) in AVM patients.
   c) Intubated patients may be returned directly to the ICU.
   d) Carry vasoactive agents and/or sedation (propofol) during transport in case of hypo- or hypertension.



It is usually desirable for the patient to be awake upon completion of surgery in order to assess neurologic status.



Angioplasty with adjuvant intra-arterial nimodipine, and hypertensive therapy are currently the most medically effective treatments for cerebral vasospasm.[8]

11) **Potential complications**
   a) **Vasospasm**
      i) Diagnosed on cerebral digital subtraction and CT angiography, as routine postop follow-up or if patient becomes symptomatic (change in neuro examination). Transcranial Doppler may also be useful.
      ii) Most likely a multifactorial problem that will require more than one therapeutic option. Research into involvement of inflammatory pathways, immunosuppressant therapy, and hyperdynamic therapy (milrinone) is ongoing.
      iii) Current best therapies are hypertensive medications, systemic or direct intra-arterial administration of calcium channel blockers (injected by the surgeon, or via radiologic catheter), and angioplasty.[7]
      iv) Triple H therapy (hypertension, hypervolemia, and hemodilution) has fallen out of favor due to systemic problems that can be caused by hypervolemia and hemodilution; however, induced hypertension is still considered to be effective in increasing CBF.
   b) **Bleeding**
      i) Because the posterior fossa is a small space, even a small amount of bleeding can cause serious problems, including compression and herniation.
      ii) Watch for somnolence, decreased CN signs such as loss of gag reflex, and altered respiratory patterns.
      iii) Intraop bleeding from an AVM can be extremely difficult to control, and extensive blood loss can occur in a matter of minutes.
   c) **Venous air embolus (VAE)**
      i) Sitting craniotomies and any other case where the operative site is higher than the right atrium carry a clinically significant risk of VAE. Preoperative echocardiogram ruling out PFO is required for sitting position cases.
      ii) During cases with higher risk of VAE
         (1) Intraoperative monitoring should include precordial Doppler, as well as multiorifice CVC; transesophageal echo (TEE) may be used if available.
         (2) Sensitivity for detection of VAE: TEE > Doppler > PAP and $ETCO_2$ > CVC > BP > ECG
         (3) Be alert for VAE particularly during turning of flap and bone work, when entry into venous sinuses is most likely.
         (4) It is preferable to use air instead of N_2O during the case.
      iii) Signs and symptoms
         (1) Consider VAE if hypotension is accompanied by decrease in $ETCO_2$; $ETCO_2$ does not need to decrease to zero to be significant.
         (2) Rapid entrainment of 100-300 mL of air may be quickly lethal due to right cardiac inflow/outflow obstruction (similar to an "air lock" in IV lines). Pulmonary HTN and right-sided heart failure can also develop if a longer slower process of entrainment is ongoing.
      iv) Treatment
         (1) Immediate treatment of VAE
            (a) Notify surgeons.
            (b) Call for help.
            (c) Surgeons flood operative field with saline.
            (d) Ensure 100% O_2 with controlled ventilation.
            (e) Attempt to aspirate air from CVP.
            (f) Change patient's position, putting operative site lower than right atrium.
            (g) Consider jugular venous compression.
            (h) Support blood pressure.
            (i) Prepare for cardiac arrest.

(2) Rapid administration of fluids through the central line to increase CVP.
   (a) May help prevent further air entry and force the air into the pulmonary circulation where it can be absorbed.
   (b) Must be weighed against the potential increased risk of paradoxical (arterial) air embolism and the possibility of worsening pulmonary HTN.

d) **Syndrome of Inappropriate ADH (SIADH)**
   i) Seen with many malignancies as well as intracranial processes.
   ii) Be suspicious if urine output (UOP) is low relative to IV fluids given, $Na^+ < 124$, serum Osm < 280, and high urine Osm.
   iii) Treat with restriction of fluids, administration of furosemide, and possibly HS if severe.

e) **Diabetes insipidus**
   i) Rarely seen intraoperatively but may develop during surgeries of the pituitary or hypothalamus.
   ii) Large quantity of dilute urine (<200 mOsm, SG 1.001-1.005) with normal or elevated serum Osm and Na^+
   iii) Treat with vigorous rehydration with 0.5 NS to match UOP, and 5-10 units vasopressin or 1-4 μg desmopressin IV.

f) **Neurogenic pulmonary edema (NPE)**
   i) Probably due to massive sympathetic activation caused by acute and extreme spike in ICP[8]
   ii) Not well understood and likely underreported but can be seen in severe TBI, SAH, intraparenchymal hemorrhage (IPH), status epilepticus, as well as many other conditions
   iii) May be acute or delayed onset after initial injury and often resolves quickly though may have relapsing course.
   iv) Treatment is generally symptomatic; α blockade has worked in animals and some human patients. Ruling out cardiogenic pulmonary edema may be helpful.
   v) Frequently accompanied by pulmonary hemorrhage, probably due to increased pulmonary pressures during the sympathetic release.

g) **Pneumocephalus**
   i) Most common in frontal craniotomy but can be seen in any position where most of head is above surgical site
   ii) Can be seen even without N_2O use
   iii) Should be ruled out if unexplained delayed awakening
   iv) Can be seen on XR or CT

h) **Postoperative respiratory failure**
   i) Long posterior fossa surgeries
   ii) Surgeries where CN IX & X involved or are near the lesion
   iii) Consider leaving intubated: older patients with CAD, decreased LOC, long surgeries, posterior fossa surgery

i) **Eye injury**
   i) Known risk with any surgery on the head and neck
   ii) Initial discussion of anesthetic risks may include the potential for postoperative ischemic optic neuropathy especially in patients who will be placed prone.
      (1) Risk highest in patients who have substantial blood loss and/or undergo prolonged procedures (>6.5 hours)
      (2) Patients who are diabetic, obese, hypertensive, and smoke may have an incidence as high as 1%.
      (3) Avoid pressure on the globe, increased venous pressure (a slight head-up position may assist), and overhydration.

iii) Ophthalmic ointment may protect against corneal abrasions.

12) **Intraoperative: Intracranial procedures**

a) **Neuroendovascular (neuroradiology) procedures**

i) Indications

(1) Diagnostic

(a) Delineation of size and location of lesions to plan further therapy

(2) Therapeutic

(a) Detection and treatment of vasospasm

(i) Transluminal balloon angioplasty is one of the most effective treatments for vasospasm, whether it is the reason for the angiogram (eg, post-SAH) or occurs unexpectedly during an angiogram.

(ii) Treatment may include intra-arterial injection of vasodilators (eg, calcium channel blockers).

(b) Coiling and embolization

(i) May be definitive therapy for vascular lesions such as AVM or aneurysm.

(ii) More often used as a staging procedure to decrease size, and minimize blood loss and possibility of rupture during a planned resection to follow

(c) Placement of stents for stenotic lesions, across ruptured vessels, or to direct flow away from a lesion

(d) Stroke thrombectomy

(i) The treatment window for thrombectomy for ischemic stroke was extended to 24 hours in the 2018 American Heart Association/American Stroke Association guidelines.

(ii) In conjunction with thrombolytic agents (tPA)

(iii) No significant difference in outcome in acute stroke patients between GA and MAC anesthetic techniques[9]

ii) General considerations

(1) Patient immobility is crucial.

(2) Minimal blood loss is expected.

(3) Monitor I/O: Patients may receive several liters of heparinized flush solution.

(4) An arterial line can be placed if treatment is planned.

(5) Do not hyperventilate as this may predispose to vasospasm.

(6) Vasopressor and vasodilator therapy should be immediately available: phenylephrine and nicardipine.

(7) Give heparin (70 units/kg) to maintain ACT > 250 if treatment of a lesion is planned; check ACT every hour and redose with partial heparin dose as needed.

(8) It is desirable to have a rapid but smooth awakening at the end of these procedures.

(9) Postoperative pain is minimal.

iii) Procedures

(1) Diagnostic angiograms

(a) Consider placing an arterial line and a foley catheter prior to beginning the procedure if treatment of lesion might become possible or necessary.

(b) Have vasopressor ready to administer, as extremely rapid systemic vasodilatory effect may be seen with intra-arterial vasodilator injection by surgeons.

(2) Aneurysm coiling

(a) The catheter travels from the femoral or radial artery to the intracranial circulation.

(b) A pusher wire is threaded through the catheter and numerous platinum coils individually inserted into the aneurysm where they remain after being disengaged.

(3) AVMs and dural AV fistulas

(a) Feeding arteries of large vascular malformations (and other very vascular lesions) may be embolized preoperatively to decrease bleeding during subsequent resection.

(b) Neuro-embolic agents include particulates and polymer glues, and nonpolymerizing liquid agents.

iv) Potential complications

(1) Vasospasm

(2) Vessel perforation or rupture with hemorrhage

(a) Treatment is to decrease BP and reverse heparin immediately.

(b) Emergency craniotomy may be required if a stent cannot be placed.

(3) Vessel occlusion

(a) Treatment is to increase BP to high normal.

(4) Contrast-induced nephropathy

(a) *N*-acetylcysteine, 600-1200 mg before and after the procedure may be effective in reducing the incidence in patients at risk.

b) **Carotid endarterectomy (CEA)**

i) Indications: removal of a stenotic plaque from the carotid artery to improve blood flow to the brain

ii) Surgical Procedure

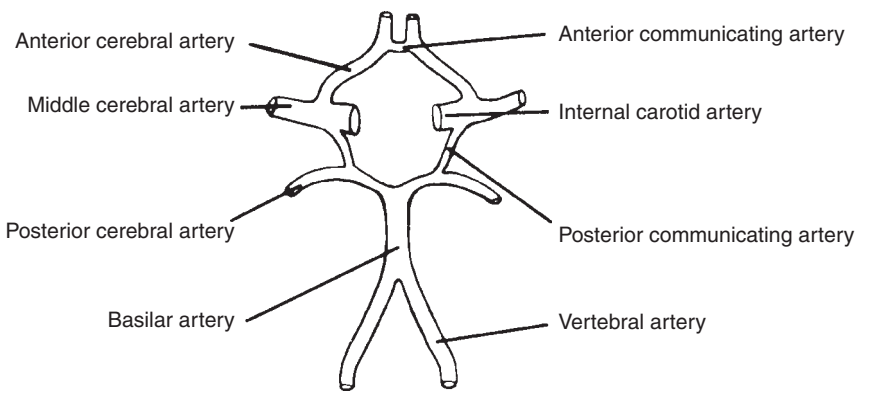
(1) After the carotid artery is exposed, vessel loops are passed around the carotid divisions (common, external or ECA, internal or ICA).

(2) The common carotid artery is clamped below the lesion; next the ECA and ICA are clamped.

(3) Before clamping the ICA, the surgeon may elect to measure perfusion pressure ("stump pressure") transmitted to the ICA via the Circle of Willis (**Fig. 123-4**).

(a) Surgeon will place needle into the ICA, and connect a (saline-flushed) sterile high-pressure line to the needle.

FIGURE 123-4 Circle of Willis



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(b) The distal end of the line is passed to the anesthesiologist to attach to a stopcock in the arterial line monitoring system.
(c) If the pressure is <20-40 mm Hg (actual number is controversial) or if other monitors (eg, EEG) indicate ischemia when the ICA clamp is applied, the surgeon may elect to insert a temporary shunt from the common carotid to the ICA distal to the lesion to ensure perfusion pressures are adequate during clamping.

(4) The common carotid artery is opened with a linear incision, and plaque is stripped off the endothelium at the bifurcation of the common carotid artery into the external and internal branches.
(5) The arteriotomy is partially sutured, backflow is allowed in order to flush the artery of air and particulate matter, and the final sutures are placed.
(6) The external and internal carotid arteries are unclamped, then the common carotid, and finally the incision is closed after strict hemostasis is obtained.



Bradycardia may be observed during dissection in region of carotid body, which may be ameliorated by the surgeon injecting 1-2 mL of lidocaine.

iii) Preoperative considerations
(1) Studies support early operation in symptomatic patients, and possibly some asymptomatic patients as well. Several studies have shown that urgent endarterectomy within 48 hours of an ischemic event carries a higher risk of stroke and/or death than surgeries performed more than 48 hours after the event.[10]
(2) Preoperative BP should be optimized. A 2017 study found that patients with preoperative baroreceptor dysfunction and poorly controlled BP were more likely to have postoperative HTN, and a higher rate of stroke and ICH.[11]
(3) It may be appropriate to proceed in patients with severe bilateral stenosis or frequent symptoms (TIAs) even if HTN is not well-controlled.

iv) Intraoperative anesthetic considerations
(1) Choice of anesthesia
(a) Studies conflict as to whether there is difference in outcome between general and local anesthesia for CEA, but many cases are safely conducted with either.
(2) BP control
(a) These patients often have impaired baroreceptor function and labile BP and should be well-hydrated.
(b) Maintain BP high normal (MAP 20% over baseline, especially if a shunt is used) with phenylephrine infusion. Once the carotid arteriotomy is sutured closed, systolic BP may be allowed to normalize at <120-150 mm Hg. Any higher risks cerebral hyperperfusion with associated complications of ICH.[12]
(3) Maintain normocarbia
(4) Clamping of the carotid artery
(a) Give heparin 70 units/kg just before clamping; reversal of heparin is not usually necessary but choice can be based on ACT results.
(b) A neuroprotective agent (usually propofol) may be infused for burst suppression before clamping if the surgeon requests.
(5) The EEG reflects only cortical events and not ischemia of deeper structures.

v) Postoperative considerations
(1) Patients with carotid artery disease are often hypertensive postoperatively.
(a) Meticulous BP control must be pursued to prevent postoperative stroke.

(b) Postoperative BP control can be difficult; aim for a postoperative systolic pressure of 120-150 mm Hg.
(c) Nicardipine infusion started just before emergence may help prevent BP swings.
(2) Coughing should be treated with small doses of lidocaine or narcotics.

vi) Potential complications
(1) CVA (more common in stenting)
(2) MI (more common in endarterectomy)
(3) Nerve injuries (most commonly hypoglossal, sublingual, or recurrent laryngeal) are almost always due to surgical retraction.
(4) Wound hematoma
(a) If there is any stridor or tracheal deviation, the best course of action may be to bring the airway cart into the OR, prep the neck, and immediately evacuate the hematoma with the patient awake, then perform rapid sequence induction and intubation for the surgeon to reopen the incision and find the bleeding vessel.


Wound hematoma after CEA has the potential to rapidly deteriorate into an airway disaster.

c) **Craniotomy**

i) Tumors
(1) Limit preoperative sedation if ICP is high, there is midline shift, or the patient has signs of impending herniation, and take care to not increase ICP during induction (do not hypoventilate).
(2) Mannitol (0.25-1 g/kg) infused slowly prior to opening the dura.
(3) Moderate hyperventilation ($PaCO_2$ 25-28) will decrease brain swelling. Normalize CO_2 once bone flap has been replaced to minimize space available for accumulation of blood, fluid, or air.
(4) Acoustic neuromas and tumors of the cerebellopontine angle may compress the brain stem and affect cardiovascular status.
(5) During orbitozygomatic approach, the surgeon may exert pressure on the globe, activating the oculocardiac reflex. Treat with anticholinergic agent.
(6) Anticipate extensive blood loss in the resection of large, highly vascular tumors (eg, hemangiomas, some glioblastomas, renal cell carcinoma metastases).


Acoustic neuromas and tumors of the cerebellopontine angle may compress the brain stem and affect cardiovascular status.

ii) Vascular lesions
(1) Aneurysm
(a) Hyperventilation can worsen vasospasm: do not use in aneurysm cases unless ICP is increased (eg, ruptured aneurysm).
(b) Keep BP lower than normal to prevent intraoperative rupture or rerupture.
(c) Surgeon may apply a temporary clip proximally to stop arterial flow through the aneurysm in order to ease placement of the permanent titanium clip. Other areas supplied by this artery will be at risk for ischemia until the clip is removed: "Time is brain."
(d) Once the permanent clip has been applied, minimize potential for vasospasm.


For all craniotomies, it is important to determine the size and location of lesion (which will determine site and size of craniotomy as well as position of patient), potential problems (crossing a sinus on opening, etc.), need for dexamethasone, mannitol, and neuroprotection.


Aneurysm cases may be an emergency if ruptured.

(i) Ensure normal $ETCO_2$.
(ii) Allow BP to rise to normal-to-increased levels (MAP of 90-100 mm Hg).

(e) Adenosine administration to cause hypotension or even momentary asystole; see above for more info on adenosine use.

(f) If clip location causes occlusion of a perforator artery to the brain stem, thalamus, or other structures, cardiovascular instability or loss of neurologic function may follow.

(g) An intraoperative angiogram may be performed to confirm exclusion of the aneurysm. Microscope-integrated fluorescence angiography is commonly used, with indocyanine green (25 mg in 10 mL saline) given IV.



Nonspecific ST changes mimicking ischemia are common EKG findings in patients with intracranial aneurysm rupture.

(h) If intraoperative aneurysm rupture occurs, ensure burst suppression and immediately control BP at MAP 40-60 mm Hg until the surgeon regains control of the vessel. Use shorter-acting agents such as esmolol, nicardipine, sevoflurane, remifentanil, and propofol. Adenosine can also be extremely useful.

(2) Arteriovenous malformation (AVM)

(a) Defined as mass of thin-walled blood vessels with direct arterial-to-venous connection without intervening capillaries.
(i) Acts as low resistance circuit (shunt), causing chronic vasodilation with loss of autoregulation in surrounding normal tissue (in order to maintain flow).
(ii) Up to 10% have an associated aneurysm.

(b) Preoperative planning is critical, as these can be extremely complicated and dangerous lesions to resect.
(i) Surgeon will map arterial supply and venous drainage: arterial inflow vessels will be resected first, venous outflow after.
(ii) Usually the craniotomy will be very large.
1. Almost 50% of AVM's cross the midline and associated venous sinuses.[13]
2. Risk of intraoperative bleeding is higher in AVM than in aneurysm, due to difficulty accessing the many deep vessels.
(iii) At least two large bore IV's and an arterial line are appropriate; consider CVP.
(iv) Often these lesions dive deep in the brain, and positioning must be methodical in case the surgery is long.

(c) After AVM resection, the surrounding brain tissue that may have been hypoperfused due to shunting is now exposed to higher CBF.
(i) This higher CBF in normal tissue may lead to edema or hemorrhage, called normal pressure perfusion breakthrough (NPPB), which can occur intra- or postoperatively.
(ii) Successful preoperative embolization can decrease blood loss and risk of intraoperative hemorrhage, shortens the duration of resection, and decreases the chance of NPPB.
(iii) Treatment of NPPB includes scrupulous control of systolic BP < 110 mm Hg (dexmedetomidine may be a good choice), hyperventilation, increased depth of anesthesia or sedation, mannitol, and diuretics.

(d) Slow, calm wakeup of these patients is helpful but must be balanced against the surgeon's need for neurologic examination at end of case.

READ MORE
Trauma, Chapter 125

iii) Traumatic brain injury (TBI)

(1) Can behave like a mass lesion; 20% mortality.

(2) Glasgow Coma Scale score of 13 or higher correlates with mild brain injury, 9-12 is moderate injury, 8 or less is severe injury; if <8, patient needs immediate intubation.
(3) Consider awake FOI; patients with trauma above the level of the clavicles have a 15% incidence of cervical spine injury.
(4) If acute, use full stomach precautions and RSI for induction.
(5) Cautious induction if hypotensive
(6) Maintain BP preferentially over ICP concerns.
(7) Do not hyperventilate unless needed for operating conditions because decreased perfusion of ischemic tissue may follow hyperventilation-induced vasoconstriction. Autoregulation may be disrupted, and there is not yet consensus on CPP management.
(8) Vasospasm is seen in >30%.
(9) Higher mortality level at 24 months in severe TBI patients resuscitated with 4% albumin vs with saline.
(10) Changes in ECG morphology and rhythm are common after head trauma and not necessarily due to cardiac issues.

iv) Craniotomy for IPH, aka "strokectomy"
(1) Removal of clot and infarcted tissue can decrease pressures and improve prognosis. Recent studies show that better outcomes may be obtained with strokectomy procedures vs decompressive craniectomy in MCA strokes.
(2) Risk of rehemorrhage is high, making surgery urgent.
(3) Patient may need reversal of antithrombotic therapy preoperatively; any coagulopathy must be reversed before surgery.[14]
(4) Volume of clot is a determining factor in outcome.
(5) Outcome in patients without upper brain stem reflexes is poor (50% die, only 20% recover to be independent).
(6) The posterior fossa is smaller and less compliant than the supratentorial compartment.
(a) ICP will increase more for a smaller change in volume due to hemorrhage.
(b) Increased infratentorial pressure is more life threatening due to the possibility of early herniation of the brain stem.
(7) Maintain systolic BP < 160 mm Hg (higher in chronically hypertensive patients).
(8) Use nicardipine to treat for HTN and risk of vasospasm.

v) Decompressive craniectomy (DC):
(1) Used primarily for large middle cerebral artery infarction. Greatly reduces mortality, but patients usually are left with severe postoperative disabilities.
(2) Sometimes done for TBI with uncontrollable ICP; bone flap will likely remain off and be replaced later.

vi) Other procedures
(1) Pituitary tumor
(a) Removed via transnasal transsphenoidal approach using endoscope, or via craniotomy if tumor quite large or invasive.
(b) Location is just medial to the cavernous sinuses and intracranial carotid arteries. If these are entered, the bleeding will be quite difficult to stop. Radiologic intervention may be requested early if necessary.
(c) Place arterial line and additional large-bore IV. Consider CVC if craniotomy planned or very large tumor.
(d) Have oral airway adjuncts ready at end of case in case patient obstructs, as nares may be packed by surgeon.

(e) Postoperative nausea and vomiting are not uncommon due to blood in stomach, despite presence of a throat pack during surgery. Preemptively treat with anti-emetics.

READ MORE
Pituitary disorders, Chapter 62

(2) Subdural hematoma (SDH) evacuation
   (a) SDH is due to venous bleeding.
   (b) Acute SDH can have high mortality (50%-80%).
   (c) Surgery will be via burr hole if acute (liquid), craniotomy if chronic (organized clot).
   (d) Insert arterial line and large bore IV; consider CVC if surgeon plans large craniotomy.
(3) Epidural hematoma (EDH) evacuation
   (a) EDH is almost always caused by skull fracture above middle meningeal artery.
   (b) Surgery is usually urgent or emergent. Craniotomy often fairly large due to urgency and uncertainty as to exact location of source of bleeding.
   (c) Vital signs may be unstable if hematoma is large.
   (d) Place arterial line, large bore IV, and consider CVC.
(4) Brain biopsy
   (a) Usually a short, low-risk procedure done via either burr hole with a needle, or a minicraniotomy.
   (b) Can be done with local or general anesthetic; no lines necessary other than IV.
(5) Chiari malformation
   (a) In adults, there is disruption of CSF flow through the foramen magnum (FM) and possible herniation of the cerebellar tonsils below the FM. Medullary and cervical cord compression can also occur. Presenting symptoms include headache and weakness.
   (b) Procedure is posterior fossa decompression and dural patch via midline suboccipital incision, with possible concurrent C1-C2 laminectomy.
   (c) Place arterial line and extra IV.
   (d) As in any posterior craniotomy, venous sinus entry is possible during opening, with sudden extensive blood loss.
   (e) Watch for postoperative respiratory depression and increased risk of aspiration.
(6) Intracerebral abscess or empyema
   (a) Similar to SDH and EDH
   (b) Consider arterial line; consider CVC for postoperative antibiotic therapy. Surgeon may request delay in antibiotic administration until cultures have been obtained.
(7) Resection of seizure focus
   (a) Demonstrable discrete focus may be resected (eg, temporal lobectomy, amygdalohippocampectomy).
   (b) If awake surgery is planned (lesion in eloquent area), patient must be able to communicate and cooperate.
      (i) Oversedation, with resultant respiratory depression, must be avoided.
      (ii) $ETCO_2$ should be monitored via nasal cannula CO_2 channel.
   (c) Remifentanil, propofol, and/or dexmedetomidine infusions may be useful.
   (d) Hyperventilation can decrease seizure threshold, while methohexital and etomidate can increase activity of seizure focus.
(8) Surgeries for trigeminal (CN V) neuralgia
   (a) Trigeminal stimulation during surgery can cause extreme adrenergic response with severe HTN if not treated expectantly. Consider arterial line. Have at least one large bore IV.

(b) Suboccipital craniotomy for microvascular decompression requires GA; surgeon will place Gore-tex or Teflon patch between nerve and artery.
(c) Rhizotomy (thermal or radiofrequency), percutaneous balloon compression, or glycerol or alcohol injection may all be accomplished with the patient intermittently awake but sedated during placement of probe or needle.
(d) Potential intraoperative complications include impaling nearby vascular structures including carotid artery.

(9) Hemifacial spasm (CN VII)
(a) Microvascular decompression similar to that for CN V
(b) Recommend arterial line placement; other monitoring will include facial nerve and BAERs.

(10) Stereotactic radiosurgery
(a) Delivery of radiation to a lesion (tumor, trigeminal neuralgia) via cyber knife or gamma knife
(b) Frame placement (used as the guide) requires sedation or "light" general anesthesia for 5-20 minutes, during which the patient will be sitting upright with spontaneous ventilation maintained.

(11) Ommaya reservoir
(a) Short procedure in which a chemotherapy reservoir is implanted under scalp, with distal end inserted into lateral ventricle via burr hole, similar to VP shunt.

(12) Endoscopic procedures
(a) Current uses are fenestration of third ventricular and other arachnoid cysts, transsphenoidal resection of pituitary tumors, and in some cranial base surgeries.
(b) Becoming more common in neurosurgery, and can greatly increase visualization of the surgical site with minimal retraction in tight spaces.
(c) Watch patient carefully for signs of herniation, as fluid inflow may be greater than outflow.
(d) Recommend arterial line and large bore IV.

READ MORE
Anesthesia and diseases of the nervous system, Chapter 55

(13) DBS electrode placement
(a) Placement of an electrode into deep brain structures (eg, hypothalamus, subthalamic nucleus) to treat various medical disorders, the most common being Parkinson disease and essential tremor.
(b) Usually done under GA (for patient comfort) with portable CT scanner in the OR.
(c) Electrodes connected via wires to a battery, usually in the subclavicular area.

SUMMARY FOR THE PATIENT FOR INTRACRANIAL NEUROPHYSIOLOGY AND SURGERY

Definition of ICP	ICP is the pressure exerted throughout the cranium by tissues and fluids inside the skull.
General Considerations	The cranium is a fixed compartment consisting of brain, blood, and CSF. CPP = MAP − ICP Increases in any of these may result in severe decrease in CPP and potentially herniation of the brain through the foramen magnum, which is life threatening. Intracranial surgery cases span a wide variety of problems and can be very challenging. The anesthesiologist must be meticulous in preparing the patient for what may be a multih-our procedure.

Preoperative	Consult with the surgeons on planning for any special issues that may exist with the airway, location of lesion, positioning, and monitoring. Do not over-sedate patients with ICH.
Intraoperative	Blood should be immediately available for patients with vascular lesions when potential for rapid blood loss exists. Hyperventilate to $ETCO_2$ 25–30 mm Hg until ICH resolved. Other treatments for acutely increased ICP are head up position, placement of CSF drain, osmotic diuretics.
Postoperative	Meticulous attention to BP control is critical. Follow serial neurologic examinations in the recovery room and immediately investigate any change. Craniotomy patients usually do not have severe postoperative pain. Watch patient for signs of increased ICP.

ICP, intracranial pressure; BP, blood pressure; CPP, cerebral perfusion pressure; CSF, cerebrospinal fluid; MAP, mean arterial pressure; ICH, intracranial hypertension; $ETCO_2$, end-tidal carbon dioxide.

FURTHER READING

Excellent references on neurosurgical emergencies from the Society for Anesthesiology and Critical Care:

Hoefnagel A, et al. Cognitive aids for the diagnosis and treatment of neuroanesthetic emergencies: consensus guidelines on behalf of the Society for Neuroscience in Anesthesiology and Critical Care (SNACC) Education committee. *Neurosurg Anesthesiol*. 2019;31(10):17.

Discussion of quick reference evidence-based guidelines included for aneurysm rupture, increased ICP, loss of evoked potentials, VAE, etc., with link: http://links.lww.com/JNA/A80 Annually, in the April issue of the Neurosurgical Anesthesiology, there is a comprehensive review of a series of journal articles from the last year that are pertinent to the practice of neuroanesthesia.

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124 Thoracic Anesthesia

Antasia Giebler • Ana Fernandez-Bustamante

Thoracic anesthesia is one of the most challenging areas of anesthetic practice. Operations range from lung biopsies or small segmental resections, to complete pneumonectomies, tracheal resections, or esophageal surgery. Surgery involving the airway or lung requires lung isolation, often by different techniques based on the patient's unique anatomy. The anesthesiologist should be prepared to manage airway abnormalities, difficult or unusual ventilation strategies, along with postoperative pain control strategies for open thoracotomy procedures. The recent advancement of robotic surgery aims to reduce complications and hasten recovery, though requires additional considerations for positioning and muscle relaxation. Enhanced Recovery After Surgery (ERAS) protocols in thoracic surgery include optimization of fluid status, pain control, and early mobilization.

1) **Respiratory physiology**

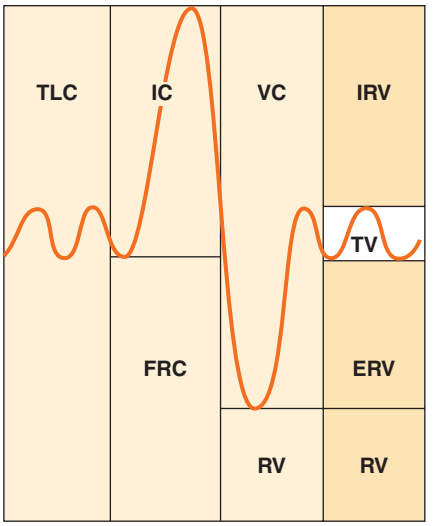
An understanding of basic respiratory physiology is important in all aspects of anesthesia care and of paramount importance in thoracic surgery.[1]

a) **Lung volumes and capacities (Fig. 124-1)**
   i) Tidal volume (TV) is the volume of gas exchanged during normal breathing.
   ii) Vital capacity is the volume of gas expired when one takes a maximal inspiration and maximal expiration.
   iii) Residual volume (RV) is the amount of gas remaining in the lung after maximal expiration.
   iv) Functional residual capacity (FRC) is the amount of gas remaining in the lung after normal expiration.

b) **Pulmonary ventilation and perfusion (Fig. 124-2)**
   i) Regardless of body position, gravitationally dependent regions of the lung (bases in the upright position, dorsal regions in the supine position) receive greater ventilation (*V*) and perfusion (*Q*).
   ii) Although the overall *V*/*Q* ratio of the lung is 0.8, gravitationally nondependent alveolar units have greater regional *V*/*Q* ratios than dependent ones.
   iii) Gas exchange efficiency is directly related to the regional *V*/*Q* ratios and is reduced significantly in Zone 1 in the supine position.
   iv) Overall *V*/*Q* (and oxygenation) decreases when moving from the upright to the supine position.

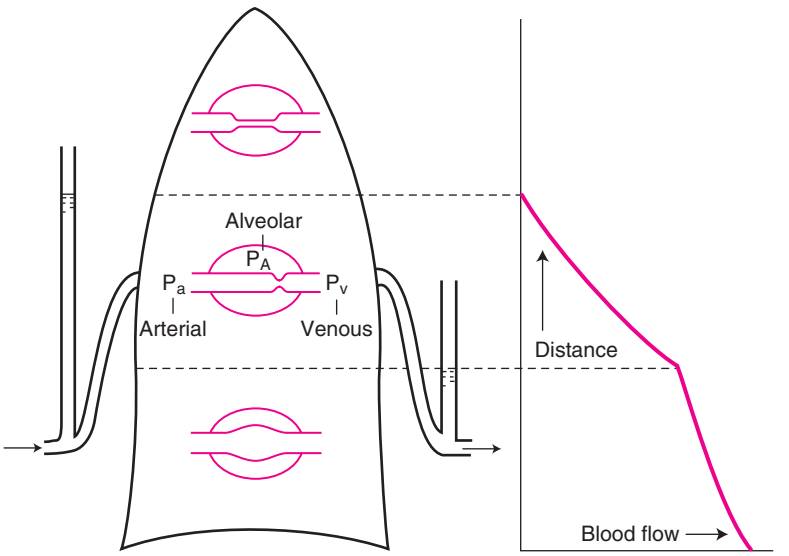
c) **Mechanisms of hypoxemia**
   i) Reduced inspired oxygen partial pressure, P_iO_2, or fraction, F_iO_2
      (1) Occurs with lower atmospheric pressure (ie, high altitude) or oxygen fraction <0.21 (ie, toxic fumes in closed environment).
   ii) Alveolar hypoventilation
      (1) Impairment in respiratory drive or ability that reduces inspired volume and/or respiratory rate (ie, sedatives, neuromuscular disorders, chest trauma)
   iii) *V*/*Q* mismatch
      (1) One of the most common O_2-treatment-resistant hypoxemic mechanisms
      (2) Dissociation between ventilation and perfusion in alveolar units

FIGURE 124-1 Lung Capacities and Volumes



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FIGURE 124-2 West Zones of the Lungs



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(3) Occurs when some units receive proportionally less ventilation than perfusion due to pathology such as secretions or edema.
(4) Right-to-left shunts cause an extreme treatment-resistant *V/Q* mismatch that occurs in alveoli that are perfused but not ventilated and therefore do not contribute to gas exchange.
(a) Examples include pulmonary arteriovenous malformations, hepatopulmonary syndrome, and alveolar consolidation.
(5) General anesthesia increases *V/Q* mismatch because of increased airway closure, inhibition of hypoxic pulmonary vasoconstriction, and shunt effect due to atelectasis.
(a) Lateral position and one-lung ventilation (OLV) enhance this *V/Q* mismatch.



The lateral position and one lung ventilation worsen the *V/Q* mismatch that occurs during general anesthesia.

iv) Impairment of O_2 diffusion through the alveolar-capillary barrier
(1) An uncommon mechanism of hypoxemia
(2) Occurs in pulmonary fibrosis and acute respiratory distress syndrome (ARDS).

d) **Physiological dead space**
i) Physiological dead space (V_D) is the fraction of inspired volume that does not contribute to gas exchange.
ii) Includes the air remaining in the respiratory tree excluding the alveolar spaces (anatomical V_D, ~2 mL/kg) and the volume that reaches poorly or not ventilated alveoli (ie, functional V_D, variable volume that increases with age, COPD, etc.).
iii) In a healthy adult, $V_D/V_T < 30\%$
iv) Physiological V_D is the expired-to-arterial PCO_2 ratio [$(PaCO_2 - P_{ET}CO_2)/PaCO_2$].



Increases in dead space, such as during low cardiac output or pulmonary embolism, may result in increasing the $PaCO_2$-to-$P_{ET}CO_2$ difference and a greater $PaCO_2$ for the same $P_{ET}CO_2$ level.

e) **Compliance**
i) The ability of the respiratory system (chest wall, lung, or both) to change in volume with a change in distending pressure.
ii) Respiratory compliance is usually measured as:

$$C = \Delta\text{Volume}/\Delta\text{Pressure} = V_T/(P_{plateau} - \text{Positive End Expiratory Pressure})$$



General anesthesia reduces respiratory compliance and increases airway resistance and work of breathing.

iii) In a spontaneously breathing healthy adult, the compliance of the respiratory system or total compliance (lung + chest wall) is ~100 mL/cmH_2O.
(1) Compliance decreases significantly in the supine position, under anesthesia, and with respiratory diseases (eg, pulmonary fibrosis, inflammation, or edema).
iv) As compliance decreases, higher ventilatory pressures are required to maintain lung expansion and oxygenation, resulting in increased work of breathing (WOB), less efficient alveolar–capillary gas exchange, and greater risk of barotrauma.



Decreasing compliance is associated with an increasing risk of barotrauma during positive pressure ventilation.

f) **Airway resistance**
i) Resistance to airflow is inversely related to lung volume and airway diameter.
ii) Resistance determines whether air flow is laminar or turbulent.
iii) Airway resistant is measured as:

$$R = \Delta\text{pressure}/\Delta\text{flow} = (P_{peak} - P_{plateau})/\text{flow}$$

g) **Work of breathing (WOB)**
i) Reflects the effort performed by respiratory muscles to overcome the resistance and elastic recoil of the chest and lung, and is estimated as a product of mean airway pressure (Paw) times tidal volume: WOB = Paw × V_T.

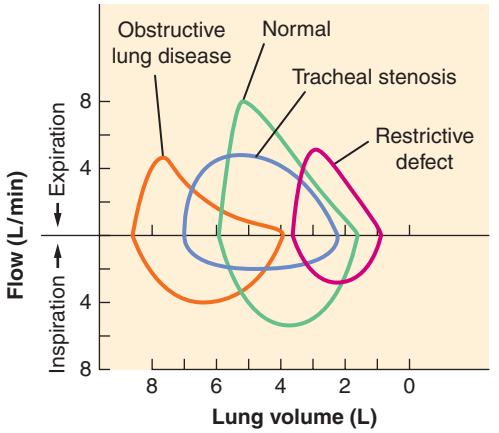
TABLE 124-1 Pulmonary Function Test (PFT) PFT Results

Description	Normal[a]	Obstructive Disease	Restrictive Disease
TLC	5.5 L	↑	↓
FVC	4 L	N/↓	↓
FEV_1	3.2 L	↓	N/↓
FEV_1/FVC	80%	↓	↑
FRC	2.5 L	↑	↓
RV	1.5 L	↑	↓
FEF_{25-75}	>65%	↓	N/↓

[a]Normal PFT values for a healthy 70-kg adult.
N, normal.

2) **Preoperative assessment and management**
   a) **General considerations**
      i) A thorough preoperative evaluation should be performed with a focus on the cardiac and respiratory function.
      ii) Pulmonary function must be optimized before surgery.
         (1) Consider pulmonary rehabilitation, treatment of underlying infections, and palliative relief of airway obstruction if needed.
         (2) Scheduled treatments (including inhalation therapy) should be continued.
      iii) The preoperative interview is an important time for discussing postoperative management, including emergence and extubation, risk of postoperative mechanical ventilation, critical role of patient controlled analgesia (PCA), etc.
      iv) If epidural anesthesia is indicated, obtain informed consent and strongly consider placement prior to surgery to optimize postoperative pain control.
   b) **Pulmonary function tests (PFTs) should be reviewed.**
      i) **Table 124-1** shows normal and pathological PFTs.
      ii) Pressure-flow curves for different respiratory conditions are described in **Figure 124-3**.
         (1) In obstructive lung disease, the expiratory curve is concave and lung volumes are increased.
         (2) Restrictive disease results in a narrow and steep expiratory curve with decreased lung volumes.
         (3) Fixed upper airway obstructions such as tracheal stenosis result in flattened inspiratory and expiratory and curves.
      iii) Preoperative findings described in **Table 124-2** indicate a high risk for postoperative complications after a pneumonectomy.[2]
      iv) Postoperative respiratory complications are rare if the preoperative FEV_1 is ≥2 L or ≥50% of predicted value.
      v) A high risk of postoperative respiratory failure is indicated by an estimated postoperative FEV_1 <0.8 L (postoperative FEV_1 = preoperative FEV_1 × perfusion % of remaining lung after resection, estimated by ^{133}Xe or ^{99}Tc V/Q scanning).
   c) **Other tests**
      i) Arterial blood gases are not routinely indicated but may be considered to assess the severity of the pulmonary condition.
      ii) CT imaging may be required in tracheobronchial stenosis for evaluation of disease severity, determination of appropriate size and length of the endotracheal tube, or to modify the airway management plan.

FIGURE 124-3 Flow Volume Loops in Various Lung Diseases



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iii) Split-lung function studies can be used to evaluate differential lung ventilation and perfusion to predict postoperative lung function and tolerance after a lung resection.

d) **Preoperative sedation**
   i) Sedation should be individually customized to avoid respiratory depression, hypoxemia, and loss of airway protection.
   ii) A delicate balance is required in patients with tracheobronchial stenosis, in whom anxiety may lead to tachypnea, increased airway resistance, and turbulent flow.
   iii) Supplemental oxygen and continuous pulse oximetry should be considered in all thoracic patients.

e) **Aspiration precautions should be considered in patients with esophageal diseases or undergoing major thoracic surgery.**

f) **An anesthesia machine capable of different ventilatory modes (including pressure-controlled ventilation) and high gas flows is needed. A fiberoptic bronchoscope should also be available for all thoracic anesthesia cases.**

TABLE 124-2 High-Risk Preoperative Test Results for Pneumonectomy

Test	High-Risk Results
Arterial blood gas (room air)	PaO_2 < 50 mm Hg
Preoperative FEV_1	<2 L
Predicted postoperative FEV_1	<0.8 L or <40% of predicted
FEV_1/FVC	<50% of predicted
Maximum breathing capacity	<50% of predicted
Maximum VO_2 during exercise	<10 mL/kg/min

FEV_1, forced expiratory volume in 1 second; FVC, forced vital capacity; VO_2, oxygen consumption.

g) **Venous access and fluid therapy**
   i) At least one large-bore IV is recommended for all patients undergoing major thoracic surgery. IV fluids are usually restricted, especially in cases with OLV in the lateral position, to minimize pulmonary edema (postthoracotomy acute lung injury or ARDS.[3] Goal-directed fluid therapy is recommended when feasible.
   ii) Consider invasive arterial BP monitoring for open thoracic procedures or as needed based on cardiac risk.



To minimize risk of rebound pulmonary edema following lung resection, especially pneumonectomy, limit IV fluids to 1-2 L provided there is no ongoing blood loss.

h) Preoperative analgesics are recommended in most enhanced recovery after surgery (ERAS) protocols including acetaminophen and gabapentin to decrease postoperative narcotic requirements. Many of the protocols also recommend a carbohydrate load (8 oz of apple juice or electrolyte containing sports drinks) 2 hours before surgery.

3) **Intraoperative management** will be discussed in the following sections.
4) **Postoperative management**
   a) **General considerations**
      i) Despite an older population undergoing elective thoracic surgery with their concurrent comorbidities, operative mortality after thoracic surgery remains unchanged from historic controls (around 2.5%).[4,5]
      ii) Factors influencing choice of single-lumen vs double-lumen tubes (DLTs) (**Table 124-3**):
         (1) DLTs should be used when lung isolation is absolutely indicated, such as preventing contamination of the contralateral lung from blood, infectious secretions, or lung lavage fluid. In other instances of absolute or relative indications for lung isolation, DLTs or bronchial blockers can be used, though DLTs allow each lung to be isolated, ventilated, or suctioned.
         (2) In patients with a known or suspected difficult airway, or in patients who would need to be kept intubated postoperatively, a bronchial blocker and single-lumen tube can be considered; likewise, a single-lumen tube can be advanced into either mainstem bronchus to achieve lung isolation.

READ MORE
Atlas of clinical procedures, Insertion of left-sided double lumen tube, Chapter 165

   b) **Independent risk factors of perioperative mortality**
      i) Increased age, male gender, dyspnea score, functional performance status, the American Society of Anesthesiologists (ASA) score, and the extent of surgical resection.[4–6]
   c) **Complications**
      i) The most common postoperative fatal complications are infections and ARDS.
      ii) Nonfatal complications are more frequent: inadequate analgesia, atelectasis, pneumothorax, bleeding (>200 mL/h through chest tube), hemoptysis, supraventricular tachyarrhythmias, and nerve injury (ie, left recurrent laryngeal, vagus, or phrenic nerve injury after mediastinal surgery).

TABLE 124-3 Lung Isolation Methods and Indications

Double-lumen tube (DLT)	• Absolute indication for lung isolation: prevention of contamination of contralateral lung (eg, from blood, infectious secretions, lavage fluid) • Main benefit: it allows each lung to be isolated, ventilated, and/or suctioned
Bronchial blocker or intentional selective stem intubation with single-lumen tube	• Relative indication for lung isolation • To be considered if known or suspected difficult airway or patients needing postoperative intubation and ventilation

d) **Postoperative management**
   i) Patients should be positioned semiupright (>30 degrees), receive close ECG and hemodynamic monitoring, supplemental O_2 as needed, incentive spirometry, a postoperative chest x-ray, and aggressive pain treatment.



Ideally, an epidural catheter should be checked for efficacy before induction of general anesthesia, and dosed during the case and at thoracotomy closure for a smoother emergence.

5) **Anesthetic management of specific procedures**
   a) **Pulmonary resection via open thoracotomy**
      i) Regional analgesia
         (1) Patients undergoing a thoracotomy benefit from a thoracic epidural catheter (T4-T7) due to superior postoperative pain control and decreased narcotic secondary effects.
         (2) A local anesthetic-opioid mixture is most commonly selected (eg, bupivacaine 0.06% with fentanyl 2 µg/mL, usually at 5-8 mL/h).
         (3) Intercostal nerve blocks may be used when epidural analgesia is not an option or not effective.
            (a) Under sterile conditions, a 22-gauge needle is inserted perpendicular to the skin at the posterior axillary line on the inferior edge of the rib. The needle is advanced until it is touching the rib, then slid downward to slip off the rib. After confirmation of no blood or air upon aspiration, 4-5 mL of local anesthetic (eg, bupivacaine 0.5% with epinephrine 1:200 000) is injected. The process is repeated at each intercostal space involved in innervation of the thoracotomy site.
         (4) Paravertebral block
            (a) Can be accomplished either preoperatively or postoperatively and provide supplemental pain control for 12-24 hours.



Epidural medications will not blunt vagal-related airway manipulation, lung reexpansion reflexes, or referred shoulder pain from diaphragmatic irritation.

READ MORE
Paravertebral blocks, Chapter 194

      ii) General anesthesia
         (1) Monitoring
            (a) Standard ASA monitoring plus invasive arterial monitoring are recommended in all patients undergoing major thoracic surgery.
            (b) Blood flow to the dependent arm in a lateral-positioned patient, and to the right arm during mediastinal surgery, should be monitored with either the arterial line or pulse oximeter. Further invasive monitoring will depend on the patient's condition.
            (c) Although specific guidelines on the use of transesophageal echocardiograph (TEE) in patients with pulmonary hypertension (PH) undergoing noncardiac surgery do not exist, TEE should be considered in patients with severe PH. TEE provides dynamic and continuous monitoring of right ventricular (RV) function to aid in optimizing RV mechanics and detecting RV failure, as well as RV filling to help guide fluid management.[7]
         (2) Induction
            (a) Can be achieved using various induction agent/muscle relaxant combinations as guided by the preoperative evaluation.
         (3) Maintenance
            (a) Can be achieved with a volatile agent or total intravenous anesthesia (TIVA).
            (b) Muscle relaxation is needed during the procedure.
            (c) Nitrous oxide is typically not used due to the possibility of increased atelectasis and pulmonary vascular resistance.

(4) Emergence
   (a) Smooth emergence is ideal to avoid stressing anastomotic lines following pulmonary resection.
   (b) Verify two-lung ventilation with manual recruitment of the lungs (inspiratory pressure <30 cmH_2O maintained for 2-3 seconds).
   (c) Use FiO_2 1.0 and verify patency of chest tubes.
   (d) A supine position with head raised 45 degrees is helpful.
   (e) If postoperative mechanical ventilation is needed, the DLT must be exchanged for a conventional ETT prior to emergence.
   (f) Chest tubes are connected to water seal alone after a pneumonectomy, or with 20 cmH_2O suction in all other resections.



Vagal-mediated bradycardia may occur with airway surgical manipulation, so atropine should always be available.

iii) Positioning
   (1) Pulmonary resection is usually performed with the patient in the lateral position (operative, or nondependent side, up), and the bed sharply flexed.
   (2) Extreme care should be taken to assure proper positioning, including pressure-relieving padding below the head and neck (neck in neutral position and dependent ear and eye free of pressure), shoulders (brachial plexus), arms (radial and ulnar nerves), legs, and scrotum in males.
   (3) Vital signs and oxygenation should be monitored during positioning.
   (4) After positioning and before preparation of the surgical field, reassessment of vitals, oxygenation, patency of IV catheters, epidural catheter, and position of endobronchial tube or bronchial blocker are mandatory.

iv) One-lung ventilation (OLV)
   (1) Physiological implications
      (a) General anesthesia, mechanical ventilation, lateral position, open chest, and OLV alter ventilation and perfusion (*V*/*Q* matching).
      (b) Oxygenation depends on blood flow to the nonventilated lung (intrapulmonary shunt).
      (c) The shunt fraction is reduced in the lateral position due to the effects of gravity, along with hypoxic pulmonary vasoconstriction, which reduces perfusion to nonventilated areas.
      (d) Additionally, chronically diseased lungs have diminished perfusion overall, which is beneficial if the operative side is the most severely injured lung.
   (2) Multiple devices are available for lung isolation, including DLTs, bronchial blockers, and Univent tubes.
   (3) Management
      (a) When switching from two-lung to OLV or vice versa, a brief period of manual ventilation is recommended for assessing compliance and recruiting collapsed alveoli.
      (b) A lung protective ventilation strategy is recommended to reduce the risk of lung injury, including low V_T (4-6 mL/kg of predicted body weight with a higher respiratory rate to optimize minute ventilation), positive PEEP, plateau pressure <30 cmH_2O, and the lowest FiO_2 to ensure adequate oxygenation.[8,9]
      Predicted body weight:
         i) *Male = 50 + 2.3 [height (in) – 60]*
         ii) *Female = 45.5 + 2.3 [height (in) – 60]*
      (c) Neuromuscular relaxation and controlled ventilation are mandatory during OLV, with the goal of maintaining adequate oxygenation and a $PaCO_2$

TABLE 124-4 Steps to Take When Oxygenation or Ventilation Difficulties Occur During OLV

- Ensure FiO_2 of 1.0.
- Confirm adequate tube position
- Suction secretions if needed
- Apply CPAP to the nonventilated lung (open thoracotomy) and notify the surgeon
- Add PEEP to the ventilated lung
- Apneic oxygenation of the nonventilated lung
- Periodic manual double-lung ventilation
- Pulmonary artery clamp
- Cardiopulmonary bypass (extreme situations)

similar to two-lung ventilation. The latter will depend largely on the dependent lung compliance, and mild permissive hypercapnia may be tolerated.

(4) Management of hypoxemia during OLV (**Table 124-4**):
   (a) Ensure FiO_2 of 1.0.
   (b) Confirm adequate tube position by fiberoptic bronchoscopy (**Figs. 124-4 and 124-5**).
   (c) Suction secretions if needed
   (d) Apply continuous positive airway pressure (CPAP) to the nonventilated lung (in open thoracotomy cases) and notify the surgeon.



Lung isolation should be confirmed whenever any changes are made in patient position.

FIGURE 124-4 Fiberoptic View of the Distal Trachea and Carina. Fiberoptic view of carina anatomy is labeled. It is common practice to identify the right upper lobe in the right mainstem bronchus (usually 2-3 cm past carina) to verify the right from left bronchus. (Image by N. Weitzel, MD.)

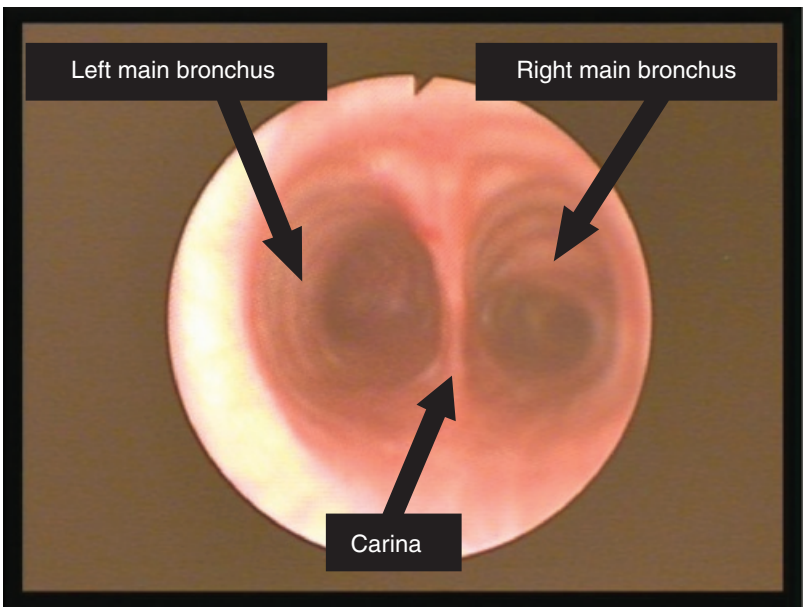
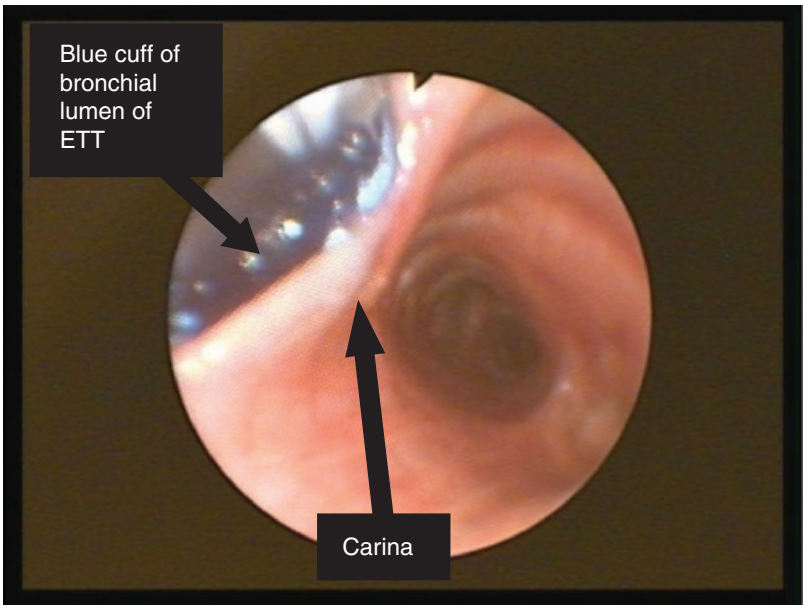


FIGURE 124-5 Fiberoptic View of Appropriately Placed Double-Lumen ETT. The bronchial lumen is in the left bronchus—with the edge of the bronchial cuff (*blue*) just visible at the carina when viewed from the tracheal lumen. (Image by N. Weitzel, MD.)



Under direct visualization, the nonventilated lung is inflated through a separate circuit and then allowed to deflate and maintained at a volume that does not interfere with the surgical procedure, usually 2-5 cmH_2O CPAP. This may not be possible in video-assisted thoracoscopic surgery (VATS) cases due to interference with the surgeon's view.

(e) Add PEEP to the ventilated lung to minimize atelectasis. This may be ineffective if high airway pressures decrease perfusion to ventilated areas, increasing the shunt fraction. Small titrations in PEEP are recommended to identify the minimum effective PEEP. Patients with obstructive lung disease can develop auto-PEEP with progressive shunting.

(f) Apneic oxygenation of the nonventilated lung: The nonventilated lung is periodically inflated with FiO_2 1.0 and then isolated by capping the exhalation valve, so that it stays motionless and slowly collapses absorbing the oxygen.

(g) Periodic manual two-lung ventilation may be required for adequate oxygenation.

(h) If hypoxemia persists, the surgeon may clamp the pulmonary artery perfusing the nonventilated lung to improve *V*/*Q* matching.

(i) Cardiopulmonary bypass can be established in extreme situations (**Table 124-4**).

b) **Video-assisted thoracoscopic surgery (VATS)**
   i) The number of procedures performed with thoracoscopic assistance is rapidly increasing.
   ii) VATS is usually performed in the lateral decubitus position with OLV and three or four small incisions.
   iii) Anesthetic management is similar to open procedures except epidural analgesia is not usually required.
   iv) CPAP to the nonventilated lung often disrupts the surgical field to a greater degree than with an open thoracotomy and may prevent adequate surgical exposure.

c) **Robotic-assisted thoracoscopic surgery (RATS)**
   i) Similar to Video-Assisted Thoracic Surgery (VATS), the use of RATS has rapidly increased and is commonly performed using OLV and without epidural analgesia.
   ii) Positioning varies with surgeon and procedure; however, the robot is typically deployed near the patient's head and requires frequent monitoring to prevent injury. Securing vascular access and ensuring that lines are patent during positioning is important as arms are usually not accessible after the robot is positioned.
   iii) Maintain adequate muscle relaxation to avoid patient movement as this can lead to tearing or puncture of vessels and organs.
   iv) In an emergency, delay in undocking the robot may prevent immediate conversion to an open procedure. Simulations can help for team-training in dealing with this crisis.

d) **Lung volume reduction surgery (LVRS)**
   i) LVRS is performed to reduce the most affected lung areas in selected patients with severe emphysema.
   ii) The major clinical trial that compared medical + surgical treatment vs medical only treatment (National Emphysema Treatment Trial, or NETT trial) showed controversial results.[10]
      (1) LVRS may improve the quality of life of patients without high operative risk who have predominantly apical disease, and a poor baseline exercise tolerance, although it does not alter their overall disease-related mortality. Postoperative mortality is increased in poorly selected patients.
   iii) Intraoperative anesthetic management
      (1) The risk of hypoxemia and pneumothorax should guide ventilator management.
      (2) Careful ventilation with individualized FiO_2, pressure limited ventilation below 30 cmH_2O, and prolonged expiratory times are mandatory.
      (3) Extubation is essential at the end of the procedure; therefore, extreme care should be paid to optimize extubation conditions before emergence: maximize SaO_2, ensure adequate analgesia, administer IV steroids and bronchodilator nebulizations, avoid shivering from hypothermia, etc.
      (4) If immediate extubation is not possible, assure an adequate level of anesthesia before replacing the DLT with a standard ETT. FiO_2 should be minimized to obtain a SpO_2 ≥88%-90%. The chronic hypoxic state of these patients alters respiratory drive such that increased FiO_2 increases the risk of hypercapnia and respiratory acidosis.
   iv) Postoperative management
      (1) Adequate analgesia is critical to avoid hypoventilation and its consequences.
      (2) Thoracic epidural analgesia is ideal, but intercostal or paravertebral regional blocks can also be considered before opting for a total IV opioid-based analgesia.
      (3) BiPAP should be considered in patients with $PaCO_2$ ≥ 70 mm Hg.



Due to chronic hypoxia in extreme COPD, patients breathing high FiO_2 are at risk for developing hypercapnia; thus, aim for SpO_2 of 88%-90% in these patients.

e) **Tracheal resection and reconstruction**



For tracheal surgeries, equipment and personnel for an emergency airway management should be immediately available before induction.

i) Preoperative considerations

(1) The major anesthetic challenge during these procedures is airway management at induction and during the case. Individualized and detailed communication with the surgical team before the procedure is critical: level and severity of airway lesion and initial airway management (oral vs tracheostomy, size of device), surgical implications on airway control (change of airway device, need of jet ventilation), postoperative plan.

ii) General anesthesia

(1) Induction

(a) IV agents can be used in patients with a preexisting tracheostomy or those without critical airway stenosis.

(b) In patients with critical stenosis, awake intubation or induction maintaining spontaneous ventilation should be considered.

(c) Neuromuscular relaxation should be delayed until the airway is instrumented under a deep plane of anesthesia and/or the use of local anesthetic at the vocal cords. TIVA (eg, propofol and remifentanil) should be considered for maintenance of anesthesia because of intermittent interruptions in ventilation during the procedure and for the facilitation of a smooth emergence.

(2) Surgical specifics

(a) Rigid bronchoscopy may be performed prior to intubation and the findings considered to guide airway management (routine oral intubation, laryngeal mask airway (LMA) with spontaneous ventilation, tracheostomy, resection of an intraluminal tracheal lesion, jet ventilation from above the lesion, etc.). In distal tracheal or carinal stenoses, a long cuffed ETT or jet ventilation through a catheter may be used.

(3) Tracheal division

(a) When the trachea is surgically divided, the ETT or jet catheter is retracted and a sterile armored ETT is placed by the surgeon in the distal airway. If the distal airway is too small to accommodate a tube (ie, carinal or mainstem bronchial resections), a jet catheter (see below) may be used.

(b) The airway resection is performed along with the posterior anastomosis around this distal tube/catheter. The anterior sutures are often placed under intermittent apnea before advancing the original ETT.

(c) The patient's neck is then flexed (extra blankets will be needed to support the head), the anastomosis finished, and skin closed. A suture may be left from the chin to the chest to remove tension at the tracheal anastomosis.

(4) Emergence and extubation

(a) Spontaneous ventilation and extubation is usually the goal to decrease strain on fresh anastomoses, but in certain cases, postoperative sedation with mechanical ventilation and a flexed neck may be preferred.

(b) A small tracheostomy may be placed below the anastomosis for safety reasons (heavy secretions, difficult anatomy, etc.).

(c) Extubation should be performed when the patient is awake enough to protect his or her airway, but without excessive head movement or coughing (low-dose sedation may be considered).

(d) Flexible bronchoscopies may be needed postoperatively to clear secretions.

(e) A small uncuffed ETT (5.0 or 6.0) and a flexible bronchoscope should be readily available for potential reintubation.

f) **Endoscopic procedures**

i) Bronchoscopy

(1) Flexible bronchoscopy

(a) Usually performed under local anesthesia (pharynx and airways) with sedation

(b) General anesthesia may be needed in certain patients and will require an LMA or ETT large enough to accommodate the bronchoscope.

(c) Consider premedication with an antisialagogue, local anesthesia in the airway, short-acting anesthetics, and/or neuromuscular relaxants.

(2) Rigid bronchoscopy

(a) Allows visualization of the larynx, trachea, and mainstem bronchi.

(b) Requires a deep level of general anesthesia and possibly neuromuscular relaxation to prevent movement and coughing.

(c) Ventilation is accomplished through the working channel of the rigid bronchoscope, attaching the respiratory circuit to the lateral port of the rigid bronchoscope. Jet ventilation may be used (see below).

(d) In cases of severe stenosis, maintenance of spontaneous ventilation is recommended.

(e) Due to the potential for large air leaks, as well as interruptions of ventilation, capnography may not be accurate. TIVA is a convenient option for maintenance.



Coordination between the surgical and anesthesia teams is critical during rigid bronchoscopy since ventilation or surgery may need to be interrupted for instrumentation or ventilation, respectively.

(f) Jet ventilation

(i) The major benefits are a continuous ventilation and a lack of movement of the surgical field.

(ii) May be applied through a thin catheter inserted through the main lumen of the rigid bronchoscope or through a small lateral lumen of a specially designed rigid bronchoscope, with a Sanders' manual jet injector or a specially designed jet ventilator.

(iii) Jet ventilation delivers very small tidal volumes (close to the anatomic dead space) at a higher frequency or respiratory rate than conventional ventilation. The oxygen-enriched gas is delivered from a high pressure source generating tidal volume that is supplemented by entraining air (entrapment). Expiration is passive due to chest wall recoil. Rate is controlled by pneumatic or electronic flow interruption.



Jet ventilation requires being familiar with the jet equipment and the administration of ventilation with small TV/high rate in an open respiratory system.

(iv) Because of the open respiratory system during jet ventilation, the exact TV and inspired oxygen fraction are not known. Similarly, capnography is rarely available or reliable.

(v) Make sure air is exhaled at the end of every breath (ensure an open respiratory system, adequate expiratory time); if not (eg, airway lumen closed by surgical instruments), the risk of auto-PEEP, barotrauma, and tension pneumothorax is high.

(vi) FiO_2 must be reduced below 0.4 during laser surgery.

(vii) Communication with surgeons is critical before (delivery method, alternative plans if jet ventilation is not well tolerated) and during (obstructed airway, laser use, desaturation) jet ventilation.



Check visually thoracic movements during jet ventilation to assure air comes in and OUT!

(3) Complications of bronchoscopy include injuries to eyes, lips, teeth, or larynx, airway rupture, pneumothorax, bleeding, and airway obstruction because of bleeding or a dislodged stent.

ii) Mediastinoscopy and mediastinal surgery

(1) Usually performed for staging tumors to facilitate subsequent planning of surgical resection, but sometimes is coupled with VATS or open resection.

(2) Preoperative evaluation of the trachea is mandatory to rule out a fixed, deviated, or compressed trachea.

(a) Large bore (≥16-gauge) venous access is highly recommended. Mediastinal surgery can be complicated by life-threatening hemorrhage due to large vessel damage, which is difficult to control.

(3) Monitoring

(a) The pulse oximeter or arterial line (if needed) should be placed in the right arm to monitor flow through the innominate artery and detect potential compression.

(b) Mediastinoscopy alone is a short procedure and short-acting hypnotics and neuromuscular relaxants should be used.

CHAPTER SUMMARY FOR THORACIC ANESTHESIA

General Population	Wide range of patients and procedures often requiring advanced airway management or one-lung ventilation
Assessment	Based on extent of procedure, but consider full laboratory workup, ± ABG, pulmonary function testing, ± VQ scanning for lung resections, CT scans. Coronary workup based on comorbidity. Aggressive pulmonary therapy preoperatively helps reduce risk of respiratory complications.
Risks	Patients are intermediate risk surgical candidates. May have secondary elevations in PAP leading to right heart dysfunction and elevated risk.
Monitoring	Arterial line for open thoracic procedures. Consider central line based on IV access/surgical risk. TEE/PAC typically not indicated unless severe cardiac disease or PH present. Fiberoptic bronchoscope recommended for ETT/DLT/bronchial blocker placement.
Anesthetic Management	General anesthesia necessary for OLV. Regional analgesia should be considered for open thoracic procedures.

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125 Trauma†

Laura M. Kirk • Alma N. Juels

Few patients offer greater challenge to the anesthesiologist than the acutely injured patient. The urgency of the situation and uncertainty as to the extent of injury requires the anesthesiologist to be methodical and vigilant. This chapter provides a systematic approach to such patients, including airway management, anesthetic technique, and concerns for injury to specific body regions.

1) **Initial assessment:** There is often little time for a standard preoperative evaluation, but all severely injured patients should be considered at risk for aspiration, cervical spine and closed-head injury, hypovolemia, substance abuse, and multisystem injury.[1,2]
   a) **Assume the patient is hypovolemic. Vital signs are insensitive for detecting moderate degrees of blood loss** (Table 125-1).
   b) **A cursory neurologic examination should be performed, noting level of consciousness (Glasgow Coma Scale, Table 125-2), cervical spine tenderness, and lateralizing defects.**
   c) **Gastroparesis and the** likelihood of a full stomach **predisposing to pulmonary aspiration are assumed.**
   d) **Respiratory drive may be impaired** due to head injury or intoxicants, and the mechanical aspects of ventilation may be compromised due to spinal cord, chest wall, or diaphragmatic injury, as well abdominal distention, pulmonary contusion, hemothorax, pneumothorax, etc.
      i) An attempt to oxygenate a hypoxic patient prior to establishing definitive airway control through endotracheal intubation is always advisable.



Trauma patients require a methodical systematic approach to ensure proper care and make sure that critical injuries are not missed.

2) **Airway management:** Establishing airway patency is an essential feature in early trauma care. The injured patient is at risk for a compromised airway for multiple reasons.
   a) **The tongue is the most common cause of an obstructed airway** and is readily treated by anterior displacement of the mandible with a jaw thrust and oral airway.

TABLE 125-1 Classification of Hemorrhagic Shock

	Class I	Class II	Class III	Class IV
Blood loss (mL)	<750	750-1500	1500-2000	>2000
Blood loss (%)	<15	15-30	30-40	>40
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure	Normal	Narrowed	Narrowed	Narrowed
Heart rate	≤100	100-120	120-140	>140
Respiratory rate	14-20	20-30	30-40	>35
Mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic

From Cannon JW. Hemorrhagic shock. *N Engl J Med*. 2018;378(4):370-379. Copyright © 2018 Massachusetts Medical Society. Reprinted with permission from Massachusetts Medical Society.

TABLE 125-2 Glasgow Coma Scale Criteria

	1	2	3	4	5	6
Eyes	Does not open eyes	Opens eyes in response to painful stimuli	Opens eyes in response to voice	Opens eyes spontaneously	N/A	N/A
Verbal	Makes no sounds	Incomprehensible sounds	Utters inappropriate words	Confused, disoriented	Oriented, converses normally	N/A
Motor	Makes no movements	Extension to painful stimuli	Abnormal flexion to painful stimuli	Flexion/ withdrawal to painful stimuli	Localizes painful stimuli	Obeys commands

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The triad of coagulopathy, metabolic acidosis, and hypothermia is lethal and should therefore be managed aggressively.

b) Direct trauma to the face may distort or obstruct the airway through disruption of the supporting bony architecture. Bilateral fractures of the mandible are particularly likely to result in loss of airway patency. Blunt trauma to the anterior neck may fracture the larynx or trachea and precipitate subcutaneous emphysema and soft tissue swelling. Penetrating neck injuries may produce hematomas sufficient to obstruct airflow.[4]
   i) Indications for definitive airway control include the need for airway protection, the need for ventilation and oxygenation, and as part of an ongoing massive resuscitation. These problems may be due to:
      (1) Loss of consciousness
      (2) Severe brain injury (GCS < 8)
      (3) Severe maxillofacial injury
      (4) Airway obstruction
      (5) Risk of pulmonary aspiration
      (6) Apnea
      (7) Chest trauma, including pneumothorax
      (8) Severe brain injury
   ii) Hoarseness, stridor, use of accessory respiratory musculature, and paradoxical motions of the chest or abdominal wall suggest some degree of airway obstruction and impending total airway collapse.
   iii) Cyanosis, pallor, declining pulse oximetry values, and apnea are signs mandating immediate airway intervention.

c) **Cervical spine stabilization.** All forms of airway control, including chin lift, jaw thrust, and oral and nasopharyngeal airway insertion, result in some cervical spine motion; therefore, immobilization in patients with C-spine injury is important.[5]
   i) Airway immobilization precautions include an appropriately sized Aspen collar, sand bags placed on each side of the head and neck, and the patient resting on a hard board with the forehead taped and secured to the board.[6]
   ii) Direct laryngoscopy, even with in-line spine stabilization, results in cervical motion, especially at atlanto-occipital and atlantoaxial levels. Straight and curved laryngoscope blades do not differ substantially in the motion they produce.[5]

d) Difficult intubations require the use of such airway adjuncts as gum elastic bougies, LMA, and fiberoptic bronchoscopes. The possibility of airway trauma must be kept in mind, especially in these situations.

e) **Video laryngoscopes** have demonstrated utility in a variety of clinical settings (emergency department, OB, ICU). These tools have a higher first attempt success rate and provide an improved view of the larynx compared to direct laryngoscopy. For the trauma patient with possible cervical spine injury, video laryngoscopy has not consistently shown to have better first attempt success rates.[7]

f) **The intubating LMA** has led to successful intubations in patients with maxillofacial injuries with possibility of difficult tracheal intubation along with difficult mask ventilation.



The cameras on fiberoptic or video-scopes may become obstructed by blood or gastric contents in the trauma patient. As with any standard intubation, suction should be readily available.

g) Fiberoptic bronchoscopy is considered by some to be the preferred method of securing the airway in patients with potential cervical spine injuries. However, the overall experience using a standard laryngoscope is satisfactory and no particular recommendation can be made based on outcome data.

h) **Failure to establish a definitive airway by intubation necessitates a surgical airway.** Cricothyroid puncture with jet ventilation is a useful temporizing measure prior to cricothyrotomy. Often performing a cricothyrotomy will be a bridging maneuver before formal tracheostomy unless oral intubation can be achieved under less stressful circumstances.

3) **Fluid resuscitation**

a) **Intravenous (IV) access.** Numerous large-gauge IV lines should be inserted (14- and 16-gauge peripheral cannulae and 9 French central introducers are ideal). Placement of IV lines above and below the diaphragm is recommended if physical findings suggest major thoracic or upper extremity vascular injury.

b) **Laboratory evaluations.** Blood should be drawn for type and crossmatch, as well as hematology, chemistry (including lactate), ABG, and coagulation profiles (including thromboelastography [TEG]).

c) **IV infusions** should be warmed, isotonic, and compatible with blood products. Unless the patient is hypoglycemic, avoid glucose-containing products to reduce the risk of CNS injury.

d) Failure to respond to crystalloid can be indicative of the need for blood.

e) **Currently, there is no consensus on the optimal ratio of blood products for damage control resuscitation.**

i) Administration of plasma, platelets, and RBCs in a 1:1:1 ratio closely approximates the components of whole blood.[8]

f) **TEG can guide product administration during massive transfusion protocol (MTP) and management of trauma-induced coagulopathy**[9] (**Fig. 125-1**, **Table 125-3**).

i) MTPs vary by institution. If cross-matched blood is unavailable, transfuse O-negative or type-specific red blood cells. Once available, it is recommended to deactivate the MTP and administer cross-matched blood.

ii) Tranexamic acid (TXA) is an antifibrinolytic that when started within three hours of injury has shown to reduce transfusion requirements and the risk of death due to bleeding.[10]

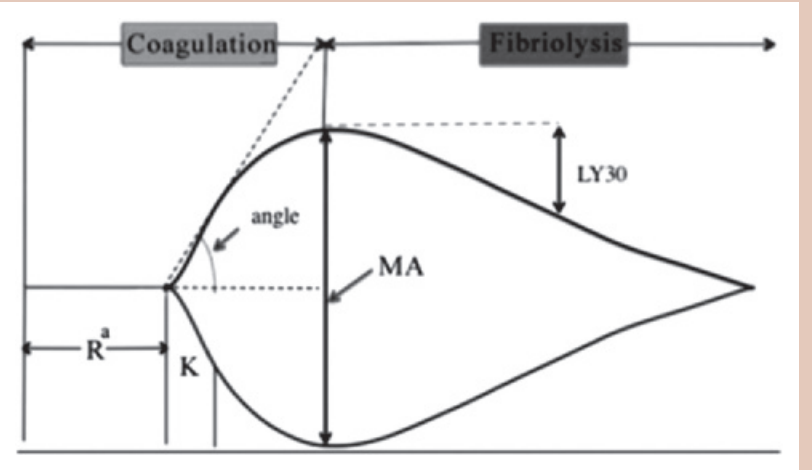
4) **Monitoring**

a) Depending on patient and surgical factors, invasive monitoring, such as intra-arterial and CVP monitoring, offer additional diagnostic information not provided by standard monitors. Patients with closed head injuries may require ICP monitoring. Urine output should also be followed.

READ MORE
Massive transfusion and resuscitation, Chapter 40

b) **Capnography.** Measuring expired CO_2 not only verifies endotracheal intubation but may assist in diagnosis of other important events.

FIGURE 125-1 Characteristic TEG Tracing. R[a]-time is substituted by ACT in rapid TEG.



c) **Trauma patients will have increased gradients between alveolar and end-tidal CO_2 due to increased dead space.** This may be due to hypovolemia, atelectasis, pulmonary contusion, pulmonary aspiration, etc.
   i) Acute, exponential increases in the CO_2 gradient are ominous and may be due to air embolism or acute decreases in cardiac output, such as might occur with myocardial ischemia, cardiac tamponade, or tension pneumothorax.

d) **Temperature monitoring**
   i) Trauma patients usually arrive to the OR hypothermic. The anesthetized state and the OR environment potentiates this problem. Core temperature monitoring is ideal, and esophageal monitoring is the norm.
   ii) Hypothermia impairs wound healing, increases the likelihood of sepsis, slows drug metabolism, and impairs coagulation. The triad of hypothermia, coagulopathy, and acidosis is a lethal combination.
   iii) The OR should be warmed, convective air warming devices employed, and all fluids should be warmed. Wrapping the head or extremities in plastic sheeting may also be helpful.

TABLE 125-3 TEG Parameters and Corresponding Guidelines for Treatment

TEG Value	Transfuse
TEG-ACT > 140	FFP
R time > 10	FFP
K time > 3	Cryoprecipitate
α Angle < 53°	Cryoprecipitate +/– platelets
Maximum amplitude < 50	Platelets
LY30 >3%	Antifibrinolytic (eg, TXA)

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iv) It is important to monitor for fevers in the trauma victim as this may be a sign of a reaction to medications or blood products. Additionally, elevated core temperatures may contribute to increased ICP and adverse CNS effects.

5) **Anesthetic agents:** Anesthetic agents should be titrated to effect, expecting that lower than normal doses may suffice where the patient is hypovolemic.
   a) **Induction agents:** While some agents are preferred for trauma patients, any agent, improperly used, can be deleterious. Two induction agents are preferable in this setting—ketamine and etomidate.
      i) Etomidate has relatively less effect on decreasing CO and SVR, though it causes adrenal depression and the impact of even a single dose in a critically injured patient remains unclear.
         (1) It is the preferred agent in a patient who may have an evolving head injury as it decreases cerebral metabolic activity and is a cerebral vasoconstrictor.
      ii) Ketamine stimulates the sympathetic nervous system and often used in the hypovolemic patient. It does have some direct myocardial depressant effects, but these are noted mostly in catechol-depleted patients.
         (1) Ketamine is a bronchodilator and a cerebral vasodilator and increases cerebral metabolic activity, contraindicating its use where head injury is suspected.

READ MORE
Intravenous induction agents, Chapter 30

      iii) Intraoperative awareness: Trauma patients are at risk for intraoperative awareness. IV benzodiazepines can reduce this risk.
      iv) Nitrous oxide: Nitrous oxide is contraindicated in trauma patients as it can rapidly diffuse into closed gas spaces, increasing the risk of tension pneumothorax, and can also expand any pneumocephalus or air embolism.
      v) Opioids: Opioids are cardiostable though they may potentiate the hypotensive effects of induction agents, volatile anesthetics, and benzodiazepines.
      vi) Muscle relaxants: Succinylcholine is routinely used to facilitate intubation and intermediate or long-acting nondepolarizing relaxants maintain good surgical conditions. Many patients are not extubated at case conclusion.



It is important to recognize the effect benzodiazepines can have on hemodynamic stability. An amnestic dose of IV scopolamine (0.6 mg in the adult patient) is another option that may have less of an impact on BP.[11]

6) **Trauma-related concerns for specific body regions**
   a) **Brain injury**
      i) Secondary CNS injury: Little can be done to reverse the primary, direct CNS effects of acute injury. However, it is absolutely clear that without attention to maintaining intravascular volume, cerebral perfusion pressure, and oxygenation, CNS injuries can be worsened.
      ii) Agents that decrease cerebral metabolic activity are preferred. At times changing from a volatile anesthetic to an IV anesthetic technique may decrease ICP as volatile anesthetics do have cerebral vasodilator effects.
      iii) In the short-term, hyperventilation decreases ICP, but over time, secondary changes in CSF pH decrease its benefit.
      iv) Elevation of the head when tolerated and perhaps loosening slightly a cervical collar (facilitating jugular venous drainage) are also useful techniques.
      v) Once a neurologic exam has been performed (looking in particular for lateralizing defects or posturing), use of muscle relaxants is common.
      vi) Coagulopathy: Patients with brain injuries have a high risk of impaired hemostasis. Serial measures of coagulation function should be performed and defects treated appropriately.

b) **Spinal cord injury**
   i) All injured patients should be considered to have cervical spine injuries until definitively disproven.
   ii) Cervical cord injuries can impair ventilation and result in hypotension due to the inability to produce distal vasoconstriction (neurogenic shock).
   iii) Spinal cord injuries can be worsened by hypotension and hypoxemia.
   iv) Steroid therapy for 24 hours is controversial but often utilized.

c) **Thoracic injury**
   i) Thoracic injuries may be penetrating or blunt in etiology, and include injuries to the heart and pericardium; lung, pleural space, or bronchus; diaphragm.
   ii) Blunt cardiac injury
      (1) Also called myocardial contusion, blunt cardiac injury is a clinical diagnosis. Associated injuries include sternal fractures, rib fractures, and pulmonary contusion.



Blunt cardiac injury can be seen following MVA resulting from the impact between the steering wheel and the driver's chest.

   iii) Dysrhythmias: Sinus tachycardia with nonspecific ST segment changes is most common. Conduction blocks and ventricular rhythms may also be observed.
   iv) Myocardial ischemia: Patients may sustain injury to valves or papillary muscles. Coronary artery thrombosis is possible and most commonly occurs in the right coronary artery (presenting with ischemic changes in inferior ECG leads). Pump failure is an ominous finding.
      (1) Cardiac enzymes add little to the evaluation of a patient suspected of having a contusion.
      (2) Echocardiography is very useful in evaluating segmental wall motion defects.



TTE/TEE has proven useful in the immediate assessment of ventricular function, volume status, and significant aortic or pericardial pathology.[12]

   v) Cardiac tamponade: May arise from either blunt or penetrating trauma.
      (1) Signs and symptoms. Bleeding into the pericardial space increases pericardial pressures and impairs cardiac filling. Positive-pressure ventilation further decreases venous return and may greatly exacerbate the reduction in CO. Stroke volume decreases, and tachycardia compensates for a time to increase CO. If feasible, consider maintenance of spontaneous ventilation through induction and intubation.
      (2) The classic signs associated with cardiac tamponade (Beck triad) are hypotension, distant heart sounds, and distended neck veins. Neck vein distention may not be observed because of hypovolemia.
      (3) Electrical alternans, where the major ECG axis is constantly changing, may be noted. This is due to the heart floating freely in the expanded pericardium.
      (4) A patient with tamponade is at risk for cardiovascular collapse with anesthetic induction. Consider draining the pericardium using local anesthesia at the operative site (a subxiphoid pericardial window) prior to general anesthetic induction.



Hemodynamic goals of the patient with cardiac tamponade include keeping the patient "full, fast, and tight." Maintaining preload (full), avoiding bradycardia (fast), and increasing or maintaining systemic vascular resistance (tight) will help the heart sustain CO.

   vi) Tension pneumothorax and hemothorax
      (1) Patients may sustain a pneumothorax in association with rib fractures, stab wounds, and central line placement. If the pleural cavity does not communicate with the ambient environment, air may accumulate between the chest wall

and lung and expand quickly with positive-pressure ventilation. Eventually, a tension pneumothorax will decrease venous return to the thorax and cause torsion of mediastinal vessels, leading to cardiovascular collapse.

(a) Signs and symptoms: The chest may rise unevenly with inspiration, breath sounds become unequal, the hemothorax is tympanitic to percussion, and the trachea may shift away from the affected side. Neck veins may become distended if the patient is normovolemic. Airway pressures rise.

(i) Treatment: Tension pneumothorax is a clinical diagnosis; do not delay treatment for radiologic confirmation of this life-threatening condition. The immediate treatment is the placement of a large-bore needle through the chest wall in the second intercostal space in the midclavicular line. A rush of air confirms the diagnosis. The needle should be left in place until a tube thoracostomy is performed.



The needle used to treat a tension pneumothorax should be left in place until a tube thoracostomy is performed.

vii) Air embolism: Penetrating lung injuries may result in systemic air entrainment via bronchovenous or alveolocapillary fistulas.

(1) Signs and symptoms: Air embolism should be suspected whenever unexpected signs of CNS or myocardial ischemia and precipitous cardiovascular collapse occur in the appropriate clinical context. When treating patients at risk, minimize inspiratory airway pressure, avoid PEEP, and administer small tidal volumes.

viii) Abdominal compartment syndrome (ACS) is regularly encountered and challenges the anesthesiologist.

(1) A victim of polytrauma with hypotension, oliguria, and respiratory failure manifesting as increasing airway pressures and decreasing oxygenation may have ACS.

(a) Evidence of end-organ damage (eg, acute kidney injury) with a concurrent bladder pressure of >20 mm Hg is diagnostic.

(2) Prompt recognition is important as the treatment, surgical decompression, is usually straightforward. Low CO may be associated with elevated pulmonary arterial occlusion pressure, somewhat similar to what might be seen in cardiac failure, calling into question optimal fluid management. A trial of volume expansion is usually indicated when a pathologic increase in abdominal pressure is suspected despite seemingly normal intravascular status as assessed by invasive hemodynamic monitors.

7) **Damage control:** Damage control is the principle of performing the minimum necessary interventions to save life and limb, leaving further procedures for a later time, after the patient has obtained hemodynamic stability. Abbreviating the initial procedure has the advantage of decreasing the incidence of metabolic acidosis, hypothermia, and coagulopathy, a triad known to have a high incidence of mortality.[13]

CHAPTER SUMMARY FOR TRAUMA

Trauma	Patients require a systematic, methodical approach.
Initial Assessment	Assume hypovolemia, full stomach, perform history and physical exam, assign GCS score.
Airway Management	Evaluate need for intubation. In-line cervical spine stabilization may be required. Have emergency airway devices ready.

Fluid Resuscitation	Establish large bore IV access, send laboratory studies (T&C, CBC, coagulation studies, electrolytes, ABG), blood transfusion may be required.
Monitoring	Invasive monitors may be required, temperature monitoring essential.
Anesthetic Management	All induction drugs can potentially cause hemodynamic compromise, etomidate and ketamine most often recommended, consider high risk of awareness, nitrous oxide contraindicated with certain injuries, neuromuscular blockade commonly used.

ABG, arterial blood gas; CBC, complete blood count; GCS, Glasgow coma scale; T&C, type and cross.

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126 Liver and Kidney Transplantation§

Kyota Fukazawa • Tzevan Poon

Major recent advancements in perioperative management have made liver or kidney transplant the definitive treatment option for patients with irreversible end-stage liver disease (ESLD) or end-stage renal disease (ESRD). The current severe shortage of donors and the increased severity of illness of patients on the waiting list have led to the increased use of extended criteria donors. Anesthetic management presents complex challenges, and understanding the pathophysiology of the donor, the recipient, as well as the surgical procedure is imperative for the safe management of liver and kidney transplant patients.

1) **Donor categorization**
   a) **Donor after brain death (DBD):** Donors are patients with catastrophic brain injury and meet strict criteria for brain death.
      i) Organs are better preserved because the heart is still beating.
      ii) This is the most common donor type for liver transplants.[1]
   b) **Donor after cardiac death (DCD):** Donors from patients who do not meet the criteria for brain death but have no meaningful chance of survival due to injury or illness and are likely to suffer cardiac arrest if life support is withdrawn.
      i) DCD organs inevitably suffer significantly more warm ischemia than DBD organs.
      ii) Examples of DCD: terminal patients with high spinal injuries, end-stage amyotrophic lateral sclerosis, and severe traumatic/anoxic brain injury in those who do not meet the criteria for brain death.
      iii) Procedure of organ procurement for DCD donors:
         (1) Withdrawal of life support by ICU team in the OR
         (2) Asystole must occur within 30 minutes or organs are not used.
         (3) Three to five-minute observation period to rule out autoresuscitation.
         (4) Transplant team enters the room to harvest the organ.
   c) **Live organ donors** are common in kidney transplantation but only make up <4% of all liver transplants in the United States.
   d) **Extended criteria donors (ECD):** the criteria for extended criteria donors by the United Network for Organ Sharing (UNOS) are as follows:
      i) Age over 60
      ii) Age over 50 with at least two of the following conditions:
         (1) Hypertension
         (2) Creatinine >1.5 mg/dL
         (3) Cause of death from cerebrovascular accident
      iii) ECD decrease the overall waitlist time mortalities, but patients may be at increased risk of delayed graft function or early/late graft failure.
   e) **Increased risk donors (IRD)**: donors carry an increased risk of infectious disease transmission such as HIV and hepatitis B and C.
      i) Donors test seronegative but may still be in the window period, testing positive at a later date.
      ii) May include IV drug users, prostitutes, recently incarcerated individuals, or those with other risk factors.

iii) A recent multivariate analysis demonstrated similar outcomes for mortality and graft failure compared to non-IRD donors.[2]

READ MORE
Diseases of the liver and biliary tract, Chapter 56.

2) **Liver transplantation**
   a) **Organ allocation**[1]
      i) Tier 1 allocation (status 1 A/B)
         (1) Expedited organ allocation for acute/fulminant liver failure without preexisting liver disease or for acute retransplant (<1% of liver transplant candidates)
      ii) Tier 2 allocation
         (1) Other conditions (such as decompensated chronic liver failure) are allocated based on MELD/PELD score.
      iii) Model for end-stage liver disease (MELD)/pediatric end-stage liver disease (PELD) score: originally developed to predict survival of cirrhotic patients without transplant. MELD was subsequently found to also predict 3-month survival while on the liver transplant waiting list.
      iv) MELD score for patients age >12 years old; PELD score for patients age <12
      v) MELD score is calculated based on:
         (1) Serum bilirubin
         (2) INR
         (3) Creatinine with/without Na^+
         (4) MELD calculator can be accessed at www.UNOS.org.
      vi) Liver grafts are typically allocated to patients within a designated geographic region with the highest MELD/PELD scores.
      vii) Patients may qualify for "MELD exception points" that raise their rank on the waiting list for certain medical conditions that benefit from early transplantation (eg, hepatocellular carcinoma, portopulmonary HTN, hepatic artery thrombosis, etc.).
   b) **Preoperative considerations**
      i) Cardiovascular
         (1) Consider using a pulmonary artery (PA) catheter and/or transesophageal echocardiography (TEE) to aid in hemodynamic decision-making.
         (2) Expand intravascular volume judiciously, while supplementing with vasoconstrictors in low systemic vascular resistance (SVR) states.
         (3) Consider techniques to preserve preload (eg, veno-venous bypass, portal caval shunt) in select situations during the anhepatic phase, as preload can significantly fall.
         (4) Cutaneous pacing/cardioversion pads should be placed as unstable arrhythmias may occur during major fluid and electrolyte shifts (eg, during reperfusion, etc.).
      ii) Pulmonary circulation
         (1) Portopulmonary hypertension (PPHTN)
            (a) Right heart cath should be performed if right ventricular systolic pressure (RVSP) estimation by TEE is >40-50 mm Hg. If PPHTN is confirmed, close coordination and discussion with other members of the transplant team is essential prior to listing and surgery.
            (b) PA catheter and TEE may also be helpful guides to therapy in patients with PPHTN.[3]
            (c) Preoperative and intraoperative pulmonary vasodilators may be beneficial.
         (2) Hepatopulmonary syndrome (HPS)
            (a) Patients with significant HPS usually require high FiO_2 in order to achieve sustainable tissue oxygenation. Continuous or serial MVO_2 and/or lactate measurements are reasonable surrogates of tissue perfusion when O_2 saturation is low or unreliable.

(b) Veno-venous bypass or veno-venous extracorporeal membrane oxygenation (VV ECMO) may be necessary in particularly severe cases. Remove all air in IV lines to prevent right-to-left air emboli.

iii) Respiratory
(1) Consider using a lung protective ventilation strategy (tidal volumes of 6-8 mL/kg, titration of PEEP, plateau pressures <30 cm H_2O).
(2) Consider draining pulmonary effusion(s) if evidence of severe compressive atelectasis.

iv) Renal
(1) Maintain renal perfusion pressure through adequate preload and judicious vasopressor use.
(2) Pretransplant dialysis and/or intraoperative dialysis may be beneficial in high-risk patients.[4]

v) Metabolic
(1) Correct significant metabolic derangements prior to surgery. Intraoperatively, use sodium bicarbonate or intraoperative dialysis for significant metabolic acidosis.

vi) Hematologic
(1) Assess for clinical signs of bleeding and treat if indicated. Efficacy of prophylactic correction of mild-moderate coagulopathy without evidence of bleeding is unproven. Use of TEG to guide therapy may be beneficial.
(2) Patients with liver disease are functionally immunosuppressed, and meticulous sterile technique should be observed (ports swabbed with alcohol, full sterile technique when placing lines, etc.).

vii) Central nervous system
(1) Avoid benzodiazepines, which may perpetuate delirium.
(2) For acute and fulminant hepatic failure, consider ICP monitoring, and maintain cerebral perfusion pressure >60 mm Hg.

c) **Intraoperative considerations**

i) Concise overview of the surgical procedure
(1) There are three major stages during liver transplant.
(a) Preanhepatic (dissection) phase: incision to portal vein clamp
(i) Dissection of the liver and isolation of major abdominal vessels
(ii) Isolation of porta hepatis
(iii) Mobilization of the liver to access IVC
1. Major hemodynamic fluctuation may occur due to compression of IVC.
(iv) Portal vein clamp
(b) Anhepatic phase: portal vein clamp to reperfusion
(i) IVC clamps placed (partial clamp for piggyback, complete clamp for traditional anastomosis)
(ii) Cirrhotic liver removed
(iii) Donor graft placed on the field (warm ischemia time begins)
(iv) Hepatic vein/IVC anastomoses
(v) Portal vein anastomosis
(vi) Flush organ (to reduce reperfusion electrolyte shifts)
(c) Neohepatic phase: reperfusion to abdominal closure
(i) Reperfusion occurs when IVC outflow tract and portal vein anastomoses are opened.
(ii) Hepatic artery anastomosis is performed.
(iii) Bile duct anastomosis is performed.
(iv) Field is checked for hemostasis and abdominal closure.



The surgical procedure for liver transplant involves three distinct stages: preanhepatic (dissection), anhepatic, and neohepatic stages.

ii) General anesthetic considerations
  (1) Infectious concerns
    (a) Due to the high prevalence of blood-borne pathogens in liver transplant recipients, especially hepatitis C, all medical personnel should use universal precautions.
    (b) Patients will require immunosuppression, so every effort must be made to avoid infectious complications. Use meticulous aseptic technique for all invasive lines and when accessing IV ports.
    (c) Administer appropriate perioperative antibiotics. Cover gram positive and gram negative organisms, and fungi.
    (d) If blood loss is massive or the procedure long, consider redosing antibiotics.
  (2) Monitoring
    (a) In addition to standard ASA monitors, an arterial catheter should be placed for serial laboratory measurements and beat-to-beat blood pressure monitoring as rapid hemodynamic change often occurs during cases.
    (b) An 8.5-9 Fr introducer central venous catheter should be used for rapid infusion of fluids, blood, drugs, and transduction of central venous pressure (CVP).
      (i) CVP is used to optimize venous return from the transplanted liver to the heart and systemic circulation.
    (c) Many transplant centers place PA catheters for continuous measurement of cardiac output, PA pressures, SVR, and MVO_2.
    (d) TEE use during liver transplantation varies widely between institutions.
  (3) Induction and maintenance
    (a) Proper preoxygenation is essential.
    (b) Propofol and etomidate are appropriate induction agents.
    (c) Rocuronium, succinylcholine, and cisatracurium are reasonable paralytic agents, depending on the clinical context.
    (d) Volatile anesthetics are suggested for maintenance.
    (e) Vasopressor infusions and bolus syringes (norepinephrine, phenylephrine, epinephrine, etc.) should be available for immediate use.
      (i) Patients with severe liver disease are typically highly vasodilated and relatively vasoplegic, so higher doses and higher potency pressors may be required.
  (4) Overall anesthetic goals
    (a) The overall goal is to maintain optimal microcirculatory tissue perfusion despite significant homeostatic changes during transplant surgery.
    (b) Monitor tissue perfusion through a variety of information streams including SpO_2, lactate trends, MVO_2, blood pressure, pulse pressure variation, cardiac output, urine output, visible blood loss, and/or TEE.
    (c) Intravascular volume management: the Frank-Starling curve is the relationship between stroke volume (or MAP as a surrogate measure) in response to increases or decreases in preload.
      (i) A central goal for fluid resuscitation is to stay on the "steep part" of the Frank-Starling curve. This means that



Liver failure patients often have full stomachs and have decreased FRC, necessitating the use of a rapid-sequence induction.



Patients with severe liver disease are typically highly vasodilated and relatively vasoplegic, so higher doses and higher potency pressors may be required.



The overall goal is to maintain optimal microcirculatory tissue perfusion despite significant homeostatic changes during transplant surgery.

reasonable volume boluses (increasing preload) should be given so far as it leads to meaningful increases in MAP (typically MAPs 50-70s mm Hg). Maintaining this delicate balance is critical to preventing complications from under-resuscitation (acidosis, ischemia, etc.) and over-resuscitation (tissue edema, pulmonary edema, coagulopathy, acidosis, etc.).

(d) Prevent and treat the "lethal triad:" hypothermia, acidemia, and coagulopathy.
  (i) Maintenance of normothermia promotes proper coagulation and optimizes many other homeostatic mechanisms.
    1. Use fluid warmers, bed warmers, forced heated air devices, control of ambient temperature, and warming via veno-venous bypass or hemodialysis as appropriate.
  (ii) Acidemia has a direct impact on coagulation, cardiac contractility, vasoactive tone, response to pressors, and a host of other essential factors. Avoid severe acidemia, and promptly treat worsening trends.
    1. Consider using balanced crystalloids (pH 7.4) over more acidic crystalloids (normal saline).

(e) Ionized calcium is a central ion for proper coagulation and cardiac and vascular contractility. Correct hypocalcemia, and be cognizant that large volume transfusions will decrease calcium levels (citrate load chelates Ca^{2+}).

(f) Management of sodium, potassium, and glucose (**Table 126-1**)
  (i) Particularly in patients with low serum Na^+ (<125 mEq/L), avoid major changes in serum Na^+ to prevent cerebral edema and delayed central pontine myelinolysis.
  (ii) Hyperkalemia should be treated aggressively.
  (iii) Closely monitor and treat hypo- and hyperglycemia during transplant.

READ MORE
Blood component therapy, Chapter 38

(5) Management of hemorrhage and coagulopathy
  (a) Management of hemorrhage and coagulopathy can be broken down into two categories: empiric therapy when data are lacking (eg, 1:1 ratio of pRBC:FFP ± platelets) and goal-directed therapy (repletion of blood components based on specific deficits) when data become more available.
  (b) Routine prophylactic full correction of PT, aPTT, and platelet count is controversial and should depend more on the clinical situation.
  (c) Draw serial labs including rapid Hb/Hct, platelet count, PT/INR, fibrinogen, electrolytes, lactate, and blood gasses.
  (d) Thromboelastography (TEG) is often used as an adjuvant to monitoring coagulation and effects of blood component therapy. TEG is currently the only commonly available rapid test to quantify hyperfibrinolysis.[5]
  (e) Bleeding is often due to highly vascular portosystemic collateral veins, adhesions, and coagulopathy. Impaired coagulation is often multifactorial,

TABLE 126-1 Medical Therapies to Treat Hyperkalemia During Liver Transplant

Medical therapies to treat hyperkalemia during liver transplant include:
1. Calcium chloride
2. Hyperventilation
3. Sodium bicarbonate
4. Albuterol
5. Early administration of furosemide in patients with intact renal function
6. Wash pRBC with cell saver machine
7. Early use of intraoperative hemodialysis

resulting from a combination of coagulopathy, thrombocytopenia, platelet dysfunction, hyperfibrinolysis, hypocalcemia, acidemia, hypothermia, and poor graft function.

(f) Drugs such as antifibrinolytic agents (α-aminocaproic acid, tranexamic acid), DDAVP (arginine vasopressin), and prothrombin complex concentrates should be considered on a case-by-case basis.

iii) Specific anesthetic management during each phase of transplant (**Fig. 126-1**)

(1) Preanhepatic phase:

(a) Pay meticulous attention to the "overall anesthetic goals" mentioned above. Close communication with transplant surgeons is essential.

(b) Draining of large volume ascites upon opening of the abdomen often leads to hypotension due to an increase in splanchnic venous capacitance and a drop in preload upon rapid decrease in intra-abdominal pressure.

(i) This can be managed with colloid loading and infusion with vasopressors (eg, norepinephrine infusion).

(c) Dissection of adhesions (from prior abdominal surgery, episodes of spontaneous bacterial peritonitis, or radioembolization therapy with radioactive isotope yttrium Y-90 for hepatocellular carcinoma), diffuse tortuous collaterals, IVC dissection, and isolation of major abdominal structures can lead to massive bleeding.

(d) Manipulation of the liver can also cause major hemodynamic fluctuations.



Major hemodynamic instability can occur during partial or full clamping of the IVC. Instability can also occur but is less common with portal vein and hepatic artery clamping as most patients have developed sufficient venous collaterals.

FIGURE 126-1 Stereotypic Hemodynamic Changes, Key Events, and Management Goals During Liver Transplant.

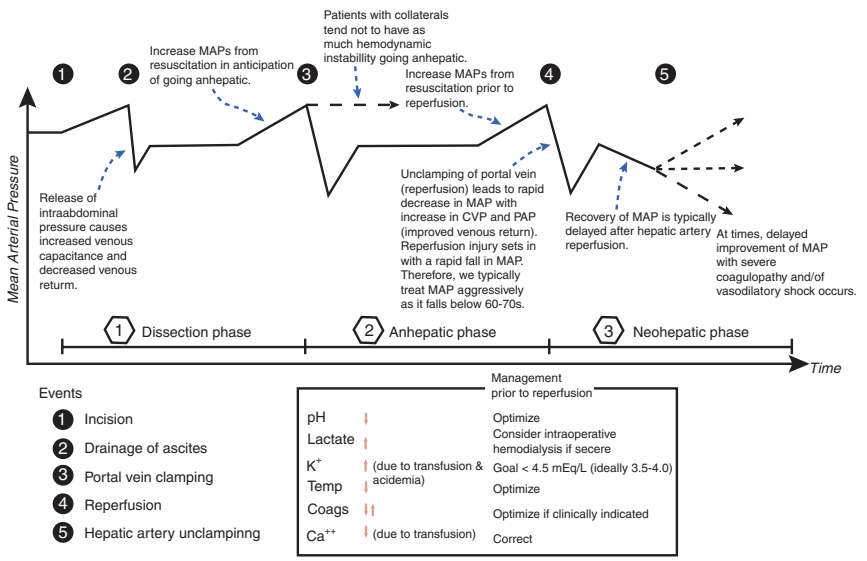


Illustration courtesy of Kyota Fukazawa, MD, PhD and Tzevan Poon, MD.

(e) It is often difficult for patients with liver failure undergoing laparotomy to maintain body temperature once they become hypothermic.

(2) Anhepatic phase
   (a) This phase has additional hemodynamic considerations related to IVC occlusion.
   (b) There are two IVC clamping techniques: full or partial clamping (**Fig. 126-2**).
   (c) There are two major caval anastomotic types.
      (i) Conventional caval replacement technique
      (ii) Piggyback caval replacement technique
   (d) Acute hypotension is common secondary to decreased preload from IVC clamping. Surgeons will typically do a temporary "test" clamp to check a patient's hemodynamic tolerance.
   (e) Judicious use of volume and pressors during this phase is recommended, as overzealous resuscitation during this period can worsen venous congestion and risk subsequent volume overload after caval unclamping.
   (f) Veno-venous bypass is used by some centers to maintain cardiac preload during the anhepatic phase and decrease hemodynamic instability (**Fig. 126-3**). Veno-venous bypass is an area of controversy. Many centers have significantly reduced or completely stopped using bypass

FIGURE 126-2 Different Types of IVC Clamping Maneuvers During Liver Transplant. Traditionally a standard full caval clamp was done, and the recipient cava was replaced *en bloc* with the donor IVC. However, partial clamping has become more common due to greater hemodynamic stability, reduced need for veno-venous bypass, an association with lower consumption of blood products, shorter warm ischemia times, and reduced hospital costs with a similar low incidence of venous drainage issues when compared to the standard technique.

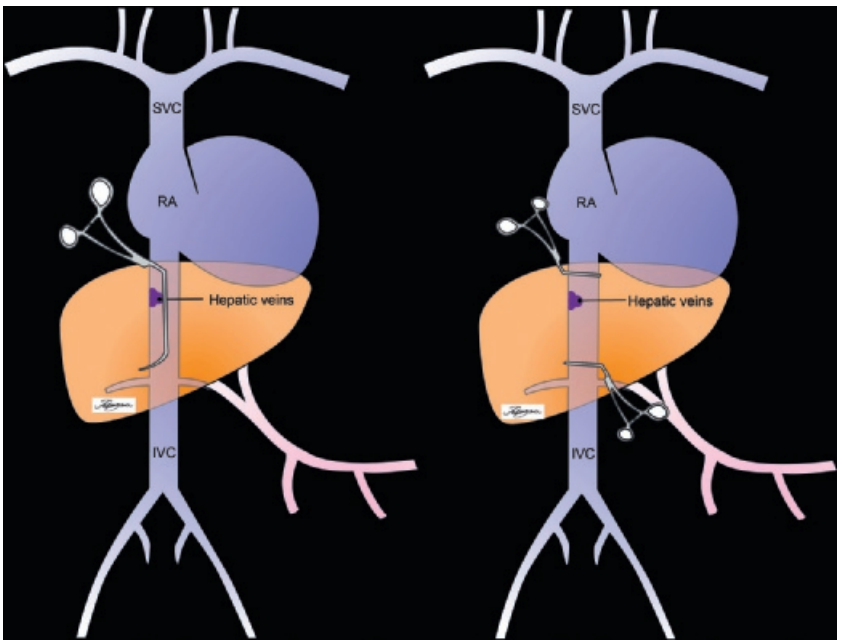


Illustration courtesy of Kyota Fukazawa, MD, PhD and Tzevan Poon, MD.

FIGURE 126-3 Veno-Venous Bypass Circuit. Blood is drained from below the inferior vena caval clamp and pumped back into (typically) the axillary vein, thereby improving venous return and reducing sequelae from the fixed caval obstruction. Complications may include bleeding, thrombosis, air embolus, cannulation issues, poor flow through the circuit ("chugging"), and injury to anatomic structures.

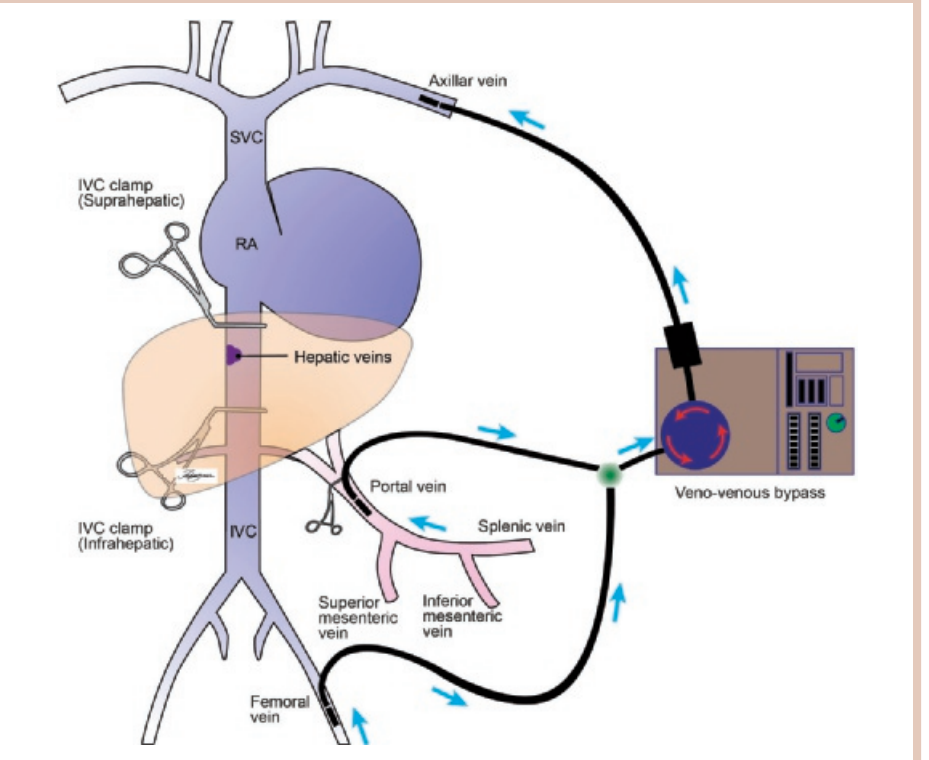


Illustration courtesy of Kyota Fukazawa, MD, PhD and Tzevan Poon, MD.

due to studies showing an association with increased complications and costs. Other techniques (eg, partial IVC clamping instead of full clamping, piggyback caval anastomotic techniques, portal caval shunt placement) have supplanted bypass at these centers.

(3) Neohepatic phase: starts with removal of vascular clamps, resulting in reperfusion of the donor graft. It encompasses surgical reconstruction of the biliary tree and the conclusion of the surgery.
   (a) Portal vein unclamping (liver reperfusion) allows oxygenated recipient blood to enter the ischemic donor graft. See Figure 126-1 on preparation prior to reperfusion.
      (i) Postreperfusion syndrome (PRS)[6]
         1. Defined as hypotension (decrease in MAP > 30%) manifesting within 5 minutes after portal vein unclamping, sustained more than 1 minute[7]



Close communication with the surgeon is necessary to allow adequate preparation for the hemodynamic consequences of unclamping.



Reperfusion syndrome occurs in up to 30% of liver transplants.

2. Caused by rapid systemic circulation of graft effluent and ischemic blood (cold, acidic, hyperkalemic, fluid with a high degree of vasoactive inflammatory mediators)
3. Symptoms include systemic vasodilation, severe vasoplegic hypotension, possible dysrhythmias, and reduced cardiac contractility.
4. Severity of symptoms is related to the donor graft, ischemia time, and recipient condition.
5. Prompt boluses of pressors such as epinephrine or (as a rescue) vasopressin may be required. Dysrhythmias may occur and require prompt pharmacologic therapy or cardioversion/defibrillation.

(ii) Hepatic artery unclamping. Retained vasoactive inflammatory mediators in the graft may be flushed out from the graft by hepatic artery reperfusion leading to hypotension.

(b) Assessment of *in situ* graft function. Markers of adequate liver graft function in the operating room include improved lactate metabolism, hyperglycemia (glycogen metabolism), maintenance of urine output (in patients with normal or mildly reduced renal function), improvement in body temperature, visible bile production, and in some cases rapidly decreasing pressor requirement.

iv) Postoperative management

(1) Extubation. With the use of short-acting agents for anesthesia, extubation is possible in the operating room or within a few hours after admission to ICU.[8]

(a) The decision to extubate should be based on patient stability, graft function, posttransplant ultrasound results, and transplant center policies.

(2) Laboratory monitoring. Serial blood counts, chemistries, liver function, and coagulation indices are routinely measured. Transfusion goals vary and are institution dependent.

(a) Transfusion of platelets for thrombocytopenia in patients without clinically significant bleeding is controversial, with a number of studies showing an association with worsened overall survival and/or decreased graft survival after platelet transfusions.[9–11]

(3) Patients with hepatic encephalopathy may take days to improve.

(4) Patients with hepatorenal syndrome on hemodialysis typically take weeks to months to regain kidney function.

(5) Pain control. Extubated patients without significant hepatic encephalopathy are often excellent candidates for patient-controlled analgesia and early mobility protocols.

3) **Kidney transplantation**

a) **Overview**

i) Offered to end-stage renal disease (ESRD) patients to allow freedom from dialysis

ii) Classified as deceased donor or living donor transplantation

(1) Living donor renal transplants are further characterized as genetically related (living-related) or nonrelated (living-unrelated) transplants.

b) **Preoperative considerations**

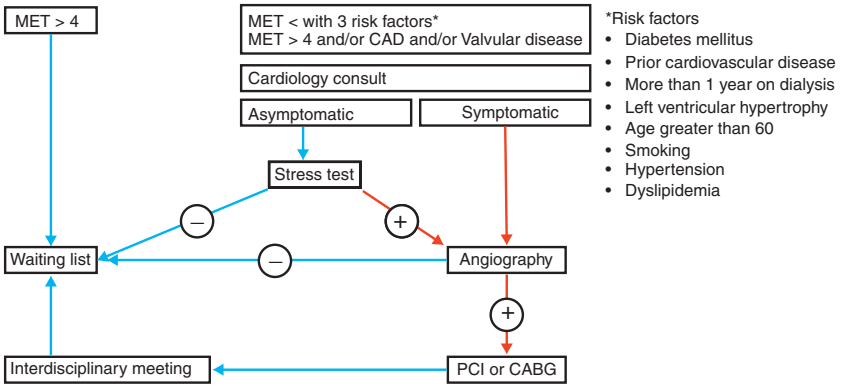
i) Preoperative evaluation

(1) Prospective candidates often have documented coronary artery disease, peripheral vascular disease, and heart failure, or have multiple risk factors (advanced age, diabetes, hypertension, and chronic renal disease).[12]

(2) A complete history and physical examination, laboratory workup, ECG, and chest x-ray are typically done.

(a) If the room air SpO_2 is <92%, arterial blood gas and/or pulmonary function tests should be obtained.

FIGURE 126-4 Flow Chart for Cardiac Evaluation in Kidney Transplant. CAD, coronary artery disease; CABG, coronary artery bypass graft surgery; PCI, percutaneous coronary intervention.



Reprinted from Lentine KL, Costa SP, Weir MR, et al. Cardiac disease evaluation and management among kidney and liver transplantation candidates: a scientific statement from the American Heart Association and the American College of Cardiology Foundation. *J Am Coll Cardiol*. 2012;60(5):434-480, with permission from Elsevier.

(3) The cardiac assessment flow sheet in **Figure 126-4** can be used as a guide.[13]
(4) Dialysis status
  (a) Patients should be queried about how often they are dialyzed, the date/time of their last dialysis, their baseline dry weight, and whether they produce urine.
  (b) If dialysis has not been performed within 6-8 hours of presenting to the operating room, serum potassium should be checked and volume status carefully assessed.



For kidney transplant patients, preoperative evaluation for coexisting cardiac disease and cardiac function is of primary importance.

c) **Intraoperative considerations**
  i) Surgical procedure
    (1) Generally performed in a heterotopic fashion, with the donor graft being placed in a nonanatomic location such as the iliac fossa
      (a) Placement of donor graft in normal (orthotopic) position is usually not performed since this anatomically challenging.
    (2) The renal vein of the new kidney is often connected to the recipient's external iliac vein.
    (3) The renal artery of the donor kidney is often connected to the external iliac artery in the recipient.
    (4) Placement of retraction may cause injury to the lateral femoral cutaneous nerve and femoral nerve.
    (5) After all anastomoses, the low pressure clamps (superior venous clamp and inferior arterial clamp) and then the high pressure clamps (inferior venous clamp and superior arterial clamp) are removed.
    (6) Reperfusion: after ensuring no bleeding at anastomotic sites, the renal vein clamp and then the renal artery clamp are removed.
    (7) Infusion of antibiotic solution is infused into the bladder for identification, and the Foley catheter is clamped.
    (8) The ureter is then anastomosed to the bladder.

ii) Intraoperative management
  (1) Monitoring
    (a) Many centers routinely use only standard ASA monitors.
    (b) Arterial blood pressure monitoring and CVP monitoring may be considered based on patient comorbidities and institutional practice.
  (2) Induction and maintenance
    (a) Induction
      (i) Propofol and etomidate are reasonable induction agents to use. The free fraction of etomidate is increased in patients with kidney disease, but the clinical effects are marginal.
      (ii) Consider rapid sequence induction depending on the NPO status and comorbidities of the patient (many recipients arrive to hospital from home on short notice).
      (iii) Paralytic agents with heavy reliance on renal metabolism are best avoided.
    (b) Maintenance
      (i) Can be achieved by a combination of opioid and volatile agents
      (ii) Paralytics, opioids, and volatile anesthetics should all be titrated with the goal of successful extubation at the conclusion of the procedure.
      (iii) Avoid use of ketorolac (renal toxicity), meperidine, and morphine (due to reduced renal clearance of these agents and their metabolites).
  (3) Intraoperative goals
    (a) One of the main goals in the perioperative management of kidney transplant patients is optimization of renal perfusion to facilitate immediate graft function.
    (b) The most important intraoperative measure that improves the probability of immediate graft function is adequate intravascular volume.
    (c) However, this goal must be balanced with the risk of fluid overload, which can lead to ventricular distention and pulmonary edema since many recipients have preexisting coronary artery disease or heart failure.
    (d) Commercially available isotonic crystalloid solutions have different effects on electrolytes and acid-base balance.
      (i) Normal saline is commonly used for kidney transplant because it is free of potassium. However, it causes hyperchloremic metabolic acidosis in larger volumes.
      (ii) Lactate Ringer solution is potentially safer option since it is associated with less hyperkalemia and acidosis compared to 0.9% normal saline.[14]
      (iii) Albumin use has been associated in some studies with improved graft perfusion and faster improvement in urine output.
    (e) Mannitol, loop diuretics, dopamine, and occasionally fenoldopam can be used to increase urine output and improve graft function. Only mannitol has been shown to decrease the incidence of acute tubular necrosis after transplant.
    (f) Immunosuppression (ie, antithymocyte globulin [ATG], basiliximab, alemtuzumab, etc.) is typically started intraoperatively as this has been shown to reduce rates of delayed graft function when compared to postoperative infusion.[15]



The most important intraoperative measure that improves the probability of immediate graft function in patients undergoing kidney transplant is adequate intravascular volume.



Immunosuppressive medications used intraoperatively can lead to a severe systemic inflammatory response: hypotension/hypertension, tachycardia, fever, anaphylaxis, etc. Patients receiving these agents should be pretreated with systemic steroids.

iii) Postoperative considerations
   (1) Most patients meet standard criteria for extubation at the conclusion of the procedure and can recover in the postanesthesia unit.
   (2) Recipients with severe respiratory or cardiac disease, or patients who become unstable intraoperatively, may need to be admitted to the ICU for further management.
   (3) Urine output should be closely monitored. Immediate urine production is seen in over 90% of living donor transplants and between 40% and 70% of deceased donor transplants. Protocols and strategies for replacing urine output with intravenous fluids vary among institutions.

CHAPTER SUMMARY FOR LIVER AND KIDNEY TRANSPLANTATION

Liver Transplant Key Points	Patients undergoing liver transplant often have severe multiorgan dysfunction and are at high risk for perioperative death and disability Liver transplant surgery is defined by three major phases: preanhepatic/dissection, anhepatic, and neohepatic phases. Figure 126-1 provides a useful template to anticipate and plan for key events during transplant Communication with the surgical team throughout the case is essential The key goal during liver transplant is to optimize tissue perfusion. This takes the form of a number of basic physiologic tenets Stay on the "steep part" of the Frank-Starling curve (MAPs 50-70s) with reasonably sized volume boluses leading to meaningful increases in MAP. This delicate balance prevents the major harmful effects from under- or over-resuscitation Prevent and treat the lethal triad of hypothermia, acidemia, and coagulopathy Proper planning will help prevent many of the most serious complications during reperfusion Massive hemorrhage is common. Empiric and goal-directed transfusion and resuscitation strategies used in trauma care can be successfully applied to liver transplantation
Kidney Transplant Key Points	Patients undergoing kidney transplant often have coronary artery disease, heart failure, or peripheral vascular disease Patients should ideally be dialyzed and close to their dry weight prior to surgery in anticipation of volume loading during surgery. Labs should be checked preoperatively and major derangements corrected prior While expansion of intravascular circulating volume is imperative for successful early renal graft function, this must be balanced with the potential for complications from volume overload Immunosuppression is often started in the OR. Be on the lookout for side effects and complications from these agents Kidney transplant recipients are often successfully managed and extubated in the OR, recovered in the postanesthesia recovery unit, and transferred to the ward for the remainder of their care A smaller subset may require ICU care

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127 Out of OR Anesthesia and Transport

Carlee A. Clark • Jeffrey McMurray

The number of anesthetics delivered at sites outside of the operating room (OR) has been steadily increasing. As procedures continue to move toward the minimally invasive, there will be a continued need for anesthesiologists to provide the quality of anesthesia expected in the OR at remote locations. With each out of OR location, there are unique challenges for the anesthesia team. The goal is to hold these sites to the standards of the OR. An easy way to prepare is to think about each case in terms of how the anesthetic is influenced by the patient, the procedure and the environment.

1) **Overview**
   a) **All patients should have a thorough preoperative anesthesia evaluation.**
   b) **An anesthetic plan should be formulated by determining the unique needs of the patient.**
      i) Take into consideration the anticipated procedure and environment.
      ii) Special equipment should be anticipated and ordered in advance.
   c) Patients requiring the presence of an anesthesiologist outside of the OR are frequently high-risk, difficult to sedate, or may have a movement disorder.
      i) A high-risk patient can have comorbidities. Some examples include:
         (1) A morbidly obese patient with obstructive sleep apnea (OSA)
         (2) An elderly patient with aortic stenosis or significant coronary artery disease (CAD)
         (3) A patient who is not a candidate for invasive surgery due to the extent of his/her disease and presents for a less invasive procedure
      ii) Difficult-to-sedate patients may have:
         (1) High levels of anxiety
         (2) Psychiatric disorders
         (3) Chronic opioid, alcohol, or benzodiazepine use
         (4) Mental status changes
         (5) Dementia
      iii) Patients with movement disorders may require an anesthesiologist to help provide patient immobility.



For out of OR anesthesia, prepare for each case by thinking about how the anesthetic is influenced by the patient, the procedure, and the environment.

READ MORE
Anesthetic plan and induction of anesthesia, Chapter 3

2) **Procedures (Table 127-1)**
   a) Most out of OR procedures are radiographic (magnetic resonance imaging [MRI]/computed tomography [CT]), percutaneous, endoscopic, or electroconvulsive in nature.
   b) These procedures usually do not involve major surgical interventions but can involve manipulation of large blood vessels (eg, transjugular intrahepatic portosystemic shunt [TIPS]).
   c) Some examples of procedures frequently performed out of the OR are summarized in **Table 127-1**.

TABLE 127-1 Procedures Frequently Requiring Out of OR Anesthesia

Neuroradiology	• Cerebral angiography • Cerebral stenting • Cerebral coiling • Mechanical thrombectomy
Radiology	• MRI • CT • Interventional radiology • TIPS
Cardiology cath lab	• Cardioversion • Radiofrequency ablations/EP studies • AICD testing • Coronary angiography/angioplasty/stenting
PACU/interventional psychiatry	• Electroconvulsive therapy
Endoscopy	• Upper endoscopy • Esophageal dilatation/stenting • ERCP • Colonoscopy/sigmoidoscopy
Intensive care unit	• Tracheostomy • Endoscopic procedures

3) **Environment**
   a) Out of OR locations range from a crowded intensive care unit (ICU) room to state-of-the-art endoscopy and fluoroscopy suites.
   b) Not all sites were developed with the anesthesiologist in mind.
      i) Adaptability and having a plan are very important.
   c) The goal is to provide a safe anesthetic for the patient.
   d) There are ASA guidelines for anesthesiologists to apply to each new environment before administering anesthesia (**Table 127-2**).
4) **Anesthetic considerations for specific procedures**
   a) **Magnetic resonance imaging**
      i) Indications for scans with anesthesia
         (1) Pediatric patients and in adult patients with movement disorders, psychiatric disorders, claustrophobia, or OSA
      ii) Technique
         (1) MRIs can take several hours, and patient immobility may be critical for adequate imaging.
            a) Can require sedation or a general anesthetic
         (2) Most patients receive propofol for sedation, with a total intravenous anesthesia technique for general anesthesia.
         (3) General anesthesia with LMA or endotracheal intubation is an acceptable alternative.



For adequate MRI imaging, the patient often needs to be immobile.

      iii) Anesthetic considerations
         (1) Patients are positioned at a distance from the anesthesiologist, and there must be access to ensure that they can be viewed at all times via a window or a video screen or the provider can remain next to the MRI scanner.
         (2) Patient monitors must be visible.
         (3) Airway equipment, medication pumps, poles, monitors, and the anesthesia machine must be MRI compatible.
            (a) MRI compatible equipment is MRI safe, works correctly in the presence of the magnet, and does not interfere with the correct operation of the MRI equipment.

TABLE 127-2 ASA Guidelines for Nonoperating Room Anesthetizing Locations[1]

Oxygen	• Reliable source and quantity • Backup cylinder—full
Suction	• Adequate and reliable
Scavenging	• Adequate and reliable when administering anesthetic gases
Anesthetic equipment	• Self-inflating bag for PPV providing at least 90% oxygen • Drugs, supplies, and equipment for intended level of anesthetic • Monitoring equipment to allow adherence to Standards for Basic Anesthetic Monitoring[2] • Administration of inhaled anesthetics requires an anesthesia machine equivalent to those in the OR and maintained with the same standards
Electrical outlets	• Sufficient for machine and monitors • Isolated electrical power or ground fault circuit interrupters if "wet location"
Adequate lighting	• Requires illumination of the patient, monitors, and equipment • Battery-powered backup available
Sufficient space	• Space for personnel and equipment • Easy access to patient, machine, and monitors
Resuscitation equipment	• Emergency drugs • Defibrillator • Cardiopulmonary resuscitation equipment
Staff	• Adequately trained staff for support • Reliable means of two-way communication to request assistance
Building and safety codes	• All should be observed
Postanesthesia care facilities	• Appropriate postanesthesia management • Adequately trained staff • Appropriate equipment for safe transport

Statement on Nonoperating Room Anesthetizing Locations. Committee of Origin: Standards and Practice Parameters; https://www.asahq.org/standards-and-guidelines/statement-on-nonoperating-room-anesthetizing-locations, is reprinted with permission of the American Society of Anesthesiologists, 1061 American Lane, Schaumburg, Illinois 60173-4973.

(b) Using non-MRI compatible equipment can have devastating consequences including the projectile effect, burns, and equipment malfunction.
(c) If using non-MRI compatible equipment, extensions for tubing, breathing circuits, or monitoring cables must be immediately available as non-MRI compatible equipment must remain in the scanner control room.[3,4]

(4) Organization of lines and cables is important, so they are not dislodged while moving the patient through the MRI.
(5) The magnet can produce heat over a long procedure. Monitoring patient temperature is helpful, especially if using a warming device.
(6) Airway management and maintenance are critical when working in the MRI suite.

b) **Computed tomography**
i) Anesthetic considerations
(1) Similar to MRI but CT is typically a shorter procedure.
(2) Risk of radiation exposure

c) **Interventional radiology**
i) Indications
(1) Placement of tunneled catheters, percutaneous drains for abscess, or drainage of other materials, TIPS procedure for refractory variceal bleeding or ascites, and embolization for acute arterial bleeding.
ii) Technique
(1) Minor IR procedures such as drain or intravenous catheter placement can be done with light sedation.

(2) Some procedures such as TIPS benefit from general anesthesia due to the often prolonged procedure time, patient comorbidities, frequent requests for breath holds and higher risk for life-threatening complications.

iii) Anesthetic considerations

(1) The TIPS procedure can involve life-threatening hemorrhage due to hepatic injury, or in the case of emergent TIPS due to refractory variceal bleeding.

(a) In addition, patients undergoing TIPS have end-stage cirrhosis and are at risk for additional comorbidities and complications such as pulmonary hypertension, cardiomyopathy, hepatic encephalopathy, and coagulopathy.[5]

(2) Patients undergoing emergent arterial embolization for active extravasation are frequently unstable, and often require massive transfusion until hemorrhage control can be achieved. General anesthesia with invasive arterial monitoring is frequently required.

(3) Radiation exposure

(a) Personal protective equipment such as a lead apron should be used in addition to screens.

d) **Neuroradiology**

i) Indications

(1) Cerebral angiography, mechanical thrombectomy for acute stroke, stenting, or coiling of cerebral aneurysms

ii) Technique

(1) Most healthy patients can tolerate cerebral angiography with nurse-administered sedation.

(2) Stenting or coiling of cerebral aneurysms or unstable, emergent procedures require a general anesthetic or deep sedation to provide patient immobility and close hemodynamic monitoring.

(3) Mechanical thrombectomy for acute stroke can be done safely under sedation or general anesthesia as long as strict blood pressure control is maintained. This should be individualized and discussed with the proceduralist.[6,7]



Stenting or coiling of cerebral aneurysms or unstable, emergent procedures require a general anesthetic or deep sedation.

iii) Anesthetic considerations

(1) Typically, the anesthesiologist does not have close proximity to the patient or the airway.

(a) All breathing circuits, IVs, and arterial line tubing must have extensions.

(2) Arterial lines are commonly used for hemodynamic monitoring.

(a) May be from the percutaneous sheath placed by the interventionalist

(3) Radiation exposure

(4) Complications include intracranial bleeding with resulting intracranial hypertension and hemodynamic instability.

e) **Cardiac catheterization laboratory**

i) Cardioversion

(1) Indications

(a) Conversion of aberrant rhythm patterns

(i) Atrial fibrillation

(ii) Atrial flutter to sinus rhythm

(2) Technique

(a) Cardioversion is painful, so patients need to be unconscious for the procedure.

(b) The procedure only lasts a few seconds.

(i) The key is to provide deep short-acting sedation.

(ii) Low doses of propofol are an option and allow for quick recovery.[8]
(c) The patient should be provided with supplemental oxygen.
(d) Phenylephrine and IV fluids should be administered to treat hypotension.
(e) Patients with or without OSA may develop brief airway obstruction. A chin lift, jaw thrust, or nasal airway may be helpful.
(f) For TEE/CV, morbidly obese patients and patients with OSA at high risk for airway obstruction may benefit from nasal CPAP use during the procedure.
(3) Anesthetic considerations
(a) These patients often have an extensive history of cardiac disease.

ii) Radiofrequency ablations
(1) Indications
(a) Treatment of refractory atrial and ventricular arrhythmias
(2) Technique
(a) Can be performed under both sedation or general anesthesia, however, given the often long procedure time and risk for life-threatening complications general anesthesia may be preferred.
(b) Invasive arterial blood pressure monitoring is recommended when prolonged tachyarrhythmias are expected, or patient has significant comorbidities necessitating invasive monitoring.
(c) Mechanical cardiac support may be required in prolonged ventricular tachycardia ablations to support cardiac output during prolonged runs of ventricular tachycardia.
(d) Esophageal temperature monitoring is often requested by the electrophysiologist due to the risk of esophageal thermal injury.
(e) Electrophysiologists may request beta-agonists, such as Isoproterenol, to help induce arrhythmias.
(3) Anesthetic considerations
(a) There is radiation exposure.
(b) Emergence and extubation of a patient with a femoral sheath warrants special attention because the patient must remain still to avoid dislodging the sheath and bleeding.
(c) Cardiac tamponade is a known complication, occurring in ~1.3% of cases.[9,10]



Patients undergoing cardiac ablations are at risk for sudden hemodynamic deterioration due to induced malignant arrhythmias, or acute cardiac tamponade from cardiac perforation.

iii) Coronary angiography
(1) Indications
(a) Angiography can be done to evaluate the patency and flow patterns of coronary arteries.
(2) Technique
(a) Most elective procedures are performed with nurse-administered sedation.
(b) Many pediatric patients require an anesthesiologist depending on the age of the patient and the cardiac lesion.
(c) An adult for emergent catheterization may require a general anesthetic if the patient is in respiratory failure or has hemodynamic compromise.
(3) Anesthetic considerations
(a) Pediatric patients with congenital lesions require understanding of their cardiac anatomy.
(b) Adults requiring general anesthesia may be unstable and have a full stomach.
(c) In emergency cases, the patient may be in a location without an anesthesia machine or a ventilator.

(d) Ask the support staff in the procedure room to obtain any additional equipment needed and call for anesthesia backup if necessary.
(e) Be prepared for the worst-case scenario and a potential transport directly to the OR or ICU.

f) **Intensive care unit**
i) Indications
(1) ICUs may require an anesthesiologist for bedside procedures.
(2) Percutaneous tracheostomies and endoscopic procedures are frequently performed at the bedside to minimize patient complications from transport.
(3) Bedside endoscopy for GI bleeding frequently requires airway protection and sedation and continuous hemodynamic monitoring.
ii) Technique
(1) Procedure and patient specific
iii) Anesthetic considerations
(1) Usually, this is a tight workspace.
(a) Plan ahead and have emergency medications and airway equipment at the head of the bed.
(2) For tracheostomies, always confirm that the tracheostomy is in the trachea with direct visualization via a fiberoptic scope or $ETCO_2$ before removing the endotracheal tube (ETT).
(3) Patients with GI bleeds can become hemodynamically unstable with intubation and sedation, so be prepared to fully support the patient.
(4) Other complications to be concerned about are loss of the airway, bleeding, and/or viscous perforation.

g) **Endoscopy suite**
i) Indications
(1) Many patients requiring monitored anesthesia care for endoscopic procedures are healthy but have requested to be "asleep."
(2) Other patients have chronic GI disorders or comorbidities that warrant close hemodynamic or airway monitoring.
(3) Invasive procedures such as ERCP, stenting, or dilatations require a deeper level of sedation or a general anesthetic.
(4) The procedure length is operator and patient dependent.
ii) Technique
(1) Deep sedation and patient position can lead to airway obstruction in any patient.
(2) Access to the patient's airway is important because patients may need a chin lift or a jaw thrust to relieve airway obstruction.
(3) Have fluids and phenylephrine available to treat hypotension.
iii) Anesthetic considerations
(1) Workspace varies, so organization is very important.
(2) Patients often are dehydrated due to their bowel preparation and NPO status.
(3) There is also a chance of bleeding and viscous perforation during the procedure.
(4) Patients undergoing emergent procedures or those with underlying pathology such as severe gastroparesis or achalasia may have an increased risk for aspiration.



Patients undergoing endoscopy procedures may be prone to hypotension due to dehydration from bowel preparation and fasting.

h) **Electroconvulsive therapy: read chapter on psychiatric diseases which covers this extensively:**

READ MORE
Psychiatric diseases, Chapter 72

i) **Transport**
   i) There are many situations in which a member of the anesthesia team will find themselves transporting a patient: from the OR to the postanesthesia care unit (PACU) to the ICU, or in reverse.
   ii) During any patient transport, a member of the anesthesia team ideally should be with the patient to continually monitor, evaluate, and treat the patient.
   iii) Transporting patients requires preparation.
      (1) Obtain the following items for transport:
         (a) A transport monitor capable of displaying pulse oximetry, invasive or noninvasive blood pressure, and ECG
         (b) Emergency airway equipment (including a bag-valve mask, laryngoscope, and ETT)
         (c) Resuscitation medications (including sedatives, pain medications, vasopressors, and antihypertensives) must be obtained before transport.
      (2) Some patients may need to be transported with a ventilator, but most intubated patients can be transported with a self-inflating bag, a person dedicated to ventilating the patient, and a full oxygen tank. A PEEP valve should also be considered to avoid alveolar collapse in intubated patients.
      (3) Patients coming to and from ICUs often have several invasive catheters and monitors with multiple intravenous infusions.
      (4) All lines and monitors should be well organized and labeled.
      (5) If the patient's condition warrants it, a defibrillator should be obtained for transport.
      (6) In order to ease the transition, report can be called to the PACU or ICU prior to leaving the procedure site to give the receiving team time to prepare.
      (7) To facilitate a safe and expedient transport, someone from the surgical or the medical service should help transport and supporting staff should aid in retrieving elevators or opening doors.

j) **Postoperative care**
   i) Some offsite facilities have their own recovery unit.
   ii) The standards for postanesthesia care should be the same as the PACU in the main OR environment.
   iii) A verbal sign out should be given to whoever is assuming care of the patient.
   iv) A member of the anesthesia team should stay with the patient until the effects of the anesthetic has worn off; allowing the patient to be extubated, spontaneously ventilating and supporting his/her airway, and hemodynamically stable with adequate pain control.[11]
   v) The offsite staff should have contact information in case of any postoperative issues relating to the anesthetic.

CHAPTER SUMMARY FOR OUT OF OR ANESTHESIA AND TRANSPORT

Definition	Out of OR anesthesia refers to anesthesia services provided in areas that may not frequently support the anesthesiologist, such as diagnostic radiology facilities. The same standards for OR anesthetic care should be applied to anesthesia practices outside of the OR.
General Considerations	For out of OR anesthetics, consider the patient, procedure, and environment. Difficult-to-sedate patients may have high levels of anxiety, psychiatric disorders, chronic opioid use, mental status changes, or dementia. Patients with movement disorders may require an anesthesiologist to help provide patient immobility.

Preoperative Considerations	All patients should have a thorough preoperative anesthesia evaluation. The anesthetic plan should be tailored to the patient's individual needs and the available facilities in the practice environment.
Intraoperative Considerations	The ASA Guidelines for Nonoperating Room Anesthetizing Locations have recommendations about availability of equipment including oxygen, suction, and resuscitative medications.[1]
Postoperative Considerations	The standards of postoperative care should be the same as those implemented in the OR perioperative environment.

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128 The Geriatric Patient Presenting for Surgery

Shamsuddin Akhtar

Humans a surviving much longer and average life expectancy continues to increase. Most adult anesthesia providers are now involved in the care of geriatric patients. Elderly patients are not only 'old or very old' patients, but they are unique phenotype comprised of aged biological system, multiple co-morbid diseases and have a spectrum of 'geriatric syndromes'. The specific preoperative assessment of elderly patient should involve assessment of geriatric syndromes, functional status, frailty, cognition, nutritional status and discussion of goals of care. Intraoperative management to postoperative care require special considerations in the elderly.

1) **Physiological Effects of Aging**[1]
   a) ***Central Nervous System***
      i) All major cell types in the brain undergo structural changes with aging. These changes include neuronal cell death, dendritic retractions and expansion, synaptic loss and remodeling, and changes in glial cell (astrocyte and microglia) reactivity.
      ii) Overall reduction in neuronal regenerative capacity
      iii) All areas of the brain do not shrink at the same rate. For example, white matter and pons are not affected by aging, while there is a significant decline in thalamic and cortical gray matter.
      iv) With time and aging, neural plasticity decreases, however, neuronal connectivity may increase.
      v) Cellular signaling transduction pathways, cytokines, and growth factors that are involved in neuronal excitability and plasticity are also affected by aging.
      vi) Aging brain also has decreased cerebral blood flow due to decreased cerebral metabolic rate and is more susceptible to metabolic stress.
      vii) Elderly patients have a high incidence of neurodegenerative diseases and dementia.



Elderly have decreased anesthetic requirements and are more prone to develop neurological dysfunction due to inflammatory, metabolic, and neuroendocrine stress.

   b) ***Cardiovascular System***[2]
      i) Blood vessels: Two broad structural effects are noticed in blood vessels with aging. These processes occur simultaneously but are different.
         (1) The vessels become stiff and thicken due to the natural change in the composition of blood vessels with decreasing amount of elastin and increasing collagen.
         (2) The second is the effect of atherosclerosis, which compounds the problem related to aging vessels.
         (3) Functionally, the arteries become less responsive due to changes in the endothelium.
            (a) Decrease in nitric oxide production has been noted.
            (b) The effect of vasoconstrictor agonists, norepinephrine, ephedrine, and phenylephrine is attenuated in the elderly.



Blood vessels become stiff and less compliant with aging.

(c) The pulse wave of the ejected blood travels faster. Stiffer arteries also allow pressure to reflect from the periphery to the heart quicker, while the aortic valve is still open and the heart is ejecting, thus effectively increasing the afterload on the heart. The net effect is increase in the systolic blood pressure and no effect on the diastolic blood pressure.

ii) Heart: The changes in the vessels affect the heart.
(1) The heart responds to increased impedance by left ventricle hypertrophy.
(2) Left ventricular mass increases by 15% from age 30 to 70 years.
(3) The chronic changes make the myocardium more prone to ischemia.
(4) The incidence of diastolic dysfunction increases with age.
(5) Annular dilatation is very common and 90% of healthy octogenarians demonstrate some form of mild multivalvular regurgitation, which is typically mild, central, and present with normal-appearing leaflets.
(6) Left atrial chamber size also increases.
(7) The incidence of aortic sclerosis and stenosis increases with age.
(8) The incidence of arrhythmia, especially atrial fibrillation, increases significantly with aging.
(9) Normal age-related changes in the cardiovascular physiology include decrease in peak heart rate, peak cardiac output, and peak ejection fraction. In addition, there are changes in autonomic tone and baroreceptor reflex activity.
(10) The function and the number of adrenergic receptors also change in the heart. Higher dose of agonist drugs are required to obtain desired hemodynamic response.



Left ventricular mass increases with aging and is more prone to ischemia.

Incidence of diastolic dysfunction increases with aging.

Higher dose of agonist drugs is required to obtain desired hemodynamic response.

c) ***Respiratory System***
i) The chest compliance decreases, while the lung compliance and the respiratory system resistance increases.
ii) With aging, total lung capacity remains the same; however, residual volume increases, while vital capacity decreases with aging.
iii) Due to progressive decline in diaphragmatic strength (25%) in healthy elderly person and changes in the airway, expiratory flows (FEV1, FEV1/FVC, and FEF 75%) all decrease.
iv) Increased degree of ventilation/perfusion (V/Q) mismatching and to a lesser extent, pulmonary shunting. The arterial partial pressure of oxygen (PaO_2) decreases by an average rate of 0.35 mm Hg/y from the age of 20 years.
v) Protective cough mechanisms may be attenuated, which may decrease their ability to clear secretions and increase risk of aspiration.
vi) Elderly patients are more prone to respiratory failure, increase risk of atelectasis and aspiration.



Elderly patients are more prone to respiratory failure, increase risk of atelectasis and aspiration.

d) ***Renal, Fluids, and Electrolytes***
Various aspects of renal function decline with normal aging. The salient changes that accompany normal renal aging are as follows:
i) Renal vascular dysautonomy: This term reflects attenuation of the autonomic renal vascular reflexes to protect the kidney from hypotensive or hypertensive states.
ii) Senile hypofiltration: Which describes progressive decline in number of glomerular filtration rate (GFR) at about 1 mL/y after 30 years

iii) Tubular dysfunction: which leads to the reduction in maximal tubular capacity to reabsorb and excrete solutes, especially sodium
iv) Medullary hypotonicity: This phenomenon describes lower tonicity of the renal medulla.
v) Elderly patients are unable to concentrate or dilute urine to the maximum.
vi) The aging kidney is more susceptible to injury, less able to accommodate hemodynamic changes, and it is also not able to handle water and salt perturbations.
vii) Thirst response and drinking behavior in the elderly is altered.


Elderly patients are unable to concentrate or dilute urine to the maximum and can become dehydrated easily.

e) ***Gastrointestinal System***
Aging process significantly affects the oropharyngeal upper esophageal motility, colonic function, gastrointestinal immunity, and gastrointestinal drug metabolism.

f) ***Immune systems***
Both innate immune system (macrophages, neutrophils, natural killer cells, etc.) and the adaptive immune systems are affected by aging. Bactericidal activity of immune cells is decreased.


Elderly patients are immunocompromised and less able to fight infections.

g) ***Endocrine Function Changes***
i) Endocrine glands tend to atrophy in the elderly.
ii) Hormone production is frequently decreased in the elderly leads to impaired endocrine function.
iii) Deficiencies of compounds like insulin, thyroxine, growth hormone, renin, aldosterone, and testosterone are often present causing diabetes, hypothyroidism, impotence, and osteoporosis are common, along with chronic electrolyte abnormalities.

h) ***Sarcopenia and body composition***
i) Though the total fat content goes down, the percentage of fat per total body weight increases. Weight tends to decline with aging.
ii) Lean body mass, which is predominantly composed of muscle and visceral organs, decreases steadily after the third decade.
iii) Muscle strength declines with aging. The elderly become progressively frail.

2) **Frailty**[3]
a) Incidence of frailty increases with aging.
b) Frailty is defined as a state of reduced physiologic reserve which is associated with increased susceptibility to disability and failure of the body to respond to additional stresses such as surgical intervention or infection.
c) Frailty is characterized by weight loss, fatigue, impaired grip strength, low physical activity, and slow gait speed and in some patients, cognitive decline. All these changes lead to decreased reserve capacity.
d) Frailty is an independent predictor of in-hospital mortality and postoperative disposition.


Frailty is defined as a state of reduced physiologic reserve which is associated with increased susceptibility to disability and failure of the body to respond to additional stresses such as surgical intervention or infection.

3) **Geriatric Syndromes**[3,4]
Geriatric syndrome encompasses clinical conditions that are frequently encountered in older people. The pathophysiology of geriatric syndrome is usually multifactorial and involved multiple unrelated organs. They have deleterious effects on function and quality of life. Virtually all geriatric syndromes are characterized by changes of four domains signified by alteration in body composition,

TABLE 128-1 List of Geriatric Syndromes

1. Incontinence
2. Delirium
3. Falls
4. Pressure ulcers
5. Sleep disorders
6. Problems with eating or feeding
7. Pain
8. Depressed mood
9. Dementia
10. Physical disability

gaps in energy supply and demand, signaling disequilibrium, and neurodegeneration. Geriatric syndromes should be considered as phenotypic consequences of frailty. Geriatric syndromes are listed in **Table 128-1**.

4) **Pharmacokinetic and Pharmacodynamic Changes in the Elderly**[5,6]
Polypharmacy is the norm in the elderly. Effects of drug interactions are substantially magnified with advanced age. It is also well established that due to pharmacokinetic and pharmacodynamic changes, anesthetic requirements decrease with aging (**Table 128-2**).



Anesthetic requirements decrease with aging.

MAC values decrease by 6% per decade after 40 years for volatile anesthetics and about 7.7% per decade for nitrous oxide.

Dose of intravenous anesthetics and opioids should be decreased by 25%-50% in elderly patients.

a) ***Inhalational anesthetics***
   i) The minimum alveolar concentration (MAC) required to achieve adequate anesthetic depth progressively decreases with age.
   ii) MAC values decrease by 6% per decade after 40 years for volatile anesthetics and about 7.7% per decade for nitrous oxide.

b) ***Propofol***
   i) Pharmacology of propofol is significantly altered with aging.
   ii) Propofol should be decreased by 25%-50% in the elderly.
   iii) For the same plasma concentration of propofol, the drop in blood pressure with propofol administration is significantly higher in the elderly than young patients.

c) ***Etomidate***
   i) Etomidate is an anesthetic and amnestic, but not an analgesic.
   ii) Like propofol, much lower induction doses are recommended in the elderly.
   iii) Elderly patients require 0.2 mg/kg for induction, as opposed to 0.3-0.4 mg/kg for young adults.

TABLE 128-2 Effect of Aging on Intravenous Anesthetics

Drug	% Decrease	PK, PD	Duration of Effect
Propofol	30%-75%	PK, PD	Prolonged
Etomidate	50%	PK	—
Midazolam	50%-60%	PK, PD	Prolonged
Fentanyl	25%-50%	PK, PD	Prolonged
Remifentanil	50%	PD	—
Dexmedetomidine	50%	PD	Prolonged

PK, pharmacokinetics; PD, pharmacodynamics.

d) ***Thiopental***
   i) Central volume of distribution of thiopental decreases in the elderly and total dose need to be reduced.
   ii) An optimal dose in an 80-year-old patient was suggested to be 2.5 mg/kg, which is 50%-80% of the dose needed for an adult patient (3-5 mg/kg).
   iii) Recovery after a bolus dose of thiopental is also delayed in older patients, because of decreased central volume of distribution.

e) ***Midazolam***
   i) Benzodiazepines should be avoided in the elderly patients.
   ii) Elderly patients are significantly more sensitive to midazolam than younger patients, primarily because of pharmacodynamics differences.
   iii) The duration of effect of midazolam may last much longer and can potentially contribute to postoperative delirium.
   iv) A 75% reduction in dose from a 20-year-old to a 90-year-old has been recommended.


The duration of effect of midazolam may last much longer and can potentially contribute to postoperative delirium.

f) ***Opioids***
   i) Age-related pharmacodynamic and pharmacokinetic changes have been noted within the opioid receptor system.
   ii) The liver mainly metabolizes opioids, and the kidneys excrete the metabolites.
   iii) The primary risk of opioids is respiratory depression, the incidence of which is markedly increased with age.
   iv) *Fentanyl*: Approximately 50% increase in potency of fentanyl has been reported in the octogenarians. Though clearance of fentanyl is not affected, transdermal absorption of fentanyl is delayed in the elderly.
   v) *Remifentanil*: Remifentanil is metabolized by nonspecific tissue and plasma esterases, has a short half-life, which makes it an ideal drug for use in the elderly. However, elderly patients are also more sensitive to remifentanil. Elderly subjects need about half of the bolus dose and require an infusion rate about one-third dose as younger subjects for the same drug effect.
   vi) *Meperidine*: Meperidine also has negative inotropic properties and intrinsic anticholinergic properties, and its use has been associated with development of postoperative delirium in elderly patients. Meperidine is not recommended in the elderly patients except in very small doses to manage postoperative shivering.


Opioids increase the risk of respiratory depression in the elderly.

g) ***Neuromuscular blocking drugs***
   i) The pharmacodynamics of neuromuscular blocking drugs is not significantly altered with age.
   ii) The ED_{95} of neuromuscular blocking is essentially the same for young and old patients.
   iii) The recovery time from the neuromuscular blockade could be increased by 50%. Incidence of residual muscular blockade is increased in elderly patients.
   iv) As cisatracurium is not dependent on hepatic or renal function for clearance, it is considered neuromuscular blocker of choice for the elderly.


Incidence of residual muscular blockade is increased in elderly patients.

5) **Perioperative Outcomes After Cardiac and NonCardiac Surgery**[7]
   a) Elderly patients have significantly poorer outcomes than their younger counterparts.
   b) The operative mortality of octogenarians undergoing cardiac surgery is reported to be 5.8%-11%, compared with 2.7%-4.1% in younger patients.
   c) The mortality after exploratory laparotomy can be as high as 45% in the octogenarians.

d) Risk of adverse events after urgent hip fracture surgery are 5%-7%, while the risk after elective knee and hip arthroplasty is 1.5%-3%.
e) Postoperative complications, as high as 60%-63%, have been reported.
f) Pulmonary insufficiency or infection was one of the leading causes of postoperative morbidity.
g) Neurocognitive dysfunction is very common after cardiac and noncardiac surgery. The incidence of cognitive dysfunction is about 3-9 times higher after noncardiac surgery compared to their nonsurgical counterparts.
h) Delirium is very common in the elderly (5%-45%) after major surgery and the incidence of long-term decline in cognitive function is significant.
i) Functional recovery after cardiac and noncardiac surgery continues to be a significant problem in the elderly. Less than 50% of the elderly patients are discharged home, and many patients are discharged to long-term rehabilitation facilities or nursing homes.

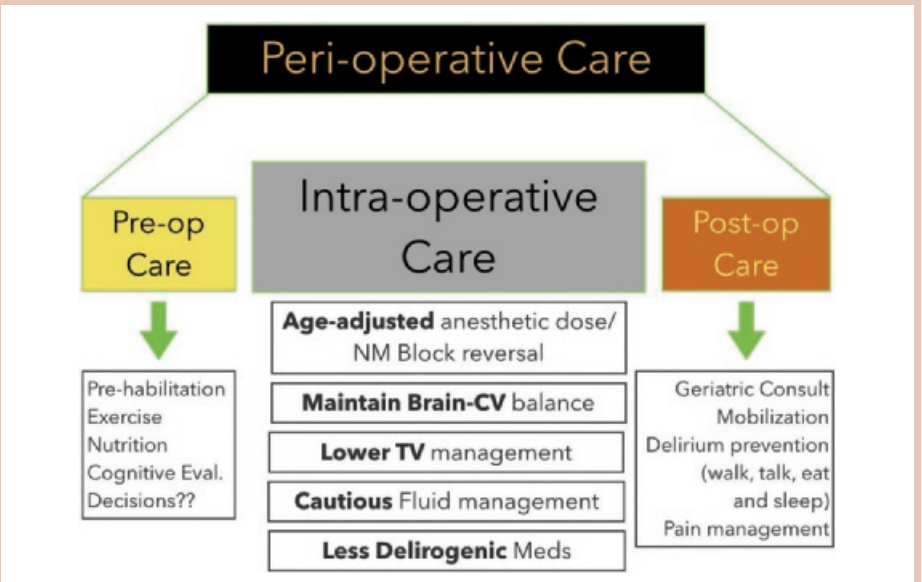


The operative mortality after cardiac and non-cardiac surgery is 2-5 times higher in octogenarians than younger patients.

Delirium is very common in the elderly (5%-45%) after major surgery.

6) **Perioperative Care of the Elderly Patient (Fig. 128-1)**
   a) ***Preoperative assessment***[8,9]
      i) The traditional risk indices are not able to predict outcomes in octogenarians.
      ii) Specific geriatric preoperative assessment should include review of geriatric syndromes, evaluation of frailty, baseline cognitive status, nutritional status, assessment of functional status, review of medications, and goals of care discussion.
      iii) ASA Brain Health Initiative suggests baseline cognitive evaluation in patients older than 65 years. Identification of baseline cognitive dysfunction can then lead

FIGURE 128-1 Summary of Goals of Perioperative Care in the Geriatric Patient.



to formal cognitive testing, formal diagnosis, and more detailed discussion of perioperative complications and goals of care.

iv) Due to complexity of geriatric patients, it is not uncommon that geriatric consultation is obtained preoperatively, or an elderly patient is comanaged with a geriatrician.



Geriatric preoperative assessment should include review of geriatric syndromes, evaluation of frailty, baseline cognitive status, nutritional status, assessment of functional status, review of medications, and discussion of goals of care.

b) ***Prehabilitation***[10]

i) Prehabilitation prior to elective surgery may improve perioperative outcomes and facilitate early recovery. However, the results of the studies have been mixed, and the data are evolving. It cannot be recommended for all elderly patients.

ii) Prehabilitation is individualized and multidisciplinary.

iii) Components of optimization of nutrition, smoking cessation, alcohol cessation, exercise, psychological support, and management of depression and anemia

iv) It needs to be instituted 4-12 weeks prior to elective surgery.

c) ***Preoperative assessment of patients undergoing urgent or emergent surgery***

d) Many elderly patients will present for urgent or emergent surgery after trauma, falls (hip fractures, intracranial bleed), and intra-abdominal and vascular emergencies. This may preclude detailed preoperative evaluation and optimization.

i) Basic evaluation of the cardiorespiratory system, especially looking for signs of acute heart failure, fat embolism, acute lung injury, and signs of dehydration should be sought. Increased oxygen requirements and low oxygen saturation may denote worsening LV dysfunction, acute lung injury, aspiration, and pneumonia.

ii) Acute delirium may be evident even before the surgery and should be investigated (if possible) prior to surgery to rule out new intracranial processes.

iii) Patients and their caregivers should be given realistic information regarding intraoperative risk and postoperative outcomes (prolonged postoperative course, possible postoperative ventilation, and ICU care).

e) ***Intraoperative management***[1]

i) Monitoring

(1) Age alone is not an indication for invasive monitoring such as transesophageal echocardiography or pulmonary artery catheterization. The decision to use specific monitoring methods should be based on the potential benefits and risks in a particular patient undergoing specific surgery.

(2) Use of cerebral function monitors to titrate anesthetics has been recommended by some organizations. However, due to inherent limitations of these monitors, it is not clear if use of EEG monitors reduces the incidence of delirium and postoperative neurological dysfunction.

ii) Anesthetic management

(1) One of the key factors to keep in mind is the anesthetic requirement for anesthetic is reduced significantly in the elderly.

(2) The dose of induction agent and opioids should be decreased by 25%-50%.

(3) MAC of inhalational anesthetic for an octogenarian is 30% lower than their younger counterpart.

(4) The intravenous anesthetics have more pronounced hemodynamic effect, and smaller doses are required to achieve the same anesthetic level.

(5) Benzodiazepines should be avoided as much as possible. If benzodiazepines are used, their dose should be lowered in the elderly.

(6) Meperidine should not be used in the elderly.

(7) Prudent to use cisatracurium in patients with renal and liver dysfunction

(8) Bony prominences must be protected and padded.

(9) Elderly patients are often dehydrated because of a diminished sensation of thirst.

(10) Measures to conserve body heat and decrease the risk of hypothermia should be implemented.

(11) Fluid therapy should not be considered routine. Due to advanced atherosclerosis, stiff ventricles, diastolic dysfunction, and occult coronary artery disease, elderly patients do not tolerate hypovolemia or hypervolemia. Imprecise fluid administration in the elderly can have dire consequences. Hypovolemia leads to severe hypotension and organ hypoperfusion, while over hydration can lead to congestive heart failure.

(12) Higher hemoglobin and hematocrit values may be more desirable in the elderly patients. It is appropriate to keep hematocrit levels above 24 mg/dL in the elderly.

(13) Regional anesthesia (if possible) may be desirable in patients undergoing noncardiac surgeries, especially in orthopedic surgeries. However, many elderly patients are on anticoagulants and antiplatelet agents, which may preclude regional anesthesia based on ASRA guidelines. Regional anesthesia can reduce administration of systemic medications intraoperatively. Pain management is facilitated by regional anesthesia and reduces use of systemic opioids, which can predispose to delirium. Large prospective studies are underway to address this ongoing controversy.

f) ***Postoperative management***

i) Postoperative delirium and cognitive dysfunction[11,12]

(1) Neurocognitive dysfunction is very common in the elderly after cardiac and noncardiac surgery (20%-40%).

(2) Delirium is an acute disorder of disturbed attention that fluctuates with time. It effects 15%-55% of hospitalized older patients. It is characterized by the following features: (1) rapid decline in the level of consciousness with difficulty focusing, shifting, or sustaining attention; (2) cognitive change, for example, incoherent speech, memory gaps, disorientation, hallucination, which are not explained by dementia and a medical history suggestive of preexisting cognitive impairment frailty and comorbidity.

(3) The strongest predisposing factor for delirium is preexisting dementia.

(4) The mechanism of postoperative delirium remains elusive but may be related to the inflammatory response resulting in leukocyte migration into the central nervous system. Most patients with postoperative delirium experience complete recovery.

(5) Early identification, supportive measures, and symptomatic treatment for delirium are the rule.

(6) Prophylactic use of antipsychotic medications has not shown to improve outcomes and are not recommended for hypoactive delirium. Antipsychotics should only be used to control symptoms of agitation.



Antipsychotics should only be used to control symptoms of agitation in delirious patients.

ii) Postoperative pain control

(1) Many elderly patients may suffer from chronic pain.

(2) Acute procedural pain should be differentiated from chronic pain or pain due to complications of a procedure (eg, new pain, increased intensity of pain, pain not relieved by previously effective strategies), and treatment should be directed accordingly. Conducting a pain history before a procedure can help discriminate procedural from chronic pain.

(3) Principles of pain management in the elderly are the same as for nonelderly population; however, the tools for assessing pain have to be adapted to compensate for the cognitive and sensory impairments seen in the elderly.
(4) The cognitive status of the older adult impacts the approach to pain assessment, patient and family education, as well as pain treatment options. Older adults with mild to moderate cognitive impairment are often able to rate pain.
(5) Many severely impaired persons can respond to simple questioning about presence of pain.
(6) For older adults with cognitive impairment unable to report pain, it is important to assess for the presence of factors that cause pain.
(7) Observing behavior when the patient is engaged in activity (eg, transfers, ambulation, repositioning) can provide clues to the level of pain the patient may be experiencing. Observation at rest, to assess pain, can be misleading.
(8) Whenever an older adult with cognitive impairment shows a change in mental status, pain should be considered a potential etiology.
(9) Potential sources of pain in the elderly include distended bladder, incision, infection, inflammation, fracture, positioning, urinary tract infection (UTI), and constipation. Treating the underlying cause of pain using etiology-specific interventions is important.
(10) Beer criteria which list drugs potentially harmful to the elderly patients should be kept in mind.[13]
(11) The use of meperidine is not recommended in older individuals.
(12) The use of transdermal fentanyl is not recommended for acute pain management in opioid-naive older adults because of its potential for delirium and respiratory depression.
(13) Agonist–antagonist opioids should be avoided in older adults.
(14) Butorphanol and pentazocine produce psychotomimetic effects and may lead to delirium. Pentazocine causes hallucinations, dysphoria, delirium, and agitation in older adults and has been shown to be no more effective than aspirin or acetaminophen.
(15) Analgesics with a long, highly variable half-life (eg, opioids, such as methadone and levorphanol) should also be avoided.



Potential sources of pain in the elderly include distended bladder, incision, infection, inflammation, fracture, positioning, urinary tract infection (UTI), and constipation and should be treated accordingly.

iii) Care of the elderly in the ICU
iv) It is not uncommon to expect elderly patients to be transferred to the intensive care unit on mechanical ventilation or close postoperative hemodynamic monitoring after major surgeries.
(1) Presence of comorbidities and intraoperative course determine the intensity of postoperative monitoring.
(2) For sedation in the critical units, dexmedetomidine may be a better drug than benzodiazepines in the elderly.
(3) Long-acting benzodiazepines should definitely be avoided.
(4) Dealing with social, ethical, and end-of-life issues should be addressed as soon as possible. Physicians need to be mindful of the sensitivities and wishes of the patient and provide realistic prognosis to the family members and care takers to achieve best possible outcomes.

SUMMARY

Physiological Effects of Aging	CNS: neural plasticity decreases, neuronal cell death, dendritic retractions and expansion, synaptic loss and remodeling, and changes in glial cell (astrocyte and microglia) reactivity occur with aging CVS: The heart and the vasculature become stiff. Increased systolic pressure and pulse pressure. Diastolic dysfunction increases Respiratory: lung volumes change with aging. Ventilation/perfusion mismatch and shunt fraction increases with aging Renal: elderly patients are unable to concentrate or dilute urine to the maximum and can become dehydrated easily
Frailty	Frailty is characterized by weight loss, fatigue, impaired grip strength, low physical activity, and slow gait speed and in some patients, cognitive decline. All these changes lead to decreased reserve capacity
Geriatric Syndromes	Geriatric syndrome encompass clinical conditions that are frequently encountered in older people, for example, incontinence, delirium, falls, pressure ulcers, sleep disorders, problems with eating or feeding, dementia, physical disability
Pharmacokinetic and Pharmacodynamics Changes in the Elderly	MAC values decrease by 6% per decade after 40 years for volatile anesthetics and about 7.7% per decade for nitrous oxide Dose of intravenous anesthetics and opioids should be decreased by 25%-50% in elderly patients
Preoperative Assessment	Specific geriatric preoperative assessment should include review of geriatric syndromes, evaluation of frailty, baseline cognitive status, nutritional status, assessment of functional status, review of medications, and goals of care
Intraoperative Care	Anesthetic requirement for anesthetic is reduced significantly in the elderly The dose of induction agent and opioids should be decreased by 25%-50% Age alone is not an indication for invasive monitoring
Postoperative Delirium and Cognitive Dysfunction	Delirium is an acute disorder of disturbed attention that fluctuates with time. Delirium is an acute disorder of disturbed attention that fluctuates with time. It is multifactorial and very common postoperatively

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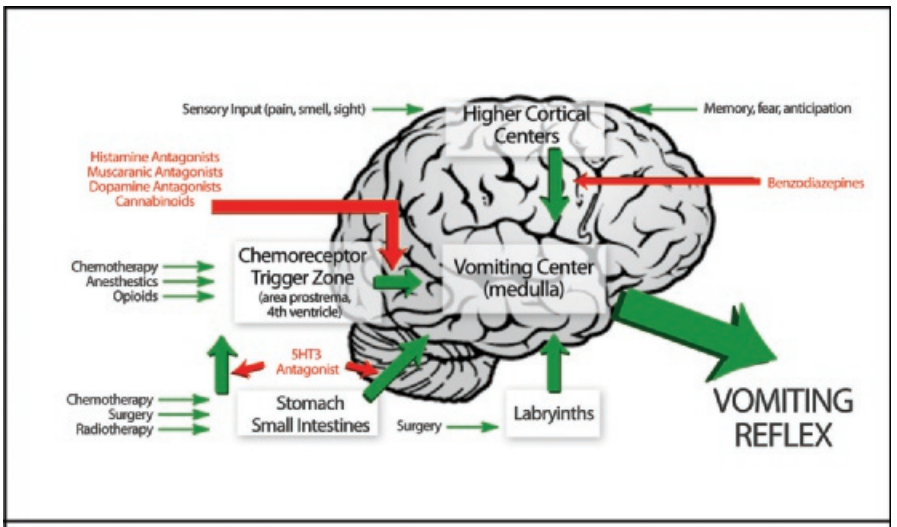
129 Postoperative Nausea and Vomiting§

Gregory J. Moy • Bassam Kadry

Postoperative nausea and vomiting (PONV) is a common problem in patients recovering from surgery and anesthesia. Several risk factors have been associated with increased risk of PONV. In the current anesthetic practice, an effective strategy to prevent PONV is vitally important in order to ensure efficient patient throughput, reduce costs, and improve patient satisfaction.

1) **Pathophysiology**
   a) **Prevalence and risk stratification**
      i) Estimated to occur in 30% of all anesthetics
         (1) In high-risk patients, the prevalence approaches 80%.[1]
      ii) Postoperative nausea and vomiting (PONV) can adversely affect patient satisfaction and lead to:
         (1) Increased cost due to extended postanesthesia care unit (PACU) times
         (2) Unplanned admission to the hospital
         (3) Increased drug use to treat PONV once it develops[2]
   b) **Molecular mechanism (Fig. 129-1)**
      i) The etiology of PONV is multifactorial.
      ii) The emetic center of the brain
         (1) Located in the lateral reticular formation of the medulla, receiving input from[1,2]
            (a) Chemoreceptor trigger zone
            (b) Vestibular apparatus
            (c) Cerebellum
            (d) Solitary tract nucleus
            (e) Cerebral cortex
         (2) Receptors that are related to and serve as a basis for the treatment and prevention of PONV include:[1]
            (a) Dopamine (D_2)
            (b) Acetylcholine (M_1)
            (c) Histamine (H_1)
            (d) Serotonin ($5HT_3$)
            (e) Neurokinin (NK_1) receptors
   c) **Risk factors**[2,3]
      i) Surgery related:
         (1) Prolonged surgical duration
         (2) Procedure type (neurological, ENT, breast, strabismus, abdominal, and laparoscopic surgeries)
      ii) Anesthesia related:
         (1) Nitrous oxide
         (2) Volatile anesthetic
         (3) Opioids
         (4) General anesthesia

FIGURE 129-1 Mechanisms for PONV and Recommended Strategies for Minimizing the Incidence of PONV



Recommended Strategies for Minimizing the Incidence of PONV

1. Identify high-risk patients
2. Avoid emetogenic stimuli
   - Etomidate
   - Inhalational Anesthetics
   - Opioids*
3. Multimodal Therapy
   - Antiemetics (consider combination therapy)
   - TIVA with Propofol
   - Adequate Hydration
   - Effective Analgesia with local anesthetics and Cox-2 inhibitors
   - Anxiolytics
   - Intraop O_2 (FIO2 0.8)
   - Nonpharmacologic techniques

* Although opioids are emetogenic, optimal analgesia should be the goal and can be achieved by incorporating preop education, local anesthetics, and Cox-2 inhibitors. Optimal analgesia may include an opioid.

Risk Factors for PONV and Guidelines for Prophylactic Antiemetic Therapy

Patient Factors	Surgical Factors
Female Sex	Laparoscopy
History of PONV or Motion Sickness	Laparotomy
Nonsmoker	Plastic Surgery
Post-op Opioid Use	Major Breast Surgery
	Craniotomy
	ENT Procedures
	Strabismus Surgery

Mild to Mod 20-40% Risk 1-2 Factors	Mod to High 40-80% Risk 3-4 Factors	Very High >80% Risk >4 Factors
Any 1 of the following: Droperidol Dexamethasone Scopolamine 5HT3 Antag.	Droperidol Plus 5HT3 Antag. or Dexamethasone Plus 5HT3 Antag.	Combination Antiemetics Plus TIVA with Propofol

iii) Patient related:
(1) Female
(2) History of PONV/motion sickness
(3) Nonsmoker
(4) Young age
(5) Disease with GI involvement, for example, ileus
(6) Dehydration
(7) Pain

2) **Treatment and prevention of PONV**[4–8]
a) **Pharmacology**
i) Several classes of drugs have been studied for the prevention and/or treatment of PONV:
(1) Corticosteroids
(a) Dexamethasone
(i) Should be administered at induction of anesthesia to have maximal effect
(ii) Recommended prophylactic dose is 4-5 mg IV for patients at increased risk of PONV.
(iii) A 4-mg IV dose has similar efficacy to ondansetron 4 mg IV and droperidol 1.25 mg IV.
(iv) Doses <8 mg have not been shown to have significant side effects.
(v) Use with caution in diabetic patients.


Dexamethasone can cause hyperglycemia and should be used with caution in diabetic patients.

(2) D_2-antagonists
(a) Droperidol
(i) Recommended prophylactic dose is 0.625-1.25 mg IV.
(ii) Similar efficacy as ondansetron for PONV prophylaxis
(iii) Most effective when administered at the end of surgery
(iv) FDA black box warning
1. Associated with prolonging the QTc interval
2. Possible fatal cardiac arrhythmias
3. Unlikely to occur at the extremely low dosing levels used of PONV prophylaxis


Droperidol is associated with prolonging QTc interval and possible fatal cardiac arrhythmias in susceptible patients.

(b) Haloperidol
(i) Has been used as an alternative to droperidol.
(ii) Meta-analysis shows doses of 0.5-2 mg IM or IV reduces PONV risk.
1. Sedation and cardiac arrhythmias were not reported at these doses.

(3) H_1-antagonists
(a) Dimenhydrinate
(i) Antihistamine with antiemetic effects
(ii) Antiemetic efficacy may be similar to $5HT_3$ receptor antagonists.
(iii) Recommended dose is 1 mg/kg IV.

(4) M_1-antagonist
(a) Transdermal scopolamine
(i) Useful adjunct to other antiemetic treatments
(ii) Onset of effect is 2-4 hours.
1. Apply in evening prior to surgery or 4 hours prior to end of anesthesia.
(iii) Adverse effects are mild and can include:
1. Visual disturbances
2. Dry mouth
3. Dizziness

(iv) Should be used with caution in elderly patients, particularly those with mild cognitive impairment (MCI), glaucoma, and urinary problems
1. May trigger delirium or worsen glaucoma and urinary retention

(5) NK_1-antagonist
(a) Aprepitant
(i) A 40-mg po dose equivalent to 4 mg ondansetron IV in incidence of nausea during initial 24 hours
(ii) Superior to ondansetron in preventing vomiting 24-48 hours postoperatively

(6) Serotonin ($5HT_3$) receptor antagonists
(a) Favorable side effect profile
(b) All considered equally safe and antiemetic for the treatment of established PONV
(c) $5HT_3$ receptor antagonists used in clinical practice
(i) Ondansetron
1. Recommended prophylactic dose is 4 mg IV.
2. Most effective when given at the end of surgery.
(ii) Dolasetron (**Table 129-1**)
(iii) Granisetron (**Table 129-1**)
(iv) Tropisetron (**Table 129-1**)

(7) Unknown mechanisms
(a) Propofol
(i) Low-dose infusions, such as 5-15 µg/kg/min, given at emergence may be given to prevent PONV.
(ii) Total intravenous anesthesia (TIVA) with higher doses of propofol avoids the use of nitrous oxide and volatile anesthetics and can prevent PONV.



When evaluating PONV, consider underlying etiology by reviewing surgery type, anesthetics administered, and patient-specific factors.

b) **Approaches to PONV prophylaxis**[2,7]

i) Once PONV develops, it can be difficult to treat. Therefore, an effective preventive strategy should be employed for patients most at risk of developing PONV.

ii) Identify patients at risk for PONV.

(1) Apfel risk score can be used to quantify a patient's risk profile based on a point system, 1 point for each of the following risks:
(a) Female gender
(b) History of PONV
(c) Nonsmoking status
(d) Use of postoperative opioids
(e) For points 0, 1, 2, 3, or 4, adults have a PONV risk of 10%, 20%, 40%, 60%, and 80%, respectively.[3]



Once PONV develops, it can be difficult to treat. At-risk patients should receive a preventive strategy for PONV.

(2) Low risk
(a) Wait and see.[2]

(3) Medium risk
(a) Select one to two interventions for adults.

(4) High risk
(a) Select two or more interventions for adults.
(b) Consider a multimodal approach.

iii) Reduce baseline risk of PONV.

(1) Several factors reduce risk, including avoiding volatile anesthetic agents, nitrous oxide, general anesthesia, and emetogenic drugs such as etomidate (**Table 129-2**).

TABLE 129-1 PONV Treatment Options[4–10]

Drug Name	Adult Dose	Time of Dosing	Receptor Site
Aprepitant	40-125 mg po	Before surgery	NK_1
Dexamethasone	4-8 mg IV	Induction	?
Diphenhydramine (QT)	25-50 mg IV		H_1, M_1
Dimenhydrinate	1 mg/kg IV		H_1
Dolasetron (QT)	12.5 mg IV	Emergence	$5HT_3$
Droperidol[a] (QT)	0.625-1.25 mg IV	Emergence	D_2
Ephedrine	35-50 mg IM		?
Granisetron	0.35-1 mg IV	Emergence	$5HT_3$
Haloperidol	0.5-2 mg IV/IM	Emergence	D_2
Methylprednisolone	40 mg IV		?
Metoclopramide[b]	25 mg IV		D_2
Ondansetron (QT)	4-8 mg IV	Emergence	$5HT_3$
Palonosetron	0.075 mg IV	Induction	$5HT_3$
Prochlorperazine[b]	2.5-10 mg IV	Emergence	D_2
Promethazine[c]	6.25-12.5 mg IV	Emergence	D_2, H_1, M_1
Propofol	5-15 µg/kg/min	Emergence	?
Scopolamine	0.4 mg patch	Before surgery	M_1
Tropisetron	2 mg IV	Emergence	$5HT_3$

Alternative therapies: adequate hydration; aromatherapy with isopropyl alcohol vapor and peppermint; acupressure.
[a]Black box warning for QT prolongation.
[b]Risk of extrapyramidal reaction. Reverse with diphenhydramine.
[c]Do not give IV push. Must be diluted.
QT, risk of QT prolongation; $5HT_3$, serotonin; D_2, dopamine; H_1, histamine; M_1, muscarinic; NK_1, neurokinin; ?, exact mechanism unknown.

TABLE 129-2 Strategies to Reduce Baseline PONV Risk[2]

1. Avoidance of general anesthesia by the use of regional anesthesia
2. Use of propofol for induction and maintenance of anesthesia
3. Avoidance of nitrous oxide
4. Avoidance of volatile anesthetics
5. Minimization of intraoperative and postoperative opioids
6. Adequate hydration

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TABLE 129-3 Combination Therapy for PONV Prophylaxis in Adults[2]

1. Droperidol + dexamethasone
2. $5HT_3$ receptor antagonist + dexamethasone
3. $5HT_3$ receptor antagonist + droperidol
4. $5HT_3$ receptor antagonist + dexamethasone + droperidol

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TABLE 129-4 Risks Associated With Treating PONV

1. Droperidol has black box warning for QTc prolongation.
2. Other drugs with QTc prolongation (see Table 129-1 for QT).
3. Metoclopramide has extrapyramidal side effects reversed with diphenhydramine.
4. Corticosteroid use may significantly increase blood glucose.
5. Do not administer promethazine via IV push.

iv) Administer PONV prophylaxis when appropriate.
  (1) Single-agent therapy (see Section A above)
  (2) Combination therapy (Table 129-3)
    (a) Consider for adults at moderate risk for PONV.
    (b) Combine treatments using drugs from different classes.
    (c) Generally, combination therapy has superior efficacy compared with monotherapy for PONV prophylaxis.
    (d) Optimal antiemetic dosing with combination therapy has not been established. Most anesthesiologists use dosing recommendations for single agents.
  (3) Failed prophylaxis
    (a) If prophylaxis fails or was not received, use drug from different class than prophylactic agent.[2]
    (b) Redose antiemetic only after more than 6 hours in PACU.
      (i) Do not redose dexamethasone or scopolamine.



Metoclopramide when used in standard 10-mg IV doses is ineffective for PONV prophylaxis.

c) **Nonpharmacologic approaches and alternative therapies to PONV prophylaxis.**[2,10]
  i) Adequate hydration
  ii) Aromatherapy with isopropyl alcohol and peppermint oil
  iii) Acupressure at P6 acupoint, located 4 cm proximal to the transverse crease between the tendons of the carpi radialis and palmaris longus
  iv) Acupuncture (APu) and Electro-Acupuncture (EAPu) at P6 acupoint
d) Appropriate treatment depends on a careful assessment of risk versus benefit for each treatment modality (Table 129-4).
  i) Some risks of treatment depending on the medication include
    (1) QT prolongation (Table 129-4)
    (2) Drug reactions (metoclopramide)
    (3) Hyperglycemia (dexamethasone)
    (4) Thrombophlebitis (promethazine, hydroxyzine)

CHAPTER SUMMARY FOR PONV

General Considerations	PONV is a common problem that is best treated with prevention. It is important to understand the side effect profile for each drug.
Approach	1. Identify patient's risk for PONV. 2. Reduce baseline risk for PONV. 3. Administer PONV prophylaxis when appropriate.
Risk Factors	Risk factors can be surgery related, anesthesia related, or patient related. Most important risk factors include: History of PONV/motion sickness Nitrous oxide Opioids Volatile anesthetic Female gender Nonsmoker

Treatment	The drug classes used to treat PONV include dopamine, serotonin, muscarinic, histamine, and neurokinin receptor antagonists. Appropriate treatment depends on a careful assessment of risk versus benefit for each treatment modality.
Prevention Strategy	For patients at moderate risk, use 1-2 interventions. For patients at high risk, use a multimodal approach.

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130 Postanesthesia Care Unit

Tina Tran • Danielle B. Ludwin • In Kim • Jamie Murphy

The postanesthesia care unit (PACU) provides close monitoring of patients immediately following anesthesia. It facilitates the transition between the operating room and discharge home or transfer to a hospital ward. Common PACU concerns include hypotension, hypertension, dysrhythmias, airway obstruction, hypoxemia, hypercarbia, delayed emergence, and oliguria. Equipment and medications should be readily available for routine and critical care situations.

1) **Overview**
   a) The PACU is a high-acuity environment where physicians and nurses provide expert postoperative care of patients immediately following surgery.
   b) Staffing models
      i) Nurses[1]
         (1) The American Society of Perianesthesia Nurses state that the minimum nursing staffing requirement for Phase I PACU is two registered nurses, one competent in Phase I postanesthesia nursing, in the same room where the patient is receiving Phase I level of care.[1]
         (2) Many nurses have advanced certifications in critical care (CCRN) and/or postoperative care (CPAN or CAPA).
      ii) Physicians
         (1) According to the ASA standards and practice guidelines for postoperative care, an anesthesiologist should be responsible for general medical supervision and coordination of patient care in the PACU.[2]
   c) **ASA standards for postoperative care**[2]
      i) Standard I: All patients who have received general anesthesia, regional anesthesia, or monitored anesthesia care (MAC) shall receive appropriate postanesthesia management.
         (1) The setting should be a PACU or ICU or equivalent unless waived by the anesthesiologist responsible for the patient's care.
         (2) The care provided in this area should follow the policies and procedures reviewed by the Anesthesiology Department or the individual anesthesiologist responsible for the patient's care.
         (3) The PACU design, equipment, and staffing should meet expectations of the facility's accrediting and licensing bodies.
      ii) Standard II: A patient transported to the PACU shall be accompanied by a member of the anesthesia care team who is knowledgeable about the patient's condition. The patient shall be continually evaluated and treated during transport with monitoring and support appropriate to the patient's condition.
      iii) Standard III: Upon arrival in the PACU, the patient shall be reevaluated and a verbal report provided to the responsible PACU nurse by the member of the anesthesia care team who accompanies the patient.
         (1) The patient's preoperative history and a plan of care should be relayed to the PACU nurse.

(2) A member of the anesthesia care team should remain at the patient's side until reconnected to oxygen and monitors, a set of vital signs has been recorded, the patient's condition is reassessed, and the PACU nurse accepts responsibility for the patient.

(3) If additional physician care is required in the PACU, a thorough care transition should also occur between physicians.

iv) Standard IV: The patient's condition shall be evaluated continually in the PACU.

(1) Observation and monitoring should include oxygenation, ventilation, circulation, level of consciousness, and temperature.

(2) Documentation should be done to record the patient's vital signs and notable events while in the PACU.

v) Standard V: A physician is responsible for the discharge of the patient from the PACU.

(1) In the absence of the physician responsible for the discharge and contingent on facility guidelines, the PACU nurse may determine that the patient meets the discharge criteria. The name of the physician accepting responsibility for discharge will be noted on the record.



When transferring a patient to the PACU, a member of the anesthesia care team should remain at the patient's side until reconnected to oxygen and monitors, a set of vital signs is recorded, the patient condition is reassessed, and the PACU nurse accepts responsibility for the patient.

The patient's condition shall be evaluated continually in the PACU.

2) **Phases of postoperative care**:[1]

a) **Phase 1**

i) Nursing is focused on providing care in the immediate postoperative period and transitions the patient to Phase 2 recovery, the inpatient general floors, or step-down or ICU settings.

(1) Vital signs are typically recorded every 5-15 minutes but frequency should depend on the patient's condition.

(2) Nursing is typically staffed one nurse to two patients.

b) **Phase 2**

i) Nursing is focused on preparing the patient/family/significant others for care at home, extended observation level of care or the extended care environment.

(1) Nursing is typically staffed one nurse to three patients.

(2) "Fast-tracking" refers to having a patient bypass Phase 1 recovery and go directly to Phase 2 recovery.

(a) Fast-tracking may assist in cost reduction and reduce recovery room time. It is important to appropriately select patients for fast-tracking. ASA 1 or 2 patients undergoing local or MAC anesthesia are potentially good candidates.

c) **Phase 3/extended care**

i) Nursing is focused on ongoing care after transfer or discharge from Phase 1 and Phase 2.

(1) Nursing is typically staffed one nurse to three to five patients.

3) **Admission and monitoring**[3-6]

a) Role of the anesthesia provider

i) The anesthesia provider typically transports the patient from operating room to PACU and is responsible for patient care until the PACU nurse receives sign-out. An anesthesiology provider should be readily available to address patient issues.

ii) Most patients are transported to the PACU with nasal cannula or face mask oxygen delivered via a supplemental oxygen tank.

TABLE 130-1 The PACU Report

Patient
• Patient identification
• Surgical procedure and reason for surgery
• Past medical and surgical history
• Medications and allergies
• ASA scoring
• Preoperative vital signs and cognitive function
• Pertinent perioperative labs
Perioperative course
• Type of anesthesia
• Intubation conditions
• Anesthetic drugs
• Anesthetic course
• Lines/catheters
• Fluid management
• Postoperative pain management and possible complications

iii) Some patients require pulse oximetry, ECG, BP, or other monitoring for transport due an unstable condition.

iv) Upon PACU arrival, a complete report consists of the following (Table 130-1):

(1) Background information (name, age, procedure performed, diagnosis, past medical history, medications, allergies, preoperative vital signs, pertinent labs)

(2) Perioperative course

(a) Type of anesthetic

(b) Drugs given (premedication, induction, maintenance, opioids, muscle relaxants and reversal agents, antiemetics, vasoactive agents, antibiotics)

(c) Anesthetic course (complications, difficult IV access, difficult airway management, hemodynamic instability)

(d) Presence of lines, tubes, or drains (location, sizes, specific instructions on use or care)

(e) Fluid input and output (estimated blood and fluid losses, urine output, and fluids administered), and any other specific needs or instructions



PACU sign-out should include a pertinent summary of the patient's history and perioperative events.

b) PACU monitoring

i) Vital signs measured in the PACU include BP, respiratory rate, continuous ECG, pulse oximetry, and temperature.

ii) Typically, vital signs are recorded at intervals no greater than every 15 minutes.

iii) Level of consciousness, airway patency, breathing patterns, and peripheral perfusion should also be routinely assessed.



Hypotension in the PACU can be due to hypovolemia, impaired venous return, vasodilation, and/or decreased cardiac output.

c) Common PACU problems include cardiac, respiratory, and renal complications (Table 130-2).

4) **Cardiac complications in the PACU**[7,8]

a) **Hypotension**—Causes include hypovolemia, impaired venous return, vasodilation, and decreased cardiac output.

i) Hypovolemia

(1) Causes include inadequate intraoperative fluid resuscitation, third-space loss, osmotic polyuria, or ongoing surgical bleeding.

TABLE 130-2 Common Problems Observed in the PACU

Problem	Differential Diagnosis
Hypotension	Hypovolemia, ↓venous return, vasodilation, ↓CO, medications
Hypertension	Preexisting HTN, pain, fluid overload, hypercarbia, hypoxemia, hypothermia, ↑ICP, bladder distention, medications
Dysrhythmias	Hypercarbia, hypoxemia, acidosis, residual anesthetics, myocardial ischemia, ↑sympathetic discharge, metabolic abnormalities, preexisting cardiac/respiratory disease, medications
Airway obstruction	Mechanical, ↑secretions, vomitus/blood, glottic swelling, external compression
Hypoxemia	Atelectasis, hypoventilation, diffusion hypoxia, obstruction, bronchospasm, aspiration, pulmonary edema, pneumothorax, pulmonary embolus, medications
Hypercarbia	Respiratory depression from anesthetics/opioids, hypothermia, inadequate reversal of neuromuscular blockade, pain, splinting, abdominal distention
Delayed awakening	Residual anesthetics, ↓cerebral perfusion, stroke, hypoglycemia, sepsis, encephalopathy, metabolic abnormalities
Oliguria	Prerenal—hypovolemia; intrarenal—kidney injury; postrenal—obstruction

(2) Position the patient supine or in Trendelenburg, examine surgical drains and urine output, and evaluate for any related signs and symptoms, including lightheadedness, altered mental status, and changes in vital signs. Consider fluid boluses and vasopressor agents if symptomatic.
(3) Potential sources of ongoing bleeding should be further evaluated and surgical colleagues consulted.
(4) Persistent hypotension despite resuscitative attempts should prompt further investigation into etiology, consideration of urinary catheter placement, additional intravenous access and invasive monitoring, and additional vasopressor support.

ii) Impaired venous return
(1) Causes include positive end-expiratory pressure (PEEP), pneumothorax, and pericardial tamponade.
(2) Clinically, impaired venous return may present as increased jugular venous distention, elevated CVP, and decreased breath or heart sounds.
(3) Treatment involves addressing the underlying cause.

iii) Vasodilation
(1) Causes include residual inhalational anesthetic agents, adrenal insufficiency, neuraxial anesthesia, rewarming, anaphylaxis, transfusion reactions, drug effects, liver failure, systemic inflammation, and sepsis.
(2) Treatment addresses the underlying cause.
(3) Consider administration of α-adrenergic drugs (phenylephrine, norepinephrine, epinephrine).

iv) Decreased cardiac output
(1) Causes include congestive heart failure, arrhythmias, myocardial ischemia or infarction, negative inotropic drugs, sepsis, hypothyroidism, malignant hyperthermia.
(2) Labs (Basic Metabolic Profile, complete blood count [CBC]), 12-lead electrocardiogram (ECG), and CXR should be considered in the initial workup.
(3) Consider treatment with diuresis, antiarrhythmic agents, afterload reducers, inotropic agents, and cardioversion.

TABLE 130-3 Common Dysrhythmias[8-10]

Common supraventricular dysrhythmias • Sinus bradycardia, sinus tachycardia
Paroxysmal supraventricular tachydysrhythmias • Consider synchronized cardioversion, adenosine, verapamil, amiodarone, β-blockers, digoxin, ibutilide, procainamide • Consider administration of glycopyrrolate, atropine, or ephedrine for treatment of symptomatic bradycardia • Consider administration of B-blockade or phenyephrine for treatment of symptomatic sinus tachycardia
Stable ventricular dysrhythmias • Premature ventricular contractions (PVCs), stable nonsustained ventricular tachycardia • Usually do not require further intervention • Assess for reversible causes (hypoxemia, hypercarbia, acidosis, hypotension, electrolyte imbalances, mechanical irritation, hypothermia, adrenergic stimulation, proarrhythmic drugs, micro/macro shock, cardiac ischemia)
Unstable ventricular dysrhythmias • Ventricular tachycardia, ventricular fibrillation<Insert the following 2 additional bullet points in next line> • Consider adminstration of amiodarone or external cardioversion/defibrillation for treatment of unstable ventricular tachycardia • Consider defibrillation and administration of epinephrine or amiodarone for treatment of unstable ventricular fibrillation
Myocardial ischemia and infarction • T-wave changes (such as inversions, flattening), ST-segment changes (elevation or depression) • In patients at high risk for cardiac events, perioperative β-blockade has been shown to decrease risk • Consider cardiology consultation, 12-lead EKG, cardiac enzymes (CK, CK-MB, troponins). Supplemental O_2, β-blockade, nitrates, opioids, aspirin been shown to decrease risk

b) **Hypertension**—Causes include pain, fluid overload, preexisting hypertension, medications, hypercarbia, hypoxemia, hypothermia, increased intracranial pressure (ICP), and bladder distention.
   i) Although patients are usually asymptomatic, physical manifestations of hypertension may include visual changes, headache, restlessness, or dyspnea.
   ii) The goal of management is to identify the underlying cause and, in most cases, to restore the BP to the patient's baseline.
   iii) Postoperative hypertension (HTN) can be more pronounced if patients have held antihypertensive medications preoperatively.
   iv) β-Adrenergic antagonists, calcium channel antagonists, hydralazine, and nitrates can be used as antihypertensives.
   v) If pain is the etiology of the hypertension, opioids as well as nonsteroidal anti-inflammatory agents should be used.

c) **Dysrhythmias**—Causes include hypercarbia, hypoxemia, acidosis, residual anesthetic effects, increased sympathetic discharge from surgical procedures, metabolic derangements, and preexisting cardiac and respiratory disease (**Table 130-3**).
   i) Bradycardia can be a residual effect of cholinesterase inhibitors, β-blockers, or opioids.
   ii) Tachycardia can be due to anticholinergic agents, vagolytic drugs, and β-receptor agonists, in addition to fever, pain, hypovolemia, and anemia.

5) **Respiratory complications in the PACU**[3-5,9]

a) **Airway obstruction**—Causes include mechanical obstruction, increased secretions, vomitus or blood in the airway, glottic swelling, or from external compression (eg, expanding neck hematoma).
   i) In partial airway obstruction, stridor occurs due to limited air movement.



When called about a potential patient concern in the PACU, immediately go to the bedside to assess the patient.

ii) In complete airway obstruction, there may be paradoxical chest wall movement with lack of associated respiratory sounds.
iii) Any patient with a suspected airway obstruction should immediately receive supplemental O_2 and a jaw-thrust, head-tilt, chin-lift, oral/nasal airways, and suctioning, to relieve the obstruction.
iv) If these maneuvers are still ineffective, laryngospasm should be considered as a cause of airway obstruction.
(1) Laryngospasm can be treated with continuous positive airway pressure (CPAP). Succinylcholine (10-20 mg in normal adult patients) and continuation of 100% O_2 delivery via positive pressure ventilation should be considered for refractory cases. In the most severe cases, reintubation may be required.
v) For cases of glottic edema, intravenous corticosteroids, or aerosolized racemic epinephrine should be considered.
vi) For head and neck cases, the surgical site should be carefully inspected for signs of bleeding.
vii) Expanding hematomas of a surgical wound in the neck can lead to airway compression, and the incision site may have to be reopened to relieve the compression.



Treat laryngospasm with CPAP. Refractory cases can be treated with succinylcholine (10-20 mg IV for normal adult patients without contraindications) and 100% O_2 via positive pressure ventilation.

b) **Hypoxemia**—Causes include atelectasis, hypoventilation, diffusion hypoxia, upper airway obstruction, bronchospasm, aspiration, pulmonary edema, pneumothorax, and pulmonary embolism.
i) Hypoxemia is commonly encountered in the PACU as patients recover from general anesthesia.
ii) Transient mild to moderate hypoxemia (PaO_2 50-60 mm Hg) is generally tolerated well by otherwise healthy individuals.
iii) Signs and symptoms of hypoxemia include tachycardia, cyanosis, restlessness, and cardiac irritability.
iv) As hypoxemia progresses, acidosis, bradycardia, hypotension, neurologic obtundation, and cardiac arrest may occur.
v) Early detection of hypoxemia in the PACU is critical in identifying potential oxygenation and ventilation abnormalities.[10]
vi) Decreased functional residual capacity (FRC) is common following surgery especially after upper abdominal and thoracic procedures.
(1) Decreased FRC can lead to increased intrapulmonary shunting resulting in hypoxemia.
(2) Other causes of right to left intrapulmonary shunting include prolonged intraoperative low tidal volume ventilation, airway obstruction, pulmonary aspiration, and pulmonary edema.
(a) Pulmonary edema usually presents as wheezing within the first hour after surgery.
(b) It can be from cardiogenic causes, acute respiratory distress syndrome (ARDS), or a sudden relief of a prolonged airway obstruction, unintentional endobronchial intubation, and lobar collapse.



Progression of hypoxemia can lead to acidosis, bradycardia, hypotension, neurologic obtundation, and cardiac arrest.

vii) Pneumothorax may occur in patients who have central venous catheters, intercostal nerve blocks, history of rib fractures or lung disease (blebs, bullae), neck dissection, tracheostomy, retroperitoneal procedures, or other cases involving potential diaphragmatic penetration.
viii) Treatment—O_2 therapy is the mainstay treatment for hypoxemia.
(1) Treatment with 30%-60% O_2 is sufficient to prevent hypoxemia in patients with mild to moderate hypoventilation and hypercapnia.
(2) Pulse oximetry (SpO_2) and arterial blood gas measurements can be used to guide O_2 treatment.

(3) Consider 100% O_2 via nonrebreather mask, positive pressure ventilation, and intubation and mechanical ventilation for patients having severe or persistent hypoxemia.
(4) A chest radiograph (CXR) (ideally upright) can help assess for pneumothorax, infiltrates, and heart and lung volume and size.
(5) In cases of suspected atelectasis, interventions including incentive spirometry, chest physical therapy, and repositioning the patient to a semi-upright position can facilitate maintaining and improving FRC.
(6) Therapeutic bronchoscopy can help reexpand areas of atelectasis while also removing debris or mucous plugs.
(7) Persistent hypoxemia requiring >50% O_2 supplementation is an indication for the use of PEEP or CPAP.[4]

c) **Hypoventilation/hypercarbia**—Causes include respiratory depression from residual anesthetic agents or opioids, medication overdose, hypothermia, inadequate reversal of neuromuscular blockade, pharmacologic interaction (eg, mycins, magnesium), alterations in pharmacokinetics or metabolism, discomfort or splinting from pain, diaphragmatic dysfunction, abdominal distention, and restrictive abdominal dressings or surgical closures.
i) Definition: Hypoventilation is defined as $PaCO_2$ >45 mm Hg.
(1) Mild hypoventilation occurs commonly in postoperative patients after general anesthesia.
(2) Significant hypoventilation is usually only clinically appreciated when $PaCO_2$ is >60 or pH is <7.25.
(3) Signs and symptoms of hypoventilation may include labored respirations, depressed respiratory rate, rapid and shallow breathing, excessive or prolonged somnolence, or airway obstruction.
(4) Progression of respiratory acidosis can increase sympathetic stimulation resulting in tachycardia and hypertension.
(5) Severe acidosis can lead to circulatory depression.
(6) Shivering, hyperthermia, and sepsis can worsen the clinical changes associated with hypoventilation by contributing to increased CO_2 production.
ii) Treatment: The underlying cause must be addressed.
(1) Patients with obtunded mental status, circulatory depression, and severe acidosis (pH < 7.15) should be considered for emergent intubation and controlled ventilation.
(2) Consider diuretics for cases of circulatory fluid overload, aerosolized bronchodilators for bronchospasm, naloxone for cases of opioid-induced respiratory depression, and additional cholinesterase inhibitor for suspected residual muscle paralysis.
(3) With persistent residual respiratory depression despite a full dose of neuromuscular paralytic reversal, controlled ventilation should be maintained until spontaneous recovery occurs.


Progression of respiratory acidosis can increase sympathetic stimulation resulting in tachycardia and hypertension.

6) **Neurological problems in the PACU** include delayed awakening, neurologic damage, emergence delirium, peripheral neurologic lesions, and awareness/recall. Presence of residual anesthetics is the most common cause of delayed awakening.[4]
a) **Stroke**
i) The perioperative incidence of stroke ranges from 0.08% to 2.9%.[3]
ii) The incidence of stroke is higher following intracranial surgery, carotid endarterectomy, cardiac surgery, and patients with multiple traumas.[5]

iii) Initial signs and symptoms of stroke, such as slurred speech, visual disturbances, dizziness, agitation, confusion, numbness, muscular weakness, paralysis, and psychosis, can be nonspecific making early diagnosis challenging.

iv) Prompt neurologic consultation and imaging should be performed in all cases of suspected stroke.

b) **Emergence delirium**[3,5]

i) May present as agitation alternating with disorientation, lethargy, and inappropriate behavior

ii) It occurs more often in patients with history of drug dependency, dementia, psychiatric disorders, pediatric patients and the elderly.

iii) Drugs used in the perioperative period (especially benzodiazepines, ketamine, anticholinergics, opioids, droperidol, and large doses of metoclopramide) may contribute to emergence delirium.

iv) Other causes of delirium include hypoglycemia, metabolic derangements, hypoxemia, hypothermia, electrolyte and acid-base disorders, intracranial injury, preexisting encephalopathies, sepsis, pain, and drug withdrawal.

v) Treatment for emergence delirium includes supplemental O_2, pain control, fluid and electrolyte repletion, and supportive care.

vi) The use of antipsychotics (haloperidol), benzodiazepines, and physostigmine (to reverse delirium from anticholinergics) may also be considered.

READ MORE
Crisis management: delayed emergence, Chapter 144.



Prompt neurologic consultation and imaging should be performed in all cases of suspected stroke.

c) **Awareness/recall**[3,11]

i) Risk factors include ASA III-V, light anesthesia, trauma surgery, caesarean section with general anesthesia, cardiac surgery with cardiopulmonary bypass, substance abuse history, young age, and use of intraoperative muscle relaxants.

ii) The long-term sequelae of these experiences range from mild anxiety to severe posttraumatic stress disorder.

iii) Patients who note having these experiences should be provided with reassurance and additional support resources, including a referral for counseling.



Residual anesthetics, including inadequate reversal of neuromuscular blockade, is the most common cause of delayed awakening.

7) **Renal issues in the PACU**[3-5]

a) **Oliguria**

i) Is typically defined as urine output <0.5 mL/kg/h

ii) It may be attributed to prerenal, intrarenal, or postrenal causes. In the PACU, the prerenal (via hypovolemia) cause is most common.

(1) In prerenal states, urine electrolytes show low urine Na concentration (<10 mEq/L).

(2) A fluid bolus trial (eg, 250-500 mL crystalloid) should be considered and urine output reevaluated.

iii) Persistent oliguria warrants further diagnostic workup.

iv) The prognosis of acute renal failure is not improved with medication-induced diuresis to temporarily sustain urine output.

v) Intrarenal causes of oliguria may stem from acute tubular necrosis secondary to renal hypoperfusion, toxin exposure, or trauma. Granular casts may be present on urinalysis.

vi) Postrenal oliguria is commonly related to mechanical causes, such as urinary catheter obstruction, trauma, or iatrogenic injuries.

b) **Polyuria**
   i) Polyuria is a disproportionately high urine output for a given fluid intake.
   ii) The differential diagnosis includes large intraoperative volume administration, postobstructive diuresis, nonoliguric renal failure, osmotic diuresis, and diabetes insipidus.
   iii) Symptomatic treatment typically involves volume replacement to maintain hemodynamic stability. Electrolytes and acid-base status should be followed.

c) Renal insufficiency or failure can make a patient susceptible to electrolyte imbalances.
   i) Hyperkalemia, hypokalemia, hypomagnesemia, and acidemia can lead to cardiac arrhythmias.

8) **Postoperative pain**

a) When treating postoperative pain in the PACU setting, important considerations include whether the patient is an inpatient or outpatient in terms of choosing a short- or long-acting medication and its associated safety profile.

b) IV or oral medications can be given depending on the patient's ability to tolerate medications by mouth.

c) If a patient is enrolled in an ERAS (enhanced recovery after surgery) protocol, multimodal analgesic therapy should be implemented. Appropriate therapy minimizes opioids and typically consists of nonsteroid anti-inflammatories, acetaminophen, other nonopioid analgesics, and peripheral nerve blocks.

READ MORE
Acute pain management, Chapter 132.
Postoperative nausea and vomiting, Chapter 129.

9) **Postoperative nausea/vomiting (PONV)[12] (Fig. 130-1)**

a) Can be a significant factor in delayed PACU discharge and increased health care costs

b) Review antiemetic agents administered during the perioperative period.

c) Initial treatment is a low-dose 5HT3 antagonist.

d) If PONV persists, consider other agents such as dexamethasone, droperidol, or low-dose propofol.

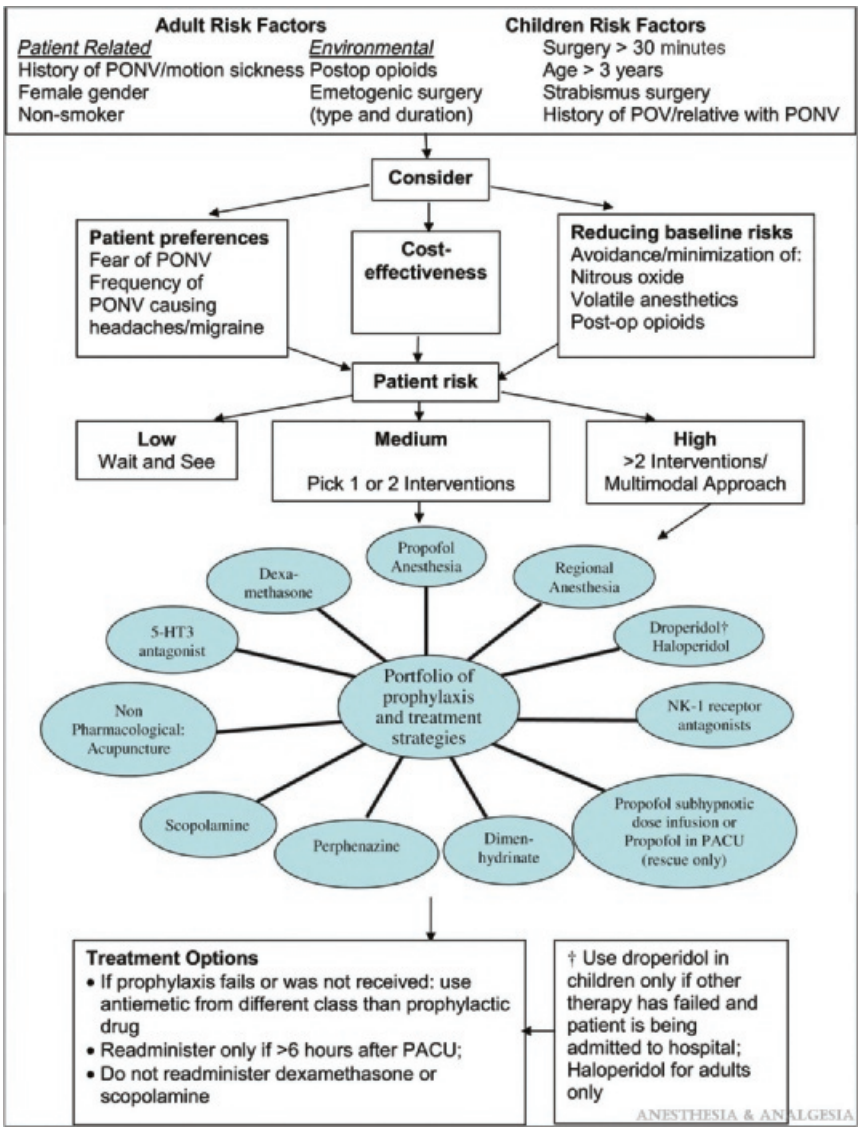
READ MORE
Postoperative nausea and vomiting, Chapter 129.

10) **Discharge criteria**[2-5]

a) According to the ASA Practice Guidelines for Postanesthetic Care, PACU discharge criteria should be based upon evaluating many factors (**Table 130-4**).
   i) Factors include neurologic status, vital signs, respiratory status, control of nausea/vomiting, and pain.
   ii) Patients should be arousable and oriented or at their baseline preoperative neurologic status.
   iii) Vital signs should be stable and within normal parameters or within the patient's preoperative baseline for at least 30 minutes.
   iv) Patients should be free from any obvious surgical complications or active bleeding and should be comfortable, without nausea and emesis, and with pain well controlled.
   v) Peripheral nerve function should be assessed and signs of returning sensory and motor function should be documented.
   vi) Patients can go home with an insensate limb if they receive appropriate counseling and support (eg, knee brace if received a femoral nerve block).
   vii) Patients that receive respiratory depressant drugs should be observed for at least 30 minutes prior to discharge.
   viii) Patients receiving supplemental O_2 in the PACU should be observed for at least 15 minutes on room air to ensure that hypoxemia does not develop.[8]

READ MORE
Crisis management: ventricular tachycardia and ventricular fibrillation, Chapter 138.
Crisis management: myocardial ischemia, Chapter 151.

FIGURE 130-1 Algorithm for Management of Postoperative Nausea and Vomiting



Reprinted with permission from Gan TJ, Diemunsch P, Habib AS, et al. Society for Ambulatory Anesthesia Consensus guidelines for the management of postoperative nausea and vomiting. *Anesth Analg*. 2014;118(1):85-113.

ix) Use of discharge scoring systems such as Postanesthesia Discharge Scoring System (PADSS) or Modified Aldrete Score may help in determining readiness for discharge.

x) Outpatients should be discharged to a responsible adult who will accompany them home and be provided with written instructions including diet, medications, activities, and an emergency phone number.

TABLE 130-4 PACU Discharge Criteria

- Stable vital signs for at least 30 min
- Ability to maintain and protect airway
- Easy arousability and full orientation
- Absence of nausea or vomiting
- Adequate postoperative pain control
- No obvious surgical complications including active bleeding
- Return of peripheral nerve function after regional anesthesia
- Ability to call for help if necessary or the presence of an adult care provider
- May use Postanesthesia Discharge Scoring System (PADSS) or Modified Aldrete Score to assist in decision-making

CHAPTER SUMMARY FOR MANAGEMENT OF PATIENTS IN THE PACU

Definition	• The PACU facilitates the transition between the operating room and discharge home or transfer to a hospital ward.
ASA Standards	• All patients shall receive appropriate postanesthesia management. • Patients transported to the PACU will be accompanied by a member of the anesthesia care team. • On arrival to the PACU, the patient will be reevaluated to ensure clinical stability and a verbal report will be given to the PACU nurse. • Patients will be evaluated continuously in the PACU. • A physician is responsible for PACU patient discharge.
Admission and Monitoring	• PACU sign-out should include a pertinent summary of the patient's history and perioperative events. • Monitoring typically includes pulse oximetry, telemetry, and blood pressure.
Common Problems	• Common PACU issues include hypotension, hypertension, dysrhythmias, airway obstruction, hypoxemia, hypercarbia, delayed emergence, PONV, and oliguria. • Hypoxemia can lead to acidosis, bradycardia, hypotension, neurologic obtundation, and cardiac arrest. • Respiratory acidosis can cause sympathetic stimulation resulting in tachycardia and hypertension. • PONV can be distressing to patients and result in prolonged PACU stay as well as increased health care costs.
Discharge Criteria	• Patients should be arousable, oriented, or at their baseline preoperative neurologic status. • Vital signs should be stable or within the patient's baseline for at least 30 min. • Patients should be free from any obvious surgical complications. • Patients' pain and PONV should be well controlled. • Patients who have received respiratory depressant drugs should be observed for at least 30 min before discharge. • Outpatients should be discharged to a responsible adult and should be provided with written instructions on outpatient care.

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131 Post-op Ventilation of the Surgical Patient§

Raymond A. Pla, Jr • Jeffrey S. Berger

Respiratory management of the postoperative patient has unique considerations. Up to 5% of patients will fail extubation following a surgical procedure.[1,2] This population typically has a short duration of intubation and rapid weaning from mechanical ventilatory support.

Postoperative pulmonary complications, including but not limited to bronchospasm, pneumonia, failed extubation, aspiration, and respiratory insufficiency/failure, are important indications for postoperative mechanical ventilation. Of these, postoperative respiratory failure is the most common.[3] This chapter will discuss management of continued mechanical ventilation from the operating room, as well as postoperative pulmonary complications that require postoperative mechanical ventilation.

1) **Discontinuation of mechanical ventilation**
   a) **General considerations**
      i) Weaning a patient from mechanical ventilation in the operating room is a rapid process.
      ii) The decision to extubate the trachea is often based on practitioner experience; however, subjective and objective data should be used.
      iii) In order to facilitate successful extubation, the patient must have the following: a patent airway, return of muscle strength, the ability to cough and protect the airway, spontaneous respiratory drive, and adequate blood oxygenation.
   b) **Extubation criteria (Table 131-1)**
      i) When evaluating a patient for discontinuation of mechanical ventilation and extubation in the operating room, it is helpful to use a systems approach.
      ii) Failure to meet these criteria will often preclude extubation and lead to intensive care unit (ICU) admission for a more gradual weaning process.
2) **Surgery-specific extubation considerations (Table 131-2)**
   a) **Cardiac**
      i) 8.5% of adult patients undergoing cardiac surgery will have postoperative respiratory complications.[4]
      ii) Cardiac fast tracking
         (1) The practice of "fast tracking" involves rapid weaning within a few hours after transfer to the ICU, as opposed to the traditional 24 hours or more of mechanical ventilation.
         (2) Postoperative respiratory complications are a significant cause of morbidity and mortality, and earlier extubation is clinically and economically favored over prolonged intubation.[4–6]
         (3) Rapid weaning should be reserved for patients with no sign of cardiac dysfunction, excess bleeding, or neurologic injury.
3) **Postoperative respiratory failure (PRF)**
   a) Inadequate exchange of oxygen (O_2) and carbon dioxide (CO_2) to maintain homeostasis.[7]
   b) Postoperative patient who have received general anesthesia for cardiac, thoracic, or upper abdominal surgery, or who have underlying pulmonary disease are at particular risk.

TABLE 131-1 Decision-Making for Extubation at the End of Surgery

Extubation Criteria/Assessment		Example
General	The acute disease process is addressed or resolved	Open wounds Impending sepsis
	Chronic systemic disease does not preclude extubation	Acute or chronic lung disease Cardiovascular disease Musculoskeletal disease
	Acceptable surgical duration	Usually <8 h
	No major surgical involvement of the chest, lungs, or muscles of respiration	
Neurologic	Arousable patient	GCS > 13 Follows commands
	Adequate pain control	Postoperative epidural or regional analgesia RR < 35
Respiratory	Sufficient respiratory drive	pCO_2 < 60 mm Hg Minute ventilation >4 L/min
	Normal mechanics of ventilation	Tidal volume 4-6 mL/kg RR < 35 Negative inspiratory force less than −20 to −30 cm H_2O
	Normal oxygenation	SpO_2 > 95% on FiO_2 1.0 PaO_2 > 60 mm Hg with FiO_2 < 0.4 PEEP < 5-10 cm H_2O PaO_2/FiO_2 > 200-300 mm Hg
	Airway protection intact	Muscle relaxants reversed Cough to eliminate secretions No upper airway edema No hemorrhage
Cardiovascular	Hemodynamic stability	Vasopressors not required for BP stability HR < 120 bpm
	No active bleeding	Hemoglobin >8 mg/dL and stable Drain output stable
Infection, fluids, and metabolic	No metabolic abnormalities	Sodium, phosphate, magnesium, and blood glucose in the normal range
	Normal pH	Range 7.35-7.45
	Normal temperature	Range 35.5°C-38°C
	No evidence of fluid overload	Absence of obvious facial or extremity edema Normal cuff leak test

Successful extubation after mechanical ventilation depends on a variety of parameters, including the patient's ability to protect the airway, and maintain adequate ventilation and oxygenation.

c) Any patient who has undergone surgery under general anesthesia is at risk for PRF due to the effects of residual anesthetic agents on neuromuscular function, pulmonary mechanics, and ventilatory responses to hypoxia and hypercarbia.

d) **Causes of postoperative respiratory failure:**

i) General anesthesia alters pulmonary mechanics.

(1) Reduction in lung capacities: vital capacity (VC), functional residual capacity, and total lung capacity (TLC)

(a) Airway closure and atelectasis result in ventilation-perfusion (V-Q) mismatch and shunt.

(b) Impaired mucous/secretion clearance

TABLE 131-2 Special Considerations With Subspecialty Surgery[3–7]

Special Patient Population	Extubation and Ventilation Considerations
Otolaryngology	Tumors may compress airway. Airway stenosis may be present. Caution should be taken following surgery in the neck, pharynx, mandible, maxilla, or adjacent structures.
Thoracic Surgery	Many patients have severe lung disease. Up to 30% of patients with severe emphysema undergoing thoracotomy will have postoperative respiratory complications. Patients undergoing lung volume reduction surgery, pneumonectomy, and lung transplant benefit from early discontinuation from mechanical ventilation due to effects of positive pressure on suture lines and prevention of reintubation.
Cardiac Surgery	Respiratory complications are usually due to cardiac dysfunction. Consider rapid weaning of ventilation in patients with no sign of cardiac dysfunction, excess bleeding, or neurologic injury.
Neurosurgery	GCS > 8 is a strong predictor of extubation success in the ICU. Possibility of ICP manipulation Prolonged mechanical ventilation may be necessary.
Trauma	Consider the possibility of undiagnosed head injury.

GCS, Glasgow Coma Scale; ICP, intracranial pressure; ICU, intensive care unit.
Strongly consider the use of noninvasive positive-pressure ventilation patients with emphysema undergoing thoracotomy to avoid reintubation.
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ii) Hypoventilation due to residual anesthetic agents
  (1) Residual neuromuscular blocking agent
  (2) Inhibition of hypoxic and hypercarbic ventilatory drive by anesthetic drugs, such as narcotics or sedatives
iii) Postoperative respiratory muscle dysfunction
  (1) Upper airway collapsibility
  (2) Phrenic nerve or recurrent laryngeal nerve injury in select (ie, neck) surgeries
iv) Exacerbation of preexisting medical conditions, such as obstructive sleep apnea or COPD



Administration of high FiO_2 (>80%) can cause absorption atelectasis.[3]

Upper abdominal surgery can reduce vital capacity by up to 60%.[7]

e) **Prevention of postoperative respiratory failure (PRF)**
  i) Identify those at greatest risk (Table 131-3).[3]
  ii) Ventilatory strategies
    (1) Monitor FiO_2.
    (2) Prevent atelectasis.
      (a) Employ positive end-expiratory pressure (PEEP).
      (b) Intermittently use recruitment maneuvers.
    (3) Prevent volutrauma.
      (a) Maintain tidal volumes between 6 and 10 mL/kg ideal body weight.
      (b) Limit airway plateau pressures to 20 cm H_2O.
  iii) Nonventilatory strategies
    (1) Anesthetic technique
      (a) Choose regional or neuraxial techniques when feasible.
      (b) Judicious use of muscle relaxants and careful attention to complete reversal prior to extubation

TABLE 131-3 Patient and Surgical-Based Risk Factors for Postoperative Respiratory Failure

Patient-Based Risk Factors	Surgical-Based Risk Factors
Advanced age	Thoracic, cardiac, or upper abdominal surgery
Male	Prolonged surgical duration
Smoking	Patient positioning: prone, Trendelenburg
Obesity	
Alcohol abuse	
COPD	
Chronic kidney disease	
Advanced liver disease	
Congestive heart failure	
Neuromuscular disease	
ASA Physical Status Class 3+	

(c) Adequate analgesia without overreliance upon narcotics
(d) Goal-directed fluid and blood administration
(e) Ensure that neurologic/neuromuscular/pulmonary/cardiovascular extubation criteria are met prior to extubation.
(f) Consider the use of advanced extubation techniques:
  (i) Bailey maneuver involves the placement of an oral supraglottic airway prior to tracheal extubation.
  (ii) Cuff-leak test: Once spontaneous ventilator parameters are adequate and suctioning has been accomplished, the pilot balloon is completely deflated and the endotracheal tube is occluded by the operator (ie, placing one's thumb over the proximal orifice). A positive test reflects audible breath sounds heard emanating from the oral cavity; alternatively, end-tidal CO_2 sampling can be detached from the circuit and placed in the mouth—a waveform would also indicate a positive cuff-leak test and confirm airway patency in advance of endotracheal tube removal.
  (iii) Staged extubation: Place a Cook exchange catheter through the endotracheal tube, extubate the patient, leaving the Cook catheter in the trachea as an aide to facilitate reintubation should it be necessary.
  (iv) The Difficult Airway Society (U.K.) recommends awake extubation or staged extubation (Bailey maneuver or tube exchange catheter) in patients with difficult airway or with risk factors for PRF.[8]
(g) The direct consequences of PRF are arterial hypoxemia, with or without hypercarbia.
  (i) Hypoxemia
    1. Partial pressure of oxygen in arterial blood (PaO_2) <60 mm Hg
    2. Severity of PRF is categorized according to the PaO_2/FiO_2 ratio[2]:
      a. Mild: PaO_2/FiO_2 ratio <300 but >200
      b. Moderate: PaO_2/FiO_2 < 200 but >100
      c. Severe: PaO_2/FiO_2 < 100
  (ii) Hypercarbia
    1. Partial pressure of carbon dioxide in arterial blood ($PaCO_2$) >45 mm Hg

(h) PRF must be treated quickly and decisively. In addition to supplemental oxygen, airway adjuvants such as nasal/oral airways, noninvasive ventilation (NIV), and mechanical ventilation (MV) may be required to address PRF.



Indirect consequences of untreated postoperative respiratory failure include, but are not limited to, tachycardia/dysrhythmia, anoxic encephalopathy, and death.

4) **Management of acute respiratory distress following surgery**

a) Noninvasive means of treatment are usually sufficient to improve pulmonary function and, thus, gas exchange.[9] These include the following:
   i) Supplemental oxygen (O_2) administration
   ii) Incentive spirometry
   iii) Chest physiotherapy
   iv) Adequate analgesia
   v) Noninvasive ventilation (NIV)
      (1) Continuous positive airway pressure (CPAP)
      (2) Bidirectional positive airway pressure (BiPAP)
      (3) Noninvasive positive pressure ventilation (NPPV)



Low flow systems produce FiO_2 values from 21% to 80%.

b) Two types of supplemental oxygen delivery systems: low flow vs high flow
   i) Low flow systems (nasal cannula, simple face masks, partial/nonrebreathing masks)
      (1) Depend on room-air entrainment to help meet the patient's minute ventilation demands
      (2) FiO_2 may vary with the size of the O_2 reservoir or become unpredictable with abnormal or changing ventilatory patterns.
      (3) Simple masks have a reservoir to provide a higher FiO_2 than that of a nasal cannula and two side ports to allow room air entrainment and exit for exhaled gases.
      (4) Partial rebreathing masks: two side ports for RA entrainment and exit of exhaled gases. The first 33% of exhaled gases fills the reservoir bag; thus, partial rebreathing occurs.
      (5) Nonrebreathing masks: similar to partial rebreathing masks but have one-way valves on each side of the mask to prevent room air entrainment and a one-way valve to prevent exhaled gas from entering the reservoir bag (**Fig. 131-1; Tables 131-4** and **131-5**).



O_2 supports combustion and is a potential fire hazard.

Avoid undue cutaneous pressure around the face from the nasal cannula or mask straps.

   ii) High flow systems (Venturi masks): Nonrebreathing systems that provide the entire inspiratory flow rate and FiO_2 to meet the patient's entire minute ventilatory demands
      (1) Flows of 30-40 L are often necessary (**Fig. 131-2; Table 131-6**).



The reservoir bag should be inflated to maximize the FiO_2 delivery.

c) **Incentive spirometer (IS):**
   i) Provides a visual goal for a patient to achieve sustained maximal inspiration
   ii) Generation of a more negative intrapleural pressure promotes alveolar hyperinflation
   iii) Compensates for atelectasis induced by a shallow tidal volume breathing pattern that often results from residual anesthesia/sedatives or pain from abdominal or thoracic surgery



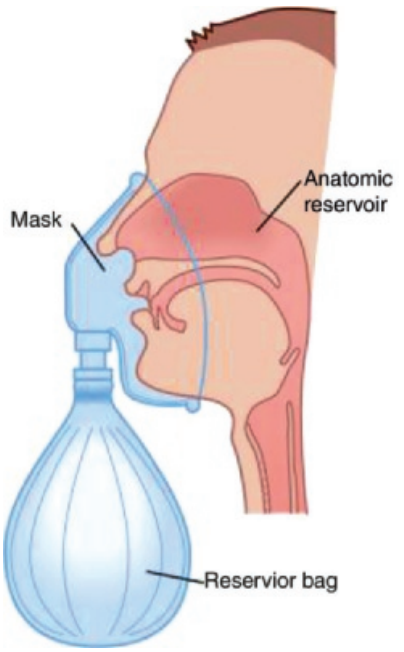
High flow systems are indicated for those with a high minute ventilation or inconsistent ventilatory pattern who require predictable FiO_2 to reverse hypoxemia.



For incentive spirometry to be effective the patient should have a vital capacity of >15 mL/kg and a respiratory rate <25.

d) **Chest physical therapy**
   i) Postural drainage and position changes move secretions toward the proximal airway for removal.

FIGURE 131-1 The Three Reservoirs of Low-Flow Oxygen Therapy.[10]



Reprinted with permission from Vender JS, Clemency MV. Oxygen delivery systems, inhalational therapy, and respiratory care. In: Benumof JL, ed. *Clinical Procedures in Anesthesia and Intensive Care*. Philadelphia, PA: Lippincott, 1992:63-87.

ii) Effective in patients with pulmonary infections or recovering from abdominal or thoracic surgery
iii) Gentle percussion and vibration therapy to the chest aid in mobilizing secretions that are adherent to bronchial walls.

e) **Noninvasive ventilation (NIV)**[12]
i) Delivery of ventilator support to the upper airway
ii) Proven alternative to tracheal intubation and mechanical ventilation (with its attendant risks) in select patients.



Rib fractures, coagulopathy, and bone metastases are relative contraindications to percussion therapy.

TABLE 131-4 Approximate FiO_2 Delivered by Nasal Cannula[a][11]

Flow Rate (L/min)	Approximate FiO_2
1	0.24
2	0.28
3	0.32
4	0.36
5	0.40
6	0.44

[a]Based on normal ventilatory patterns.
FiO_2, fraction of inspired oxygen.
Reprinted from Tokarcyzk AJ, Katz J, Vender JS. Oxygen delivery systems, inhalation, and respiratory therapy. In: Hagberg, ed. *Hagberg and Benumof's Airway Management*. 4th ed. Philadelphia, PA: Elsevier; 2018:287-308, with permission from Elsevier.

TABLE 131-5 Approximate FiO_2 Delivered by Simple Face Mask[a][11]

Flow Rate (L/min)	FiO_2
5-6	0.4
6-7	0.5
7-8	0.6

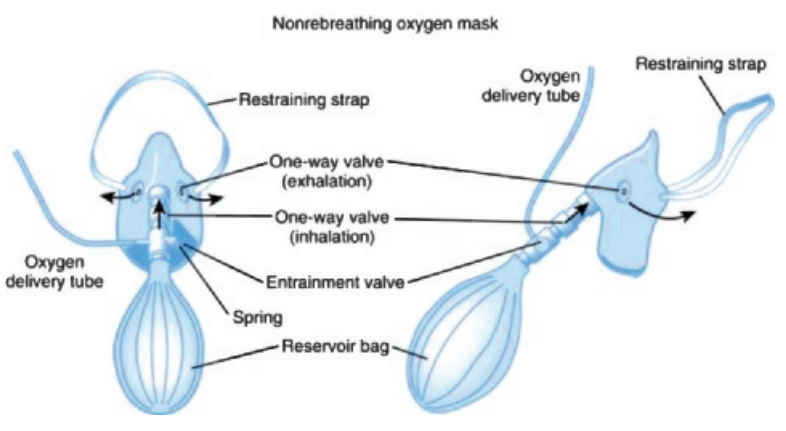
[a]Based on normal ventilatory patterns.
FiO_2, fraction of inspired oxygen.
Reprinted from Tokarcyzk AJ, Katz J, Vender JS. Oxygen delivery systems, inhalation, and respiratory therapy. In: Hagberg, ed. *Hagberg and Benumof's Airway Management*. 4th ed. Philadelphia, PA: Elsevier; 2018:287-308, with permission from Elsevier.

iii) Bilevel positive airway pressure (BiPAP)
   (1) BiPAP refers to provision of inspiratory positive airway pressure (IPAP) along with expiratory positive airway pressure.
   (2) In adjusting BiPAP, an IPAP level and a positive end-expiratory airway pressure (PEEP) are set.
   (3) The patient's spontaneous breaths trigger the machine to deliver the IPAP.
   (4) During expiration, PEEP is maintained.

iv) Continuous positive airway pressure (CPAP)
   (1) CPAP is the provision of a constant positive airway pressure, which acts to maintain positive pressure in the airway; CPAP does not offer pressure support ventilation.
   (2) Adjusted by changing the level of positive pressure to the patient, typically 4-6 cm H_2O
   (3) Useful modality to increase the functional reserve capacity and improve oxygenation
   (4) Consider CPAP (8-12 cm H_2O) for postoperative respiratory failure in patients with hypoxemia. Titrate FiO_2 to $SpO_2 > 92\%$.

v) Consider NPPV for patients with hypercarbia.

FIGURE 131-2 A Nonrebreathing Oxygen Mask. In addition to the exhalation valve, the mask has a one-way inhalation valve.[10]



Reprinted with permission from Vender JS, Clemency MV. Oxygen delivery systems, inhalational therapy, and respiratory care. In: Benumof JL, ed. *Clinical Procedures in Anesthesia and Intensive Care*. Philadelphia, PA: Lippincott, 1992:63-87.

TABLE 131-6 Approximate FiO_2 Delivered by Mask With Reservoir Bag[a][11]

Row Rate (L/min)	FiO_2
6	0.6
7	0.7
8	0.8
9	0.8+
10	0.8+

[a]Based on normal ventilatory patterns.
FiO_2, fraction of inspired oxygen.
Reprinted from Tokarcyzk AJ, Katz J, Vender JS. Oxygen delivery systems, inhalation, and respiratory therapy. In: Hagberg, ed. *Hagberg and Benumof's Airway Management*. 4th ed. Philadelphia, PA: Elsevier; 2018:287-308, with permission from Elsevier.

(1) Inspiratory pressure should be set to achieve a tidal volume of 4-8 mL/kg.
(2) Titrate respiratory rate to "normalize" the $PaCO_2$ (<45 mm Hg).
(3) PEEP 5-8 cm H_2O

f) **Tracheal intubation/mechanical ventilation**
i) Total ventilator support: minimize patient work of breathing
(1) Pressure control: preset inspiratory pressure (cm H_2O)
(2) Volume control: preset tidal volume
ii) Partial ventilatory support: allows spontaneous patient breaths
(1) Synchronized intermittent mandatory ventilation (SIMV): spontaneous breaths interspersed between machine breaths
(2) Airway pressure release ventilation (APRV): spontaneous breaths superimposed on machine breaths
(3) Modes that supply partial support with every spontaneous breath (ie, Pressure-support, pressure-assist, or proportional-assist ventilation)
(4) Newer "closed loop" modes
(a) Pressure-regulated volume-control: Pressure-targeted time-cycled breaths capable of automatically adjusting pressure to guarantee a certain tidal volume

g) **Mechanical ventilator parameter settings:**
i) Tidal volume: 6-8 mL/kg ideal body weight
ii) Respiratory rate: 12-18 breaths/min but adjust to desired $PaCO_2$
iii) FiO_2: Titrate to PaO_2 > 80 mm Hg.
iv) PEEP: 5-8 cm H_2O (Table 131-7)



Increase PEEP to improve oxygenation when FiO_2 of 1.0 is reached with persistent hypoxemia.



Consider reducing minute ventilation (TV, RR) in hypothermic patients to prevent hypocarbia/alkalosis.

Avoid NIV with significant airway swelling, severe GERD/airway secretions/blood, altered consciousness, and acute facial trauma

TABLE 131-7 The Reservoir Volume and Fraction of Attainable O_2 With Various O_2 Delivery Devices[13]

Device	Reservoir Volume (mL)	Oxygen Flow (L/min)	FiO_2
Nasal cannula	50	1-6	0.21-0.46
Simple face mask	150-250	5-10	0.4-0.6
Partial rebreather mask with reservoir bag	750-1250	5-7	0.35-0.75
Nonrebreather mask with reservoir bag	750-1250	5-10	0.4-1.0

Reprinted with permission from Marino PL. Oxygen inhalation therapy. In: *The ICU Book*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2007:403-418.

TABLE 131-8 Clinical Extubation Criteria

- Adequate SpO_2 (>92%) with $FiO_2 < 0.50$
- Awake/alert/follows commands
- Intact airway protective reflexes
- Demonstrable neuromuscular strength (head lift)
- Adequate pulmonary mechanics
- Spontaneous tidal volume >5 mL/kg
- Respiratory rate <25
- Negative inspiratory force less than −25 cm H_2O
- Vital capacity >10 mL/kg
- Hemodynamic stability

5) **Monitoring of Ventilated Patients**
   h) The following data should be obtained to ensure adequacy of corrective measures:
      i) SpO_2
      ii) Arterial blood gas measurements
      iii) Level of consciousness
      iv) Adequacy of pain control
      v) Pulmonary mechanics
      vi) Hemodynamics
      vii) Temperature
      viii) Fluid balance



Pressure mode ventilation may result in reduced tidal volumes as lung compliance decreases.



Auto-PEEP manifests as increased plateau pressure. It is important to monitor this in patients with preexisting airway obstructive disease (ie, COPD), and inadequate pulmonary mechanics may result barotrauma due to breath stacking.

6) **Extubation From Mechanical Ventilation/Extubation**
   a) Extubation should not be considered a benign procedure. If not timed and executed carefully it can lead to a number of conditions such as[14]:
      i) Hypercarbia (eg, residual anesthesia decreasing CO_2 ventilatory responsiveness)
      ii) Hypoxemia (eg, atelectasis)
      iii) Upper airway obstruction (eg, vocal cord dysfunction/edema, laryngospasm)
      iv) Tracheal obstruction (eg, subglottic edema, tracheomalacia)
      v) Aspiration (resulting from diminished or absent airway protective reflexes)
      vi) Hypertension/tachycardia/diaphoresis (from respiratory distress)
   b) Meticulous attention must be paid to generally accepted extubation criteria (**Table 131-8**):



Surgery involving the neck (ie, carotid endarterectomy, thyroid/parathyroid, cervical spine) can cause hematomas of the neck, causing airway occlusion that presents as postoperative respiratory failure.



Great caution should be taken when extubating patients following surgery on the neck, pharynx, maxilla, mandible, and adjacent structures.

CHAPTER SUMMARY: POSTOPERATIVE VENTILATION OF THE SURGICAL PATIENT

Extubation Criteria	Patient should be awake, with a patent airway, have return of muscle strength, be able to cough and protect the airway, have the drive to breathe spontaneously, and be able to oxygenate the blood
Surgical Considerations	Surgical procedure may determine ability to extubate. Take great caution with patients following **otolaryngologic**, **thoracic, cardiac, trauma, and neurological surgeries** Reintubation of patients with emphysema significantly elevates risk of barotraumas
Postoperative Respiratory Failure	A common and potentially lethal complication of surgery and anesthesia is multifactoral in etiology Both surgical and patient-based predictors help to identify who may be at elevated risk. Once identified, preventive steps such as use of minimally invasive surgical techniques, employing regional anesthesia, minimizing narcotics, and careful adherence to well established extubation criteria can be effective in prevention. Treatment options range from low flow oxygen supplementation to noninvasive ventilation strategies to tracheal intubation and mechanical ventilation

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132 Acute Pain Management

A. Katharine Hindle

The American Society of Anesthesiologists (ASA) Task Force on Acute Pain Management currently recommends a choice of three modalities forming the cornerstone of acute pain management: systemic opioids, neuraxial techniques, and regional anesthesia. The focus of this chapter will be to address these recommendations from the perspective of an acute pain service, with a focus on pharmacologic treatment of acute postoperative surgical pain. Within the current context of the opioid crisis/epidemic, an informed perspective on pain management may allow for an improvement in patients' pain control with fewer medication risks and side effects.

1) **Overview of acute pain management**
   a) Pharmacologic advancements and the concept of multimodal analgesia have improved the management of acute pain.
   b) **The goal of multimodal analgesia is to improve pain control and minimize adverse side effects of medications.**
   c) **Effective use of multimodal analgesia may minimize opioid with improved pain control.**
   d) A longer duration of prescribed opioids has been found to be predictive of prolonged opioid use, which is one of the many factors associated with the opioid epidemic in the United States.[1–5]
      i) Poor pain management in the acute postsurgical setting remains a problem.
         (1) Inadequately controlled acute pain has adverse effects on many physiologic systems including the cardiovascular, pulmonary, gastrointestinal, immunologic, renal, and hematologic systems.[6]
         (2) Most agree regarding the ethical mandate to provide adequate pain control.
            (a) Growing evidence suggests that inadequate pain control may lead to increases in morbidity, mortality, and the development of chronic pain syndromes.[6–8]



Inadequately controlled acute pain has adverse effects on cardiovascular, pulmonary, gastrointestinal, immune, renal, and hematologic systems.

      ii) The ASA Task Force on Acute Pain Management currently recommends a choice of three modalities forming the cornerstone of acute pain management. These cornerstone modalities include:
         (1) Systemic opioid therapy via patient-controlled analgesia (PCA)
         (2) Neuraxial techniques, such as spinal or epidural anesthesia
         (3) Regional anesthesia in the form of peripheral nerve blocks (PNBs)[9]
      iii) The Task Force also recommends the use of multimodal analgesia and specifically discusses the use of scheduled NSAIDs, COX-2 inhibitors, or acetaminophen.[9] Furthermore, others advocate for the use of a nonopioid as part of any analgesic regimen.[10] These types of interventions can be described as adjuvant therapy.
2) **Pathophysiology and neurobiology of pain transmission.** Formulating an effective pain control plan requires an understanding of pain transmission. Pain perception is a complex interaction involving neural, biochemical, and emotional inputs. However, a reasonable model of acute pain can be described at the most basic level.
   a) **Acute tissue damage leads to activation of specialized neurons termed nociceptors.** Two types of nociceptors, A-delta and C, have been described.

i) A-delta nociceptors respond to thermal and mechanical stimuli.
   (1) They are responsible for the fast pain response to tissue injury and the sharp sensation of acute pain.
ii) C nociceptors respond to thermal, mechanical, and chemical stimuli.
   (1) C nociceptors are involved with a slower response to tissue injury and the release of chemical mediators of pain.
   (2) They are also responsible for the dull, throbbing aspect of acute pain and may play a role in chronic pain development.

b) **Afferent nerve signals travel from these nociceptors via peripheral nerves to the spinal cord where they synapse on second-order neurons.**
   i) The nerve signal travels up spinal tracts where it is processed in higher brain centers.
   ii) The efferent nerve response travels down descending spinal tracts when it can interact and modulate further ascending pain signals or it can travel and terminate at peripheral motor neurons.



A-delta nociceptors are responsible for fast pain response and sharp sensation of acute pain. C nociceptors are responsible for dull throbbing pain.

c) In addition, the initial tissue injury leads to the release of chemical mediators including leukotrienes, bradykinins, serotonin, histamine, potassium, substance P, thromboxanes, and prostaglandin. These mediators serve to activate or modulate further nociceptive afferent signals.[11]



Understanding this neurobiological pathway involved in pain signal processing provides the clinician with various cellular targets for pharmacologic intervention in an effort to control pain.

3) **Receptor targets**
   a) Examples include opioids, which bind to mu opioid receptors located primarily in the spinal cord.
   b) Local anesthetics bind and inhibit nerve signal transmission via inactivation of sodium channels at the peripheral nerve (PNBs) or at the spinal roots (neuraxial block).
   c) NSAIDs and acetaminophen have analgesic effects via inhibition of prostaglandin synthesis with various chemical mediators mentioned above involved in pain transmission.
   d) Ketamine binds and inhibits the *N*-methyl-D-aspartate (NMDA) receptor located in the brain.

READ MORE
Opioids, Chapter 215

4) **Medications used for acute pain management**
   a) **Opioids**
      i) Mechanism of action—Opioids bind and activate opioid receptors.
         (1) Opioid receptors are mostly located in lamina I and the substantia gelatinosa of the spinal cord.
         (2) However, they have also been identified in the brain and in the periphery, and they are activated in the presence of inflammation.[12]
      ii) Adverse effects Side effects of opioids include respiratory depression, impaired gastric motility, nausea, vomiting, sedation, pruritus, and urinary retention.
      iii) Dosing
         (1) Opioids should be dosed for each patient's individual needs.
         (2) The proper dose is the dose that produces the desired effect with minimal adverse side effects.[7,12]
      iv) Specific agents
         (1) Morphine
            (a) Morphine is the most commonly used and studied opioid in the United States.
            (b) An appropriate initial choice for somatic pain but has low efficacy in the treatment of neuropathic pain.[7]

(c) Metabolized in the liver
  (i) Principal metabolites include morphine-3-glucuronide and morphine-6-glucuronide.
  (ii) Metabolites are excreted by the kidneys. Morphine-6-glucuronide is an active metabolite possessing sedating and analgesic properties.[12,13]
  (iii) Therefore, morphine may be a poor choice for a patient with renal failure.



Morphine may be a poor choice in patients with renal failure due to accumulation of morphine-6-glucuronide metabolites.

(d) Appropriate initial IV PCA settings for morphine in the opioid-naive patient should be 1-2 mg every 8-10 minutes.

(2) Hydromorphone
  (a) Hydromorphone is a commonly used opioid, which is 3-8 times more potent than morphine.[12,13]
  (b) If the potency of hydromorphone is considered as being five times greater than morphine; conversions between the two medications can be easily accomplished.
    (i) For example, 1 mg of morphine would be equianalgesic to 0.2 mg of hydromorphone.
    (ii) Hydromorphone is metabolized in the liver to inactive metabolites.[13] Therefore, hydromorphone may be a better choice in patients with renal insufficiency.
  (c) Initial intravenous PCA settings for hydromorphone should be 0.2-0.4 mg every 8-10 minutes.

(3) Fentanyl
  (a) Fentanyl is a synthetic opioid and anywhere from 80 to 125 times more potent than morphine.[13]
  (b) It is metabolized in the liver to inactive metabolites, which are excreted in the urine.[12]
  (c) Fentanyl is administered in micrograms.
    (i) If the potency of fentanyl is considered to be 100 times greater than morphine, conversions become easy to accomplish.
    (ii) For example, 10 µg of fentanyl would equal 1000 µg (1 mg) of morphine.
  (d) Appropriate initial IV PCA settings for fentanyl should be 10-20 µg every 6-8 minutes.

(4) Meperidine
  (a) Meperidine is 7-10 times *less* potent than morphine.[12,13]
  (b) Meperidine's mechanism of action is at opioid receptors, but it also possesses some antagonist activity at NMDA receptors.[12]
  (c) In addition, meperidine inhibits norepinephrine (NE) and serotonin reuptake.[12]
  (d) Metabolized in the liver[12]
    (i) An active metabolite normeperidine is formed, which relies on renal excretion.[13]
    (ii) Normeperidine lowers the seizure threshold.
    (iii) Caution should be used with patients who have seizure disorders or with renal failure.
    (iv) Concomitant use with monoamine oxidase inhibitors has been associated with malignant hyperpyrexia.[13]
  (e) Meperidine is not recommended for use in PCA.



Meperidine should be used with caution in patients with renal failure or seizure disorders or in those who are taking monoamine oxidase inhibitors.

(5) Methadone
(a) Methadone has been described as being anywhere from equipotent or up to seven times more potent than morphine.[12,14]
(b) It is metabolized in the liver by the cytochrome P-450 system to primarily inactive metabolites that are excreted in the urine and bile (fecal route).[14]
(c) Methadone is unique in that is has an extremely long half-life. The t1/2 alpha is 2-12 hours, while the t1/2 beta is anywhere between 15 and 60 hours.[14]
(d) Primary action is at opioid receptors
(i) However, methadone exhibits some antagonism at NMDA receptors.[12,14]
(ii) Methadone also inhibits NE and serotonin reuptake.[12,14]
(iii) Therefore, methadone is a useful agent for patients with both somatic and neuropathic pain components.
(e) Methadone is associated with the risk of QT interval prolongation and torsade de pointes, and serial EKGs should be monitored in patients with prescribed methadone.[14]


Methadone is associated with risk of QT interval prolongation and torsade de pointes.

b) **Nonsteroidal anti-inflammatory drugs (NSAIDs)**
i) NSAIDs can be used solely for the treatment of mild to moderate pain.[7,15]
ii) Additive and/or synergistic effects have been shown when used with other agents.[15]
iii) Therefore, NSAIDs represent a useful component of a multimodal analgesic plan for severe postoperative pain. The use of NSAIDs is specifically mentioned by the ASA Task Force on Acute Pain Management.
iv) NSAIDs inhibit prostaglandin synthesis through the arachidonic acid pathway.
(1) Prostaglandins are important mediators of pain, inflammation, and fever.
(2) Specifically, NSAIDs reversibly inhibit the enzyme cyclooxygenase (COX) that converts arachidonic acid to prostaglandins.[7,10,15,16]
(3) There are two major isoforms of the COX enzyme, COX-1 and COX-2.
(a) COX-1 is constitutively expressed and is involved in platelet aggregation.
(b) COX-1 also exerts protective effects within the kidneys and gastrointestinal tract.
(c) The COX-2 isoform is inducible and is involved in pain and inflammatory states.[7,15,16]
(4) Traditional NSAIDs inhibit both isoforms of the COX enzyme and are termed nonselective NSAIDs.
(5) Newer COX-2–specific agents, termed coxibs, were developed in an effort to minimize side effects associated with COX-1 inhibition.[10,17]


Risks of NSAIDs include bleeding from gastroduodenal ulceration and renal failure.

v) Adverse effects
(1) Side effects attributed to NSAIDs are associated with inhibition of the COX enzymes.
(2) Patients utilizing nonselective NSAIDs are at risk from bleeding, gastroduodenal ulceration, and renal failure.[10]
(3) COX-2–selective agents have a lower risk of bleeding and ulceration.[10]
(a) However, the risk of renal failure is unchanged with these agents.[10] Information suggests that the COX-2 enzyme may play a role in cardiovascular protection.[15]
(b) COX-2 inhibitors have been associated with an increased risk of myocardial infarction and stroke.[10]


COX-2 inhibitors have been associated with an increased risk of MI and stroke.

vi) Specific agents
(1) Ketorolac
(a) Ketorolac is a potent nonselective NSAID and should be considered part of a multimodal analgesic plan.
(b) Ketorolac is very effective in managing pain associated with recovery following thoracotomy.
(c) Dosing is 30 mg IV followed by 15 mg IV every 6 hours for 3 days.[10]
(d) Ketorolac can be associated with renal failure and should be used with caution in the elderly or in patients with impaired kidney function.[10] Concerns have been raised regarding an increased risk of perioperative bleeding and an association with ketorolac administration. There is evidence that bleeding time is prolonged with ketorolac administration, but it has not been shown to have a clinically significant increase in perioperative bleeding risk.[19]
(2) Celecoxib
(a) Celecoxib is a COX-2–specific inhibitor used for mild to moderate pain control or as an adjuvant as part of a multimodal analgesic plan. Celecoxib is administered orally.
(b) Dosing is 200 mg orally every 12 hours with a maximum dose of 400 mg/d.[10]
(c) Celecoxib is metabolized in the liver to inactive metabolites.[12] The $t_{1/2}$ beta is ~11 hours.[15]
(d) Celecoxib is contraindicated in those with an allergy to sulfa-containing medications.[15]
(e) As mentioned previously, coxibs have been associated with an increased risk of myocardial infarction and stroke.
(i) The other two COX-2–specific medications, rofecoxib and valdecoxib, were initially approved and later withdrawn from the US market.[10]
(ii) However, the use of celecoxib is considered appropriate in those without a significant cardiovascular history who may derive benefit from its COX-2 specificity.

c) **Acetaminophen**
i) Acetaminophen is often overlooked as part of an effective postoperative pain management plan.
ii) Synergistic and/or additive effects have been suggested when it has been used in combination with opioids, NSAIDs, and tramadol.[17]
iii) Therefore, this agent may be considered as an adjuvant in the setting of multimodal analgesia.
iv) Mechanism of action
(1) Acetaminophen's mechanism of action is unclear, but research suggests that it acts as a central inhibitor of prostaglandin synthesis through COX inhibition.
(2) In addition, it has been demonstrated to inhibit spinal transmission of nociception in the dorsal horn via serotonergic interactions.[17]
(3) Some evidence points to inhibition of a third isoform of the COX enzyme (COX-3).[10]


Acetaminophen has been associated with hepatic necrosis during acute overdose.

v) Adverse effects
(1) Acetaminophen has been associated with hepatic necrosis during an acute overdose.[10]
(2) Risk factors include those with known liver disease, excessive alcohol consumption, and those with decreased glutathione stores (malnutrition, starvation).[10]
(3) Acetaminophen has no adverse effects on platelets or upon the gastrointestinal mucosa.[10]

vi) Dosing
(1) Current dosing is focused on the total amount of acetaminophen ingested per day.
(2) 4000 mg orally per day has been traditionally considered safe for healthy individuals.
(3) A maximum dose of 3000 mg orally per day should be utilized in those with mild liver impairment and in the elderly.[10]


The daily dose of acetaminophen should be limited to 4000 g/d for healthy individuals.

d) **Ketamine**
i) Ketamine is used as an intravenous general anesthetic and is also a potent analgesic and can be used at subanesthetic doses for the treatment of acute pain.
(1) It is useful as an adjuvant to opioid therapy and should be considered when severe pain is resistant to opioid therapy.[8]
(2) In addition, ketamine is effective for the treatment of neuropathic and chronic pain states.[10]


Ketamine can be associated with psychomimetic side effects, including hallucinations, bad dreams, and delirium.

ii) Mechanism of action
(1) Ketamine is an NMDA receptor antagonist.[8]
(2) Ketamine also may have some effects at opioid receptors.[8]
iii) Adverse effects
(1) Ketamine can be associated with psychomimetic side effects, including hallucinations, dreams, and delirium.[8] These effects may be mitigated with a benzodiazepine.
(2) Ketamine is a potent sialagogue, and these effects may be minimized with a dose of an anticholinergic medication (eg, glycopyrrolate).
iv) Dosing
(1) Low-dose ketamine is defined as intravenous dosing of <1 mg/kg.[8]
(2) Doses of 12.5 mg intravenously can be administered at the bedside with proper hemodynamic and respiratory monitoring (blood pressure cuff, pulse oximeter).
(3) Do not exceed a dose of 1 mg/kg total.
(4) Ketamine is metabolized in the liver to norketamine.

e) **Tramadol**[18]
i) Tramadol is useful in the treatment of mild to moderate pain or as an adjuvant.
ii) Mechanism of action
(1) Tramadol possesses opioid agonist properties, although it is not classified as an opioid.
(2) Tramadol also acts as an inhibitor of serotonin and NE reuptake.[12]
iii) Metabolism occurs in the liver with renal elimination of metabolites.
(1) The $t_{1/2}$ beta is ~6 hours.
(2) Unlike traditional opioid receptor agonist, tramadol has no effects on respiration and a low incidence of constipation.[12]

5) **Clinical management.** When formulating a specific analgesic plan, there are several key concepts to consider.
a) **A detailed patient history must be obtained.**
i) Classify—Classify the pain as acute or chronic. Remember, it is possible to have acute postoperative pain while also having an ongoing chronic pain syndrome.
ii) Qualify—Qualify the pain as being somatic, visceral, or neuropathic in nature. Again, it is possible for multiple types of pain to exist in the same patient at the same time.
iii) Quantify—Quantify the level of baseline pain. It is unreasonable to expect a pain-free postoperative recovery period for the chronic pain patient who experiences 7/10 pain on a daily basis. On the other hand, a healthy patient can probably expect to have postoperative surgical pain kept to a minimum during his/her recovery period.

iv) Medication intake—It is important to note the types of pain medications the patient requires to maintain baseline functioning prior to surgery. This will help in choosing appropriate initial PCA settings. An opioid-naive patient will respond differently than the patient taking chronic opioid therapy at home.

b) **Cornerstone therapy**. Patients should be placed on at least one of the cornerstone therapies outlined by the ASA Task Force on Acute Pain Management. Some patients will be candidates for two or more of the cornerstone therapies.
   i) Opioids
      (1) In addition to a well-rounded multimodal approach, opioid therapy may be considered for patients with moderate to severe pain.
      (2) Morphine, hydromorphone, or fentanyl should be considered for most patients.
      (3) Methadone should be reserved for those with coexisting chronic pain syndromes and those with significant neuropathic components to their pain.
   ii) Peripheral nerve blocks
      (1) Local anesthetic single injection or continuous block techniques should be considered for all procedures involving the upper and lower extremities.
      (2) PNBs are considered standard of care at many institutions for hand, elbow, hip, knee, ankle, and foot procedures.
   iii) Neuraxial techniques
      (1) Epidural catheter placement with a continuous infusion of local anesthetic is the modality of choice for thoracotomy, upper and lower abdominal, and gynecologic procedures.
      (2) Along with local anesthetics, opioids can be administered directly through a properly functioning epidural catheter resulting in improved pain control at lower medication doses.

c) **Adjuvant medications**
   i) Following the initiation of appropriate cornerstone therapy, adjuvant medications should be considered for most patients expected to experience moderate to severe postoperative pain.
   ii) NSAIDs
      (1) Scheduled ibuprofen, 200 mg orally three times per day, can be an effective adjuvant for healthy patients.
      (2) A short course of IV ketorolac is very helpful for the management of severe pain particularly those recovering after a thoracotomy.
      (3) Celecoxib is effective for those with a history of gastrointestinal ulceration.
   iii) Acetaminophen
      (1) Scheduled acetaminophen is also an effective adjuvant when used at the appropriate strength.
      (2) Remember to limit the daily dose under 4000 mg/d for healthy adult patients.
   iv) Ketamine
      (1) Ketamine should be reserved to help with severe pain that has been unresponsive to opioid therapy.
      (2) Ketamine is particularly helpful in those with coexisting neuropathic and chronic pain syndromes.
   v) Tramadol
      (1) Tramadol can also be helpful for the acute pain patient with coexisting neuropathic and chronic pain syndromes.
      (2) It should be considered for the patient with mild to moderate pain complaints despite opioid and other adjuvant therapy use.



Ketamine is particularly helpful in those with coexisting neuropathic and chronic pain syndromes.

CHAPTER SUMMARY FOR ACUTE PAIN MANAGEMENT

Overview	Effective treatment of acute pain requires an understanding of the pathophysiology and receptor targets involved with pain transmission.
Acute Pain Management Approach	A thorough history should be taken and physical exam performed. Pain should be classified, qualified, and quantified. Current pain medications should be noted. Together, these factors should be considered when implementing cornerstone therapies.
Cornerstone Therapies	The ASA Task Force on acute pain recommends that patients be given at least one cornerstone therapy: systemic opioids, neuraxial anesthesia, or PNB. Some patients may qualify for more than one therapy, and multimodal pain management is recommended to minimize adverse effects of medications.
Adjuvant Therapies	Adjuvant therapy should be considered and added as necessary. Adjuvant therapies include NSAIDs, acetaminophen, ketamine, tramadol.

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133 Using Emergency Manuals: Helping Teams to Care for Patients More Effectively

Sara N. Goldhaber-Fiebert

This chapter introduces the concept of emergency manuals, along with implementation and use, followed by the contents of the Stanford Emergency Manual (SEM): Cognitive Aids for Perioperative Critical Events by Stanford Anesthesia Cognitive Aid Group. The most updated version of the SEM is available for cost-free electronic download, in multiple languages, at http://emergencymanual.stanford.edu. Like all tools, emergency manuals must be used appropriately.

1) **Overview**
   a) When rare crises occur, we strive to deliver appropriate patient care both efficiently and accurately, yet patients die because of frequent omissions or delays in key actions.[1]
   b) Stress and chaos impair cognitive abilities of expert professionals across diverse safety-critical industries.[2]
   c) No clinician is expert in managing all rare crises.[3]
   d) Multiple simulation-based studies show that cognitive aids can help clinicians to perform better in delivering appropriate patient care during crises.[4–9]
   e) Emergency manuals (EMs), crisis checklists, and similar terms refer to context-relevant sets of cognitive aids, designed for use in a certain clinical context, such as operating rooms and procedural settings.
   f) This chapter provides perioperative teams with tools for educational and clinical use as well as institutional implementation, with the caveat that, like all tools, EMs must be used appropriately.



No clinician is expert in managing all rare crises.

2) **Emergency manuals (EMs)**
   a) EMs complement, rather than replace, good training, clinical judgment, and teamwork, and should *never* interfere with immediate clinical actions such as chest compressions.
   b) These tools do not jump off the wall to treat a patient, so content, design, and access to them are a good start but usually insufficient.
   c) Effective use also requires understanding of *why* to use them as well as skills for *how* to use them.[9] Good design and training can both help to support effective use.
3) **Toolkit and tools**
   a) A practical and cost-free toolkit for implementation of any EM, including a road map, adaptable training resources, customizable templates, and annotated references, is available at www.implementingemergencychecklists.org/.
   b) Hyperlinked toolkit sections include Get started; Get Buy-In; Create a Multidisciplinary Team; Select, Customize and Test the Checklists; Train Staff; Start Using the Checklists; Monitor Use; Spread Beyond the OR, each with links to relevant resources and references to support the implementation team throughout the process.

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c) Multiple variants of cost-free tools, including for special contexts such as pediatric or obstetric patients, as well as further resources, are available from the Emergency Manuals Implementation Collaborative at www.emergencymanuals.org/.

4) **Customization**
   a) Local customization of resources is extremely helpful: at a minimum provide local emergency phone numbers; try to adapt key details to be consistent with local practice.
   b) Assembling an interprofessional team for local customization increases broad commitment to the intervention because team members develop a sense of ownership.
   c) Local institutions are welcome to customize these cognitive aids for local use and to print "Adapted by INSTITUTION NAME" while retaining credit line from the developing group.
   d) The SEM is covered by Creative Commons licensing for noncommercial use, with attribution, and allows local derivatives (for local clinical use, not for broad distribution). If you contact us, via http://emergencymanual.stanford.edu with your intended use, we can provide a modifiable word document for this purpose.
   e) When customizing, beware that it is easy to introduce content errors (eg, via autocorrect changing dose abbreviations) and that adding more information on a page may make key content less usable under stress.
5) **Implementations and clinical uses**
   a) Content in the toolkit above was developed from research studying effective implementations.
   b) There is a dose-response relationship between the number of implementation steps completed and the success of implementation.[10]
   c) While crises are rare, institutions that have implemented EMs have shown significant EM use, with positive impacts.
      i) In survey studies, up to 45% of respondents reported that they had used an EM during a clinical crisis, and almost all stated it was very helpful.[11,12]
      ii) Both simulation and early clinical studies suggest that mechanisms for improved delivery of patient care include improved team communication, as well as content accessibility and decreased stress for individuals.[13,14]
6) **Disclaimer**
   a) The material in this Manual is not intended to be a substitute for sound medical knowledge and training.
   b) Clinicians should always use their clinical judgment and decision-making for patient management.
   c) Since medical conditions described in this Manual can have variable presentations, departure from the information presented is encouraged when appropriate.

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ACLS (For Perioperative Setting)
Asystole/PEA
Bradycardia–Unstable
Supraventricular Tachycardia–Stable
Supraventricular Tachycardia–Unstable
VF/VT–Pulseless

BROAD DIFFERENTIAL DIAGNOSIS
Hypotension
Hypoxemia

SPECIFIC CRITICAL EVENTS
Anaphylaxis
Amniotic Fluid Embolism
Bronchospasm (Intubated Patient)
Delayed Emergence
Difficult Airway Unanticipated
Fire–Airway
Fire–Patient
Hemorrhage
Local Anesthetic Toxicity
Malignant Hyperthermia
Myocardial Ischemia
Oxygen Failure
Pneumothorax
Power Failure
Total Spinal Anesthesia
Transfusion Reactions
Venous Air Embolus

CRISIS RESOURCE MANAGEMENT
Crisis Resource Management

EMERGENCY MANUAL

COGNITIVE AIDS FOR PERIOPERATIVE CRITICAL EVENTS 2016, V3.1

STANFORD ANESTHESIA COGNITIVE AID GROUP

134 Part A: ACLS (For Perioperative Setting)

Asystole/PEA

SIGNS

FLAT LINE: 

or

+

PULSE

CPR:

1. **100-120** compressions/minute;
   ≥ **2** in deep
   Allow complete chest recoil.
2. **Minimize breaks** in CPR.
3. Rotate compressors q2min.

Assess CPR quality, improve IF:

- **$ETCO_2$ < 10 mm Hg**
- **Arterial line Diastolic < 20 mm Hg**

1. CALL FOR HELP.
2. CALL FOR CODE CART.
3. INFORM TEAM.

IMMEDIATE

1. Turn **OFF** vasodilating volatile & IV drips; increase to 100% O_2, high flow.
2. Ventilate **10 breaths/minute**; do not overventilate.
3. Ensure **IV access** (or consider intraosseous).
4. **Epinephrine**—1 mg IV push q3-5min
5. If **rhythm changes** to **VF/VT** (shockable rhythm) → immediate defibrillation. **Go To VF/VT.**
6. Consider **ECMO** if available and reversible cause.
7. Consider TTE or TEE **Echocardiography** to evaluate cause.

DIAGNOSIS

Consider common perioperative Ddx:

1. Hemorrhage
2. Anesthetic overdose
3. Septic or other shock states
4. Auto-PEEP
5. Anaphylaxis
6. Medication error
7. High spinal
8. Pneumothorax
9. Local anesthetic toxicity
10. Vagal stimulus
11. Pulmonary embolus

Find and Treat Causes—H's and T's: Expanded on next page.

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DETAILS

1. **Hypovolemia:** Give rapid bolus of IV fluid. Check hemoglobin/hematocrit. If anemia or massive hemorrhage, give blood. Consider relative hypovolemia: Auto-PEEP (disconnect circuit); high spinal; or shock states (eg, anaphylaxis). **Go To relevant event.**
2. **Hypoxemia:** Increase O_2, to 100% high flow. Confirm connections. Check for bilateral breath sounds. Suction ET tube and reconfirm placement. Consider chest x-ray. **Go To Hypoxemia.**
3. **Tension pneumothorax:** Unilateral breath sounds, possible distended neck veins, and deviated trachea (late signs). Perform emergent needle decompression (2nd intercostal space at mid-clavicular line) then chest tube placement. Call for chest x-ray, but do NOT delay treatment. **Go To Pneumothorax.**
4. **Thrombosis—Coronary:** Consider transesophageal (TEE) or transthoracic (TTE) echocardiography to evaluate ventricle wall motion abnormalities of the ventricles. Consider emergent coronary revascularization. **Go To Myocardial Ischemia.**
5. **Thrombosis—Pulmonary:** Consider TEE or TTE to evaluate right ventricle. Consider fibrinolytic agents or pulmonary thrombectomy.
6. **Toxins (eg, infusions):** Consider medication error. Confirm no infusions running and volatile anesthetic off. If local anesthetic toxicity **Go To Local Anesthetic Toxicity.**
7. **Tamponade—Cardiac:** Consider placing TEE or TTE to rule out tamponade. Treat with pericardiocentesis.
8. **Hypothermia ↓:** Active warming by forced air blanket, warm IV fluid, raise room temperature. Consider cardiopulmonary bypass.
9. **Hyperthermia ↑:** If Malignant Hyperthermia, call for MH Cart. Give dantrolene immediately: start at 2.5 mg/kg. MH Hotline: (800) 644-9737. **Go To Malignant Hyperthermia.**
10. **Obtain ABG to rule out:**
    - **Hyperkalemia ↑:** Give calcium chloride 1 g IV; D50 1 amp IV (25 g Dextrose) + Regular Insulin 10 units IV. Monitor glucose. Sodium bicarbonate 1 amp IV (50 mEq)
    - **Hypokalemia ↓:** Controlled infusion of potassium and magnesium
    - **Hypoglycemia:** If ABG delay, check fingerstick. Give D50 1 amp IV (25 g dextrose). Monitor glucose.
    - **H+ Acidosis:** If profound, consider sodium bicarbonate 1 amp IV (50 mEq). May consider increasing ventilation rate (but can decrease CPR effectiveness so monitor)
    - **Hypocalcemia:** Give calcium chloride 1 g IV.

END

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135 | Bradycardia–Unstable

SIGNS

1. CHECK FOR PULSE
- If NO pulse, **Go To PEA/Asystole.**
- If pulse present but hypotensive, proceed with treatment.

1. CALL FOR HELP.
2. CALL FOR CODE CART.
3. HALT SURGICAL STIMULATION.

TREATMENT

1. Increase to **100% O_2**, high flow.
2. Confirm adequate **ventilation** and **oxygenation**.
3. Consider turning down or **OFF** all anesthetics.
4. **Atropine**: 0.5-1 mg IV, may repeat up to 3 mg. Consider infusions below.
5. Consider transcutaneous **pacing**:
   - ↑ • Set rate to at least 80 bpm.
   - **OR** • Increase current until capture achieved.
   - ↓ • Confirm patient has pulse with capture.
6. Consider infusions:
   - **Dopamine**: 2-20 µg/kg/min
   - **Epinephrine**: 2-10 µg/min

SECONDARY

1. Place **arterial line**.
2. Send labs: ABG, hemoglobin, electrolytes.
3. Rule out **ischemia**: Consider EKG, troponins.

END

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136 Supraventricular Tachycardia–Stable

SIGNS

1. **CHECK FOR PULSE.**
   - If NO pulse, **Go To PEA/Asystole.**
   - If **Unstable, Go To SVT—UNSTABLE.**
     Prepare for synchronized cardioversion.
     UNSTABLE = ANY OF: Sudden and/or continuing sharp decrease in BP; acute ischemia; SBP < 75.
2. **Sinus tachycardia** is **NOT SVT**. May be compensatory. Search for and treat underlying cause(s).
3. More likely **SVT THAN SINUS** if any of:
   - Rate >150
   - Irregular
   - Sudden onset

1. CALL FOR HELP.
2. CALL FOR CODE CART?
3. INFORM TEAM.

IMMEDIATE

1. Increase to **100% O_2**, high flow.
2. Confirm adequate ventilation, oxygenation.
3. Consider **12-lead EKG or Print Rhythm Strip**, then treat per rhythm (**Go To** next page).
4. **If UNSTABLE at any point: Go To SVT—UNSTABLE.**
5. Consider placing defibrillator pads.
5. If still STABLE supraventricular tachycardia consider:
   - arterial line
   - Check ABG and electrolytes.
7. Consider STAT cardiology consult.
8. **Go To** next page.

Go To Next Page

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Narrow Complex and Regular

1. **Adenosine** 6 mg IV push with flush. May give 2nd dose: 12 mg IV (avoid adenosine if asthma or WPW).
2. If NOT converted, may rate control.
   Choose beta-blocker or calcium channel blocker:
   - Beta-Blocker: (consider avoiding if asthma)
     - **Esmolol**: Start 0.5 mg/kg IV over 1 minute. May repeat after 1 minute and may start infusion 50 μg/kg/min
     - **Metoprolol**: Start 1-2.5 mg IV. May repeat or double after 2.5 minutes
   - Calcium Channel Blocker:
     - **Diltiazem**: 5-10 mg IV over 2 minutes. May repeat after 5 minutes

Narrow Complex and Irregular

1. Choose beta-blocker or calcium channel blocker:
   - Beta-Blocker: (Consider avoiding if asthma)
     - **Esmolol**: Start 0.5 mg/kg IV over 1 minute. May repeat after 1 minute and may start infusion 50 μg/kg/min
     - **Metoprolol**: Start 1-2.5 mg IV. May repeat or double after 2.5 minutes
   - Calcium Channel Blocker:
     - **Diltiazem**: 5-10 mg IV over 2 minutes. May repeat after 5 minutes
2. **Amiodarone**: 150 mg IV **SLOWLY** over 10 minutes. May repeat once. Start infusion 1 mg/min for first 6 hours.

Wide Complex and Regular (monomorphic)

1. If SVT with aberrancy **Adenosine**: 6 mg IV push with flush. May give 2nd dose: 12 mg IV (avoid adenosine if asthma or WPW)
2. If VT or uncertain VT versus SVT with aberrancy:

Amiodarone: 150 mg IV SLOWLY over 10 minutes. May repeat once. Start infusion 1 mg/min for first 6 hours.

May also consider procainamide or sotalol

Wide Complex and Irregular (Likely Polymorphic VT)

If unstable immediate defibrillation

If stable have defibrillator pads on and consult cardiology.

END

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137 Supraventricular Tachycardia–Unstable

SIGNS

1. **CHECK FOR PULSE.**
   - If NO pulse, **Go To PEA/Asystole.**
2. **UNSTABLE** = ANY OF: Sudden and/or continuing sharp decrease in BP; acute ischemia; SBP < 75.
3. Sinus tachycardia is **NOT SVT**. May be compensatory. Search for and treat underlying cause(s).
4. More likely **SVT THAN SINUS** if any of the following:
   - Rate >150
   - Irregular
   - Sudden onset

1. CALL FOR HELP.
2. CALL FOR CODE CART.
3. INFORM TEAM.

TREATMENT

1. Increase to **100% O_2**, high flow. Decrease volatile anesthetic.
2. Confirm adequate ventilation, oxygenation.
3. If unstable SVT, **IMMEDIATE SYNCHRONIZED CARDIOVERSION**—biphasic doses
   - Narrow complex and Regular: 50-100 J
   - Narrow complex and Irregular: 120-200 J
   - Wide complex and Regular: 100 J
   - Wide complex and Irregular requires Unsynchronized Defibrillation: 200 J
4. **If unsuccessful cardioversion**: Re-SYNC and increase Joules incrementally for Synchronized Cardioversion.
5. While preparing to cardiovert (do NOT delay), if narrow-complex and regular, consider **adenosine** 6 mg rapid IV push with flush; via access closest to heart. May give 2nd dose of 12 mg IV

END

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138 | Ventricular Fibrillation Ventricular Tachycardia–Pulseless

SIGNS

V-TACH:



V-FIB:



CPR:

1. **100-120** compressions/minute; ≥ 2 in deep
   Allow complete chest recoil.
2. **Minimize breaks in CPR.**
3. Rotate compressors q2min.

Assess CPR quality, improve IF:
- $ETCO_2$ < 10 mm Hg
- Arterial line Diastolic < 20 mm Hg

1. CALL FOR HELP.
2. CALL FOR CODE CART.
3. INFORM TEAM.

TREATMENT

1. **DEFIBRILLATE**: 120-200 J (biphasic, per manufacturer)
2. **RESUME CPR IMMEDIATELY.**
3. **REPEAT SHOCK q2min,** reasonable to increase energy with subsequent shocks, resume CPR.
4. **AFTER 2nd SHOCK EPINEPHRINE:** 1 mg IV push q3-5min

CHECK

1. In OR: Turn **OFF** volatile; increase to **100% O_2,** high flow.
2. Ventilate **10 breaths/minute**; do **not** overventilate.
3. Ensure IV access (or consider intraosseous).

CONSIDER

Consider Antiarrhythmics:

- **If pulseless:** Amiodarone 300 mg IV PUSH or lidocaine 100 mg IV PUSH
- **If HypoMg or torsades + prolonged QT:** Magnesium sulfate 2 g IV
- **If HyperK:** Calcium, insulin and glucose, sodium bicarbonate

Search for Treatable Causes (H's & T's on next page).

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If still VF/VT, keep shocking q2min.

DETAILS

1. **Hypovolemia:** Give rapid bolus of IV fluid. Check hemoglobin/hematocrit. If anemia or massive hemorrhage, give blood. Consider relative hypovolemia: Auto-PEEP (disconnect circuit); high spinal; or shock states (eg, anaphylaxis). **Go To relevant event.**
2. **Hypoxemia:** Increase O_2, to 100% high flow. Confirm connections. Check for bilateral breath sounds. Suction ET tube and reconfirm placement. Consider chest x-ray. **Go To Hypoxemia.**
3. **Tension pneumothorax:** Unilateral breath sounds, possible distended neck veins, and deviated trachea (late signs). Perform emergent needle decompression (second intercostal space at mid-clavicular line) then chest tube placement. Call for chest x-ray, but do NOT delay treatment. **Go To Pneumothorax.**
4. **Thrombosis—Coronary:** Consider transesophageal (TEE) or transthoracic (TTE) echocardiography to evaluate ventricle wall motion abnormalities of the ventricles. Consider emergent coronary revascularization. **Go To Myocardial Ischemia.**
5. **Thrombosis—Pulmonary:** Consider TEE or TTE to evaluate right ventricle. Consider fibrinolytic agents or pulmonary thrombectomy.
6. **Toxins (eg, infusions):** Consider medication error. Confirm no infusions running and volatile anesthetic off. If local anesthetic toxicity **Go To Local Anesthetic Toxicity.**
7. **Tamponade—Cardiac:** Consider placing TEE or TTE to rule out tamponade. Treat with pericardiocentesis.
8. **Hypothermia ↓:** Active warming by forced air blanket, warm IV fluid, raise room temperature. Consider cardiopulmonary bypass.
9. **Hyperthermia ↑:** If Malignant Hyperthermia, call for MH Cart. Give dantrolene immediately: start at 2.5 mg/kg. MH Hotline: (800) 644-9737. **Go To Malignant Hyperthermia.**
10. **Obtain ABG to rule out:**
    - **Hyperkalemia ↑:** Give calcium chloride 1 g IV; D50 1 amp IV (25 g dextrose) + regular insulin 10 units IV. Monitor glucose. Sodium bicarbonate 1 amp IV (50 mEq)
    - **Hypokalemia ↓:** Controlled infusion of potassium and magnesium
    - **Hypoglycemia:** If ABG delay, check fingerstick. Give D50 1 amp IV (25 g Dextrose). Monitor glucose.
    - **H+ Acidosis:** If profound, consider sodium bicarbonate 1 amp IV (50 mEq). May consider increasing ventilation rate (but can decrease CPR effectiveness so monitor)
    - **Hypocalcemia:** Give calcium chloride 1 g IV.

If still VF/VT, keep shocking q2min.

END

139 | Part B: Broad Differential Diagnosis
Hypotension§

1. CALL FOR HELP.
2. CALL FOR CODE CART?
3. INFORM TEAM.

IMMEDIATE

Immediate Actions:

1. Feel for **pulse** and check **monitors**. If no pulse, slow or abnormal rhythm, **Go To appropriate ACLS event.**
2. Inspect **surgical field** for blood loss or manipulation. Consider pausing surgery if nonbleeding cause.
3. Give **IV fluid** bolus. Ensure IV working
4. Give **phenylephrine** or **ephedrine** to temporize.
   - If severe refractory hypotension, consider: **epinephrine** 10-100 g and/or **vasopressin** 1-4 units.
5. **If bleeding**, consider lower normal MAP until surgeon controls source. Consider ordering blood.
6. Turn down or off **anesthetic agent**.
7. Consider **Trendelenburg** or elevation of patient's legs.
8. Increase to **100% O_2**, high flow.
9. Consider **terminating surgical procedure** or getting **surgical help**.
10. Consider **code cart** if severe. Monitor all **vitals** continuously.
11. If **pulseless**: alert team, start CPR, **Go To PEA.**

RULE OUT

First Rule out Rapidly Lethal Causes:

1. Hemorrhage ?occult (**Go to Hemorrhage—MTG**)
2. **Vasodilators** (volatile, IV anesthetics, or drips)
3. **Auto-PEEP** (disconnect circuit)
4. Pneumothorax (**Go to Pneumothorax**)
5. Anaphylaxis (**Go to Anaphylaxis**)
6. **Cardiac event:** Myocardial infarction/ischemia (**Go to Myocardial Ischemia),** Low Ejection Fraction, Systolic Anterior Motion of mitral valve, Hypertrophic Obstructive Cardiomyopathy. TEE to assess
7. **Pneumoperitoneum** or surgical manipulation
8. **IVC Compression** for example, prone, obese, pregnant, or surgical
9. **Expand Ddx** using **Physiologic approach on next page.**

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DDX

Physiological Differential Diagnosis of Hypotension

MAP = **CO** × SVR **CO** = SV × HR
(SV components: preload, contractility, afterload)

1. **Decreased Preload:** For example, Auto-PEEP, hypovolemia including hemorrhage, arrhythmias, IVC compression, embolism (air, blood, fat, AFE), pneumothorax, pericardial tamponade, venodilators
2. **Low SVR:** For example, vasodilation (medications, neuraxial block), shock (anaphylaxis, sepsis, spinal, neurogenic), endocrine abnormalities
3. **Decreased Contractility:** For example, medications, low EF, myocardial ischemia, valvular disease, increased afterload, hypoxemia, local anesthetic toxicity
4. **Low HR**: Including vagal stimulus

SECONDARY

Depending on likely diagnosis, consider:

1. Treat the problem, if diagnosed. **Go To relevant event** if ACLS, Anaphylaxis, Hemorrhage, Hypoxemia, Local Anesthetic Toxicity, Myocardial Ischemia, Pneumothorax, Total Spinal Anesthesia, Transfusion Reaction, Venous Air Embolism. For sepsis: refer to local guidelines (IV fluids, invasive monitoring?, send lactate, blood cultures, appropriate antibiotics).
2. **Transesophageal echo** if unclear cause
3. More **IV access**
4. Place **arterial line**.
5. Steroid for adrenal insufficiency (eg, **hydrocortisone** 100 mg IV)
6. Send labs: ABG, Hgb, electrolytes, calcium, lactate, type and cross.
7. **Foley catheter** if not present. Monitor urine output.

END

140 | Hypoxemia§

1. CALL FOR HELP.
2. CALL FOR CODE CART?
3. INFORM TEAM.

IMMEDIATE

Immediate actions:

1. Increase to **100% O_2**, high flow.
2. Check gas analyzer to **rule out low FiO_2** or **high N_2O**. If concerned, **Go To Oxygen Failure.**
3. Check other **vitals** (cycle NIBP) and PIP. Feel for **pulse**.
4. Check for **$ETCO_2$** (?extubated, disconnected, low BP).
5. **Hand-ventilate**: Check compliance. Rule out leaks, machine factors.
6. Listen for **breath sounds** (bilateral? clear?). Check **position ETT**.
7. **Suction** catheter via ETT (to clear secretions and check obstructions).
8. Consider **Pneumothorax.**
9. Consider **Code Cart** if severe.

DDX

Differential diagnosis: See next page for details.

1. Hypoventilation
2. Low FiO_2
3. V/Q mismatch or shunt
4. Diffusion problem
5. Increased metabolic O_2 demand

SECONDARY

Depending on likely diagnosis, consider:

1. Large **recruitment breaths**, consider PEEP—caution if hypotensive.
2. **Bronchodilators** (eg, albuterol MDI or nebulizer)
3. Additional **neuromuscular blockade** if indicated
4. Increase FRC: head up (if BP ok), desufflate abdomen.
5. **Check placement of ETT**:
   - **Fiberoptic** to confirm tracheal rings, rule out mainstem intubation or ETT obstruction
   - **Ultrasound**: bilateral sliding pleura are reassuring.
6. **ABG** and/or **CXR**
7. Consider **terminating procedure** for refractory hypoxemia.
8. Plan for **postop care**: remain intubated? ICU bed?
9. **Artifacts**: See next page, consider after Ddx.

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DDX

Physiological differential diagnosis of hypoxemia:

1. **Low FiO_2**: If gas analyzer states low FiO_2 while on "100% O_2" likely have O_2 failure or pipeline crossover of gases. **Go To Oxygen Failure,** immediately.
2. **Hypoventilation**: Check for **signs** of low minute ventilation:
   - Low TV or RR
   - High or low $ETCO_2$
   - Poor chest rise
   - Decreased breath sounds
   - Patient bucking ventilator

   Rule out or fix equipment and patient causes:
   - Circuit leak
   - Obstructed or kinked ETT
   - High PIP
   - Residual neuromuscular blockade
   - Patient breathing asynchronously with ventilator

   Postoperative respiratory failure common causes: residual NMB, opioid, anesthetic, laryngospasm (sudden), bronchospasm, pulmonary edema, high spinal, pain
3. **V/Q Mismatch or Shunt**: A-a Gradient common causes:
   - Mainstem intubation (+?Anaphylaxis)
   - Atelectasis
   - Aspiration
   - Bronchospasm
   - Mucus plug
   - Pleural effusion

   CONSIDER rare but critical:
   - **Pneumothorax**
   - **Hypotension—any cause of poor perfusion**
   - **Embolus—air, blood, fat, AFE**
4. **Diffusion abnormality**: Usually chronic lung disease
5. **Methemoglobinemia** (O_2 Sat ~85%), **COHgb** (O_2 Sat often normal). **If suspect, send for co-oximetry.**
6. **Increased metabolic O_2 demand**: MH, thyrotoxicosis, sepsis, hyperthermia, neuroleptic malignant syndrome
7. **Artifacts**: finally, confirm by ABG. For example, poor waveform (probe malposition, cold extremity, light interference, cautery), dyes (methylene blue, indigo carmine, blue nail polish)

END

141 | Part C: Specific Critical Events
Anaphylaxis

SIGNS

Some signs may be absent in an anesthetized patient:

1. Hypoxemia, difficulty breathing, tachypnea
2. Rash/hives
3. Hypotension (may be severe)
4. Tachycardia
5. Bronchospasm/wheezing
6. Increase in peak inspiratory pressure (PIP)
7. Angioedema (potential airway swelling)

1. CALL FOR HELP.
2. CALL FOR CODE CART.
3. INFORM TEAM.
4. CONSIDER PAUSING SURGERY.

1. **If patient becomes pulseless, start CPR, continue epinephrine 1 mg IV boluses and large volume IV fluid.**
2. **Also Go To PEA/Asystole.**

RULE OUT

Consider and rule out other causes:

- Pulmonary embolus
- Myocardial infarction
- Anesthetic overdose
- Pneumothorax
- Hemorrhage
- Aspiration

For anaphylaxis treatment, Go To Next Page ➔

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TREATMENT

1. **Discontinue potential allergens:** muscle relaxants, latex, antibiotics, colloids, protamine, blood, contrast, chlorhexidine
2. **Discontinue volatile anesthetic** if hypotensive. Consider amnestic agent.
3. Increase to **100% O_2**, high flow.
4. **Give IV fluid bolus.** May require many liters!
5. **Give epinephrine IV in escalating doses** every 2 minutes. Start at 10-100 μg IV and increase dose every 2 minutes until clinical improvement is noted. **Start early epinephrine infusion.** May require large doses > 1 mg
6. **IF no improvement:** continue treatment, but consider other causes (**Go To Hypotension, and Hypoxemia**—consider Differential Diagnoses).
7. Consider **vasopressin** bolus IV or **norepinephrine** infusion.
8. Treat **bronchospasm** with **albuterol** and **epinephrine** (if severe).
9. Consider **additional IV access** and **invasive monitors** (arterial line).
10. If signs of **angioedema**, consider **early intubation** to secure airway.
11. After stable consider **H_1 antagonist** (eg, diphenhydramine 25-50 mg IV), H_2 antagonist (eg, ranitidine 50 mg IV), and **corticosteroids** (eg, methylprednisolone 125 mg IV).

POST EVENT

Consider the following interventions when patient stable:

1. Send serum tryptase level (peaks <60 minutes postevent).
2. Send serum histamine (peaks <30 minutes postevent).
3. If the event was severe, consider keeping patient intubated and sedated.
4. Can recur after initial treatment: Consider monitoring patient for 24 hours postrecovery.
5. Refer the patient for postoperative allergy testing

END

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142 Amniotic Fluid Embolism

SIGNS

Consider amniotic fluid embolism if there is the sudden onset of the following in a pregnant or post-partum patient:

1. Respiratory distress, decreased O_2 saturation
2. Cardiovascular collapse: hypotension, tachycardia, arrhythmias, cardiac arrest
3. Coagulopathy ± Disseminated intravascular coagulation (DIC)
4. Seizures
5. Altered mental status
6. Unexplained fetal compromise

1. CALL FOR HELP.
2. CALL FOR CODE CART.
3. INFORM TEAM.

TREATMENT

1. Anticipate possible **cardiopulmonary arrest** and **emergent C-section.**
2. Place patient in left uterine displacement (**LUD**).
3. Increase to **100% O_2**, high flow.
4. Establish large-volume **IV access** (upper body best).
5. Support circulation with **IV fluid, vasopressors, and inotropes.**
6. **Prepare for emergent intubation.**
7. When possible, place arterial line. Consider central venous access or IO line in humerus.
8. Anticipate **massive hemorrhage** and DIC. **Go To Hemorrhage—MTG.**
9. Consider **circulatory support**: IABP/ECMO/CPB.

RULE OUT

Rule out other causes that might present in a similar fashion:

1. Eclampsia
2. Hemorrhage
3. Air embolism
4. Aspiration
5. Anaphylaxis
6. Pulmonary embolism
7. Anesthetic overdose
8. Sepsis
9. Cardiomyopathy/cardiac valvular abnormality/MI
10. Local anesthetic toxicity
11. Total spinal

END

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143 | Bronchospasm (Intubated Patient)

SIGNS

1. Increased peak airway pressures
2. Wheezing on lung exam
3. Increased expiratory time
4. Increased $ETCO_2$ with upsloping $ETCO_2$ waveform
5. Decreased tidal volumes if pressure control



1. CALL FOR HELP.
2. CALL FOR CODE CART?
3. INFORM TEAM.

Bronchospastic patients who develop sudden hypotension may be airtrapping—disconnect patient from circuit to allow for complete exhalation.

TREATMENT

1. Increase to **100% O_2**, high flow.
2. Change **I:E ratio** to allow for adequate exhalation.
3. **Deepen** anesthetic (sevoflurane or propofol).
4. **Rule out problems with ETT** via auscultation and suction catheter (mainstem intubation, kinked ETT, mucus plug).
5. Give inhaled agents: Beta 2 agonist (**albuterol,** multiple puffs required) ± anticholinergic (**Ipratropium**).
6. If severe consider **epinephrine** (start with 10 µg IV and escalate, monitor for tachycardia and hypertension).
7. Consider **ketamine**: 0.2-1.0 mg/kg IV.
8. Consider **hydrocortisone** 100 mg IV.
9. Consider **nebulized racemic epinephrine**.
10. Rule out anaphylaxis (hypotension/tachycardia/rash). **Go To Anaphylaxis.**
11. Consider ABG.

END

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144 | Delayed Emergence

CHECK

1. Confirm that all anesthetic agents (inhalation/IV) are **OFF**.
2. Check for residual muscular paralysis (if patient is asleep, use twitch monitor), and reverse accordingly.

CONSIDER

Consider:

1. Opioid reversal: Start with **naloxone** 40 g IV; repeat every 2 minutes, increasing up to 400 g.
2. Benzodiazepine reversal: Start with **flumazenil** 0.2 mg IV every 1 minute; max dose = 1 mg.
3. Scopolamine reversal (eg, Patch): **Physostigmine** 1 mg IV (Potential cholinergic crisis, including severe bradycardia, so have atropine ready).

CHECK

1. Monitors: Check **Hypoxemia**? **Hypercarbia**? **Hypothermia**?
2. Complete **Neuro exam**, as able, for focal neurologic deficits (if intubated look for: pupils, asymmetric movement, gagging, etc.)

   If abnormal exam or **suspect stroke**, obtain **stat Head CT scan** and consult neurology/neurosurgery.
3. **Hypoglycemia**: Check glucose (glucometer).
4. Labs: **ABG plus electrolytes**. Rule out CO_2 narcosis from hypercarbia, hyponatremia, or hypernatremia.
5. Check for **medication swap** or dosing error.

TREATMENT

1. Correct any abnormalities in oxygenation, ventilation, laboratory values, or temperature.
2. If residual mental status abnormalities, monitor the patient in the ICU with **neurological follow up**, including serial exams. Repeat head CT or MRI as needed.

END

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145 | Difficult Airway Unanticipated§

If unable to see vocal cords or pass ET tube during first direct laryngoscopy (DL):

1. Consider external laryngeal manipulation, BURP (Backwards Upwards Rightwards Pressure).
2. Consider placing Bougie introducer.
3. **Limit total number of DL attempts to 2.**
4. Recommend video assisted laryngoscopy.
5. Before repeating DL, consider mask ventilation with oral/nasal airways.
6. Consider optimizing patient position and/or blade selection.
7. **If successful, confirm placement with $ETCO_2$ and bilateral breath sounds.**

Can NOT Intubate

1. Attempt face mask ventilation—consider oral airway.
2. **Call for Difficult Airway cart.**

Can NOT Ventilate

CALL FOR HELP!
Place oral, nasal airway switch to two-handed mask ventilation.

Can NOT Ventilate

1. **Place LMA if feasible.**
2. Consider any SGA, Intubating LMA, Combitube, or laryngeal tube.

Can NOT Ventilate

Emergency Airway Ventilation

1. Call for surgical help.
2. Perform cricothyrotomy.
3. Confirm successful placement with $ETCO_2$ and bilateral breath sounds.

Successful Ventilation

If at any point inadequate ventilation by mask or LMA,

Go To Red Box.

If ventilation remains adequate, CONSIDER:

1. Awakening patient
2. Complete case with LMA or face mask
3. Video assisted laryngoscopy
4. Asleep fiberoptic bronchoscopy
5. LMA as conduit for intubation or intubating LMA
6. Retrograde wire intubation

Successful Ventilation

For more details, see latest ASA Practice Guidelines for the Management of Difficult Airway

END

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146 | Fire–Airway§

FOR NONAIRWAY FIRE: Go To Fire—Patient

SIGNS

SUSPECT FIRE if:
Sudden pop, spark, flame, smoke, heat, or odor

1. CALL FOR HELP.
2. INFORM TEAM.

IMMEDIATE

SURGEON:

1. **REMOVE ENDOTRACHEAL TUBE.**
2. **Remove** airway foreign bodies for example, ETT pieces.
3. **Pour** saline or water into patient's airway.
4. **Examine** entire airway (including bronchoscopy) to assess injury and remove residual debris.

ANESTHESIOLOGIST:

1. **STOP ALL AIRWAY GAS FLOW BY DISCONNECTING THE BREATHING CIRCUIT FROM THE ANESTHESIA MACHINE.**
2. When sure fire is extinguished: **Reestablish** ventilation; avoid supplemental O_2 if possible.
3. Consider prompt **reintubation** prior to swelling and coordinated with surgeon's bronchoscopy.
4. **Inspect** ETT pieces to verify none left in airway.
5. **Save** all materials for later investigation.

For prevention of airway fires, see next page.

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FOR NONAIRWAY FIRE: Go To Fire—Patient

PREVENTION

If high-risk procedures, including those listed below:

- Discuss fire prevention and management with team during time-out.
- Avoid $FiO_2 > 0.3$ and avoid N_2O.

For **laser** surgery of vocal cord or larynx:

- Use laser resistant ETT (single or double cuff).
- Assure ETT cuff sufficiently deep below vocal cords.
- Fill proximal ETT cuff with methylene blue-tinted saline.
- Ensure Laser in STANDBY when not in active use.
- Surgeon protects ETT cuff with WET gauze.
- Surgeon confirms $FiO_2 < 0.3$ and no N_2O prior to laser use.

For **nonlaser** surgery in oropharynx:

- Regular PVC ETT may be used.
- Consider packing wet gauze around ETT to minimize O_2 leakage.
- Consider continuous suctioning of the operating field inside oropharynx.

END

147 | Fire–Patient§

FOR AIRWAY FIRE: Go To Fire—Airway

SIGNS

SUSPECT FIRE if:
Sudden pop, spark, flame, smoke, heat, or odor

1. INFORM TEAM.
2. CALL FOR HELP.
3. CALL FOR FIRE EXTINGUISHER.

IMMEDIATE

1. **Stop flow** of all airway gases to patient.
2. **Remove** burning or flammable materials from patient immediately for other team member to extinguish.
3. **Extinguish patient fire:**
   - If electrical equipment burning, use only **CO_2 fire extinguisher** (safe in wounds).
   - If nonelectrical, attempt to extinguish with saline and soaked gauze.
4. **Care for the patient:** Ventilate with room air; control bleeding; assess injuries and vital signs.
5. Consider **evacuating** patient and OR if smoke or continued fire, per local protocol.
6. Close OR doors.
7. **Turn OFF** external gas supply to OR.
8. **Alert fire department.**

For prevention of airway fires, see next page.

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FOR AIRWAY FIRE: Go To Fire—Airway

PREVENTION

- Team Communication at time-out if high-risk procedure
- Highest risk in MAC head and neck procedures
  - Use nasal cannula instead of face mask, if able.
  - Configure drapes to avoid O_2 buildup, consider active scavenging if required.
  - Use minimum O_2 concentration for adequate SpO_2.
- If high O_2 concentration required, use an LMA or ETT.
- Allow complete drying of alcohol skin prep solutions.
- Consider coating patient's head hair and facial hair with water-soluble surgical lubricating jelly.

Remember: Fuel Source + Oxidizer + Spark = FIRE

END

148 | Hemorrhage Massive Transfusion Guidelines

1. CALL FOR HELP.
2. CALL FOR CODE CART?
3. INFORM TEAM.

IMMEDIATE

1. Follow local protocol to order Massive Transfusion Guideline (**MTG**) or equivalent.
2. Increase to **100% O_2**, high flow.
3. **Treat hypotension with IV fluid bolus.**
4. Consider **Trendelenburg** or **elevation of patient's legs**.
5. Use **vasopressor boluses** (ephedrine, phenylephrine, epinephrine) as a temporizing measure. Consider accepting low normal blood pressure until bleeding is controlled.
6. Call for **rapid infuser**.
7. Establish **additional IV access** as needed. Consider intraosseous if needed.
8. Ask surgeon: "Should we page a **vascular surgeon** or other additional help for you?"
9. **Send Type and Cross sample.** TS will provide emergency release Type O PRBC until crossmatched blood is available.
10. **Maintain normothermia.** Use fluid warming devices for IV and blood products. Use forced air warmers.
11. Place **arterial line** as indicated.
12. Follow patient's acid/base status by **ABG as indicator of adequate resuscitation. Monitor for hypocalcemia.**
13. Place **Foley catheter** when able.
14. Call for **cell-saver** (if noncontaminated, nonmalignant case).

Replace products EARLY! until current lab data available:

- If >1 blood volume of loss expected: give **1 unit FFP for every 1 unit PRBC. Give 1 apheresis unit of platelets** (= old "6-pack") for every 6 units PRBC.
- When labs back: Replace factors, platelets, fibrinogen as indicated on next page, but do not wait if blood loss is too rapid.

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COMPONENTS

PRBC: Give for Hgb < 7-10 (CAD? Rate of blood loss?) Each unit PRBC raises Hgb ~1 g/dL.

PLATELETS: Give for <50000-100000 per μL with signs of ongoing bleeding. Each apheresis unit raises platelets ~50000 per μL.

FRESH FROZEN PLASMA: Give for INR (PT) or PTT >1.5 times normal. Give 10-15 cc FFP per kg body weight, then recheck labs and continue with 1:1 FFP:PRBC ratio.

CRYOPRECIPITATE: Give for fibrinogen <80-100 mg/dL. Each 10 units of cryoprecipitate raises fibrinogen ~50 mg/dL.

VOLUMES

$$\text{Est. Blood Loss} = \text{EBV} \times \frac{HCT_{starting} - HCT_{measured}}{HCT_{starting}}$$

Estimated Blood Volume (EBV) ~65-70 mL/kg body weight (~4.5 L for 70 kg)

END

149 | Local Anesthetic Toxicity

SIGNS

1. Symptoms:
   Tinnitus, metallic taste, or circumoral numbness
2. Altered mental status
3. Seizures
4. Hypotension
5. Bradycardia
6. Ventricular arrhythmias
7. Cardiovascular collapse

1. CALL FOR HELP.
2. Alert possible CPB.
3. CALL FOR CODE CART.
4. INFORM TEAM.

TREATMENT

1. **Call for Intralipid kit.**
2. **If pulseless, start CPR** and give **<1 µg/kg epinephrine**.
3. **Avoid vasopressin**.
4. **Stop local anesthetic** injection and/or infusion.
5. Establish airway—ensure adequate **ventilation and oxygenation**. Consider endotracheal intubation.
6. Treat seizure activity with **benzodiazepines**.
7. If signs persist or patient unstable: **Rapidly give 1.5 mL/kg bolus of 20% Intralipid IV (70 kg adult gets 105 mL fast)** then start infusion at 0.25 mL/kg/min. May repeat loading dose (max 3 total doses). May increase infusion rate (max 0.5 mL/kg/min)
8. Monitor for hemodynamic instability—**treat hypotension** (see next page for details).

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SECONDARY

1. **Variable arrhythmias: Go to** appropriate ACLS event with the following **modifications per ASRA** Practice Advisory:
   - **CONSIDER** reducing epinephrine doses <1 µg/kg IV.
   - **AVOID**: Vasopressin, calcium channel blockers, beta-blockers, and local anesthetics.
2. If refractory to treatment, alert personnel for **potential cardiopulmonary bypass.**
3. May require **prolonged resuscitation**
4. Monitor the patient post event in **ICU.**

For latest recommendations, see ASRA Web site (http://www.asra.com).

CPR:

1. **100-120** compressions/minute; **≥ 2 in** deep. Allow complete chest recoil.
2. **Minimize breaks in CPR.**
3. Rotate compressors q2min.

Assess CPR quality, improve IF:

- $ETCO_2$ < 10 mm Hg
- Arterial line Diastolic < 20 mm Hg

END

150 | Malignant Hyperthermia§

SIGNS

EARLY:
1. Increased $ETCO_2$
2. Tachycardia
3. Tachypnea
4. Mixed Acidosis (ABG)
5. Masseter spasm/trismus
6. Sudden cardiac arrest in young person due to hyperkalemia

May be LATER
1. Hyperthermia
2. Muscle rigidity
3. Myoglobinuria
4. Arrhythmias
5. Cardiac arrest

1. CALL FOR HELP.
2. CALL FOR MH CART.
3. INFORM TEAM.
4. START PREPARING DANTROLENE or RYANODEX!

DDX

- Light anesthesia
- Hypoventilation
- Insufflation of CO_2
- Overheating (external)
- Hypoxemia
- Thyroid Storm
- Pheochromocytoma
- Neuroleptic malignant syndrome (NMS)
- Serotonin syndrome

TREATMENT

1. **Discontinue** anesthetic triggers (volatiles and succinylcholine). Do **NOT** change machine or circuit.
2. Increase to **100% O_2, high flow** 10 L/min.
3. **Halt procedure** if possible. If emergent, continue with nontriggering anesthetic.
4. **Increase minute ventilation** (but avoid air trapping).
5. **Assign several people to prepare 2.5 mg/kg IV dantrolene or Ryanodex bolus:**
   - **Dantrolene**: Dilute **each 20 mg dantrolene vial in 60 mL** preservative-free **sterile water** (for **70 kg person give 175 mg so prepare 9 vials** of 20 mg dantrolene each as above).
   - **Ryanodex** (new formulation of dantrolene): Dilute **250 mg Ryanodex vial in 5 mL** preservative-free **sterile water** (for **70 kg person give 175 mg**).
6. **Rapidly give dantrolene** or **Ryanodex**. Continue giving until patient stable (may need >10 mg/kg, call MHAUS 800-644-9737 for advice).
7. For metabolic acidosis, give **sodium bicarbonate** 1-2 mEq/kg.

MH Treatment continued on next page.

GO TO NEXT PAGE ➔

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TREATMENT

8. **Hyperkalemia** – or suspect from EKG, treat with:
   - **Calcium chloride** 10 mg/kg IV; max dose 2000 mg or **Calcium gluconate** 30 mg/kg IV, max dose 3000 mg
   - **D50** 1 amp IV (25 g or 50 mL Dextrose) + **regular insulin** 10 units IV (monitor glucose).
   - **Sodium bicarbonate** 1-2 mEq/kg, max dose 50 mEq.
9. Arrhythmias are usually secondary to hyperkalemia. Treat as needed except **avoid calcium channel blockers. Go to ACLS events** as relevant and return.
10. Actively **cool patient** with ice packs, lavage if open abdomen. Stop cooling at 38°C.
11. Send **labs** for ABG, Potassium, CK, urine myoglobin, coagulation studies, lactate.
12. Place Foley catheter. **Monitor UO. Goal 1-2 mL/kg/h** Can give IV fluid and diuretics
13. **Consider alkalinizing urine** if CK or urine myoglobin elevated (sodium bicarbonate 1 mEq/kg/h).
14. Arrange **ICU** bed. Mechanical ventilation usually required
15. **Continue dantrolene** or **Ryanodex**: 1 mg/kg every 4-6 hours or 0.25 mg/kg/h infusion for at least 24 hours (**25% of MH events relapse**). Observe patient in ICU for at least 24 hours.
16. Call **MH hotline** (below) for any suspected case with any questions.

Contact the Malignant Hyperthermia Association of the United States (MHAUS hotline) at any time for consultation if MH is suspected:

1-800-MH-HYPER (1-800-644-9737)

or see suggestions online at http://www.mhaus.org

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151 | Myocardial Ischemia

SIGNS

Suspect myocardial ischemia if:

1. Depression or elevation of ST segment
2. **Arrhythmias**: conduction abnormalities, **unexplained tachycardia, bradycardia, or hypotension**
3. Regional wall motion abnormalities or new/worse mitral regurgitation on TEE/TTE
4. In **awake patient:** chest pain, etc

1. CALL FOR HELP.
2. CALL FOR CODE CART.
3. INFORM TEAM.

TREATMENT

1. **If hypoxemic**, increase to 100% O_2, high flow.
2. Verify ischemia (expanded monitor view vs 12-lead EKG).
3. Treat **hypotension** or hypertension.
4. Be prepared for **arrhythmias** and have **Code Cart** at bedside. Consider applying pads.
5. **Beta-blocker** to slow heart rate. Hold for bradycardia or hypotension.
6. Discuss with surgeon: **aspirin** 160-325 mg PR, PO, NG.
7. Consider **STEMI team** or **consult Cardiology**—stat. Discuss among cardiology, surgery, anesthesia:
   - Heparin ± clopidogrel.
8. Treat pain with **narcotics** (fentanyl or morphine).
9. Consider **nitroglycerin** infusion (hold until hypotension treated).
10. Place **arterial line** and send **Labs**: ABG, CBC, troponin.
11. **If anemic,** treat with packed red blood cells.
12. Consider TTE or TEE **Echocardiography** for monitoring volume status and regional wall motion abnormalities.
13. Consider central venous access.
14. If hemodynamically unstable, consider **Intra-Aortic Balloon Pump.**

END

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152 Oxygen Failure: O_2 Cross Over/ Pipeline Failure§

SIGNS

Hear O_2 failure alarm **OR** Inappropriately low FiO_2 value on gas analyzer

IMMEDIATE

Immediate actions:

1. **Disconnect the patient from the machine and ventilate with an Ambu bag on Room Air.** Do **not** connect the patient to auxiliary flowmeter on machine—comes from SAME central source!
2. **Open O_2 tank** on back of anesthesia machine (check not empty) and disconnect pipeline oxygen to force flow from tank into circuit.

   Alternative: Obtain full E cylinder of O_2 with a regulator. Ventilate with Ambu bag or Jackson-Rees circuit attached to new O_2 tank.
3. Connect gas sampling adaptor to allow monitoring of respiratory gases:

   Is the patient receiving 100% oxygen?
4. Maintain anesthesia (if necessary) with IV drugs.

1. CALL FOR HELP.
2. CALL FOR CODE CART?
3. INFORM TEAM.

SECONDARY

1. Reduce O_2 flow rates to minimum needed to conserve oxygen.
2. Obtain extra backup sources of O_2.
3. When patient more stable, contact Bioengineers to alert them to problem and enlist help with machine diagnosis while you focus on patient.
4. Inform OR leadership, ICU, hospital of potential large-scale O_2 problem.
5. Discuss with surgeon implications of O_2 failure for this patient's management and OR schedule.

END

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153 | Pneumothorax

SIGNS

1. Increased peak inspiratory pressures
2. Tachycardia
3. Hypotension
4. Hypoxemia
5. Decreased or asymmetric breath sounds
6. Hyperresonance of chest to percussion
7. Tracheal deviation (late sign)
8. Increased JVD/CVP
9. Have **high index of suspicion** for pneumothorax in **trauma patients** and **COPD patients.**

1. CALL FOR HELP.
2. CALL FOR CODE CART?
3. INFORM TEAM.

TREATMENT

1. Increase to **100% O_2**, high flow.
2. **Rule out** mainstem intubation.
3. Consider **ultrasound** or stat CXR.
4. **Do not delay treatment if hemodynamically unstable.**
5. **Place 14 or 16 gauge needle mid clavicular line 2nd intercostal space on affected side;** may hear a whoosh of air if under tension.
6. Immediately follow up needle decompression with thoracostomy (chest tube).

END

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154 | Power Failure

IMMEDIATE LIFESAVING ACTIONS:

1. **Get additional light sources:**
   - Laryngoscopes, cell phones, flashlights, etc
2. **Open doors and shades** to let in ambient light.
3. **Confirm ventilator is working** and **if not, ventilate patient with Ambu bag and switch to TIVA.**
4. If monitors fail, **check pulse and manual blood pressure.**
5. **Request transport monitor** or defibrillator monitor.
6. **Confirm adequate backup O_2 supply:**
   - Power failure may affect oxygen supply or alarms.
7. **Check extent of power failure:**
   - Call bio-med or engineering.
   - Is the problem in one OR, all ORs, or hospital-wide?
   - If only in your OR, check if circuit breaker has been tripped.

END

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155 | Total Spinal Anesthesia

SIGNS

AFTER NEURAXIAL ANESTHESIA BLOCK:

1. Unexpected rapid rise in sensory blockade
2. Numbness or weakness in upper extremities (check hand grip)
3. Dyspnea
4. Bradycardia
5. Hypotension (or nausea/vomiting)
6. Loss of consciousness
7. Apnea
8. Cardiac arrest

1. CALL FOR HELP.
2. CALL FOR CODE CART.
3. INFORM TEAM.

TREATMENT

1. **If Cardiac Arrest:** Start CPR, immediate epinephrine, **Go To appropriate ACLS event.**
2. Support **ventilation** and intubate if necessary.
3. Treat significant **bradycardia or hypotension** with immediate **epinephrine** (start 10-100 g, increase as needed). If mild bradycardia, consider **atropine** (0.5-1 mg), but progress quickly to epinephrine if needed.
4. Give **IV fluid** bolus.
5. **If parturient:** Left uterine displacement, call OB and neonatology, prepare for possible emergent C-section, monitor fetal heart rate.

END

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156 Transfusion Reactions

SIGNS

Hemolytic Reaction	Febrile	Anaphylactic
1. Fever 2. Back/flank pain 3. Tachycardia 4. Tachypnea 5. Hypotension 6. Dark urine 7. Oozing—DIC?	1. Fever 2. Chills/rigors 3. Headache 4. Vomiting	1. Hypotension 2. Urticaria/hives 3. Wheezing 4. Tachycardia

1. CALL FOR HELP.
2. CALL FOR CODE CART?
3. INFORM TEAM.

TREATMENT

1. **Stop the transfusion.**
2. Support blood pressure with IV fluids and vasoactive medications if needed.
3. Retain blood product bag and notify Transfusion Service. Additional patient samples will need to be drawn.
4. Consult Transfusion Medicine MD if advice needed.
5. Consider TRALI or volume overload if evidence of lung injury (hypoxemia, pulmonary edema). May require postoperative ventilation

Hemolytic Reaction	Febrile	Anaphylactic
• Maintain urine output —IV fluids, diuretics, renal dose dopamine. • Monitor for signs of DIC.	• Treat with antipyretics • Rule out hemolysis. • Rule out bacterial contamination.	• Epinephrine infusion • Give antihistamines. • **Go To Anaphylaxis.**

END

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157 | Venous Air Embolus

SIGNS

OBSERVE SUDDEN:

1. Air on TEE or change in Doppler tone (if monitoring)
2. Decrease in $ETCO_2$
3. Decrease in BP
4. Decrease in SpO_2
5. Rise in CVP
6. Onset of dyspnea and respiratory distress or cough in awake patient

1. CALL FOR HELP.
2. CALL FOR CODE CART?
3. INFORM TEAM.

TREATMENT

1. Increase to **100% O_2**, high flow.
2. **Flood** surgical field with saline.
3. Place **surgical site below heart** (if able).
4. **Aspirate air** from the central line if present.
5. Give rapid fluid bolus to increase CVP.
6. Turn down or **off volatile anesthetic**.
7. Give **epinephrine** (start 10-100 μg) to maintain cardiac output.
8. Start **CPR** if BP catastrophically low.
9. Consider TTE or TEE **Echocardiography** to assess air and RV function.
10. Consider left lateral decubitus.
11. If severe, terminate procedure if able.

END

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158 Part D: Crisis Resource Management

Crisis Resource Management



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Call for Help Early
• Call for help early enough to make a difference.
• Err on the side of getting more help.
• Mobilize early personnel with special skills if they may be needed.
Designate Leadership
• Establish clear leadership.
• Inform team members who is in charge.
• "Followers" should be active in asking who is leading.
Anticipate and Plan
• Plan and prepare for high work-load periods during low work-load periods.
• Know where you are likely headed during the crisis and make backup plans early.
Know the Environment
• Maintain situational awareness.
• Know how things work and where things are.
• Be aware of strengths and vulnerabilities of environment.
Establish Role Clarity
• Determine who will do what.
• Assign areas of responsibility appropriate to knowledge, skills, and training.
• Active followers may offer specific roles.
Use All Available Information
• Monitor multiple streams of data and information.
• Check and cross-check information.
Distribute the Workload
• Assign specific tasks to team members according to their abilities.
• Revise the distribution if there is task overload or failure.
Allocate Attention Wisely
• Eliminate or reduce distractions.
• Monitor for task saturation and data overload.
• Avoid getting fixated.
• Recruit others to help w/ monitoring.
Mobilize Resources
• Activate all helpful resources including equipment and additional personnel.
Communicate Effectively
• Command and request clearly.
• Seek confirmation of request (close the loop).
• Avoid "thin air" statements.
• Foster input and atmosphere of open information exchange among all personnel.
Use Cognitive Aids
• Be familiar with content, format, and location.
• Support the effective use of cognitive aids.

COGNITIVE AID FOR INDUCTION OF GENERAL ANESTHESIA

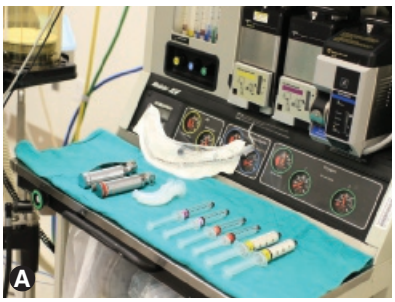
159 Standard Induction of General Anesthesia

Larry F. Chu • T. Kyle Harrison

Ⓜ (**M**achine checked, high-flow O_2)
Ⓢ (**S**uction on, Yankauer catheter at patient's head)
Ⓜ (**M**onitors on, NIBP every minute, baseline measurement)
Ⓐ (**A**irway equipment ready and available)
Ⓘ (**I**V access and free flow IV with adequate fluid in bag)
Ⓓ (**D**rugs for induction of anesthesia ready and available)
Ⓢ (**S**pecial—extra equipment for case)

Ⓐ Recheck anesthesia machine and OR setup (see MSMAIDS).

Ⓑ Place ASA standard monitors on patient.





A. Check the anesthesia machine; verify high-flow O_2, suction, airway equipment, drugs according to the MSMAIDS mnemonic above. **B.** Place ASA standard monitors (EKG, NIBP cuff, pulse oximeter). Avoid placing pulse oximeter probe on the index finger as patients can scratch their eyes inadvertently.

Ⓒ Reassure patient and explain induction. Preoxygenate.

Ⓓ Confirm vital signs every minute. **Titrate** induction agent.





C. Reassure patient and explain induction. Preoxygenate with 100% O_2 3 minutes or 8 deep breaths over 60 seconds. **D.** Obtain baseline vitals, and check every 1 minute. Titrate IV induction agent to effect.

Ⓔ Confirm induction of anesthesia by testing eyelash reflex. Tape eyelids with eye tape.

Ⓕ Confirm ability to mask ventilate patient. Consider insertion of oral or nasal airways to improve mask ventilation.





E. Test eyelash reflex to confirm patient is unconscious. Tape eyelids shut to protect eyes from corneal abrasion during airway manipulation and surgery. **F.** Confirm ability to mask ventilate patient.



If mask ventilation is not possible, call for help!
Implement ASA Difficult Airway Algorithm.





G. Administer neuromuscular blocking agent through the IV. **H.** Attach nerve simulator leads to ulnar aspect of the patient's arm and monitor twitches continuously. Mask ventilate patient while awaiting full neuromuscular blockade in order to produce ideal intubation conditions.

Ⓖ Administer neuromuscular blocking agent.

Ⓗ Mask ventilate patient and monitor neuromuscular function. Proceed with intubation when neuromuscular blockade is adequate.

PATIENT CONSIDERATIONS DURING INDUCTION OF GA

1. **Make patient comfortable** (warm room temperature, apply warm blankets when moved to OR table, introduce OR staff).
2. **Reassure patient** during this anxious period of time. Maintain patient modesty by draping body while positioning and applying monitors.

160 Mask Ventilation

Larry F. Chu • T. Kyle Harrison

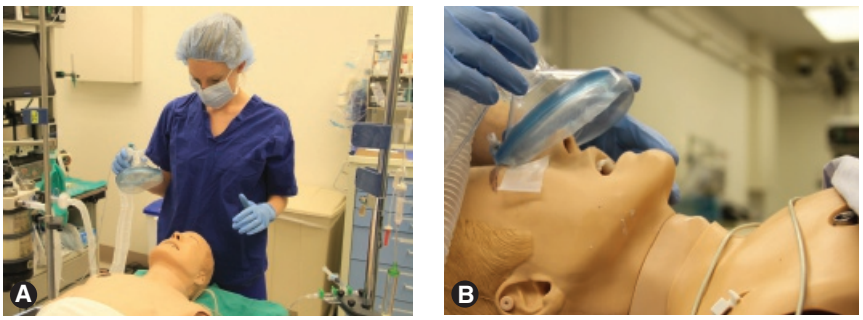
Equipment: Anesthesia machine, airway supplies including oral and/or nasal airways, face mask, ventilation system.

Mask ventilation is a vital skill for anesthesiologists.

Mask ventilation allows the anesthesiologist time to safely manage and instrument the airway. Confirm MSMAIDS mnemonic (see Induction of Anesthesia Cognitive Aid, Chapter 159).

Ⓐ Preoxygenate patient and induce general anesthesia.

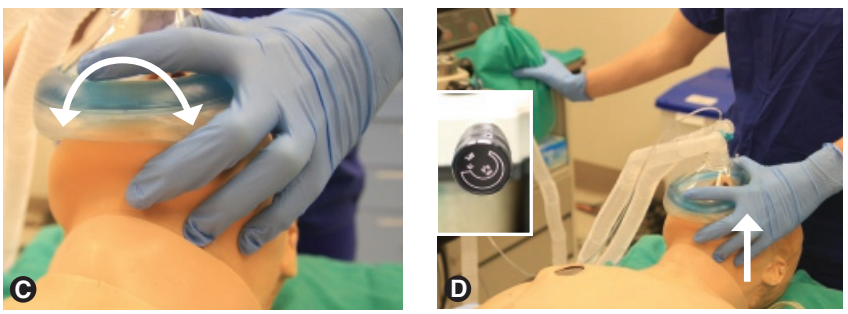
Ⓑ Place nasal part of mask on the nose and lever mask down on the face.



A. Preoxygenation and induction of anesthesia should proceed as previously described (see Induction of Anesthesia Cognitive Aid). **B.** The nasal aspect of mask is placed on the bridge of the nose, and the body of the mask is levered down onto the face, covering the nose and mouth.

Ⓒ The thumb and index finger hold the mask.

Ⓓ The remaining fingers pull the mandible into mask to open airway.



C. The nondominant thumb and index finger hold the mask and can rock gently side to side to achieve the best mask seal. **D.** The remaining fingers pull upward on the mandible to open the airway and ease bag ventilation. The machine pop-off valve (**inset**) can be rotated to adjust airway pressures if needed.

Ⓔ An oral airway can be inserted to facilitate mask ventilation. Insert the curved tip toward the patient's face.

Ⓕ Rotate the airway 180 degrees as it is inserted into the oropharynx.



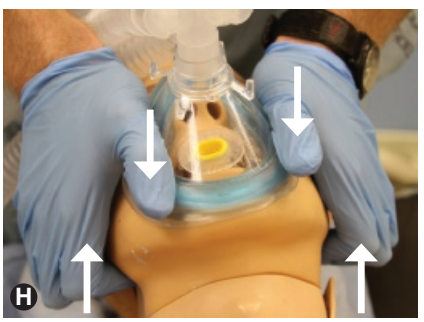


E. An oral airway can be inserted to open airway structures and ease mask ventilation. The airway is inserted with the curved tip pointing toward the patient's face. **F.** The airway is rotated 180 degrees as it is fully inserted into the patient's oropharynx.

Ⓖ A single-handed mask hold with oral airway is a common technique for mask ventilation of patients in the operating room.

Ⓗ If the single-handed method is inadequate, institute the two-handed mask technique. Place both hands on the mask, thumbs opposite the mask connector. Create firm mask seal with jaw-thrust and chin-lift maneuvers.





G. Single-handed mask hold with oral airway. **H.** A two-handed mask technique can be employed if difficult mask ventilation is encountered using the single-handed mask hold technique. The metacarpophalangeal (MCP) joints of both thumbs are placed opposite the mask connector. This allows four fingers to create a firm mask seal, while maintaining jaw-thrust and chin-lift maneuvers. This position can be maintained comfortably for prolonged ventilation.



If mask ventilation is not possible, call for help!
Implement ASA Difficult Airway Algorithm.

161 Laryngeal Mask Airway Insertion

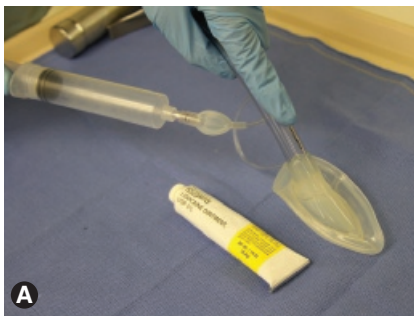
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Equipment: Laryngeal mask airway, 30-cc air syringe, lubricant, adhesive tape, airway management equipment including face mask and oral airways.

Confirm MSMAIDS mnemonic (see Induction of Anesthesia Cognitive Aid, Chapter 159).

Ⓐ Assemble equipment, and deflate LMA cuff with air syringe.

Ⓑ Lubricate LMA cuff.

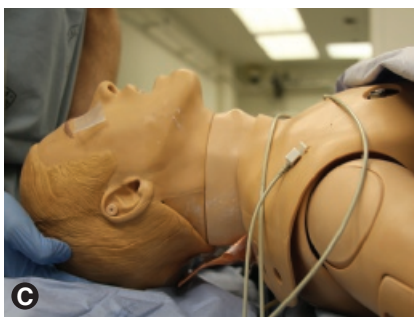




A. Assemble the components necessary for LMA insertion. Deflate the LMA cuff according to the manufacturer's guidelines so that the leading edge is smooth. **B.** Lubricate the LMA with lubricant, such as lidocaine ointment.

Ⓒ Explain the procedure and reassure patient. Induce general anesthesia (see Induction of Anesthesia Cognitive Aid, Chapter 159). Place patient in "sniffing" position.

Ⓓ Open mouth with "scissor" technique using nondominant hand.





C. Explain procedure, and induce general anesthesia. Neuromuscular blockade is usually unnecessary for LMA insertion. Place patient in proper "sniffing" position. **D.** Tilt the patient's head backward (caution in patients with uncleared C-spine injuries) or open mouth with "scissor" technique.

Ⓔ Grasp LMA in dominant hand. Flat side of the cuff should face patient's head. Place first finger in space between the tube and cuff.

Ⓕ The leading edge of the cuff should be flat and pressed upward against the hard palate during insertion. Guide LMA above tongue and down oropharynx in a smooth continuous motion.





E. Grasp the LMA in your dominant hand with the curved tube and flat side of cuff facing the patient. Place your index finger in the space between the tube and the LMA cuff. **F.** Insert the LMA into the mouth and press the cuff upward against the hard palate. Guide the cuff above the tongue and down the oropharynx in a smooth motion. Inadequate anesthesia may cause difficulty with insertion of the LMA.

Ⓖ Stop when resistance is met (7-10 cm of LMA should protrude).

Ⓗ Inflate cuff with ~30 cc air. The LMA may slide out of the mouth 1-2 cm during inflation. This is normal.

Confirm proper placement of the LMA with bilateral auscultation of breath sounds and capnography. The neck should be auscultated, and air leakage should not be heard below 20 cm H_2O, indicating proper positioning of the device. Secure the LMA with adhesive tape.





G. Stop advancing LMA when resistance is met. Inflate the cuff with about 30 cc air. The LMA may slide out of the mouth 1-2 cm, which is normal. **H.** Confirm proper placement by auscultation and capnography. A leak pressure should be assessed by insufflating 20 cm H_2O of air pressure through LMA. No air leakage at the patient's neck should be observed.

162 Endotracheal Intubation

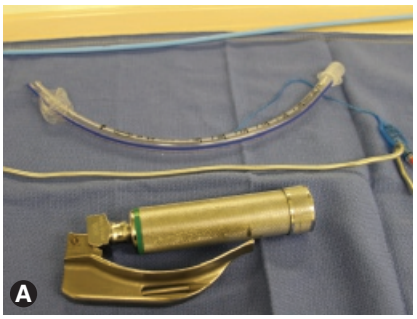
Larry F. Chu • T. Kyle Harrison

Equipment: Endotracheal tube (ETT), stylet, air syringe, stethoscope, bag mask ventilation device, adhesive tape.

Confirm MSMAIDS mnemonic (see Induction of Anesthesia Cognitive Aid, Chapter 159).

Ⓐ Assemble equipment for airway manipulation and intubation.

Ⓑ Explain procedure and reassure patient. Induce general anesthesia (see Induction of Anesthesia Cognitive Aid, Chapter 159). Place patient in "sniffing" position.





A. Assemble the airway equipment needed for endotracheal intubation, including an ETT, stylet, laryngoscope, and Macintosh 3 blade. **B.** Place the patient in the "sniffing" position after induction of anesthesia and establishing optimal neuromuscular blockade and intubating conditions.

Ⓒ Use right thumb and third finger to "scissor" open the mouth widely.

Ⓓ Insert laryngoscope sweeping tongue aside from right to left.



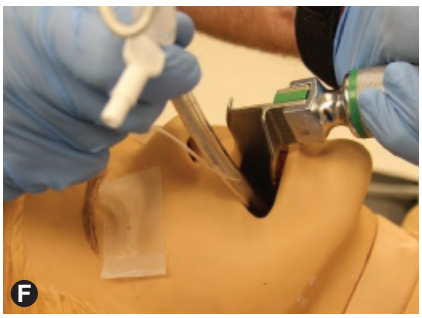


C. Use the right hand to scissor open the mouth with the thumb and third finger. **D.** Insert the Macintosh laryngoscope blade into the mouth, sweeping the tongue to the side (right to left).

Ⓔ Advance laryngoscope blade into the airway. Lift in an upward and forward motion toward the corner of the room.

Ⓕ Once VCs are visualized, advance ETT through glottic opening. Stop advancing when ETT cuff is past the VCs.





E. Advance the laryngoscope blade into the airway and gently lift in an upward and forward motion. Do not tilt the laryngoscope backward and do not use excessive force. Be careful to avoid dental damage during laryngoscopy. Beginners often do not advance the blade far enough into the Vallecula. Cricoid pressure can assist airway visualization. **F.** Once the vocal cords (VC) are visualized, do not take your eyes off glottic opening—have an assistant hand you the ETT. Advance ETT through glottic opening. Stop when cuff is past VCs.

Ⓖ Inflate the ETT with 2-6 cc air to achieve 20 cm H_2O pressure.

Ⓗ Attach anesthesia circuit to airway connector at the end of the ETT.

Proper placement should be confirmed by capnography and bilateral auscultation of breath sounds on lung examination.
Secure the ETT with adhesive tape.





G. Remove ETT stylet. Inflate the ETT cuff with 2-6 cc air (cuff pressure can be measured and adjusted to a minimum of 20 cm H_2O). **H.** Attach anesthesia circuit to the airway connector at the end of the ETT. Confirm proper placement of the ETT by capnography and auscultation of bilateral breath sounds.

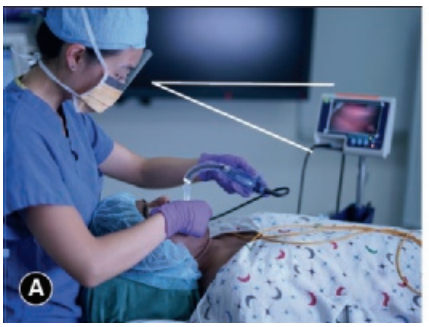
163 Video Laryngoscopy (VL) Intubation

Remi Wolf • Juliette Piot • Howard Ching • Janak T. Chandrasoma • T. Kyle Harrison • Vladimir Nekhendzy • Larry F. Chu

Equipment: Endotracheal tube, video laryngoscope, lubricant, adhesive tape, airway management equipment including face mask and oral airways.

Ⓐ Position the video screen in front of you at a comfortable height.

Ⓑ Prepare equipment and prewarm the optical system.





A. Position the video screen in front of you at a comfortable height. **B.** Insert rigid curved stylet into endotracheal tube. Prewarm the optical system by leaving the blade in place and camera light on. For demonstration purposes, the GlideScope Cobalt system is used for this cognitive aid.

Ⓒ Induce general anesthesia (see Induction of General Anesthesia Cognitive Aid, Chapter 159).

Ⓓ Open mouth with "scissor" technique using nondominant hand.

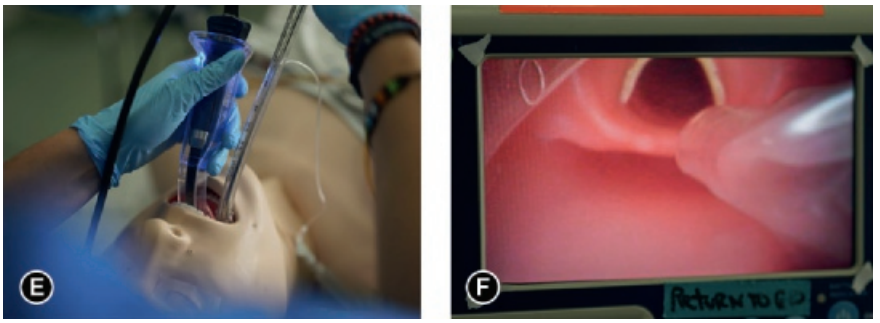




C. Induce general anesthesia. The patient's head should be in a neutral position. A sniffing position is an independent risk factor for acute angle VL failure. **D.** Open mouth with "scissor" technique.

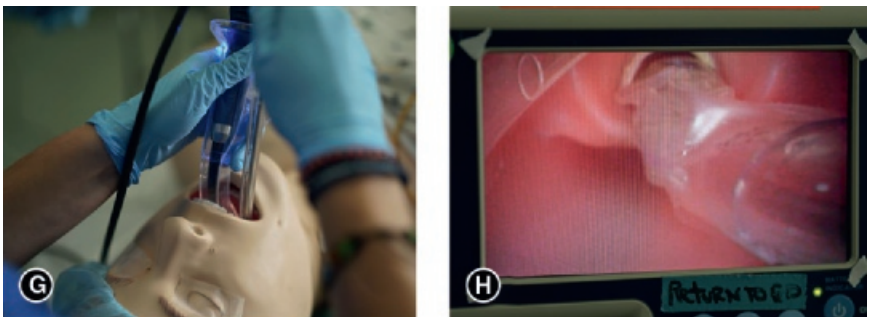
Ⓔ First, look in the mouth, and under direct vision, introduce the ETT.

Ⓕ Then, look at the screen. Gently advance ETT toward the larynx.



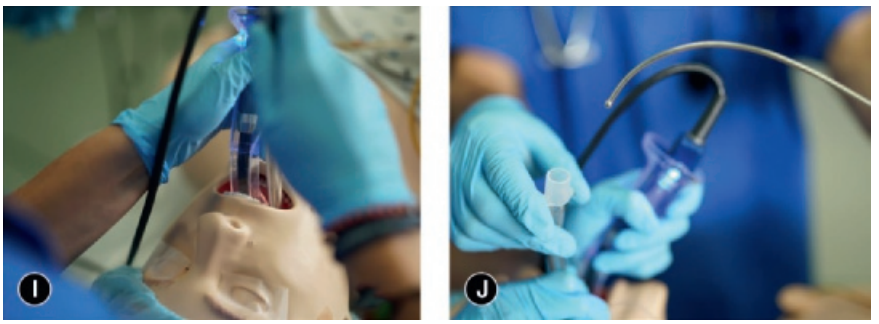
E. First, look in the mouth, and under direct vision, introduce the tube. **F.** Then, look at the screen.

Ⓖ, Ⓗ While watching the screen, advance the tube between the vocal cords.



G. Take care to avoid damage to tonsillar pillars as you advance the styletted tube. **H.** Watch the screen carefully as you guide the tube between the vocal cords. Never advance against resistance.

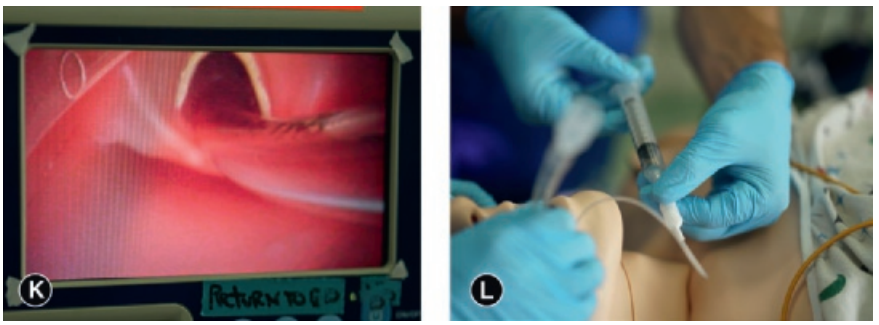
Ⓘ, Ⓙ Remove the stylet once ETT tip has passed between the cords.



I. Remove the stylet once the tip of the tube has passed between the vocal cords. **J.** Removing the stylet is facilitated by placing your thumb on the stylet thumbwheel to dislodge the stylet.

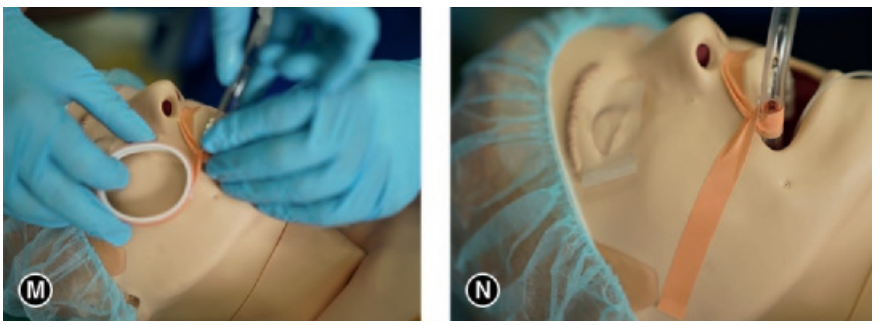
Ⓚ With the vocal cords still in view, advance the tube until its tip extends a few centimeters under the vocal cords.

Ⓛ Inflate ETT cuff, confirm ETT position, and secure per routine.



K. Confirm the final position of the tube as it is advanced until its tip extends a few centimeters under the vocal cords. **L.** Complete intubation by inflating the cuff of the endotracheal tube and connecting the airway in the standard manner.

Ⓜ, Ⓝ Once the patient's trachea has been successfully intubated, check the placement of the tube and secure it, as recommended in direct laryngoscopy procedures.



M. Properly secure the airway in the standard manner to ensure that the tube is not accidentally dislodged or removed **(N).**

164 Awake Fiberoptic Intubation

Larry F. Chu • T. Kyle Harrison

Equipment: Airway topicalization supplies, including 4% lidocaine solution. Fiberoptic bronchoscope (FOB), endotracheal tube (ETT), air syringe, and Magill forceps.

Confirm MSMAIDS mnemonic (see Induction of Anesthesia Cognitive Aid, Chapter 159). An assistant should be available throughout procedure.

Ⓐ Assemble equipment for airway anesthesia and intubation.

Ⓑ Explain procedure and reassure patient. Sedate as appropriate. Administer nebulized lidocaine. Consider antisialagogue.





A. Assemble equipment including 4% lidocaine solution, topicalization devices such as spray wand or nebulizer, oral airway, ETT, and air syringe. **B.** Nebulized lidocaine is an effective method for topical anesthesia of the airway to tolerate awake fiberoptic intubation (FOI). Ensure total topical lidocaine does not exceed maximum dose of 5 mg/kg. Consider 0.2 mg IV glycopyrrolate antisialagogue.

Ⓒ Lidocaine should also be applied directly to the airway.

Ⓓ Insert an oral airway. The patient should not react or "gag."





C. Lidocaine should also be directly administered to the airway with a spray wand. **D.** Test adequate airway topicalization by placing oral airway with lidocaine ointment. The patient should not react or "gag."

Ⓔ Test the fiberoptic bronchoscope (FOB) to ensure that it is functioning properly.

Ⓕ Remove the airway connector from the end of an ETT and apply lidocaine ointment for lubrication.

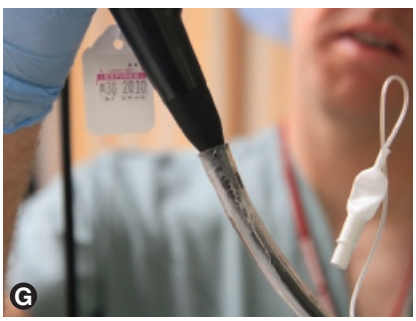




E. Assemble and test the FOB according to the manufacturer's instructions. **F.** Remove the airway connector from the end of an ETT and apply a small amount of lidocaine ointment for lubrication so the ETT will glide smoothly over the FOB.

Ⓖ Slide lubricated ETT over the FOB. Remove any excess ointment from the FOB. Consider applying anti-fog spray to FOB.

Ⓗ Elevate the head of the bed. Adjust bed height for easy positioning.





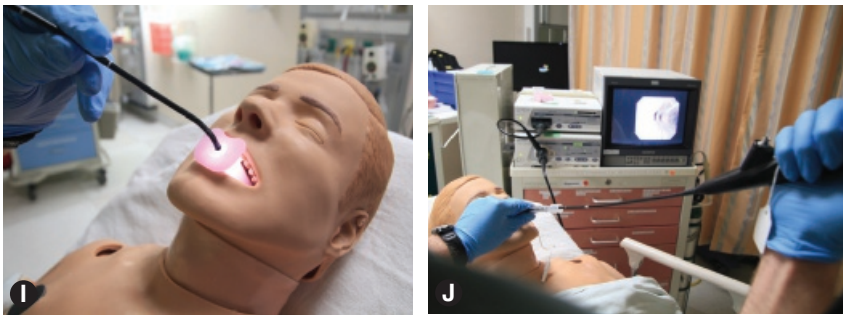
G. Slide the lubricated ETT over the FOB. Remove any excess ointment from the end of the FOB. Consider applying anti-fog spray or liquid to the end of the FOB. **H.** For awake FOI, stand in front of the patient and place video monitor within easy viewing distance. Elevate the head of the patient's bed 45 degrees.

IMPORTANT TASKS DURING FOI

1. An assistant should be present to **continuously monitor** the patient's vital signs during FOI. **Help should be immediately available.**
2. An assistant can titrate sedation, as appropriate during the procedure, under the direction of an anesthesiologist. **AVOID APNEA.**
3. Patient should **breathe spontaneously at all times** during the awake FOI procedure. Supplemental oxygen should be administered via nasal cannula.

Ⓘ Insert the tip of the FOB through the airway into the mouth.

Ⓙ Look through the eyepiece or video monitor of the FOB.



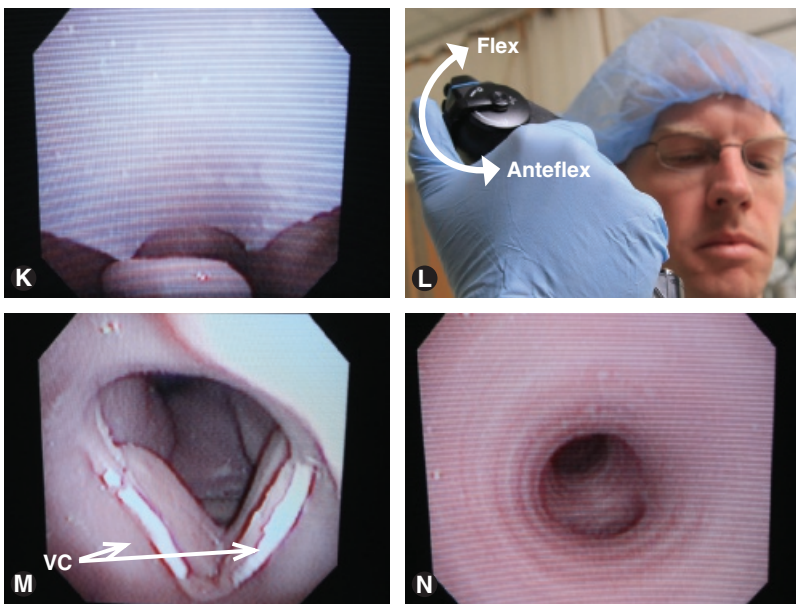
I. Insert tip of the FOB through the airway. **J.** Once the FOB is inserted, focus your attention on the video monitor (or FOB eyepiece) to visualize the airway structures as you advance the FOB.

Ⓚ Tip of airway and patient's soft palate will come into view as the FOB is advanced into the airway.

Ⓛ Anteflex (or flex) the tip of the FOB and advance until epiglottis and vocal cords (VCs) come into view.

Ⓜ Advance through cords.

Ⓝ Advance the scope past the vocal cords.



K. The tip of the airway and soft palate come into view. **L.** Gently anteflex the tip to assist visualization of the glottis or epiglottis. You may occasionally need to flex (thumb up) to assist visualization. **M.** Advance the FOB under the epiglottis, rotating the scope or flexing the tip to keep the VCs in the middle of the screen. **N.** Advance the scope past the VCs.

Ⓞ Advance to midtrachea.

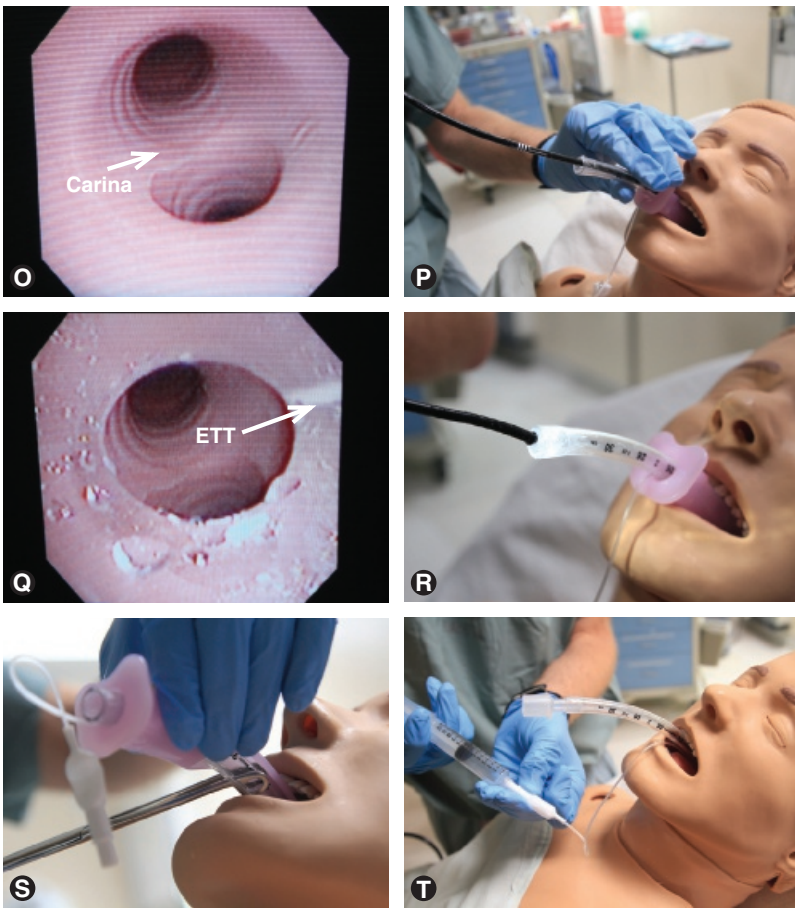
Ⓟ Advance ETT over FOB into airway in a smooth motion.

Ⓠ Confirm placement as FOB is withdrawn from airway.

Ⓡ Remove FOB from the airway.

Ⓢ Secure ETT with Magill forceps, while oral airway is removed over the ETT.

Ⓣ Reattach airway connector to ETT. Inflate the ETT cuff, and reconfirm position by auscultation and capnography.



O. Advance FOB until the carina is visualized and stop. **P.** Advance the ETT over the FOB into the airway. If difficulty is encountered, grasp the ETT and rotate 90 degrees counter clockwise to minimize impingement behind arytenoid. **Q.** Confirm proper ETT placement above carina as FOB is withdrawn. **R.** Remove FOB from ETT. **S.** Grasp ETT with Magill forceps as the oral airway is removed. **T.** Reattach airway connector to ETT. The cuff is inflated, and position is confirmed by the presence of exhaled CO_2 gas by capnography and auscultation of bilateral breath sounds.

165 Insertion of Left-Sided Double-Lumen Tube

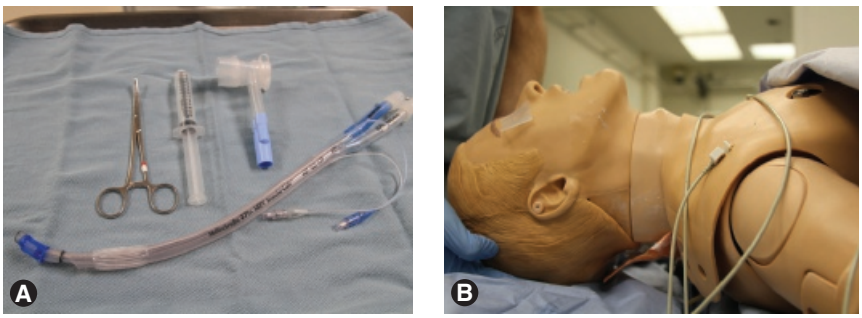
Larry F. Chu • Vivekanand Kulkarni • T. Kyle Harrison

Equipment: Appropriately sized double-lumen tube (DLT), clamp, fiberoptic bronchoscope, laryngoscope, stethoscope, and standard airway management equipment (see Intubation and Mask Ventilation Cognitive Aids).

Confirm MSMAIDS mnemonic (see Induction of General Anesthesia Cognitive Aid, Chapter 159).

Ⓐ Assemble equipment for airway instrumentation and intubation (**Table 165-1**).

Ⓑ Explain procedure and reassure patient. Induce general anesthesia (see Induction of General Anesthesia Cognitive Aid, Chapter 159). Place patient in "sniffing" position.



A. Assemble equipment including appropriately sized DLT (Table 165-1). **B.** Explain the procedure to the patient. Confirm MSMAIDS mnemonic. After confirming normal stable vital signs, proceed with induction (see Induction of Anesthesia Cognitive Aid). Place the patient in the proper "sniffing position."

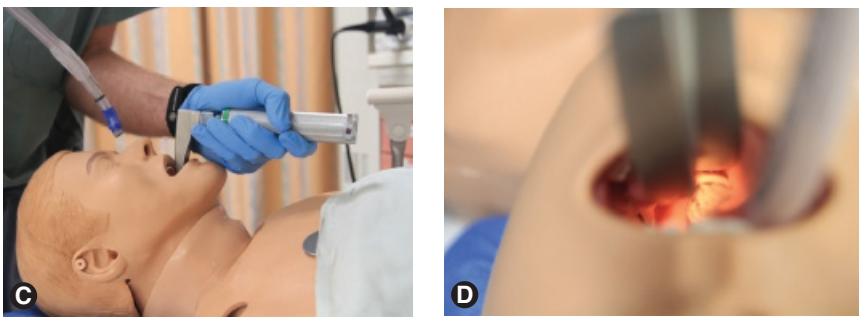
TABLE 165-1 Guidelines for Left Double-Lumen Tube Selection

Tracheal Width (mm)	Recommended Size
>18	**41 Fr** (M,R,S,P)
>17	**41 Fr** (M,S) **39 Fr** (R,P)
>16	**39 Fr** (M,S) **37 Fr** (R,P)
>15.5	**37 Fr** (M,S) **35 Fr** (R,P)
>15	**35 Fr** (M,R,S,P)
>14	**32 Fr** (M)
>13	**32 Fr** (M)
>12	**28 Fr** (M)
>11	**26 Fr** (R)

Manufacturer: M, Mallinckrodt (St. Louis, MO); P, Portex (Keene, NH); R, Rusch (Duluth, GA); S, Sheridan (Argyle, NY).

Ⓒ Perform direct laryngoscopy under ideal intubating conditions.

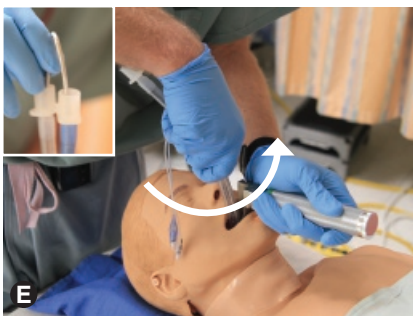
Ⓓ Advance DLT into the airway. Stop when blue cuff passes vocal cords.



C. Perform direct laryngoscopy to visualize the glottic opening under optimized intubating conditions.
D. Advance the DLT under direct visualization into the airway and stop advancing the DLT when the blue bronchial cuff passes the vocal cords. The tracheal (clear) cuff should be above the vocal cords.

Ⓔ Remove stylet from the DLT. Rotate DLT 90 degrees counterclockwise and advance with a smooth motion into the airway.

Ⓕ Attach connectors to end of bronchial (blue) and tracheal (clear) lumens.



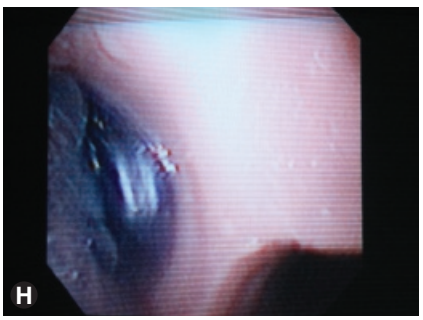


E. Remove stylet from DLT. Rotate DLT 90 degrees counterclockwise as you advance the tracheal cuff through the DLT. This will help direct the end of the DLT with the bronchial cuff into the left mainstem bronchus. **F.** When the tube is inserted to a depth of 29 cm at the lips, attach the tracheal and bronchial lumens to the airway connectors.

Ⓖ Confirm proper placement by auscultation.

Ⓗ Confirm proper placement by fiberoptic bronchoscope (FOB) through tracheal lumen.





G. Inflate both tracheal and bronchial cuffs and auscultate bilateral breath sounds. Occlude bronchial lumen and auscultate right-side only breath sounds. Occlude tracheal lumen and auscultate left-side only breath sounds, confirming placement. **H.** Insert FOB through tracheal lumen and visualize only the rim of the blue cuff in the left main bronchus, just beyond the carina, confirming proper placement. If the blue cuff herniates across the carina, it is shallow and needs to be advanced until only the blue rim is visible.

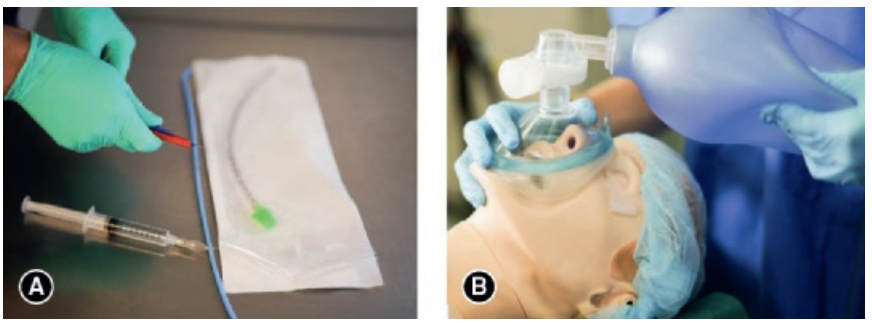
166 Scalpel-Bougie Technique for Emergency Front of Neck Access (FONA)

Janak T. Chandrasoma • T. Kyle Harrison • Vladimir Nekhendzy • Larry F. Chu

Equipment: Endotracheal tube, scalpel, bougie, lubricant, adhesive tape, airway management equipment including face mask and oral airways.

Ⓐ Call for help. Prepare equipment for the procedure.

Ⓑ Abandon bag-mask ventilation if previous attempts have failed.



A. Call for help while you assemble your equipment. Ensure your scalpel and ETT are working correctly. The scalpel should be round for blunt stab incision and the cutting edge of the blade shall be wider than the tip of the bougie. Usually a no. 10 blade is used. **B.** Abandon bag-mask ventilation if previous attempts failed.

Ⓒ Grasp the top of the larynx with your index finger and thumb. Use "laryngeal handshake" to locate cricothyroid membrane.



C. Perform the "laryngeal handshake" using your nondominant hand. Grasp the top of the larynx. Slide your hand down, identifying the hyoid and thyroid laminae as you go. Stabilize the larynx between your thumb and middle finger. Finally, move down the neck and palpate the cricothyroid membrane (CTM) with your index finger.

SCALPEL-BOUGIE TECHNIQUE FOR EMERGENCY FRONT OF NECK ACCESS (FONA)

Ⓓ Stabilize the larynx with your nondominant hand. Make a vertical skin cut with your dominant hand using the scalpel.

Ⓔ Palpate the cricothyroid membrane using your nondominant hand. Make a horizontal stab incision into the membrane.





D. Stabilize the larynx between your fingers with your nondominant hand. Rest your dominant hand on the sternum and make a vertical incision over cricothyroid membrane, keeping the blade midline. **E.** Insert your nondominant hand and palpate the cricothyroid membrane. Make a horizontal stab incision into the cricothyroid membrane using the scalpel. Then, rotate the scalpel 90 degrees with the sharp end pointed toward the patient and with a slight tug on the trachea to maintain a tracheal hole for bougie entry.

Ⓕ Advance a bougie into the opening.

Ⓖ Advance a 6.0 endotracheal tube over the bougie (for an adult). The size of the endotracheal tube should be adjusted smaller for children as appropriate, as should the size of the bougie.

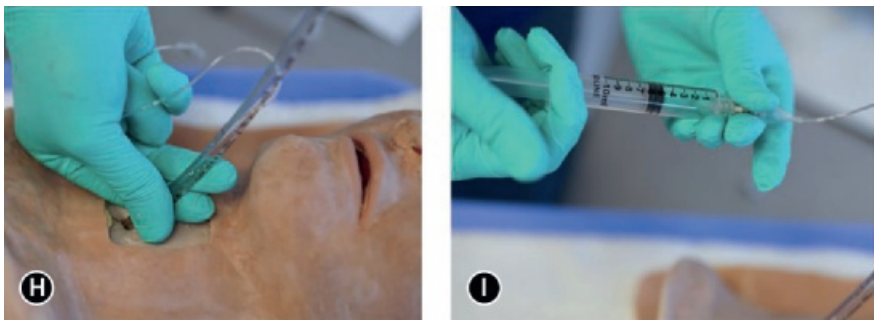




F. Advance a bougie into the opening. **G.** Advance a 6.0 endotracheal tube over the bougie. Advancement can sometimes be facilitated with corkscrew motions.

Ⓗ Advance the endotracheal tube until the cuff disappears into the trachea.

Ⓘ Inflate the endotracheal tube cuff with air.



H. Advance the endotracheal tube until you no longer can see the cuff and it disappears into the tracheal opening. **I.** Inflate the endotracheal tube cuff with air.

Ⓙ Ventilate through the endotracheal tube and confirm proper placement.



J. Ventilate through the endotracheal tube while monitoring end-tidal carbon dioxide to confirm proper placement of the endotracheal tube. Auscultate while ventilating to confirm bilateral breath sounds and to rule out mainstem intubation.

SUGGESTED READINGS

Brofeldt BT, Panacek EA, Richards JR. An easy cricothyrotomy approach: the rapid four-step technique. *Acad Emerg Med*. 1996;3:1060-1063.
Davis DP, Bramwell KJ, Hamilton RS, Chan TC, Vilke GM. Safety and efficacy of the rapid four-step technique for cricothyrotomy using a Bair Claw. *J Emerg Med*. 2000;19:125-129.
Drew T, et al. Laryngeal handshake technique in locating the cricothyroid membrane: a non-randomised comparative study. *Br J Anaesth*. 2018;121(5):1173-1178.
Holmes JF, Panacek EA, Sakles JC, Brofeldt BT. Comparison of 2 cricothyrotomy techniques: standard method versus rapid 4-step technique. *Ann Emerg Med*. 1998;32:442-446.
Morris A, Lockey D, Coats T. Fat necks: modification of a standard surgical airway protocol in the pre-hospital environmental. *Resuscitation*. 1997;35: 253-254.

167 Radial Artery Catheterization

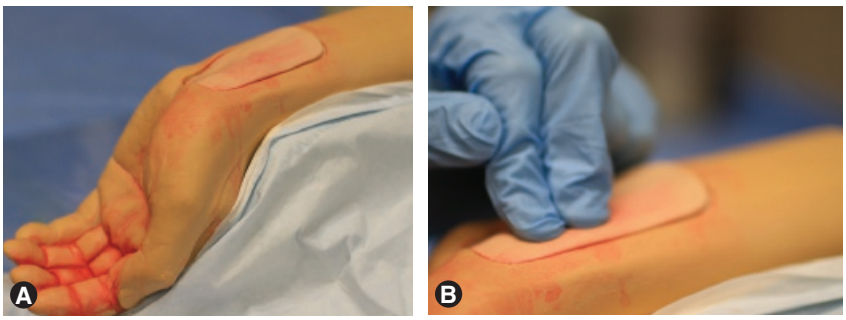
Larry F. Chu • T. Kyle Harrison

Equipment: Radial artery catheter, gauze, alcohol pad, suture material, adhesive dressing, and tape.

Explain the procedure to the patient and obtain consent. Always wear gloves and use universal precautions.

Ⓐ Place the wrist in extension as shown. Wrist splints can assist with proper positioning.

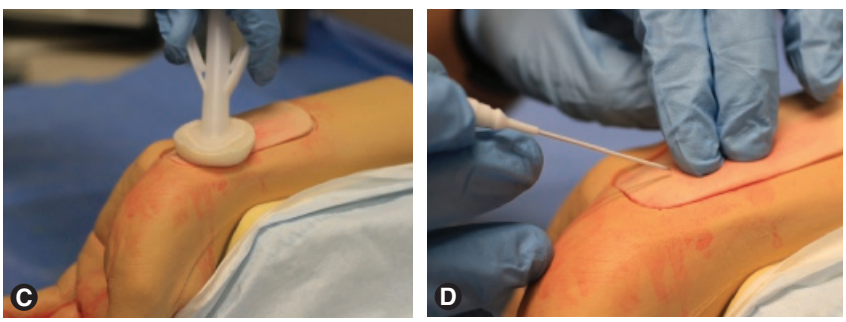
Ⓑ Palpate the radial artery pulse using the fingertips of your nondominant hand.



A. Place the wrist in extension as shown. The use of wrist splints can assist with proper positioning during placement. The splint may be removed after placement is accomplished. **B.** Palpate the radial artery pulse located 1-2 cm from the wrist, between the bony head of the distal radius and the flexor carpi radialis tendon.

Ⓒ Sterile prep the wrist at the catheter insertion site.

Ⓓ Insert the needle/catheter assembly at a 45-degree angle over the site of arterial pulsation.



C. Clean the insertion site with alcohol prep. **D.** Insert the needle/catheter assembly into the wrist at the site of arterial pulsation.

Ⓔ Advance the needle/catheter assembly slowly toward arterial pulse. Stop once arterial blood is observed in the assembly barrel.

Ⓕ Advance the guidewire by sliding the black tab on the barrel down toward the catheter. Stop if resistance is encountered. Advance the catheter over the guidewire.

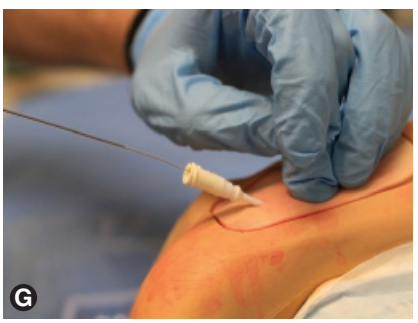


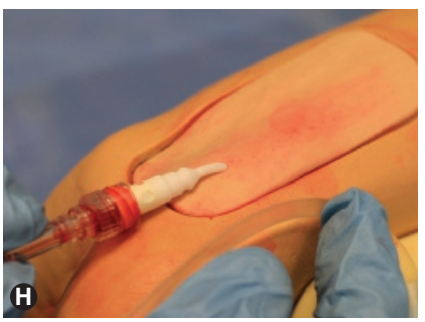


E. Advance the needle/catheter assembly slowly toward the arterial pulsations until a flash of blood is visualized. Stop advancing assembly. **F.** Free flow of arterial blood indicates proper needle placement. Advance guidewire into artery by sliding the black tab down the barrel of the assembly. Do not advance wire if resistance is encountered. Smooth and easy guidewire advancement is reassuring.

Ⓖ Apply downward pressure on the radial artery at the catheter tip. Remove the needle/wire assembly.

Ⓗ Attach arterial pressure transducer tubing to the catheter and secure the catheter with sutures to the patient's wrist. Alternatively, transparent adhesive dressing and tape can be used.





G. Apply pressure to the artery at the catheter tip and remove the needle/wire assembly. **H.** Attach arterial pressure transducer tubing to the catheter and secure with sutures or an adhesive dressing. Confirm proper placement by evaluating arterial pressure waveform on the patient monitors.

Confirm proper placement by evaluating the arterial pressure waveform on the patient monitors.

168 Central Venous Catheterization

Larry F. Chu • T. Kyle Harrison

Equipment: Central line kit and full body sterile drape. Ultrasound machine and probe with sterile transducer sheath.

Wear sterile gown, face mask, and sterile gloves. Explain procedure to patient. Always wear gloves and use universal precautions.

Ⓐ Sterile prep the patient's neck. Trendelenburg if possible.

Ⓑ Place gel on ultrasound (US) probe. Place probe into sterile sheath.

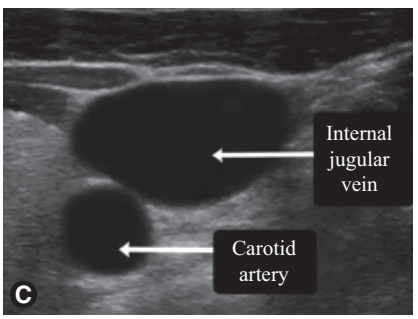




A. If needed and appropriate, Trendelenburg position (head down) can be used to increase venous return to the heart and facilitate central venous cannulation. The neck should be sterile prepped with alcohol.
B. Ultrasound gel should be placed on the probe and covered with a sterile sheath by an assistant.

Ⓒ Sterile drape neck (not shown). Place the US probe parallel and cephalad to the clavicle between the two heads of the sternocleidomastoid muscle. The internal jugular vein is visualized.

Ⓓ Puncture the skin at a 45-degree angle and aspirate until the needle is seen on US and a flash of blood is aspirated into syringe.

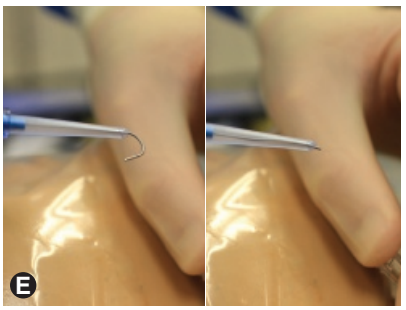


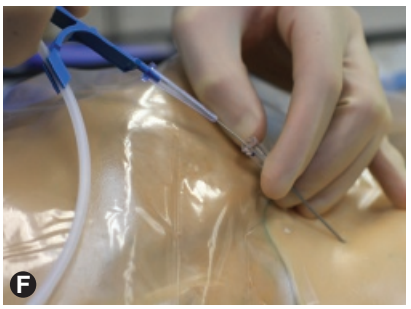


C. The internal jugular vein is easily compressible, while the common carotid artery is pulsatile and not compressible. **D.** Advance needle at a 45-degree angle and aspirate needle until blood is aspirated. Stop.

Ⓔ Prepare wire by retracting the soft tip into the holder.

Ⓕ Confirm free flow blood and advance the wire through the needle. Monitor EKG. If ventricular ectopy is observed, stop and retract wire.

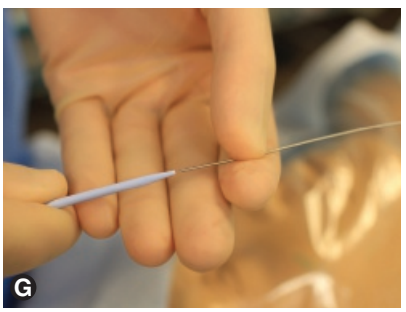


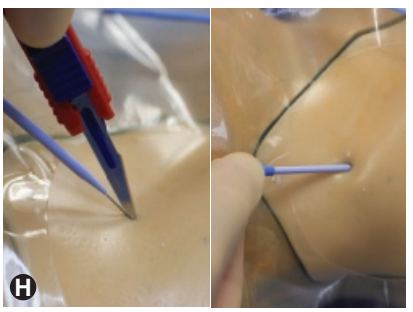


E. Retract wire into the holder. **F.** Confirm free flow venous blood and pass the wire through the needle in a smooth motion. Monitor EKG. If ventricular ectopy is observed, stop and retract wire.

Ⓖ Advance dilator over wire.

Ⓗ Nick skin and advance dilator.

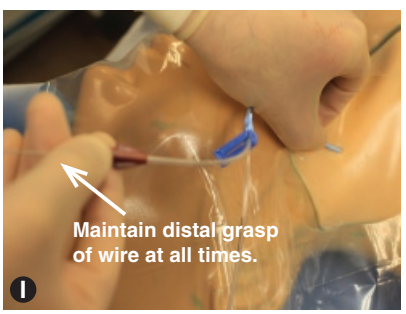


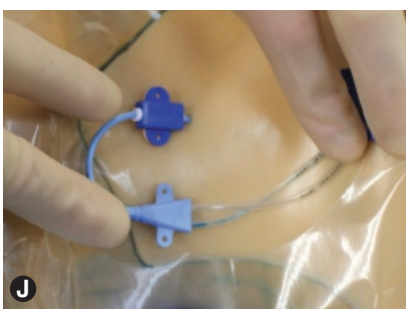


G. Advance dilator over wire, retaining control of wire at all times. **H.** Create a small skin incision at insertion site and advance dilator in a smooth motion. Remove dilator, retaining control of wire.

Ⓘ Remove dilator, advance catheter over wire.

Ⓙ Suture catheter.





I. Advance catheter over wire, grasp wire as it exits catheter. Retract wire from catheter. **J.** Flush lumens of catheter with saline. Secure the catheter with sutures and a dressing.

169 Spinal Anesthesia

Larry F. Chu • Andrea J. Traynor • T. Kyle Harrison

Equipment: Spinal anesthesia kit, sterile gloves, mask, and hat.

ASA Standard Monitors should be placed prior to spinal placement. An assistant should be available throughout procedure.

Ⓐ Assemble equipment for spinal placement.

Ⓑ Explain procedure and reassure patient. Sedation as appropriate. Patient positioning is critical for successful placement. Instruct the patient to round his or her back to facilitate spinal placement.

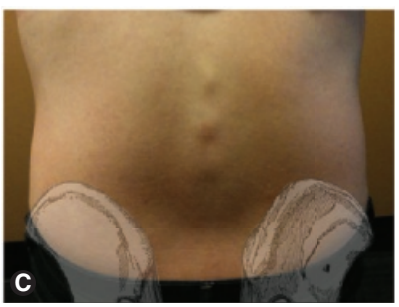


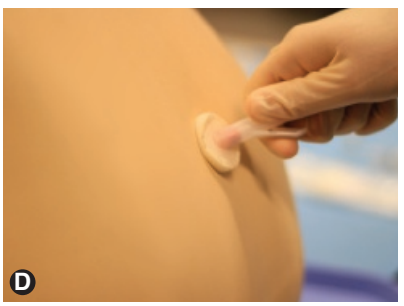


A. Assemble equipment for spinal anesthesia including spinal medications and local anesthetic for skin wheal. **B.** The patient should be instructed to sit with his or her back rounded in order to open the spaces between the spinous processes to facilitate placement of spinal anesthesia.

Ⓒ Examine surface anatomy of the patient's back. Identify the spinous processes on the top of the iliac crests (L4 spinous process).

Ⓓ Sterile prepare the back widely around the L4-L5 interspace.



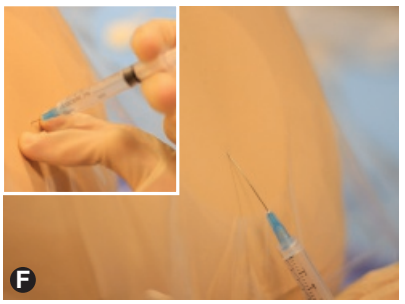


C. The surface anatomy of the back can be examined for spinous process landmarks. The superior margin of the iliac crests can be palpated and represent the approximate level of the L4 spinous process. **D.** Sterile prep the back with sterile prep solution widely around the L3-L4 or L4-L5 interspace.

Ⓔ Drape the patient. With sterile technique, reconfirm landmarks for lumbar interspace.

Ⓕ Place a skin wheal of local anesthetic. Infiltrate deeper.

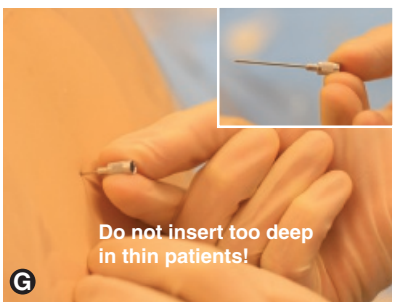


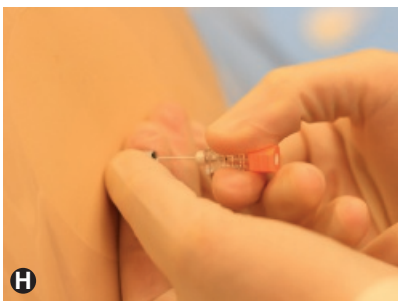


E. Drape the patient and reconfirm L3-L4 or L4-L5 level. **F.** Inject local anesthetic skin wheal and then redirect the needle through the wheal to infiltrate deeper tissues along the intended spinal needle path.

Ⓖ Insert introducer needle perpendicular to back, midline.

Ⓗ Insert spinal needle through introducer until "pop" is felt.

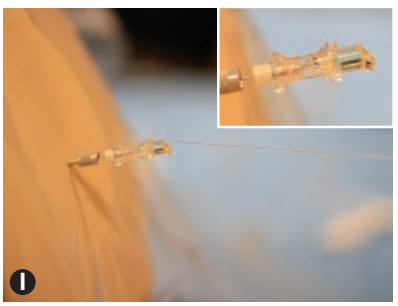




G. Place the introducer perpendicular to the back, midline at L3-L4 or L4-L5. **H.** Advance the spinal needle until a "pop" is felt, indicating dural puncture. Stop advancing the needle immediately.

Ⓘ Remove stylet. Observe free flow cerebrospinal fluid (CSF).

Ⓙ Slowly inject spinal medication.





I. Remove the stylet from the spinal needle. Observe free flow CSF fluid, noting absence of blood.
J. Attach syringe with spinal medications. Aspirate slightly to reconfirm CSF flow, then inject medication while stabilizing needle at the introducer. Aspirate at the end to reconfirm the full dose was administered.

170 Lumbar Epidural and Combined Spinal Epidural (CSE) Placement

Andrea J. Traynor • Larry F. Chu • T. Kyle Harrison

Equipment: Epidural catheterization kit, sterile gloves, mask, and hat.

ASA Standard Monitors should be placed prior to epidural placement. An assistant should be available throughout procedure.

Ⓐ Assemble equipment for epidural placement.
Explain procedure and reassure patient. Sedation as appropriate.

Ⓑ Patient positioning is critical for successful placement. Instruct the patient to round his or her back to facilitate epidural placement.



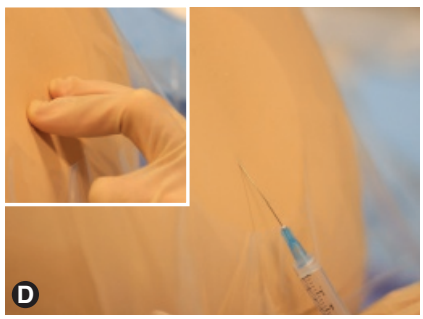


A. Assemble equipment for epidural, including saline for loss of resistance syringe and local anesthetic for skin wheal. **B.** The patient should be instructed to sit with his or her back rounded in order to open the spaces between the spinous processes to facilitate epidural catheter placement.

Ⓒ The back should be sterile prepped widely around L4-L5 interspace.

Ⓓ Place sterile drape. With sterile technique, palpate L4 spinous process (level of iliac crests). Inject local anesthetic wheal in the interspace.





C. The back should be widely sterile prepped with sterile prep solution. **D.** The L4 spinous process should be palpated (level with the top of the iliac crests) and a small local anesthetic wheal injected in the L4-L5 interspace. Local anesthetic can be infiltrated deeper through the skin wheal. 2% lidocaine can be used.

LUMBAR EPIDURAL CATHETER PLACEMENT

Ⓔ The Tuohy epidural needle should be inserted midline.

Ⓕ Once the Tuohy is seated into interspinous ligament (1-2 cm), remove stylet.



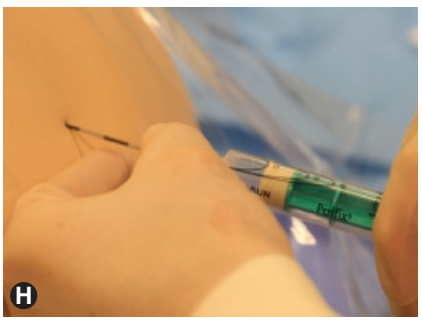


E. The Tuohy needle should be inserted perpendicular to the back, midline, through the L4-L5 interspace. **F.** Once the Tuohy needle has passed subcutaneous tissues and is firmly seated in ligament (a "crunching" sensation is felt), the stylet can be removed.

Ⓖ A saline-filled loss of resistance (LOR) syringe should be attached to the Tuohy needle. Apply gentle constant pressure to the plunger as the Tuohy needle is advanced through ligament by the nondominant hand.

Ⓗ As ligamentum flavum is encountered an **increase in resistance** may be felt, followed by a sudden LOR. Stop.





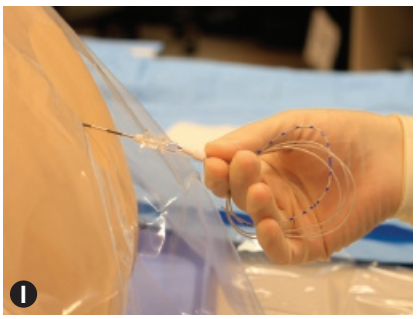
G. A saline-filled loss of resistance syringe should be attached to the Tuohy and slowly advanced while constant gentle pressure is applied to the plunger. **H.** Resistance to the plunger will suddenly decrease when the epidural space is entered (loss of resistance, LOR). **Stop**. Note the depth of LOR.

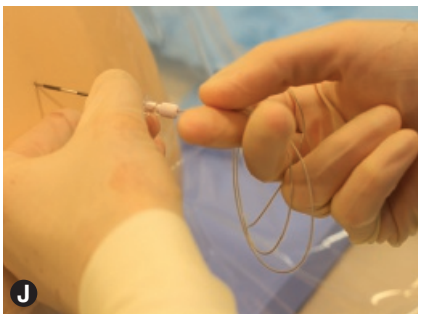
PATIENT CONSIDERATIONS DURING EPIDURAL PLACEMENT

1. **Make patient comfortable** (warm room temperature, apply warm blankets when moved to OR table, introduce OR staff).
2. **Reassure patient** during this anxious period of time. Maintain patient modesty by draping body while positioning and applying monitors.

Ⓘ Place catheter into Tuohy needle. (For CSE, first do Steps **O**-**T**.)

Ⓙ The catheter should advance smoothly into the epidural space.





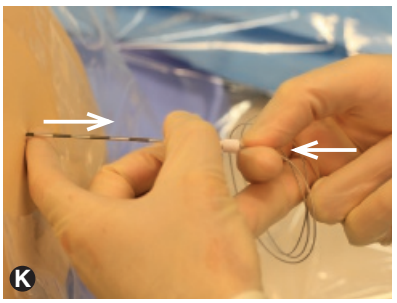
I. Epidural catheter is placed into Tuohy. **J.** The catheter should advance smoothly into the epidural space. If resistance is felt, consider dilating the epidural space with saline or repeating LOR technique.

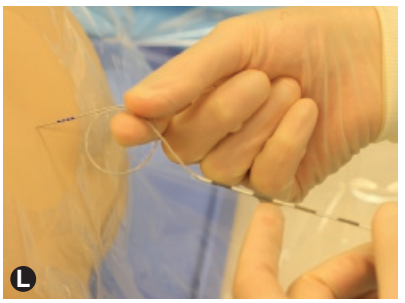
Ⓚ The needle can be removed after 15-20 cm has been threaded into the epidural space. Provide countertraction on catheter.

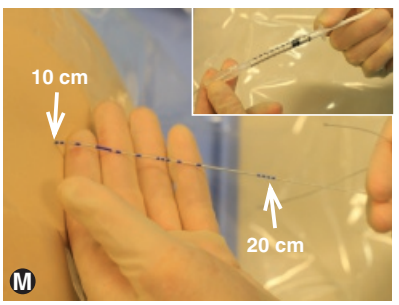
Ⓛ Once needle has been removed, pull the catheter out until 5 cm remains in the epidural space.

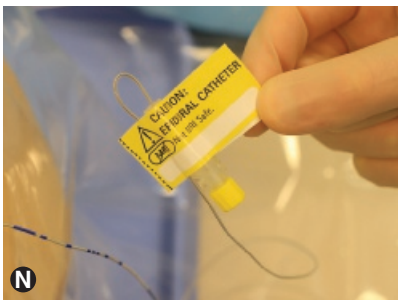
Ⓜ Aspirate the catheter.

Ⓝ Cap/label catheter.









K. The Tuohy needle is removed with countertraction on the catheter to prevent catheter migration during needle removal. **L.** Once the needle is removed, pull the catheter out until 5 cm remains in the epidural space. **M.** Aspirate the catheter with a 1-cc syringe to confirm absence of cerebrospinal fluid (CSF) (intrathecal) or blood (intravascular). **N.** Place a cap and label on the end of the catheter.

Ⓔ Place a Tuohy needle into the epidural space (Steps **A**-**E**).

Ⓞ Prepare spinal needle (SN).

Ⓟ Insert SN through Tuohy needle until a "pop" is felt

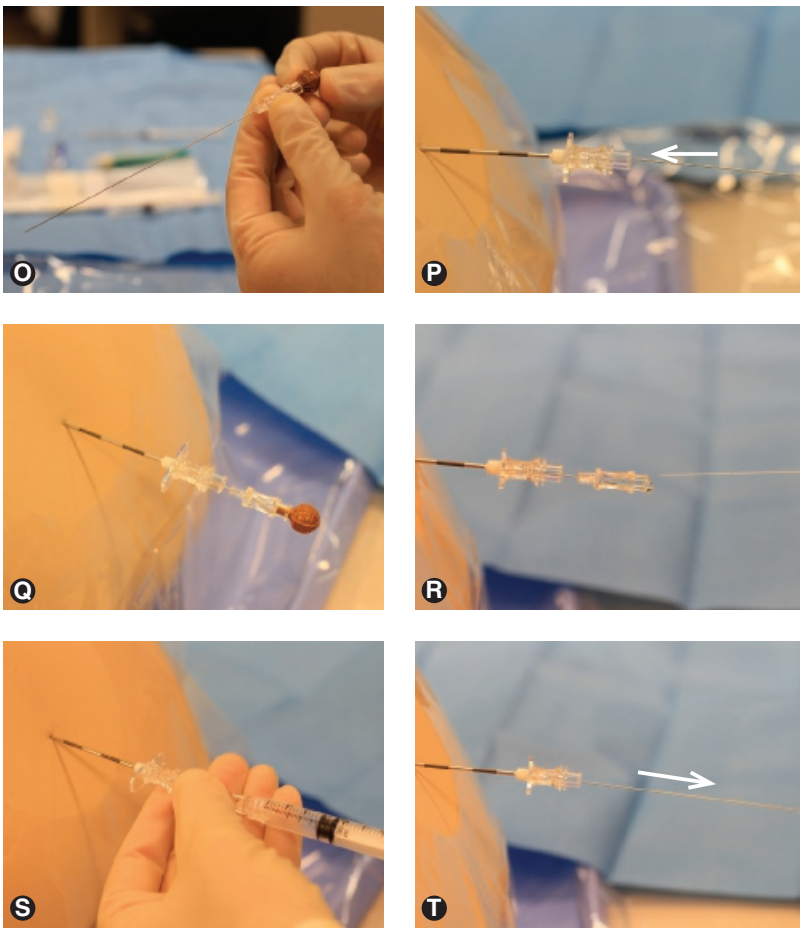
Ⓠ Remove stylet.

Ⓡ Observe CSF flow.

Ⓢ Aspirate CSF, then slowly inject spinal medication.

Ⓣ Remove SN.

Ⓖ Go to Steps **K**-**N**.



O. Prepare a 26-27 pencil point spinal needle (SN) by loosening the stylet. **P.** Advance the SN through the Tuohy from Step **E**. **Q.** Continue to advance the SN until a "pop" is felt, at which point stop advancing the needle. **R.** Remove the stylet from the SN and observe free flow CSF to gravity. **S.** Stabilize Tuohy needle and attach a syringe containing spinal medications (Luer-Lok preferable), being careful not to inadvertently reposition the SN. Gently inject the spinal medication through the SN. **T.** Remove SN from the Tuohy needle.

171 Regional Anesthesia in the Anticoagulated Patient

Patrycja Hanyen • Larry F. Chu • Bassam Kadry

Warning

The following cognitive aid was created using the 2018 guidelines from the American Society of Regional Anesthesia and Pain Medicine for patients receiving regional anesthesia and antithrombotic or thrombolytic therapy (Horlocker TT, et al. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy: American Society of Regional Anesthesia and Pain Medicine Evidence-Based Guidelines (Fourth Edition). *Reg Anesth Pain Med*. 2018;43(3):263-309). As noted in their guidelines, "An understanding of the complexity of this issue is essential to patient management; a 'cookbook' approach is not appropriate. Rather, the decision to perform spinal or epidural anesthesia/analgesia and the timing of catheter removal in a patient receiving antithrombotic therapy should be made on an individual basis, weighing the small, although definite risk of spinal hematoma with the benefits of regional anesthesia for a specific patient (and the risks of withholding these benefits)." Deriving time intervals to minimize risk of hematoma depends on the amount of time it takes the drug to undergo three elimination half-lives. Periodic neurological assessments should be conducted for minimum of 24 hours postintervention if the clinical situation is unclear.



ASRA has created a mobile app for iOS and Android to aid practitioners in navigating best practices outlined in their document. ASRA Coags 2.0 can be accessed online in the iOS and Android Play Stores (https://www.asra.com/page/150/asra-apps)

GUIDELINES FOR REGIONAL ANESTHESIA IN THE ANTICOAGULATED PATIENT[a]

Anticoagulant	Time Before Puncture/Catheter Manipulation or Removal	Time After Puncture/Catheter Manipulation or Removal	Laboratory Tests
Unfractionated heparins (UFHs) (for prophylaxis, ≤15 000 units SC once daily)	4-6 h	1 h	Platelets during treatment >5 d
UFHs (for treatment)	IV 4-6 h SC 8-12 h	1 h	Activated partial thromboplastin time (aPTT), activated clotting time (ACT), platelets
Low molecular weight heparins (LMWHs) (for prophylaxis)	12 h	1 h	Platelets during treatment >5 d
LMWHs (for treatment)	24 h	4 h	Platelets during treatment >5 d
Fondaparinux (for prophylaxis, 2.5 mg SC once daily)	36-42 h	6-12 h	(Anti-factor Xa, standardized for specific agent)
Rivaroxaban (for prophylaxis, 10 mg orally once daily)	22-26 h	4-6 h	(Anti-factor Xa, standardized for specific agent)
Apixaban (for prophylaxis, 2.5 mg orally bid)	26-30 h	4-6 h	(Anti-factor Xa, standardized for specific agent)
Dabigatran (for prophylaxis 150-220 mg orally once daily)	Contraindicated according to manufacturer	6 h	Thrombin time (TT)
Coumadin	INR ≤ 1.4	After catheter removal	International normalized ratio (INR)
Hirudins	8-10 h	2-4 h	aPTT, ecarin clotting time (ECT)
Argatroban	4 h	2 h	aPTT, ECT, ACT
Aspirin (ASA)	None	None	
Clopidogrel	7 d	After catheter removal	
Ticlopidine	10 d	After catheter removal	
Prasugrel	7-10 d	6 h after catheter removal	
Ticagrelor	5 d	6 h after catheter removal	
Cilostazol	42 h	5 h after catheter removal	
Nonsteroidal anti-inflammatory drugs (NSAIDs)	None	None	

The recommendations in the following tables were adapted from the 2018 American Society of Regional Anesthesia and Pain Medicine Evidence Based Guidelines, "Regional Anesthesia in the Patient Receiving Antithrombotic or Thrombolytic Therapy (Fourth Edition)." *Reg Anesth Pain Med*. 2018;43(3):263-309, which were developed in conjunction with the European Society of Anaesthesiology. Variances from the recommendations may be acceptable based on the judgment of the responsible anesthesiologist. The recommendations are designed to encourage quality patient care and safety but cannot guarantee a specific outcome. They are subject to revision at any time and updates can be found at http://www.asra.com.

[a]All time intervals refer to patients with normal renal function. Prolonged time interval in patients with hepatic insufficiency.

172 Part A: The Upper Extremity
Interscalene Block

Vanessa J. Loland • Matthew W. Lawrence

The interscalene block (ISB) of the brachial plexus is commonly performed for surgical procedures of the shoulder and upper arm. It may be performed with the arm in any position and provides excellent anesthesia and postoperative analgesia.[1,2] Important surface landmarks include the sternocleidomastoid (SCM), cricoid cartilage, and scalene muscles. Techniques employing ultrasound[3] or electrical stimulation[4] for localization of the brachial plexus have largely supplanted the historic paresthesia-seeking technique of Winnie,[5] although the anatomic considerations remain the same. Continuous ISBs provide prolonged analgesia following shoulder surgery, among other benefits.

1) **Benefits and indications**
   a) **Surgical anesthesia or postoperative analgesia of the shoulder or upper arm**
   b) Frequent sparing of the C8 and T1 nerve roots makes this block unreliable for hand or wrist procedures unless selective ulnar nerve and/or medial antebrachial cutaneous nerve blocks are performed.



Interscalene block frequently spares C8 and T1 nerve roots, unless specifically targeted.

2) **Risks and contraindications**
   a) **Absolute contraindications**
      i) Local infection at the site of planned needle placement
      ii) Pneumonectomy or hemidiaphragmatic paresis on the contralateral side
         (1) 100% ipsilateral hemidiaphragmatic paresis due to phrenic nerve blockade was observed with paresthesia technique using 34-52 mL 1.5% mepivacaine.[6]
         (2) Ultrasound-guided ISB using low volumes of local anesthetic carries a lower risk (33% and 60% with 5 and 10 mL, respectively) of phrenic block,[7] but the possibility of hemidiaphragmatic paresis should be considered in all patients prior to performing ISB.
         (3) Unilateral hemidiaphragmatic paresis associated with ISB results in ~25% reduction of FVC and FEV1.[8]
         (4) In patients with normal pulmonary function, phrenic nerve blockade may be asymptomatic.

READ MORE
Overview of peripheral nerve blocks, Chapter 44

   b) **Relative contraindications**
      i) Comorbid pulmonary disease
      ii) Coagulopathy
      iii) Preexisting neuropathy
   c) **Risks and complications**
      i) General risks
         (1) Nerve injury
         (2) Localized bleeding
         (3) Infection
         (4) Intravascular injection with local anesthetic toxicity

TABLE 172-1 Interscalene Brachial Plexus Block Pros and Cons

Pros	Cons
• Provides surgical anesthesia for procedures involving shoulder, proximal arm, and elbow • Better analgesia for shoulder surgery during the immediate postoperative period compared with phrenic-sparing techniques like suprascapular and axillary nerve blocks	• High incidence of hemidiaphragmatic paresis (up to 100% depending on technique) • Patients may develop hoarseness due to recurrent laryngeal nerve block or Horner syndrome due to blockade of cervical sympathetic fibers • Frequent sparing of C8/T1 nerve roots makes ISB less useful for hand surgery compared with more distal brachial plexus blocks

ii) Site-specific risks
  (1) Phrenic nerve blockade
  (2) Ipsilateral Horner syndrome (ptosis, miosis, and anhidrosis)
  (3) Hoarseness due to laryngeal vasodilation or recurrent laryngeal nerve block

iii) Potential complications
  (1) Vertebral artery injection
  (2) Epidural or intrathecal injection
  (3) Pneumothorax
  (4) Chronic diaphragmatic paralysis due to phrenic nerve injury[9]
  (5) Injury to the dorsal scapular or long thoracic nerves[10,11]

iv) Pros and cons of ISB are summarized in **Table 172-1**.



The incidence of ipsilateral hemidiaphragmatic paresis with interscalene block approaches 100% depending on technique. ISB should be avoided in patients with limited pulmonary reserve.

3) **Equipment**
  a) Common equipment for all peripheral nerve block techniques includes sterile skin cleansing solution, sterile gloves, a marking pen, and two 20-mL syringes of local anesthetic solution with extension tubing.
  b) A paresthesia technique only requires a needle attached to syringes of local anesthetic and a cooperative patient.
    i) If using a paresthesia-seeking or stimulation-guided technique, a needle of appropriate length (<5 cm for single injection) may help reduce the likelihood of serious complications.
  c) A nerve stimulator technique requires an electrical nerve stimulator with a compatible insulated needle and grounding electrode.
  d) Ultrasound-guided regional anesthesia requires portable ultrasound equipment and a high-frequency linear transducer for ISB.
4) **Technique**
  a) **Monitoring**
    i) Peripheral nerve blocks should be placed in a location where the patient's noninvasive blood pressure, oxygen saturation, EKG, and end-tidal CO_2 can be monitored.
  b) **Positioning and preparation**
    i) Position the patient supine with the back slightly elevated. A small shoulder roll may be placed for slight neck extension.
    ii) Apply monitors and supplemental O_2.
    iii) IV sedation may be administered, but the patient must be able to follow commands and volunteer sensory information.
    iv) With the head turned away from the side to be blocked, the patient should lift the head to aid in the identification of the SCM muscle.

FIGURE 172-1 Needle Position When Performing the Interscalene Block. Perpendicular to skin with an angle slightly medial, caudad, and posterior.



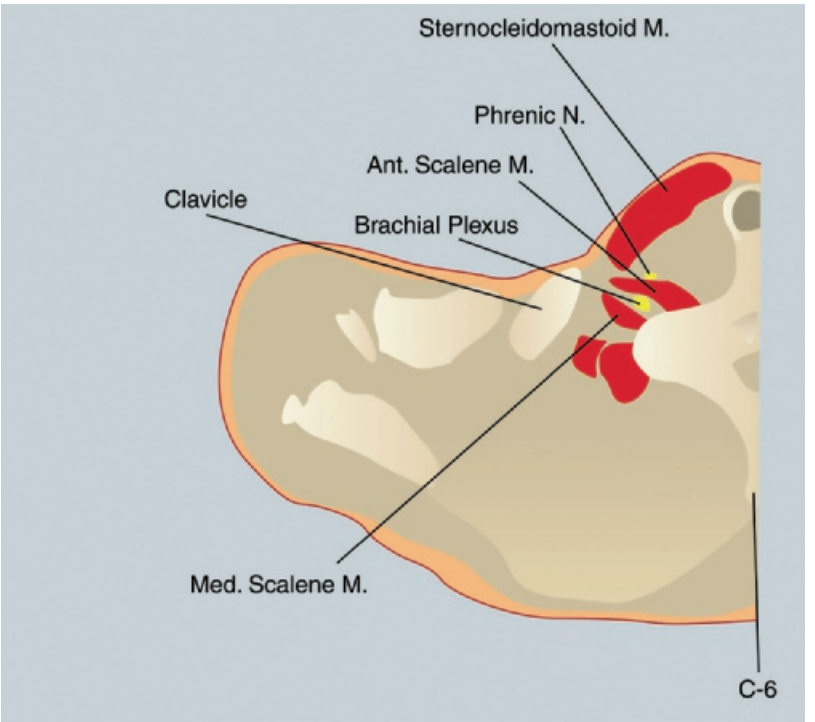
c) **Surface anatomy**
   i) Draw a line along the posterior border of the SCM.
   ii) Identify the cricothyroid cartilage and draw a line along a skin crease at this level (approximately C6).
   iii) Standing next to the head of the bed, palpate near the clavicle just posterior to the SCM. Ask the patient to inspire forcefully through the nose. This should delineate the interscalene groove and allow marking of the anterior and middle scalene muscles (Cognitive Aid: 173, Panel A).

d) **Simulating ISB**
   i) Using sterile technique and following injection of a local anesthetic skin wheal, insert a short insulated block needle at a 45-degree angle to the skin surface in the interscalene groove at the C6 level. The needle tip should be directed slightly medial, caudad, and posterior (Fig. 172-1). Avoid a perpendicular angle, as this is more likely to lead to intraforaminal needle placement.
   ii) The brachial plexus is superficial at this level and should be contacted within 2 cm of needle insertion in nearly all patients (Fig. 172-2).
   iii) Once an appropriate sensory paresthesia or motor response is elicited in the operative limb at a stimulating current <0.5 mA, slowly inject 30-40 mL of local anesthetic in incremental volumes following careful aspiration.
      (1) Look for a motor response in the hand, forearm, arm, deltoid, or pectoralis.
      (2) A deltoid response is as effective as a biceps twitch.[12]
      (3) Motor responses of the SCM or trapezius muscles are not acceptable end points.



A needle angle perpendicular to skin should be avoided as this angle is more likely to lead to intraforaminal needle placement.

FIGURE 172-2 The Safe Performance of an Interscalene Block Requires Superficial Needle Placement.



Reproduced with permission from Brown AR. Interscalene block. In: Chelly JE, ed. *Peripheral Nerve Blocks: A Color Atlas*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009:59.

e) **Ultrasound-guided ISB, in-plane technique**

i) With a high-frequency linear transducer positioned perpendicular to skin over the posterior border of SCM (Cognitive Aid: 173, Panel B), the brachial plexus is visualized in short axis between anterior and middle scalene muscles (Cognitive: 173, Panel C).

ii) After sterile skin preparation, place a local anesthetic skin wheal posterior to the edge of the ultrasound transducer.

iii) Insert the block needle through the skin wheal and direct anteriorly using in-plane guidance toward the target nerve bundle.[3]

(1) The "stoplight sign" seen on ultrasound at the level of the interscalene groove is most often formed by the C5 nerve root and the upper and lower fascicles of C6.[13]

(2) Care should be taken to avoid needle placement between the fascicles of the C6 nerve root.

iv) Once the needle tip passes through the middle scalene muscle into the interscalene groove, inject local anesthetic incrementally until satisfactory spread is visually confirmed.



Injection between the fascicles of the C6 nerve root may result in intrathecal spread of local anesthetic.

(1) Injection posterior to the "paraneural sheath" between middle scalene and superior trunk yields similar block quality and onset with less paresthesia compared with injection within the sheath between C5 and C6.[14]



Effective ISB can be achieved with placement of local anesthetic outside the paraneural sheath with less risk of paresthesia and nerve injury.

5) **Evaluation of the block**
   a) The quality of nerve blockade must be evaluated prior to surgical incision, even if intraoperative general anesthesia is planned.
   b) **Motor testing**
      i) Strength of elbow flexion against resistance tests the biceps muscle innervated by the musculocutaneous nerve.
      ii) Strength of elbow extension tests the triceps muscle innervated by the radial nerve.
      iii) Strength of shoulder abduction tests the deltoid muscle innervated by the axillary nerve.
   c) **Sensory testing**
      i) Pinching the base of the index finger, small finger, and medial aspect of the forearm tests sensation in the distribution of the median, ulnar, and medial antebrachial cutaneous nerves, respectively.
      ii) Pinching the posterior aspect of the shoulder over the scapula tests sensation in the distribution of the suprascapular nerve.



Care should be taken to avoid the long thoracic and dorsal scapular nerves within the middle scalene muscle when using the posterior in-plane approach.

6) **Postoperative management**
   a) All patients who undergo brachial plexus block should be discharged with a supportive sling.
   b) For outpatient procedures, respiratory status should be assessed prior to discharge, but a routine chest radiograph for pneumothorax or hemidiaphragmatic paresis is not recommended in asymptomatic patients.

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173 US-Guided Interscalene Block

Vanessa J. Loland • Larry F. Chu • Matthew W. Lawrence

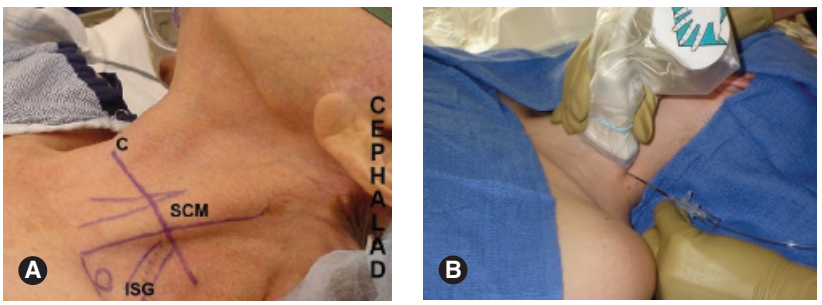
Patient Position: Position the patient supine with the back slightly elevated. A small shoulder roll may be placed for slight neck extension. With the head turned away from the side to be blocked, the patient should lift the head to aid in identification of the sternocleidomastoid (SCM) muscle.

Needle Size: 22-17 gauge, 50- to 100-mm block needle

Volume: 20-40 mL local anesthetic

Anatomic Landmarks

Ⓐ Draw a line along the posterior border of the SCM. Identify the cricothyroid cartilage and draw a line along a skin crease at this level (approximately C6). Standing next to the head of the bed, palpate near the clavicle just posterior to the SCM. Ask the patient to inspire forcefully through the nose. This should delineate the interscalene groove and allow marking of the anterior and middle scalene muscles.

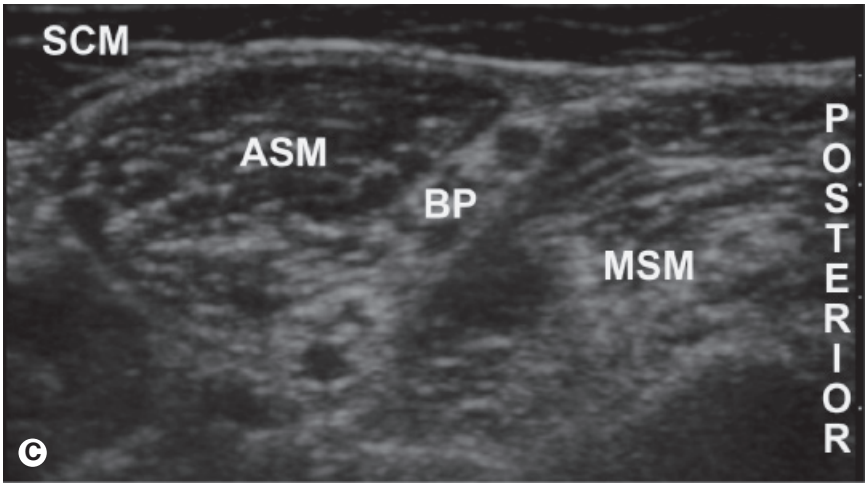


A. Surface anatomy relevant to the interscalene block. SCM, sternocleidomastoid muscle; C, cricoid cartilage; ISG, interscalene groove. **B.** A high-frequency linear transducer is placed perpendicular to skin over the posterior border of SCM. The block needle should be inserted posterior to the ultrasound transducer and directed anteromedially for in-plane guidance.

Approach and Technique

Ⓑ With a high-frequency linear transducer positioned perpendicular to skin over the posterior border of SCM, the brachial plexus is visualized in short axis between the anterior and middle scalene.

Ⓒ The "stoplight sign" seen on ultrasound at the level of the interscalene groove is most often formed by the C5 nerve root and the upper and lower fascicles of C6. After sterile skin preparation, place a local anesthetic skin wheal posterior to the edge of the ultrasound transducer. Insert the block needle through the skin wheal and direct anteriorly using in-plane guidance toward the target nerve bundle. Once the needle tip passes through the middle scalene muscle into the interscalene groove, inject local anesthetic incrementally until satisfactory spread is visually confirmed.



C. Short-axis image of the brachial plexus in the interscalene groove. SCM, sternocleidomastoid muscle; ASM, anterior scalene muscle; MSM, middle scalene muscle; BP, brachial plexus.

Evaluation of Block

Motor Testing

Strength of elbow flexion against resistance tests the biceps muscle innervated by the musculocutaneous nerve. Strength of elbow extension tests the triceps muscle innervated by the radial nerve. Strength of shoulder abduction tests the deltoid muscle innervated by the axillary nerve.

Sensory Testing

Pinching the base of the index finger, small finger, and medial aspect of the forearm tests sensation in the distribution of the median, ulnar, and medial antebrachial cutaneous nerves, respectively. Pinching the posterior aspect of the shoulder over the scapula tests sensation in the distribution of the suprascapular nerve.

174 Supraclavicular Block

Vanessa J. Loland • Ava Alamdari

The supraclavicular block anesthetizes the brachial plexus at the level of the distal trunks/divisions as the plexus crosses over the first rib between the insertion sites of the anterior and middle scalene muscles.[1] At this point, the brachial plexus is typically posterolateral and slightly cephalad to the subclavian artery and still within the interscalene groove. An advantage of this block is that it can be performed with the arm in any position. The use of real-time ultrasound guidance may significantly reduce the potential risk of pneumothorax associated with this approach[2-4] and improves the efficacy and speed of onset of this block.[5-7]

1) **Benefits and indications**
   a) Surgical anesthesia or postoperative analgesia of the upper arm, elbow, forearm, or hand.
   b) If this block is to be used for surgery extending to the shoulder, a superficial cervical plexus block and/or suprascapular nerve block may be needed to anesthetize the skin overlying the shoulder.
2) **Risks and contraindications**
   a) **Absolute contraindications**
      i) Local infection at the site of planned needle insertion
      ii) Pneumonectomy or hemidiaphragmatic paresis on the contralateral side
         (1) Risk of ipsilateral phrenic nerve block is around 50%.[8]
   b) **Relative contraindications**
      i) Comorbid pulmonary disease such as emphysema
         (1) Risk of phrenic block
         (2) Risk of pneumothorax
      ii) Coagulopathy
      iii) Preexisting neuropathy
   c) **Risks and complications**
      i) General risks
         (1) Nerve injury
         (2) Localized bleeding
         (3) Infection
         (4) Intravascular injection with local anesthetic toxicity
      ii) Site-specific risks and potential complications
         (1) The most dreaded complication of the supraclavicular block is pneumothorax with a prevalence of 0.6%-6.1%.[1,3] This quoted risk is higher than with other brachial plexus blocks but is most likely technique dependent.
            (a) The classic vertical landmark approach accounts for the upper limit of the range, followed by the plumb-bob approach.[9,10]
            (b) Ultrasound-guided techniques are at the lower limit of the risk range.[2,3]
            (c) Clinical presentation of pneumothorax may be delayed for many hours, making this technique less appealing for outpatient surgery.[1]
         (2) The proximity of the nerves to the subclavian artery introduces the risk of intravascular injection.

(3) Phrenic nerve block
(a) Less common following supraclavicular block (50%) than traditional interscalene block (100%)[8,11]
(b) Ultrasound guidance with lower injectate volume may further reduce the risk of inadvertent phrenic nerve block.[12]
(4) Horner syndrome (ptosis, miosis, and anhidrosis)
iii) Pros and cons of supraclavicular block are summarized in Table 174-1.



Supraclavicular block should not be performed in a patient with one functional lung due to the risk of ipsilateral hemidiaphragmatic paresis or possible pneumothorax.



Pneumothorax is a known risk of supraclavicular block; however, the risk has been reduced with the use of ultrasound guidance.

3) **Equipment**
a) Common equipment for all peripheral nerve block techniques include sterile skin cleansing solution, sterile gloves, a marking pen, and two 20-mL syringes of local anesthetic solution with extension tubing.
b) A nerve stimulator technique requires an electrical nerve stimulator with a compatible 1- to 2-in insulated needle and a grounding electrode.
c) Ultrasound-guided regional anesthesia requires portable ultrasound equipment and a high-frequency linear (preferred) or small curvilinear transducer for supraclavicular block.

4) **Technique**
a) **Monitoring**
i) Peripheral nerve blocks should be placed in a location where the patient's noninvasive blood pressure, EKG, oxygen saturation, and end-tidal CO_2 may be monitored.
ii) Supplemental O_2 should be provided via a face mask or nasal cannulae, especially when IV sedation is administered.
b) **Positioning and preparation**
i) Position the patient supine with back slightly elevated.
ii) The head should be turned away from the side to be blocked and the arm placed at the side.
iii) Apply monitors and supplemental O_2.
iv) IV sedation may be administered, but the patient must be able to follow commands and volunteer sensory information.
v) The anesthesiologist should stand next to the patient's shoulder on the side to be blocked.
c) **Surface anatomy**
i) Important surface landmarks include the sternocleidomastoid (SCM) and clavicle (Cognitive Aid, Chapter 175: Panel A).
ii) The SCM may be identified by asking the patient to turn the head away from the affected side and flex the neck.

TABLE 174-1 Supraclavicular Brachial Plexus Block Pros and Cons

Pros	Cons
• Reliable anesthesia of the upper limb • Block is performed at the level of distal trunks/divisions where nerves are bundled, so individual nerves do not need to be targeted • Arm can be placed in any position, unlike the axillary block, which requires the arm to be externally rotated and abducted	• Potential for ipsilateral hemidiaphragm paralysis, although risk is less than with interscalene block • Risk of pneumothorax • Potential for Horner syndrome (oculosympathetic palsy)

iii) Palpating the interscalene groove and subclavian artery pulsation posterior to the SCM is also helpful.

d) **Stimulating supraclavicular block (plumb-bob technique)**

i) After sterile skin preparation and placement of a local anesthetic skin wheal, insert a needle 1-2 in above the clavicle lateral to the insertion point of the SCM clavicular head and direct perpendicular to the bed to angled slightly cephalad (up to 45 degrees) with the patient supine.[10]

ii) Once the desired motor response is elicited (hand flexion or extension) and maintained at <0.5 mA, slowly inject 30-40 mL of local anesthetic in incremental volumes following careful aspiration.

iii) The needle should not be directed medially toward the cupola of the lung.

iv) If the subclavian artery is encountered, redirect the needle in a posterolateral direction.

e) **Ultrasound-guided supraclavicular block**

i) Supraclavicular block may be performed with real-time ultrasound guidance with or without nerve stimulation.[2,4]

ii) With a linear ultrasound transducer positioned cephalad and medial to the midpoint of the clavicle and oriented vertically (Cognitive Aid, Chapter 175: Panel B), the brachial plexus is visualized in short axis posterolateral to the subclavian artery (Cognitive Aid, Chapter 175: Panel C).

iii) After sterile skin preparation, place a local anesthetic skin wheal lateral to the ultrasound transducer.

iv) Insert the block needle through the skin wheal and direct anteromedially toward the subclavian artery.

v) To ensure blockade of the C8 and T1 division for complete upper extremity anesthesia, the needle tip should be directed into the "corner pocket" between the posterolateral portion of the subclavian artery and the first rib.[13]

vi) The total volume of local anesthetic can be injected incrementally after negative aspiration for blood, until injectate is visualized surrounding the entire plexus.

vii) For more rapid onset of a surgical block, the local anesthetic can be divided between deep, middle, and superficial areas of the plexus.[5] If satellite clusters are seen on ultrasound imaging, these can be individually targeted.[7]



The use of ultrasound allows for targeted injections resulting in faster onset for a surgical quality block.

5) **Evaluation of the block**

a) The quality of nerve blockade must be evaluated prior to surgical incision, even if intraoperative general anesthesia is planned.

b) **Motor testing**

i) Strength of elbow flexion against resistance tests the biceps muscle innervated by the musculocutaneous nerve.

ii) Strength of elbow extension tests the triceps muscle innervated by the radial nerve.

iii) Strength of shoulder abduction tests the deltoid muscle innervated by the axillary nerve.

c) **Sensory testing**

i) Pinching the base of the index finger, small finger, and medial aspect of the forearm tests sensation in the distribution of the median, ulnar, and medial antebrachial cutaneous nerves, respectively.

ii) Pinching over the medial upper arm and the elbow evaluates the block of the medial brachial cutaneous nerve.



All terminal branches of the brachial plexus should be evaluated prior to surgical incision, even if a general anesthetic is planned. Supplementation with additional local anesthetic can avoid unanticipated problems.

6) **Postoperative management**
   a) All patients who undergo brachial plexus blockade should be discharged with a supportive sling.
   b) For outpatient procedures, respiratory status should be assessed prior to discharge, but a routine chest radiograph for pneumothorax or hemidiaphragmatic paresis is not recommended in asymptomatic patients.

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175 US-Guided Supraclavicular Block

Vanessa J. Loland • Larry F. Chu • Ava Alamdari

Patient Position: Position the patient supine with back slightly elevated. The head should be turned away from the side to be blocked and the arm placed at the side with the shoulder relaxed.

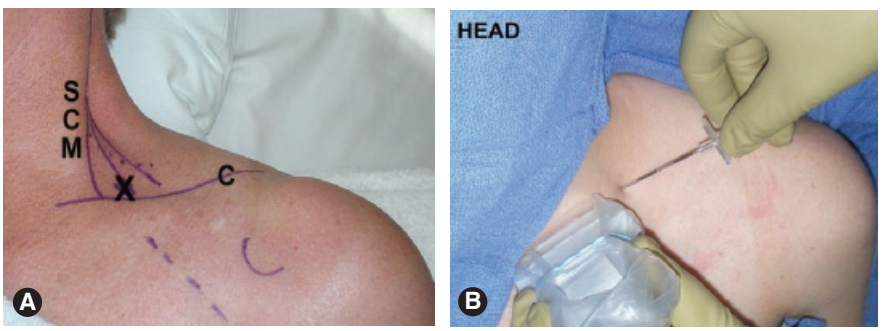
Needle Size: 22-17 gauge, 70- to 100-mm block needle

Volume: 20-40 mL local anesthetic

Anatomic Landmarks

Ⓐ Important surface landmarks include the sternocleidomastoid (SCM) and clavicle.

The SCM may be identified by asking the patient to turn the head away from the affected side and flex the neck. Palpating the interscalene groove and subclavian artery pulsation posterior to the SCM is also helpful.

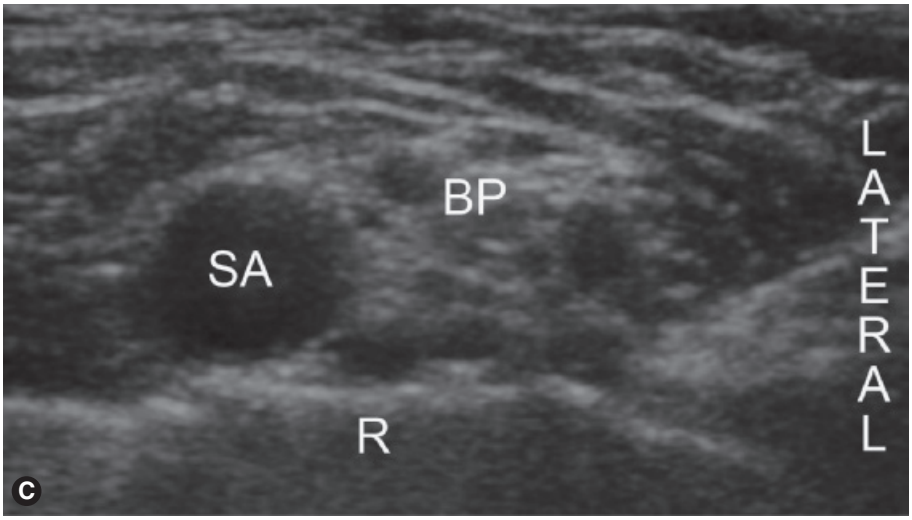


A. Surface anatomy relevant to the supraclavicular block. The "X" marks the suggested site for needle insertion when performing the plumb-bob technique with nerve stimulation. SCM, sternocleidomastoid muscle; C, clavicle. **B.** A high-frequency linear transducer placed perpendicular to skin, medial to clavicle. The block needle is inserted lateral to the transducer and directed anteromedially for in-plane needle guidance.

Approach and Technique

Ⓑ Position a high-frequency linear ultrasound transducer cephalad and medial to the midpoint of the clavicle and oriented vertically.

Ⓒ The brachial plexus may be visualized in short axis posterolateral to the subclavian artery. After sterile skin preparation, place a local anesthetic skin wheal lateral to the ultrasound transducer. Insert the block needle through the skin wheal and direct anteromedially toward the subclavian artery.



C. Short-axis image of the brachial plexus during supraclavicular block performance. SA, subclavian artery; R, first rib periosteum; BP, brachial plexus.

To ensure blockade of the C8 and T1 division for complete upper extremity anesthesia, the needle tip should be directed into the "corner pocket" between the posterolateral portion of the subclavian artery and the first rib.

The total volume of local anesthetic can be injected incrementally after negative aspiration for blood, until injectate is visualized surrounding the entire plexus.

For more rapid block onset, the local anesthetic can be divided between deep, middle, and superficial areas of the plexus, and individual satellite clusters seen on ultrasound imaging can be individually targeted.

Evaluation of Block

Motor Testing

Strength of elbow flexion against resistance tests the biceps muscle innervated by the musculocutaneous nerve. Similarly, strength of elbow extension tests the triceps muscle innervated by the radial nerve. Strength of shoulder abduction tests the deltoid muscle innervated by the axillary nerve.

Sensory Testing

Pinching the base of the index finger, small finger, and medial aspect of the forearm assesses anesthesia in the distribution of the median, ulnar, and medial antebrachial cutaneous nerves, respectively. Pinching over the medial upper arm and elbow evaluates the block of the medial brachial cutaneous nerve.

176 Infraclavicular Block

Vanessa J. Loland • Matthew W. Lawrence

The infraclavicular block may be performed using a variety of techniques to anesthetize the cords of the brachial plexus. This site is often chosen for patients requiring prolonged brachial plexus analgesia via continuous peripheral nerve catheters. An approach to this block using the coracoid process as a major landmark may be performed with the arm abducted or at the side, which may be preferable in patients unable to abduct the arm.[1,2] Nerve stimulator or ultrasound guidance is recommended for localization of the brachial plexus at this level.

1) **Benefits and indications**
   a) Surgical anesthesia or postoperative analgesia for procedures on the elbow, forearm, wrist, or hand.
   b) Infraclavicular block is functionally similar to supraclavicular block for surgery of the arm distal to the shoulder but carries a lower risk of phrenic block and Horner syndrome.

READ MORE
Overview of peripheral nerve blocks, Chapter 44

2) **Risks and contraindications**
   a) **Absolute contraindications**
      i) Local infection at the site of planned needle insertion
   b) **Relative contraindications**
      i) Coagulopathy
      ii) Preexisting nerve injury
      iii) Emphysema or other severe pulmonary disease, as there is a small risk of pneumothorax
   c) **Risks and complications (Table 176-1)**
      i) General risks
         (1) Nerve injury
         (2) Localized bleeding
         (3) Infection
         (4) Intravascular injection with local anesthetic toxicity
      ii) Site-specific risks and potential complications
         (1) Pneumothorax
            (a) Inadvertent pleural puncture with the coracoid approach is less likely when using proper technique.
            (b) MRI evaluation of the classic technique of Raj demonstrates imprecision and greater potential for pneumothorax.[3]
         (2) Hemothorax
         (3) Chylothorax (for a left-sided block)
      iii) Pros and cons of infraclavicular block are summarized in Table 176-1.



The medial, lateral, and posterior cords of the brachial plexus surround the axillary artery as it passes caudad to the coracoid process.

3) **Equipment**
   a) Common equipment for all peripheral nerve block techniques include sterile skin cleansing solution, sterile gloves, a marking pen, and two 20-mL syringes of local anesthetic solution with extension tubing.

TABLE 176-1 Infraclavicular Brachial Plexus Block Pros and Cons

Pros	Cons
• Provides anesthesia and analgesia for surgery of the upper limb below the shoulder • Low incidence of hemidiaphragmatic paresis compared with interscalene and supraclavicular approaches[10] • Favorable location for catheter placement due to stabilization from chest wall musculature	• Steep angle of approach makes needle visualization challenging • Noncompressible location requires careful consideration of coagulation status

b) A nerve stimulator technique requires an electrical nerve stimulator with a compatible insulated needle and a grounding electrode.

c) Ultrasound-guided regional anesthesia requires portable ultrasound equipment and a small low-frequency curvilinear transducer (preferred) or a high-frequency linear transducer for infraclavicular block.

4) **Technique**

a) **Monitoring**

i) Peripheral nerve blocks should be placed in a location where the patient's noninvasive blood pressure, EKG, oxygen saturation, and end-tidal CO_2 may be monitored.

ii) Supplemental O_2 should be provided via a face mask or nasal cannulae, especially when IV sedation is administered.

b) **Positioning and preparation**

i) Position the patient supine with the head turned away from the side to be blocked and the arm abducted at 90 degrees (preferred) or at the patient's side.

ii) Apply monitors and supplemental O_2.

iii) IV sedation may be administered, but the patient must be able to follow commands and volunteer sensory information.

iv) The anesthesiologist should stand next to the patient's shoulder on the side to be blocked.

c) **Surface anatomy**

i) The coracoid process is the most anterior portion of the scapula and can be palpated at the shoulder between the acromioclavicular joint and the deltopectoral groove.

ii) If the coracoid process has been correctly identified, forward elevation of the arm should rotate the scapula and displace the coracoid process superiorly, making it not palpable.

iii) After identification of this landmark, mark a point 2 cm medial and 2 cm caudad to the most anterior tip of the coracoid process (Cognitive Aid, Chapter 177: Panel A).

d) **Stimulating infraclavicular block**

i) After sterile skin preparation and injection of a local anesthetic skin wheal, insert a 2- to 4-in insulated needle using a plumb-bob approach (perpendicular to the floor), until a motor response of the wrist or hand is elicited (Fig. 176-1).

ii) The average depth from skin to brachial plexus is 4.24 ± 1.49 cm in men and 4.01 ± 1.29 cm in women.[2] The brachial plexus and blood vessels are located between the pectoralis minor anteriorly and the subscapularis muscle of the rotator cuff posteriorly.

iii) If the brachial plexus is not encountered initially, redirect cephalad-caudad within the same parasagittal plane and avoid deviation of the needle medially toward the chest wall.

FIGURE 176-1 Stimulating Infraclavicular Block Technique Employing the Coracoid Approach



A 4-in insulated needle is inserted "plumb-bob" at a site 2 cm medial and 2 cm caudad to the coracoid process.

iv) Once a motor evoked response from the desired nerve(s) is elicited and maintained at <0.5 mA of electrical current, slowly inject 30-40 mL of local anesthetic in incremental volumes following careful aspiration.

v) A multiple-injection technique (depositing local anesthetic in divided doses at two or more sites following elicitation of distinct motor responses) has been shown to increase the efficacy of this technique.[4-6]

e) **Ultrasound-guided infraclavicular block**

i) With a small curvilinear (preferred) or linear transducer positioned medial to the coracoid process and oriented parasagittally (Cognitive Aid, Chapter 177: Panel B), the brachial plexus cords can be visualized in short axis surrounding the axillary artery (Cognitive Aid, Chapter 177: Panel C).

ii) After sterile skin preparation, place a local anesthetic skin wheal cephalad to the transducer.

iii) Insert the block needle through the skin wheal and advance it caudally toward the axillary artery.



For an ultrasound-guided approach, arm abduction improves visualization and moves the clavicle away from the needle path.



Avoid deviation of the block needle medially toward the chest wall.

iv) Following careful aspiration for blood, local anesthetic can be injected around each individual cord[7] or incrementally as a single injection posterior to the axillary artery[8] until injectate spread surrounding all three cords is visually confirmed.

5) **Evaluation of the block**
   a) The quality of nerve blockade must be evaluated prior to surgical incision even if intraoperative general anesthesia is planned.
   b) **Motor testing**
      i) Strength of elbow flexion against resistance tests the biceps muscle innervated by the *musculocutaneous* nerve.
      ii) Strength of elbow extension tests the triceps muscle innervated by the *radial* nerve.
      iii) Strength of shoulder abduction tests the deltoid muscle innervated by the *axillary* nerve.
   c) **Sensory testing**
      i) Pinching the base of the index finger, small finger, and medial aspect of the forearm tests sensation in the distribution of the *median*, *ulnar*, and *medial antebrachial cutaneous* nerves, respectively.
      ii) Pinching over the medial upper arm and the elbow evaluates the block of the *medial brachial cutaneous* nerve.

6) **Postoperative management**
   a) All patients who undergo brachial plexus blockade should be discharged with a supportive sling.
   b) Routine chest radiograph for pneumothorax is not recommended in asymptomatic patients.



Consider a pregnancy test for all female patients of childbearing age.



Pressure applied to the ultrasound transducer can occlude small venous structures, masking intravascular needle placement.[9]

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177 US-Guided Infraclavicular Block

Vanessa J. Loland • Larry F. Chu

Patient Position: Position the patient supine with the head turned away from the side to be blocked and the arm abducted at 90 degrees (preferred) or at the patient's side.

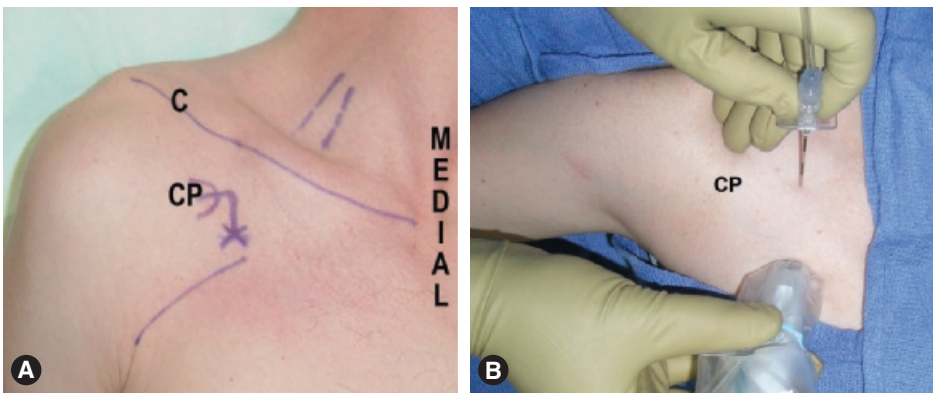
Needle Size: 22-17 gauge, 70- to 100-mm block needle

Volume: 20-40 mL local anesthetic

Anatomic Landmarks

The coracoid process is the most anterior portion of the scapula and can be palpated at the shoulder between the acromioclavicular joint and the deltopectoral groove. If the coracoid process has been correctly identified, forward elevation of the arm should rotate the scapula and displace the coracoid process superiorly, making it not palpable.

Ⓐ After identification of this landmark, mark the point 2 cm medial and 2 cm caudad to the most anterior tip of the coracoid process.

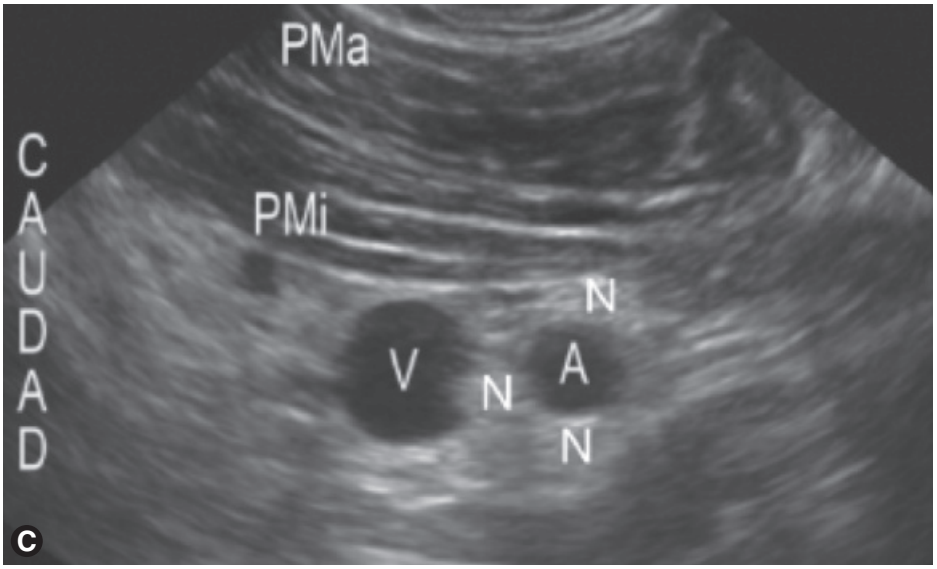


A. View of right shoulder demonstrating surface anatomy relevant to the infraclavicular block. CP, coracoid process of the scapula; C, clavicle. **B.** A small curvilinear transducer is placed medial and caudad to the coracoid process with the arm abducted 90 degrees to optimize the cross-sectional imaging of the neurovascular bundle. The block needle is inserted cephalad to the transducer and directed caudad for in-plane needle guidance.

Approach and Technique

Ⓑ With a small curvilinear (preferred) or linear transducer positioned medial and caudad to the coracoid process and oriented vertically, the brachial plexus is visualized in short axis.

Ⓒ The brachial plexus cords may be visualized in short-axis surrounding the axillary artery. After sterile skin preparation, place a local anesthetic skin wheal cephalad to the ultrasound transducer. Insert the block needle through the skin wheal and direct caudad toward the axillary artery. Inject local anesthetic around each individual cord or incrementally as a single injection posterior to the axillary artery after negative aspiration for blood, until the injectate spread surrounding all three cords is visually confirmed.



C. Short-axis image of the brachial plexus below the clavicle. PMa, pectoralis major muscle; PMi, pectoralis minor muscle; V, axillary vein; A, axillary artery; N, nerve (cords of the brachial plexus).

Evaluation of Block

Motor Testing

Strength of elbow flexion against resistance tests the biceps muscle innervated by the musculocutaneous nerve. Similarly, strength of elbow extension tests the triceps muscle innervated by the radial nerve. Strength of shoulder abduction tests the deltoid muscle innervated by the axillary nerve.

Sensory Testing

Pinching the base of the index finger, small finger, and medial aspect of the forearm assesses anesthesia in the distribution of the median, ulnar, and medial antebrachial cutaneous nerves, respectively. Pinching over the medial upper arm and elbow evaluates the block of the medial brachial cutaneous nerve.

178 Axillary Block

Vanessa J. Loland • Ava Alamdari

The axillary block is performed at the level of the terminal branches of the brachial plexus. The three major nerves that remain in close proximity to the axillary artery at this level are the median, radial, and ulnar nerves. Evaluation of the perineural sheath with magnetic resonance imaging has demonstrated incomplete spread of local anesthetic solution following large-volume single injection, which results in unsatisfactory anesthesia.[1] Therefore, a multiple compartment approach to the axillary block is recommended.[2] Ultrasound guidance for axillary block may improve onset time and initial success rates.[3,4]

1) **Benefits and indications**
   a) Surgical anesthesia or postoperative analgesia of the forearm, wrist, or hand
   b) Complete cutaneous anesthesia of the upper arm requires additional local anesthetic infiltration; however, the need for supplemental infiltration for tourniquet analgesia is controversial.[5]
      i) Medial brachial cutaneous nerve
      ii) Intercostobrachial nerve
2) **Risks and contraindications**
   a) **Absolute contraindications**
      i) Inability to abduct the arm at the shoulder
      ii) Local infection at the site of planned needle insertion
   b) **Relative contraindications**
      i) Preexisting nerve injury
      ii) Coagulopathy (although manual compression and hemostasis may be readily achieved in the event of vascular puncture)
   c) **Risks and complications**
      i) General risks
         (1) Nerve injury
         (2) Localized bleeding
         (3) Infection
         (4) Intravascular injection with local anesthetic toxicity
      ii) Site-specific risks and potential complications
         (1) Due to the proximity of the nerves to the axillary vessels, there is a risk of intravascular injection or hematoma from vascular puncture.
            (a) The risk of intravascular injection can be reduced by a multiple compartment technique, since smaller volumes of local anesthetic are deposited at multiple sites rather than a large volume injected at one site.
            (b) Hematoma formation can lead to postoperative nerve dysfunction.[6]
         (2) Since the nerves are superficial in this location, nerve impalement and intraneural injection are possible.[7]
      iii) Pros and cons of axillary block are summarized in **Table 178-1**.

TABLE 178-1 Axillary Brachial Plexus Block Pros and Cons

Pros	Cons
• Does not cause a phrenic nerve palsy and so can be used in patients with comorbid respiratory disease • The site of injection is easily compressible in the event of vascular puncture	• Does not provide anesthesia of the upper arm • Terminal branches are targeted individually, increasing the likelihood of incomplete analgesia • Requires the patient to externally rotate and abduct the arm, which may be challenging in the setting of shoulder immobility, trauma, or for patients with bulky casts • Suboptimal site for continuous peripheral nerve block[8]

3) **Equipment**
   a) Common equipment for all peripheral nerve block techniques include sterile skin cleansing solution, sterile gloves, a marking pen, and two 20-mL syringes of local anesthetic solution with extension tubing.
   b) Perivascular and transarterial techniques require a 1- to 2-in 22-gauge B-bevel needle, with or without extension tubing attached to syringes of local anesthetic solution.
   c) A nerve stimulator technique requires an electrical nerve stimulator with a compatible 1- to 2-in insulated needle and a grounding electrode.
   d) Ultrasound-guided regional anesthesia requires portable ultrasound equipment and a high-frequency linear transducer for axillary block.



There is no risk of pneumothorax with axillary block when proper technique is used.



Axillary block may be performed without the aid of nerve stimulation or ultrasound guidance to provide surgical anesthesia or postoperative analgesia for distal upper extremity surgery.

4) **Technique**
   a) **Monitoring**
      i) Peripheral nerve blocks should be placed in a location where the patient's noninvasive blood pressure, EKG, oxygen saturation, and end-tidal CO_2 may be monitored.
      ii) Supplemental O_2 should be provided via a face mask or nasal cannulae, especially when IV sedation is administered.
   b) **Positioning and preparation**
      i) Position the patient supine with his/her head turned away from the side to be blocked and the affected arm abducted 90 degrees at the shoulder and flexed 90 degrees at the elbow.
      ii) Extreme abduction of the shoulder may obscure the axillary pulse.
      iii) Apply monitors and supplemental O_2.
      iv) IV sedation may be administered, but the patient must be able to follow commands and volunteer sensory information.
      v) The anesthesiologist should stand or sit at the patient's side facing the patient's axilla.
   c) Surface anatomy
      i) The axillary artery should be located below the coracobrachialis muscle with the arm abducted (Cognitive aid, Chapter 179: Panel A).
      ii) If the pulse is difficult to palpate, Doppler or ultrasound may aid identification.
      iii) The expected locations of the nerves are median nerve superficial and lateral to the artery; radial nerve posterior to the artery; ulnar nerve medial to the artery (Cognitive aid, Chapter 179: Panel B).
         (1) At this level, the musculocutaneous nerve is located further lateral and away from the axillary artery.
         (2) The precise position of each of these terminal branch nerves is highly variable.[5]
   d) Perivascular axillary block
      i) After sterile skin preparation and injection of a local anesthetic skin wheal over the axillary artery pulse, a uniform field block of local anesthetic is deposited around the axillary artery using a 1- to 2-in B-bevel needle and three-ring control syringes of local anesthetic solution (Fig. 178-1).

FIGURE 178-1 The Perivascular Axillary Block Technique Is Performed by Injecting Local Anesthetic in a Ring Around the Axillary Artery and Does Not Require Electrical Nerve Stimulation or Ultrasound Guidance.



(1) Imagining the artery as the center of a clock face, the four major nerve branches are typically located in each of the four quadrants, and local anesthetic should be injected in each of these locations.[9]
(2) Paresthesias may occur during the performance of this block and may help confirm the location of the nerves.
(3) Neural puncture and intraneural injection occur frequently when performing techniques that involve deliberate elicitation of paresthesias.[7]

ii) Perform needle insertion in a steady, controlled manner on each pass to avoid nerve impalement.

e) Transarterial axillary block
i) After sterile skin preparation and injection of a local anesthetic skin wheal over the axillary artery pulse, direct a 1- to 2-in B-bevel needle connected to extension tubing and syringes containing local anesthetic toward the axillary artery.
ii) Confirm deliberate arterial puncture by aspiration of blood and then advance the needle until it passes through the artery.
(1) After confirming negative aspiration, incrementally inject half the volume of local anesthetic at this location.
(2) Intermittent aspiration is crucial to ensure that the needle has not migrated back into the vessel.
(3) Withdraw the needle back through the artery, confirming by aspirating blood, to a position anterior to the artery.

(4) After confirming negative aspiration for blood in this location, incrementally inject the remaining local anesthetic volume.

f) Stimulating axillary block
   i) When using an electrical nerve stimulator, the multiple-injection technique (using a peripheral nerve stimulator to elicit distinct motor responses with subsequent divided local anesthetic injections) has been shown to provide superior anesthesia compared with single injection, with lower rates of supplementation.[10,11]
   ii) After sterile skin preparation, insert a 1- to 2-in insulated needle and direct around the axillary artery in a similar fashion to the perivascular technique while seeking motor responses of at least two terminal branch nerves (median, ulnar, or radial).
   iii) For each motor response elicited at <0.5 mA of stimulating current, inject 10-20 mL of local anesthetic incrementally after negative aspiration for blood.

g) Ultrasound-guided axillary block
   i) With a linear transducer positioned perpendicular to skin over the axillary artery (Cognitive Aid, Chapter 179: Panel C), the terminal nerves can be visualized at various locations around the axillary artery (Cognitive aid, Chapter 179: Panel D).
      (1) The musculocutaneous nerve is located lateral to the axillary artery between the fascia layers of the biceps brachii and the coracobrachialis muscles.
      (2) The radial nerve is normally posterior to the axillary artery and can be traced distally as it passes into the spiral groove of the humerus.
      (3) The median and ulnar nerves are typically anterior to the axillary vessels, sometimes in close proximity to each other, and may be difficult to differentiate.
   ii) After sterile skin preparation, place a local anesthetic skin wheal lateral to the ultrasound transducer.
   iii) Insert the block needle through the skin wheal and direct medially toward the axillary artery.
      (1) Concurrent use of electrical stimulation may aid in the identification of individual nerve branches.
      (2) Multiple needle insertions may be required to avoid vascular puncture.
   iv) Local anesthetic solution should be injected around each individual nerve to ensure a successful block.[4]



Nerve impalement occurs frequently when performing deliberate paresthesia-seeking axillary blocks.

5) **Evaluation of the block**
   a) The quality of nerve blockade must be evaluated prior to surgical incision, even if intraoperative general anesthesia is planned.
   b) **Motor testing**
      i) Strength of elbow flexion against resistance tests the biceps muscle innervated by the musculocutaneous nerve.
      ii) Strength of elbow extension tests the triceps muscle innervated by the radial nerve.
      iii) Strength of shoulder abduction tests the deltoid muscle innervated by the axillary nerve.
   c) Sensory testing
      i) Pinching the base of the index finger, small finger, and medial aspect of the forearm tests sensation in the distribution of the median, ulnar, and medial antebrachial cutaneous nerves, respectively.
      ii) Pinching over the medial upper arm and the elbow evaluates the block of the medial brachial cutaneous nerve.



The musculocutaneous nerve can be found outside the coracobrachialis muscle in as many as 1 in 5 patients and is occasionally found alongside the median nerve.[12]

6) **Postoperative management**
   a) All patients who undergo brachial plexus blockade should be discharged with a supportive sling.

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179 | US-Guided Axillary Block

Larry F. Chu • Vanessa J. Loland

Patient Position: Position the patient supine with the head turned away from the side to be blocked and the affected arm abducted 90 degrees at the shoulder and flexed 90 degrees at the elbow.

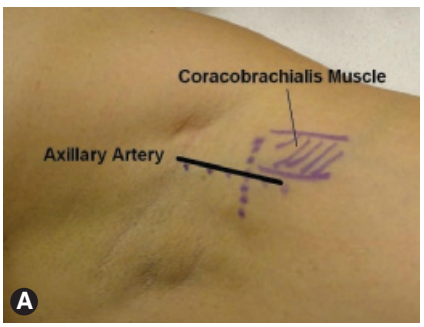
Needle Size: 22-17 gauge, 50- to 100-mm block needle

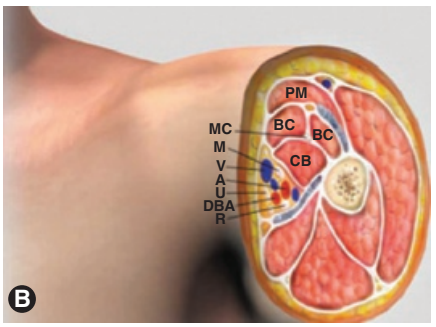
Volume: 20-40 mL local anesthetic

Anatomic Landmarks

Ⓐ The course of the axillary artery is palpated in the proximal axilla.

Ⓑ The expected locations of the nerves are median nerve superficial and lateral to the artery; radial nerve posterior to the artery; ulnar nerve medial to the artery.





A. Image of the left axilla demonstrating surface anatomy relevant to the axillary block: axillary artery and coracobrachialis muscle. **B.** Illustration of the cross-sectional anatomy through the axillary artery in the proximal axilla demonstrating the expected distribution of the terminal branch nerves in this location. PM, pectoralis major; MC, musculocutaneous; BC, biceps; CB, coracobrachialis; M, median; V, axillary vein; A, axillary artery; U, ulnar; DBA, deep brachial artery; R, radial. Reproduced with permission from Bigeleisen P, Orebaugh S. Ultrasound guided axillary block. In: Chelly JE, ed. *Peripheral Nerve Blocks: A Color Atlas*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009:286.

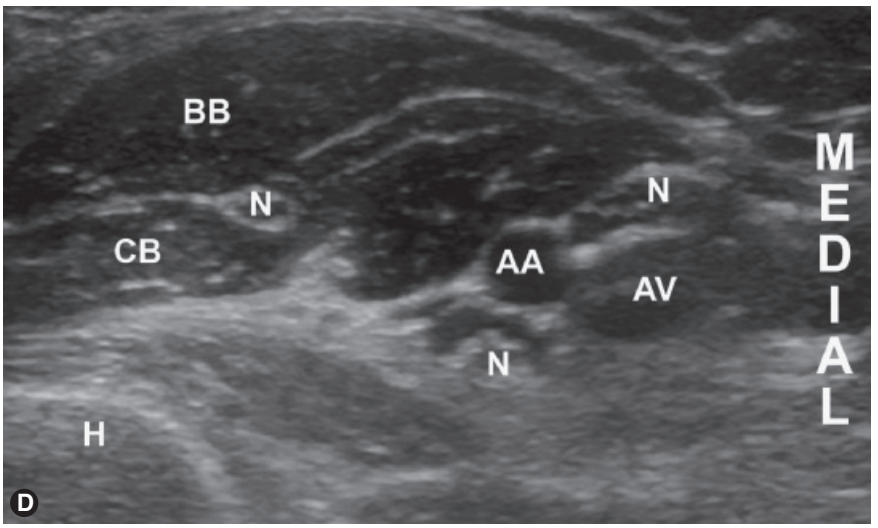
Approach and Technique

Ⓒ With a high-frequency linear transducer positioned perpendicular to skin over the axillary artery, the terminal nerves can be visualized.



C. A linear ultrasound transducer is placed perpendicular to skin in the proximal axilla to visualize the axillary vessels and terminal branch nerves in cross section.

Ⓓ The terminal nerves can be visualized at various locations around the axillary artery. After sterile skin preparation, place a local anesthetic skin wheal lateral to the ultrasound transducer. Insert the block needle through the skin wheal and direct medially toward the axillary artery. Inject 5-10 mL of local anesthetic solution around each individual nerve to ensure a successful block.



D. Short-axis image of the axillary vessels and terminal branch nerves in the proximal axilla. BB, biceps brachii muscle; CB, coracobrachialis muscle; H, humerus; AA, axillary artery; AV, axillary vein; N, nerve.

Evaluation of Block

Motor Testing

Strength of elbow flexion against resistance tests the biceps muscle innervated by the musculocutaneous nerve. Similarly, strength of elbow extension tests the triceps muscle innervated by the radial nerve. Strength of shoulder abduction tests the deltoid muscle innervated by the axillary nerve.

Sensory Testing

Pinching the base of the index finger, small finger, and medial aspect of the forearm assesses anesthesia in the distribution of the median, ulnar, and medial antebrachial cutaneous nerves, respectively. Pinching over the medial upper arm and elbow evaluates the block of the medial brachial cutaneous nerve.

180 Distal Upper Extremity Nerve Blocks

Amanda L. Downey

Selective peripheral nerve blocks of the terminal branches of the brachial plexus may be performed distal to the axilla.[1] The ulnar, radial, median, medial antebrachial cutaneous, and lateral antebrachial cutaneous nerves may be anesthetized at the elbow. The "wrist block" is composed of individual median, ulnar, and radial nerve blocks.[2] The sonoanatomy of these three distal branches in the forearm has been described,[3] and ultrasound-guided peripheral nerve blocks at this level are feasible.[4]

1) **Benefits and indications**
   a) **Indications**
      i) Individual nerve blockade of any or all of the terminal branches may be used to supplement brachial plexus anesthesia or may be used alone to provide surgical anesthesia for procedures of the distal forearm, wrist, or hand.[5]
      ii) The site of the tourniquet should be considered when planning the most appropriate regional anesthetic plan.
      iii) Although appropriate for postoperative pain management, the use of distal peripheral nerve blocks as a sole anesthetic is typically reserved for procedures of short duration limited to the hand or fingers when a tourniquet is not required or a wrist tourniquet will suffice. For surgical anesthesia, a block of the brachial plexus may be indicated.
2) **Risks and contraindications**
   a) **Absolute contraindications**
      i) Local infection at the site of planned needle insertion
   b) Relative contraindications
      i) Preexisting nerve injury
      ii) Coagulopathy (although nerve blocks distal to the elbow may have advantages over deeper blocks in the patient with bleeding tendencies since compression can be easily applied)
   c) **Risks and complications**
      i) General risks
         (1) Nerve injury
         (2) Localized bleeding
         (3) Infection at the site of needle puncture
         (4) Intravascular injection with local anesthetic toxicity
      ii) Site-specific risks and potential complications
         (1) Procedure-induced pain resulting from multiple needle insertions
         (2) Injection of local anesthetic solution into terminal arteries
            (a) Systemic toxicity is rare due to division of local anesthetic volume into multiple sites of injection.
            (b) Epinephrine-free local anesthetic solutions are commonly used for distal upper extremity blocks to avoid vasoconstriction of the terminal arteries that supply blood flow to the hands and digits.



Epinephrine-free local anesthetic solutions are used for distal upper extremity blocks to avoid vasoconstriction of the terminal arteries that supply blood flow to the hand.



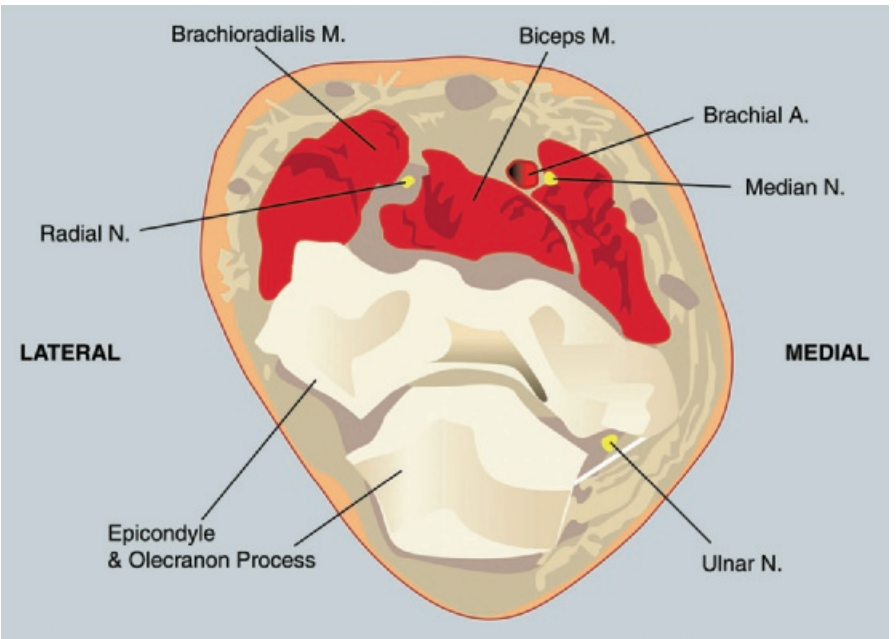
If sensory coverage of the lateral forearm is required for surgical anesthesia, the musculocutaneous nerve must also be blocked.



If surgical anesthesia is required for a long or extensive forearm or hand surgery, a more proximal brachial plexus block may be more appropriate.

3) **Equipment**
   a) Common equipment for all peripheral nerve block techniques include sterile skin cleansing solution, sterile gloves, a marking pen, and up to 30 mL of local anesthetic solution with extension tubing (may be divided into 10-mL three-ring control syringes).
   b) When performing elbow and wrist blocks with an infiltration technique, a 25-gauge needle with local anesthetic solution in three-ring control syringes should be used.
   c) A nerve stimulator technique requires an electrical nerve stimulator with a compatible 1-in insulated needle and a grounding electrode.
   d) Ultrasound-guided regional anesthesia requires portable ultrasound equipment and a high-frequency linear transducer for distal upper extremity peripheral nerve block.
4) **Technique**
   a) **Monitoring**
      i) Peripheral nerve blocks should be placed in a location where the patient's noninvasive blood pressure, EKG, oxygen saturation, and end-tidal CO_2 may be monitored.
      ii) Supplemental O_2 should be provided via a face mask or nasal cannulae, especially when IV sedation is administered.
   b) **Positioning and preparation**
      i) These blocks are most commonly performed with the patient supine.
      ii) For elbow blocks, the arm should be abducted at 90 degrees with the elbow in extension on a flat surface, except for the ulnar nerve block in which the elbow should be flexed 90 degrees over the patient's chest.
      iii) For wrist blocks, the arm should be abducted at 90 degrees and positioned initially on a flat surface with the wrist supinated, although suspending the wrist with the elbow at 90 degrees of flexion may facilitate blocks of the radial and ulnar nerves.
   c) Surface anatomy
      i) Elbow (Cognitive aid, Chapter 181: Panel A)
         (1) With the elbow extended, identify the intercondylar line at the elbow crease between the lateral and medial epicondyles of the humerus.
         (2) Palpate the brachial artery pulse medially and the tendon of the biceps brachii muscle laterally.
            (a) The median nerve lies medial to the brachial artery.
            (b) The radial nerve is located lateral to the biceps tendon and along the medial border of the brachioradialis muscle.
         (3) Elevate the shoulder 90 degrees and flex the elbow 90 degrees across the patient's chest to palpate the ulnar groove between the olecranon and the medial epicondyle of the humerus where the ulnar nerve is located.
      ii) Wrist (Cognitive aid, Chapter 181: Panel B)
         (1) With the forearm supinated, identify three tendons arranged lateral to medial on the volar surface of the wrist: flexor carpi radialis (FCR), palmaris longus (PL), and flexor carpi ulnaris (FCU).
         (2) The ulnar artery pulse is another helpful landmark since it is reliably located lateral to the ulnar nerve.
         (3) The radial artery runs medial to the radial nerve (Cognitive aid, Chapter 181: Panel B).
         (4) The PL tendon may be congenitally absent in some patients.
   d) **Landmark-based blocks**
      i) Elbow block technique
         (1) The median nerve block is performed by inserting the needle medial to the brachial artery at the elbow crease (**Fig. 180-1**).

FIGURE 180-1 Illustration Showing Cross-Sectional Anatomy at the Elbow. Individual blocks of the median, radial, and ulnar nerves may be performed at or near this level.



Reprinted with permission from Chelly JE. Terminal nerve blocks. C: blocks at the elbow. In: Chelly JE, ed. *Peripheral Nerve Blocks: A Color Atlas*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009:79.

(a) After sterile skin preparation and local anesthetic infiltration, insert the needle perpendicular to skin and inject 3-5 mL of local anesthetic solution after negative aspiration for blood.
(b) If using a nerve stimulator, inject local anesthetic when an appropriate motor response is elicited (wrist flexion and pronation with opposition of the thumb) and maintained at <0.5 mA.

(2) The radial nerve is located lateral to the biceps tendon and deep to the medial border of the brachioradialis muscle (Fig. 180-1).
(a) After sterile skin preparation and local anesthetic infiltration, insert a 1-in needle perpendicular to skin at this point and advance toward the lateral epicondyle. Inject 3-5 mL of local anesthetic after negative aspiration for blood.
(b) If using a nerve stimulator, inject local anesthetic after eliciting a radial nerve motor response (wrist and/or finger extension).

(3) The lateral antebrachial cutaneous nerve (termination of the musculocutaneous nerve providing innervation to the lateral forearm) may be blocked by infiltration of local anesthetic solution within the sheath of the biceps tendon at the elbow.

(4) The medial antebrachial cutaneous nerve may be anesthetized as a field block anterior to the medial epicondyle of the humerus at the elbow.

(5) With the shoulder elevated and the elbow flexed across the patient's chest, the ulnar nerve may be anesthetized posteriorly at a point proximal to the ulnar groove using a nerve stimulator or infiltration technique (Fig. 180-1).

ii) Wrist block technique

(1) Wrist blocks are reliably performed as infiltration techniques with 3-5 mL of plain local anesthetic solution per nerve.

(2) Special equipment is not required, although a nerve stimulator may be used as described for elbow blocks.

(3) The median nerve is typically anesthetized between the FCR and PL tendons or medial to the FCR tendon in patients who have congenital absence of the PL (Fig. 180-2).

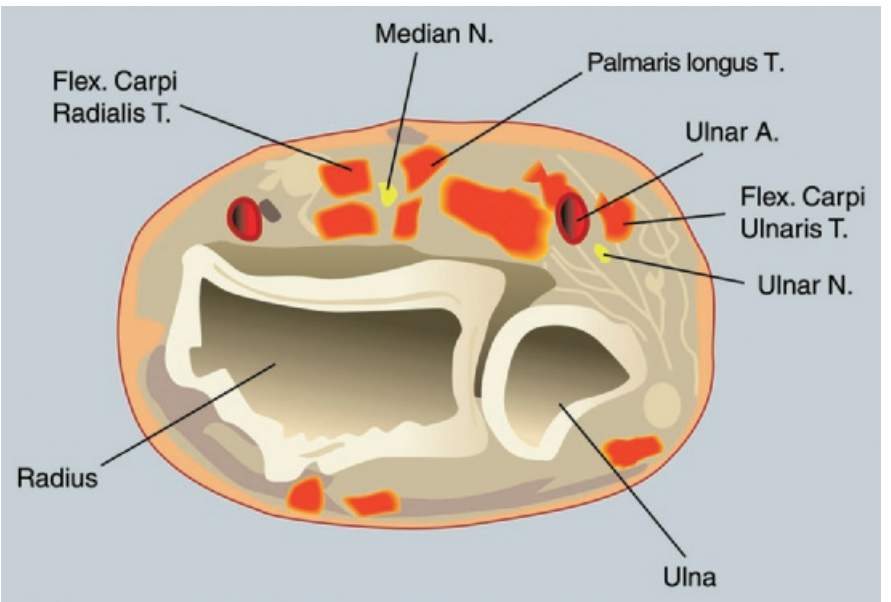
(4) The ulnar nerve is located medial to the ulnar artery between the FCU tendon and the ulna, and ulnar nerve block can be performed using either a volar or a lateral approach (Fig. 180-2).

(5) The superficial branch of the radial nerve may be anesthetized as a subcutaneous infiltration over the distal radius starting lateral to the radial artery along the dorsum of the wrist.

e) **Ultrasound-guided forearm block**

i) The advantages of an ultrasound-guided technique over nerve stimulation or field block include avoidance of vascular puncture, reduction in overall volume of local anesthetic, and minimizing movement of an injured limb.

FIGURE 180-2 Illustration Showing Cross-Sectional Anatomy at the Wrist. Individual blocks of the median, radial, and ulnar nerves may be performed at this level.



Reprinted with permission from Chelly JE. Terminal nerve blocks. D: blocks at the wrist. In: Chelly JE, ed. *Peripheral Nerve Blocks: A Color Atlas*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009:83.

ii) The terminal branches may also be anesthetized in the forearm using 3-5 mL of local anesthetic solution per nerve under ultrasound guidance.[3,4]

iii) Position the patient with the arm supinated, and place the ultrasound transducer perpendicular to skin in a transverse orientation to image the target nerves in short axis.

iv) The distal peripheral nerves are round or oval-shaped structures containing hypoechoic (neural tissue) and hyperechoic (connective tissue) elements.[3,6]

v) Scanning from distal at the wrist to the proximal forearm may help to distinguish nerve from tendon.

vi) Median nerve block

(1) The median nerve is easily visualized in the midforearm medial to the radial artery and between the flexor digitorum superficialis (FDS) and flexor digitorum profundus (FDP) muscles (Cognitive aid, Chapter 181: Panel C).

(2) After sterile skin preparation and local anesthetic infiltration, insert a 22-gauge B-bevel needle lateral to the transducer and direct it in-plane toward the median nerve (Cognitive aid, Chapter 181: Panel D).

(3) Note the position of the radial artery to prevent accidental arterial puncture.

vii) Ulnar nerve block

(1) The ulnar nerve can be identified in the midforearm deep to the FCU muscle and next to the ulnar artery (Cognitive aid, Chapter 181: Panel E).

(2) After sterile skin preparation and local anesthetic infiltration, insert a 22-gauge B-bevel needle lateral to the transducer and direct it in-plane medially toward the ulnar nerve (Cognitive aid Chapter 181: Panel F).

viii) Radial nerve block

(1) The radial nerve is best visualized immediately distal to the elbow crease and deep to the brachioradialis muscle (Cognitive aid, Chapter 181: Panel G).

(2) After sterile skin preparation and local anesthetic infiltration, insert a 22-gauge B-bevel needle lateral to the transducer and direct it in-plane medially toward the radial nerve (Cognitive aid, Chapter 181: Panel H).

5) **Evaluation of the block**

a) The quality of nerve blockade must be evaluated prior to surgical incision, even if intraoperative general anesthesia is planned.

b) Sensory testing

i) Pinching the base of the index finger, small finger, dorsum of the hand, medial aspect of the forearm, and lateral aspect of the forearm assesses anesthesia in the distribution of the median, ulnar, radial, medial antebrachial cutaneous, and lateral antebrachial cutaneous nerves, respectively.

c) Patients who receive wrist blocks will most likely not demonstrate weakness of finger flexion/extension since the responsible tendons and muscles originate more proximally in the forearm.



Avoid potential ulnar nerve impalement by inserting the block needle proximal to the ulnar groove in the upper arm and not directly into the groove.



Scanning from distal at the wrist to the proximal forearm may help to distinguish nerve from tendon.



Avoid out-of-plane technique and use a blunt tip needle (Tuohy tip or B-bevel) when performing ultrasound-guided blocks to minimize risk of nerve damage.



Wrist blocks do not result in significant motor block of the digits.



Distal forearm blocks avoid unnecessary motor/sensory block of the proximal muscles.

6) **Postoperative management**

a) All patients who undergo upper limb regional anesthesia should be discharged with a supportive sling and a protective wrist splint when indicated.

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181 US-Guided Distal Upper Extremity Nerve Blocks

Median, Ulnar, and Radial Nerve Blocks

Amanda L. Downey • Larry F. Chu

Patient Position: For elbow blocks, abduct the arm at the shoulder 90 degrees with the elbow in extension on a flat surface; for the ulnar nerve block, flex the elbow 90 degrees over the patient's chest. For wrist blocks, abduct the arm at 90 degrees and position on a flat surface with the wrist supinated.

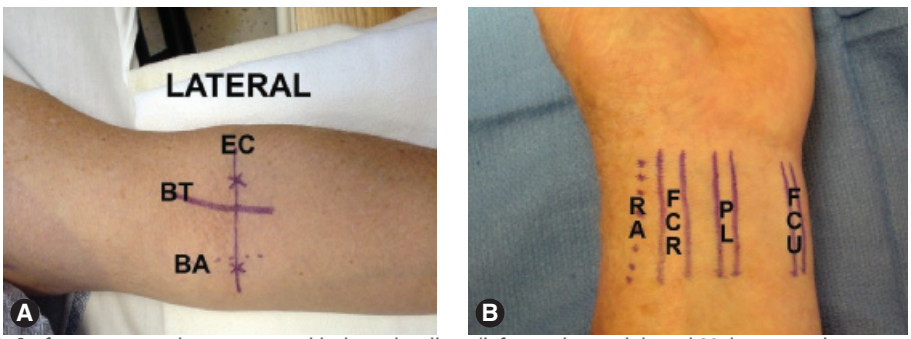
Needle Size: 22-gauge Tuohy tip or B-bevel block needle.

Volume: 5-10 mL local anesthetic per nerve.

Anatomic Landmarks

Ⓐ Elbow: Extend the elbow and identify the intercondylar line. Palpate the brachial artery pulse medially and the tendon of the biceps brachii muscle laterally. Elevate the shoulder 90 degrees and flex the elbow 90 degrees across the patient's chest to palpate the ulnar groove between the olecranon and the medial epicondyle of the humerus.

Ⓑ Wrist: Supinate the forearm and identify the flexor carpi radialis, palmaris longus, and flexor carpi ulnaris. The radial artery runs medial to the radial nerve.



A. Surface anatomy relevant to nerve blocks at the elbow (left arm shown abducted 90 degrees at the shoulder). EC, elbow crease; BT, biceps tendon; BA, brachial artery. **B.** Surface anatomy relevant to nerve blocks at the wrist (left hand and wrist shown). RA, radial artery; FCR, flexor carpi radialis tendon; PL, palmaris longus tendon; FCU, flexor carpi ulnaris tendon.

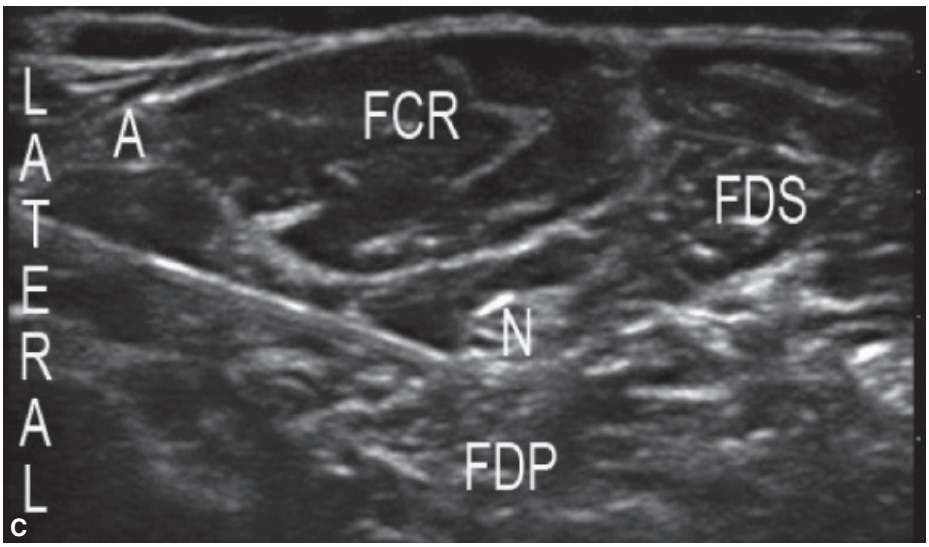
	Median	Ulnar	Radial
Origin	C5/C6/C7/C8/T1 Lateral and medial cords Lateral root from anterior divisions of superior and middle trunk Medial root from anterior division of inferior trunk	C8/T1 Medial cord from the anterior division of the inferior trunk	C5/C6/C7/C8/T1 Posterior cord from posterior divisions of all trunks
Course	Runs down arm with brachial artery, passes through antecubital fossa then divides into 3 terminal branches (anterior interosseous and deep and superficial/palmar cutaneous)	Passes behind medial epicondyle into forearm, then travels down anterior compartment with ulnar artery to enter palm of the hand	Winds around the spinal groove, enters the antecubital fossa in front of the lateral epicondyle of the humerus between the brachial and brachioradialis muscle. Divides into 2 terminal branches (superficial and deep/posterior interosseous)
Supplies	Sensory: thenar eminence, palmar aspect lateral 3½ fingers, distal dorsum of lateral 3½ fingers	Sensory: hypothenar eminence, palmar and dorsal aspect of medial 1½ fingers (pinky and ½ of ring finger)	Sensory: posterior forearm, proximal dorsal aspect of lateral 3½ fingers (spares the distal tips of the index, middle, and ½ of ring finger)
	Motor: anterior forearm compartment, wrist and finger flexors, pronator teres and quadratus, lateral 2 lumbricals, opponens pollicis, abductor pollicis brevis, flexor pollicis brevis	Motor: flexor carpi ulnaris and medial 2 parts of flexor digitorum profundus (lateral part is median), medial 2 lumbricals, adductor pollicis, muscles in hypothenar eminence, and interossei	Motor: wrist extensors, finger extensors, brachioradialis, supinator

Median Nerve Block

Approach and Technique

Supinate the arm and place the ultrasound transducer perpendicular to skin.

Ⓒ Visualize the median nerve in the midforearm medial to the radial artery and between flexor digitorum superficialis and flexor digitorum profundus muscles.



C. Short-axis ultrasound image of the midforearm demonstrating sonoanatomy relevant to the median nerve block. A, radial artery; FCR, flexor carpi radialis muscle; FDS, flexor digitorum superficialis muscle; FDP, flexor digitorum profundus muscle; N, median nerve.



D. Ultrasound-guided median nerve block technique using a high-frequency linear transducer and in-plane needle guidance.

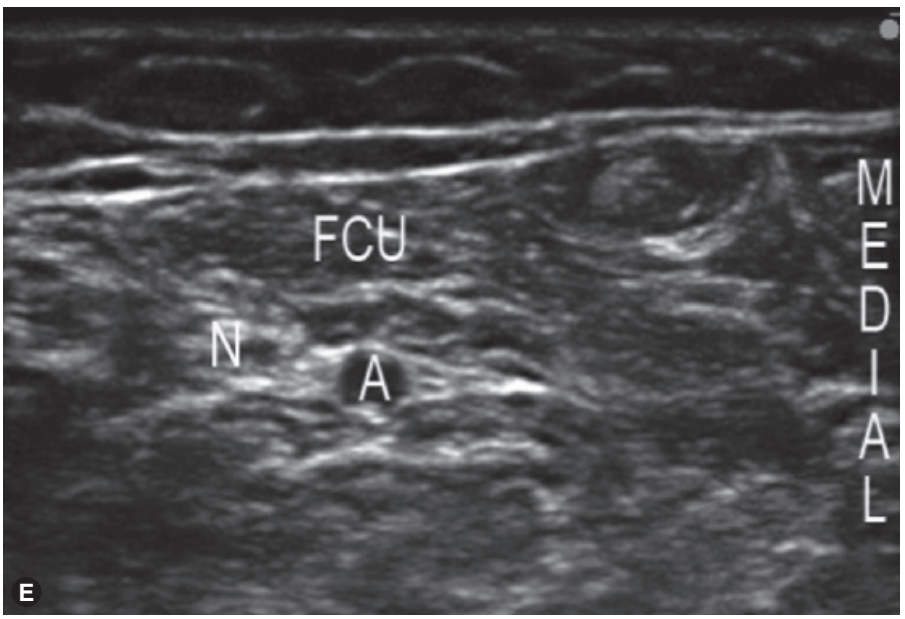
Ⓓ After sterile skin preparation and local anesthetic skin infiltration, insert the block needle lateral to the transducer and direct it medially toward the median nerve.

Ulnar Nerve Block

Approach and Technique

Supinate the arm and place the ultrasound transducer perpendicular to skin.

Ⓔ Identify the ulnar nerve in the midforearm deep to the flexor carpi ulnaris muscle and next to the ulnar artery.



E. Short-axis ultrasound image of the midforearm demonstrating sonoanatomy relevant to the ulnar nerve block. A, ulnar artery; FCU, flexor carpi ulnaris muscle; N, ulnar nerve.



F. Ultrasound-guided ulnar nerve block technique employing a high-frequency linear transducer and in-plane needle guidance.

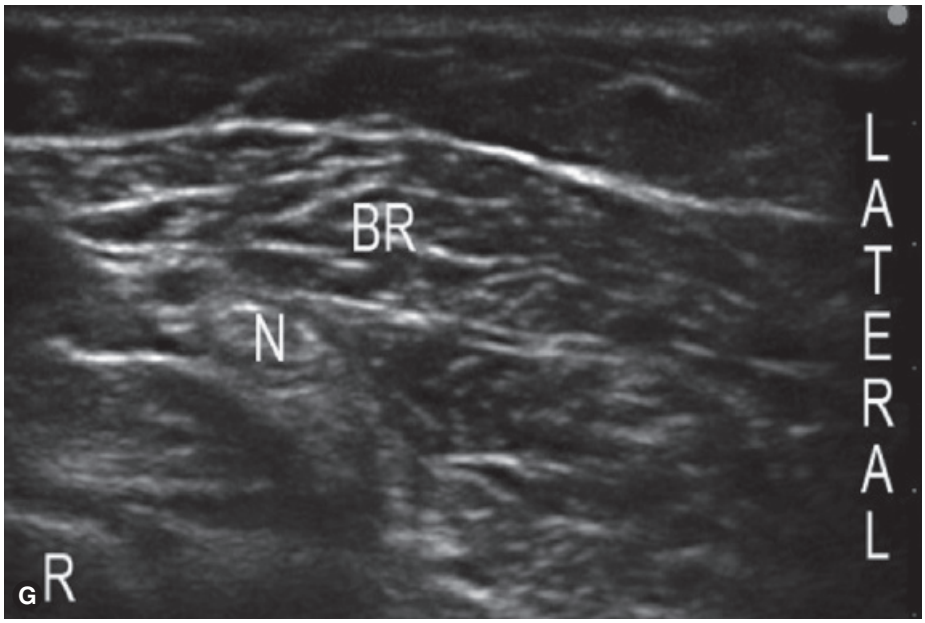
Ⓕ After sterile skin preparation and local anesthetic infiltration, insert the block needle lateral to the transducer and direct it medially toward the ulnar nerve.

Radial Nerve Block

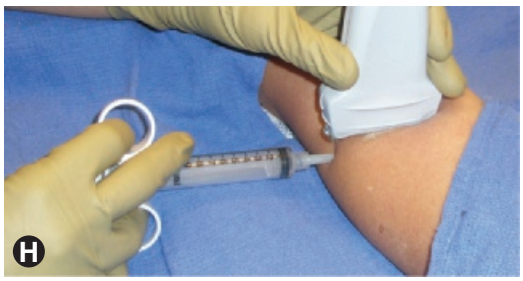
Approach and Technique

Supinate the arm and place the ultrasound transducer perpendicular to skin.

Ⓖ Visualize the radial nerve distal to the elbow crease and deep to the brachioradialis muscle.



G. Short-axis ultrasound image distal to the elbow demonstrating sonoanatomy relevant to the radial nerve block. BR, brachioradialis muscle; R, radius, N, radial nerve.



H. Ultrasound-guided radial nerve block technique employing a high-frequency linear transducer and in-plane needle guidance.

Ⓗ After sterile skin preparation and local anesthetic skin infiltration, insert the block needle lateral to the transducer and direct it medially toward the radial nerve.

Evaluation of Block

Pinch the base of the index finger, small finger, and dorsum of the hand to assess anesthesia in the distribution of median, ulnar, and radial nerves, respectively.

182 Part B: The Lower Extremity
Proximal Sciatic Nerve Block

Matthew T. Charous • Robert B. Maniker

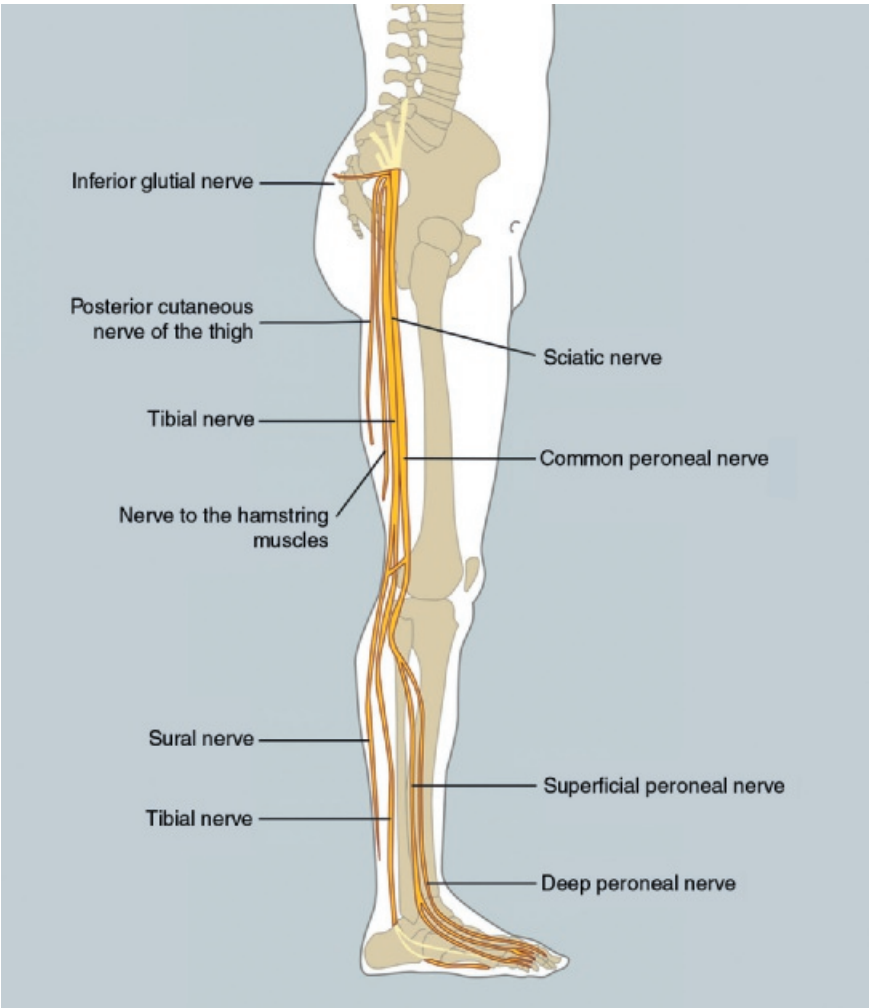
The sciatic nerve is formed by the ventral rami of nerve roots L4-S3. The roots exit the pelvis via the greater sciatic foramen and then converge under the piriformis muscle to form the sciatic nerve (Fig. 182-1). The sciatic nerve innervates the posterior aspect of the knee and the entire lower leg and foot except for the distribution of the saphenous nerve.[1] The classic transgluteal approach is a proximal sciatic nerve block that also consistently anesthetizes the posterior femoral cutaneous nerve that innervates the posterior thigh.[1] The subgluteal approach is performed distal to the gluteal crease where the sciatic nerve is more superficial and requires lower injectate volume.[2] Continuous sciatic nerve block catheters can also be readily placed using the subgluteal approach to prolong postoperative analgesia.[3]

1) **Benefits and indications**
   a) Surgical anesthesia or postoperative analgesia for knee and thigh procedures when combined with lumbar plexus blockade[4]
   b) Surgical anesthesia for procedures involving the lower leg, ankle, and foot
      i) Must be combined with a femoral or a saphenous nerve block for complete anesthesia or when the saphenous nerve distribution is involved
2) **Risks and contraindications**
   a) **Absolute contraindications**
      i) Local infection at the site of planned needle placement
   b) **Relative contraindications**
      i) Preexisting nerve injury
      ii) Coagulopathy
      iii) There is theoretical concern that a dense sciatic nerve block may mask pain produced by compartment syndrome in the lower extremity trauma patient.
         (1) Prospectively gathered data supporting this concern are lacking.[5]
         (2) A detailed discussion of the potential risks and benefits of regional anesthesia with the patient and trauma team is indicated before performing a peripheral or neuraxial block when compartment syndrome is a concern.
   c) **Risks and complications**
      i) General risks
         (1) Nerve injury
         (2) Localized bleeding
         (3) Infection at the site of needle puncture
         (4) Intravascular injection with local anesthetic toxicity
      ii) Site-specific risks and potential complications

READ MORE
Lumbar plexus block, Chapter 186; Femoral nerve and adductor canal blocks, Chapter 188

Patients with proximal tibia or tibial plateau injury should be considered high risk for postoperative compartment syndrome. A detailed discussion with the patient and trauma team should precede any regional anesthesia technique in these patients.

FIGURE 182-1 Illustration Demonstrating the Course of the Sciatic Nerve as It Exits the Greater Sciatic Foramen and Divides Into Its Terminal Branches



Reprinted with permission from Chelly JE. *Peripheral Nerve Blocks: A Color Atlas*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2004. Fig. 10-3, p. 79.

(1) Needle-induced nerve trauma
   (a) The sciatic nerve is widest at this location and may be at risk for needle-nerve contact.
(2) Hematoma formation
   (a) Inferior gluteal artery lies in close proximity to the sciatic nerve when using the transgluteal approach.
   (b) Patient procedure-related discomfort

(i) Transgluteal sciatic nerve block necessitates that the needle traverse a thick layer of muscle.
(ii) Careful titration of sedatives and analgesics helps to attenuate patients' discomfort during block performance.

3) **Equipment**
   a) Common equipment for all peripheral nerve block techniques include sterile skin cleansing solution, sterile gloves, a marking pen, and two 20-mL syringes of local anesthetic solution with extension tubing.
   b) A nerve stimulator technique requires an electrical nerve stimulator with a compatible 4-in insulated needle and a grounding electrode.
   c) Ultrasound-guided regional anesthesia requires portable ultrasound equipment and a large, low-frequency curvilinear transducer (preferred) or a high-frequency linear transducer for proximal sciatic block.
      i) The large, low-frequency (2-5 MHz) transducer is especially useful for obese or muscular patients during proximal sciatic nerve block.[6]
4) **Technique**
   a) **Monitoring**
      i) Peripheral nerve blocks should be placed in a location where the patient's noninvasive blood pressure, ECG, oxygen saturation, and end-tidal CO_2 may be monitored.
      ii) Supplemental O_2 should be provided via a face mask or nasal cannulae, especially when IV sedation is administered.
   b) **Positioning and preparation**
      i) For the classic transgluteal technique, the patient is placed in Sims position, a modified lateral decubitus position.
         (1) The nonoperative leg should be dependent and positioned straight.
         (2) The operative leg should be slightly flexed at the knee, crossing over the dependent leg.
      ii) For the subgluteal technique, the patient may be placed in the Sims position or prone with the knee slightly flexed.
      iii) Apply monitors and supplemental O_2.
      iv) IV sedation may be administered, but the patient must be able to follow commands and volunteer sensory information.
   c) **Surface anatomy**
      i) Transgluteal approach
         (1) Important landmarks for locating the sciatic nerve in this region include the greater trochanter (GT) of the femur and the posterior superior iliac spine (PSIS).
         (2) Draw a line between the GT and the PSIS, and another line (~5 cm in length) extending perpendicularly from the midpoint of the first line. The end of the perpendicular line marks the site of initial needle insertion.
         (3) Alternatively, a line can be drawn from the GT to the sacral hiatus with needle insertion at the midpoint of this line.
         (4) These two methods can be combined since they identify approximately the same point in most patients.
      ii) Subgluteal approach
         (1) Surface landmarks for the infragluteal-parabiceps approach include the lateral border of the biceps femoris and the lower border of the gluteus maximus muscle.
         (2) Caudad to the gluteal crease, the sciatic nerve lies along the lateral border of the long head of the biceps femoris as it crosses medially toward the ischial tuberosity (IT).

(3) When these landmarks are not palpable, alternative landmarks include the GT of the femur and the IT at the level of the inferior gluteal crease (Cognitive Aid: Panel A).
(4) Draw a line between the GT and the IT, and mark the midpoint of this line as the site for needle insertion.

d) **Stimulating classic transgluteal sciatic block**
   i) Using sterile skin preparation and technique, insert a 4-in insulated needle at the marked point perpendicular to skin in all planes and advance while seeking a sciatic nerve motor response.
   ii) If bone is contacted, this can represent either the IT or the lesser trochanter of the femur, and the needle should be redirected laterally or medially, respectively, and advanced another 1-2 cm.
   iii) After an acceptable evoked motor response is elicited (tibial preferred) at a stimulating current <0.5 mA, incrementally inject 20-40 mL of local anesthetic following careful aspiration.
   iv) The "double-injection" technique (identifying each branch of the sciatic nerve using nerve stimulation with separate injections of local anesthetic targeting the tibial and common peroneal contributions) results in faster onset and increased efficacy compared to the single-injection technique.[7]



For nerve stimulator–guided sciatic blocks, a plantar flexion and/or inversion evoked motor response yields the fastest onset time and the greatest likelihood of a complete block.

e) **Stimulating infragluteal-parabiceps (subgluteal) sciatic block**
   i) Using sterile skin preparation and technique, insert a 2- to 4-in insulated needle at the marked point at the intersection of the inferior border of the gluteus maximus and the lateral border of biceps femoris or at the midpoint of a line drawn between the GT and IT.
   ii) Introduce the needle at a 70- to 80-degree angle, aiming cephalad in a parasagittal plane.[8]
   iii) If bone is contacted, the femur is the most likely source, indicating that the plane of the needle is too far lateral and should be redirected medially.
   iv) If direct stimulation of the biceps femoris muscle occurs, this is an indication that the plane of the needle is too far medial and should be redirected more laterally.
   v) When performing this block as a single injection technique, injecting local anesthetic after evoking an inversion motor response (from the tibial component of the sciatic nerve) at <0.5 mA leads to the fastest onset and the greatest likelihood of a complete block.[9]



The posterior tibial nerve innervates the bony segments of the foot.



The sciatic nerve will appear flat or oval in the subgluteal region; if the nerve is difficult to identify, trace the course of the nerve cephalad from the popliteal fossa.

f) **Ultrasound-guided subgluteal sciatic block**
   i) With the patient in Sims position, place the ultrasound transducer perpendicular to skin between the IT and the GT of the femur, caudad to the gluteal crease, and oriented parallel to the crease (Cognitive Aid: Panel B).
   ii) The sciatic nerve lies deep to the gluteus maximus muscle between the IT medially and femur laterally (Cognitive Aid: Panel C).[6,10]
   iii) After sterile skin preparation, place a local anesthetic skin wheal lateral to the ultrasound transducer.
   iv) Insert the block needle through the skin wheal using in-plane guidance in a lateral-to-medial direction toward the sciatic nerve.



The ischial tuberosity and greater trochanter are easily visualized landmarks when performing an ultrasound-guided subgluteal sciatic nerve block.

v) Alternatively, the block needle may be inserted caudad to the center of the ultrasound transducer, aiming cephalad, and directed out-of-plane toward the sciatic nerve.

vi) A multiple injection technique with visual confirmation of circumferential local anesthetic spread results in a greater block success rate.[11]



The posterior cutaneous nerve of the thigh diverges from the sciatic nerve proximally and will not be reliably anesthetized with an infragluteal approach.

5) **Evaluation of the block**

a) The quality of nerve blockade must be evaluated prior to surgical incision, even if intraoperative general anesthesia is planned.

b) **Motor testing**

i) Strength of plantar flexion and dorsiflexion against resistance tests the *posterior tibial* and *common peroneal* nerves, respectively.

ii) Strength of knee flexion against resistance allows for assessment of hamstring motor blockade.



Proximal sciatic block may lead to weakness in knee flexion, which may contribute to falls.

c) **Sensory testing**

i) Decreased temperature and pinprick sensation over the lateral calf and the dorsal/plantar surfaces of the foot confirm appropriate sciatic nerve block.

6) **Postoperative management**

a) Outpatients who undergo sciatic nerve blockade should be discharged with a supportive ankle splint (due to foot drop) and weight-bearing restriction.

i) The patient is at risk for falling if ambulation is initiated prior to nerve block resolution.

b) Hospitalized patients with single-injection or continuous sciatic nerve blocks should have similar restrictions but may participate in physical therapy–directed activities (ie, continuous passive motion or ambulation with assistance).

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183 US-Guided Subgluteal Sciatic Nerve Block

Matthew T. Charous • Larry F. Chu

Patient Position: For subgluteal technique, the patient can be in Sims position or prone with the knee slightly flexed.

Needle Size: 22- to 17-gauge, 70- to 100-mm block needle

Volume: 20-40 mL local anesthetic

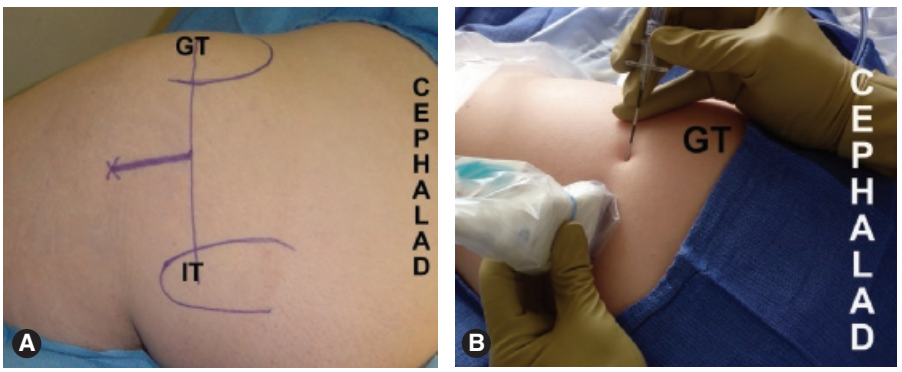
Anatomic Landmarks

Ⓐ Landmarks for the ultrasound-guided subgluteal sciatic nerve block include the greater trochanter (GT) laterally and ischial tuberosity (IT) medially. Caudad to the gluteal crease, the sciatic nerve lies along the lateral border of the long head of the biceps femoris and anterior to the gluteus maximus muscle.

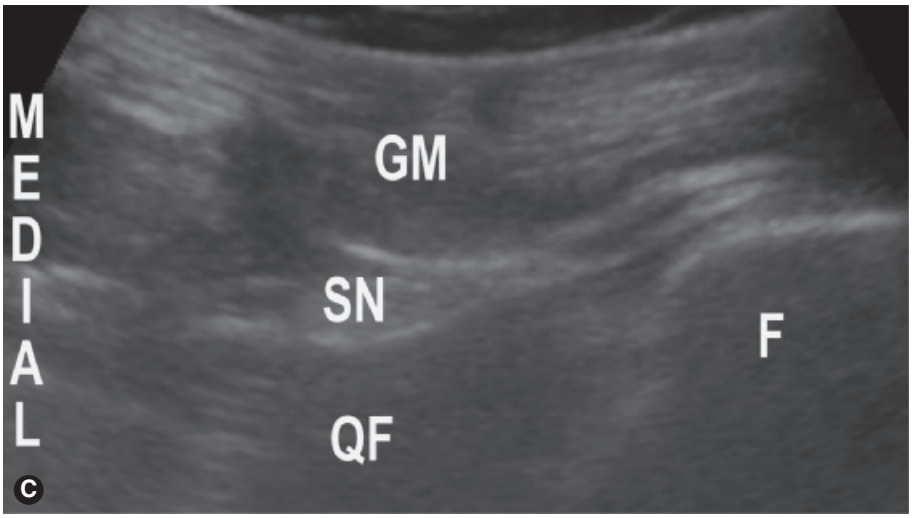
Approach and Technique

Ⓑ With the patient in Sims position, place the ultrasound transducer perpendicular to skin between the IT and the GT of the femur caudad to the gluteal crease and oriented parallel to the crease.

Ⓒ The sciatic nerve lies deep to the gluteus maximus muscle between the IT medially and the femur laterally. Place a local anesthetic skin wheal lateral to the transducer after sterile skin preparation. Insert the block needle through the skin wheal and advance in a lateral-to-medial direction toward the sciatic nerve using in-plane guidance.



A. Surface anatomic landmarks relevant to the classic subgluteal sciatic nerve block. GT, greater trochanter of the femur; IT, ischial tuberosity. **B.** Place the ultrasound transducer distal to the gluteal crease between the greater trochanter (GT) of the femur and the ischial tuberosity to image the sciatic nerve in cross section. Insert the block needle lateral to the transducer and direct medially for in-plane needle guidance (left hip shown).



C. Short-axis ultrasound image demonstrating the relevant cross-sectional anatomy for the subgluteal sciatic nerve block approach. GM, gluteus maximus muscle; QF, quadratus femoris muscle; F, femur; SN, sciatic nerve.

Evaluation of Block

Motor Testing

Test strength of plantar flexion and dorsiflexion of foot to test blockade of posterior tibial and common peroneal nerve. Asking the patient to flex the knee against resistance allows to assess the hamstring motor blockade.

Sensory Testing

Decreased temperature and pinprick sensation over the lateral calf and dorsal/plantar surfaces of the foot confirm the appropriate block.

184 Popliteal Sciatic Nerve Block

Cynthia Khoo • Sarah J. Madison

The popliteal fossa is bordered laterally by biceps femoris and medially by semimembranosus and semitendinosus.[1] Sciatic nerve block at the level of the popliteal fossa is a distal approach that preserves sensation in the posterior femoral cutaneous distribution and the motor innervation of the hamstring muscles, permitting the patient to flex the knee when crutch walking. Ultrasound-guided popliteal block with perineural catheter insertion is safe and effective, which may offer the advantages of faster placement and less patient discomfort compared to nerve stimulation techniques.[2]

1) **Benefits and indications**
   a) The sciatic nerve may be blocked in the popliteal fossa to provide surgical anesthesia and postoperative analgesia for procedures of the lower leg, ankle, and foot.
      i) A supplementary block of the femoral or saphenous nerves may be required to provide complete surgical anesthesia if the procedure will involve the medial aspect of the leg or ankle.
      ii) The site of the tourniquet should be considered when formulating the most appropriate regional anesthetic plan.
   b) A continuous peripheral nerve block catheter may be placed in the popliteal fossa to provide a continuous sciatic perineural local anesthetic infusion for prolonged postoperative analgesia.[3,4]

READ MORE
Overview of peripheral nerve blocks, Chapter 44; Femoral nerve and adductor canal blocks, Chapter 188; Ankle block, Chapter 190

2) **Risks and contraindications**
   a) **Absolute contraindications**
      i) Local infection at the site of planned needle insertion
   b) **Relative contraindications**
      i) Preexisting nerve injury
      ii) Coagulopathy
      iii) Disposition and physical limitations
         (1) Ability to ambulate on crutches should be considered prior to performing lower extremity peripheral nerve blocks in outpatients.
         (2) If a patient is unable to use crutches and has no weight-bearing restriction following foot surgery, an ankle block may be more appropriate.
   c) **Risks and complications**
      i) General risks
         (1) Nerve injury
         (2) Localized bleeding
         (3) Infection at the site of needle puncture
         (4) Intravascular injection with local anesthetic toxicity
      ii) Site-specific risks and potential complications
         (1) The sciatic nerve is in close proximity to the popliteal artery and vein in the popliteal fossa
            (a) Hematoma formation
            (b) Intravascular injection

READ MORE
Ankle block, Chapter 190.



Consider planned surgical site, tourniquet application, patient disposition, and any physical limitations when determining the right regional anesthetic technique.

3) **Equipment**
   a) Common equipment for all peripheral nerve block techniques include sterile skin cleansing solution, sterile gloves, a marking pen, and two 20-mL syringes of local anesthetic solution with extension tubing.
   b) A nerve stimulator technique requires an electrical nerve stimulator with a compatible 2-4 in insulated needle and a grounding electrode.
   c) Ultrasound-guided regional anesthesia requires portable ultrasound equipment and a high-frequency linear transducer or a low-frequency curvilinear transducer (obese patients) for sciatic nerve block at the popliteal fossa.



Neuropathy rates for the popliteal sciatic block are reported to be as high as 5% perioperatively, taking an average of 8 weeks for resolution with 0.7% of patients having prolonged neuropathy.[5]

4) **Technique**
   a) **Monitoring**
      i) Peripheral nerve blocks should be performed in a location where the patient's noninvasive blood pressure, EKG, oxygen saturation, and end-tidal CO_2 may be monitored.
      ii) Supplemental O_2 should be provided via a face mask or nasal cannulae, especially when IV sedation is administered.
   b) **Positioning and preparation**
      i) This block may be performed with the patient in the prone, lateral, or supine position.
      ii) With prone positioning, placing a towel roll under the patient's affected ankle permits passive flexion of the knee and can facilitate identification of surface landmarks.
      iii) Apply monitors and supplemental O_2.
      iv) IV sedation may be administered, but the patient must be able to follow commands and volunteer sensory information.



Performing a popliteal sciatic block at least 8 cm proximal to the popliteal crease will increase the likelihood of anesthetizing both major branches of the sciatic nerve with a single injection.

   c) **Surface anatomy**
      i) Identify and mark the popliteal crease while the patient flexes the knee.
      ii) The medial and lateral borders of the popliteal fossa are formed by the semimembranosus and biceps femoris tendons, respectively (Cognitive Aid: Panel A).
      iii) An anatomic study of 28 cadavers demonstrated division of the sciatic nerve into its tibial and common peroneal branches a mean of 60.5 ± 27.0 mm above the popliteal crease.[6]
      iv) In order to increase the likelihood of anesthetizing the sciatic nerve prior to division, the site for needle insertion should be marked ~8-10 cm cephalad to the midpoint of the popliteal crease.
      v) Alternatively, the proximal "junction" of the biceps femoris and semimembranosus tendons may also successfully predict sciatic nerve location.[7]
   d) **Stimulating popliteal sciatic nerve block**
      i) Posterior approach (prone position)
         (1) After sterile skin preparation, insert a 2-4 in insulated needle at a 45-degree angle to skin aiming cephalad, while seeking motor responses from either the tibial or the peroneal nerve branches (**Fig. 184-1**).
         (2) When a sciatic nerve motor response is elicited and maintained at a stimulating current <0.5 mA, incrementally inject 30-40 mL of local anesthetic solution following careful aspiration for blood.
         (3) Given the likelihood of proximal sciatic nerve bifurcation, this block may be performed as a double-injection technique by specifically identifying the motor responses of the tibial nerve (plantar flexion or inversion) and peroneal nerve (dorsiflexion or eversion) individually followed by separate local anesthetic injections.

FIGURE 184-1 Stimulating Technique for the Posterior Popliteal Sciatic Nerve Block



Insert a 2-4 in insulated needle at a 45-degree angle to skin aiming cephalad, while seeking motor responses from either the tibial or the peroneal nerve branches of the sciatic nerve.

ii) Lateral approach (supine or lateral position)
   (1) For patients who are unable to assume the prone position, the popliteal fossa can be approached laterally with the patient in the supine or lateral position to produce effective sciatic nerve blockade, although onset time to complete block is longer when compared to the posterior approach.[8]
   (2) With the patient supine, draw a 10-cm line cephalad along the lateral thigh from the popliteal crease between the vastus lateralis and the biceps femoris muscles.
   (3) At the top of the line, insert a 4-in insulated needle parallel to the floor and directed medially.
   (4) Upon contact with the femur, withdraw the needle and redirect ~30 degrees posteriorly while seeking motor responses produced by tibial nerve stimulation.

e) **Ultrasound-guided popliteal sciatic nerve block**
   i) With the patient in the prone position and a high-frequency linear transducer oriented perpendicular to the long axis of the femur over the intertendinous junction (Cognitive Aid: Panel B), the sciatic nerve can be identified in short axis anterior to the fascia of the biceps femoris muscle, posterior and medial to the femur, and posterior to the popliteal vessels when visible (Cognitive Aid: Panel C).
      (1) The ultrasound-guided technique may also be performed with the patient in the supine or lateral position.

(2) In supine position, the distal leg is elevated on a pillow, and the transducer is applied under the patient's thigh at the level of the intertendinous junction to image the sciatic nerve in short axis.

ii) Tracing the sciatic nerve proximally and distally from this point should reveal bifurcation of the sciatic nerve.

(1) For single-injection blocks, a dual-injection technique distal to the bifurcation provides faster onset.[9,10]

(2) For continuous blocks, a catheter placed proximal to the bifurcation provides better analgesia.[11]

iii) Place a local anesthetic skin wheal lateral to the transducer on the lateral thigh at a position corresponding to the depth of the sciatic nerve estimated by ultrasound imaging.

iv) Insert the block needle through this skin wheal using in-plane guidance in a medial direction toward the target nerve.

v) Once the needle tip passes through the biceps femoris fascia and is positioned near the sciatic nerve, incremental aspiration and injection of local anesthetic solution should result in circumferential spread around the target nerve.

(1) Injection in the paraneural sheath surrounding the tibial and common peroneal nerves hastens onset,[12] but injection inside the epineurium must be avoided.[13,14]

(2) Both intraneural and subparaneural injections are associated with a subclinical decrease in sciatic nerve action potentials at 5 weeks.[15]

vi) Following local anesthetic injection, a perineural catheter may be placed in proximity to the nerve for prolonged analgesia.[2]



Ultrasound-guided popliteal sciatic nerve block may be performed with the patient prone, lateral, or supine.



Ultrasound-guided popliteal sciatic block inside the paraneural sheath at the bifurcation of the nerve improves onset time and block efficacy.[12,15]

5) **Evaluation of the block**

a) The quality of nerve blockade must be evaluated prior to surgical incision, even if intraoperative general anesthesia is planned.

b) **Motor testing**

i) Testing strength of plantar flexion and dorsiflexion of the foot tests blockade of the posterior tibial nerve and common peroneal nerve, respectively.

ii) Asking the patient to flex the knee against resistance (by grasping the ankle) tests motor function of the hamstring muscles (should be preserved).

c) Sensory testing

i) Decreased temperature and pinprick sensation over the lateral calf and dorsal/plantar surfaces of the foot confirm appropriate sciatic nerve block.

6) **Postoperative management**

a) Outpatients who undergo popliteal sciatic nerve blockade should be discharged with a supportive ankle splint (due to foot drop) and weight-bearing restriction.

i) The patient is at risk for falling if ambulation is initiated on the anesthetized extremity prior to nerve block resolution.

ii) Hospitalized patients with single-injection or continuous sciatic nerve blocks should have similar restrictions but may participate in physical therapy–directed activities with appropriate assistance.

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185 US-Guided Popliteal Block

Sarah J. Madison • Larry F. Chu • Cynthia Khoo

Patient Position: Prone, supine, or lateral. Place a towel roll under patient's affected ankle to permit passive flexion of knee and to facilitate identification of surface landmarks. This block may also be performed in the supine or lateral position when necessary.

Needle Size: 22- to 17-gauge, 50- to 100-mm block needle

Volume: 20-40 mL local anesthetic

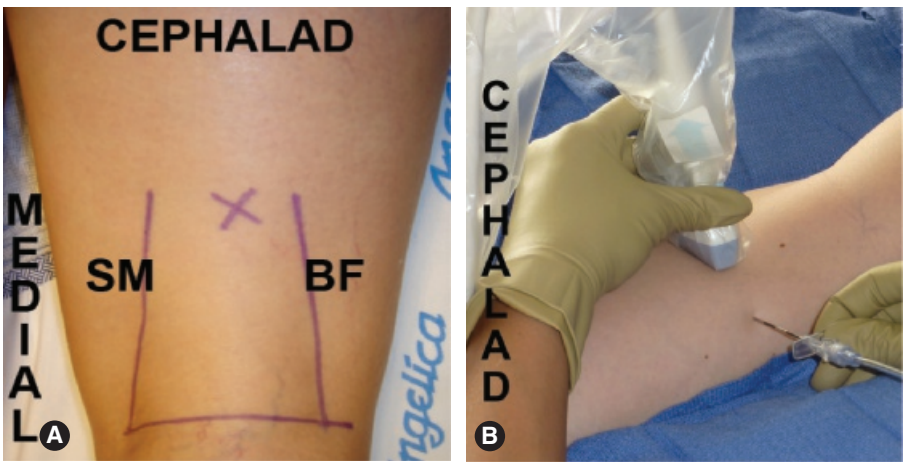
Anatomic Landmarks

Ⓐ Identify and mark the popliteal crease while the patient flexes the knee. The medial and lateral borders of popliteal fossa are formed by the semimembranosus and biceps femoris tendons, respectively. The sciatic nerve is located between these two muscle groups.

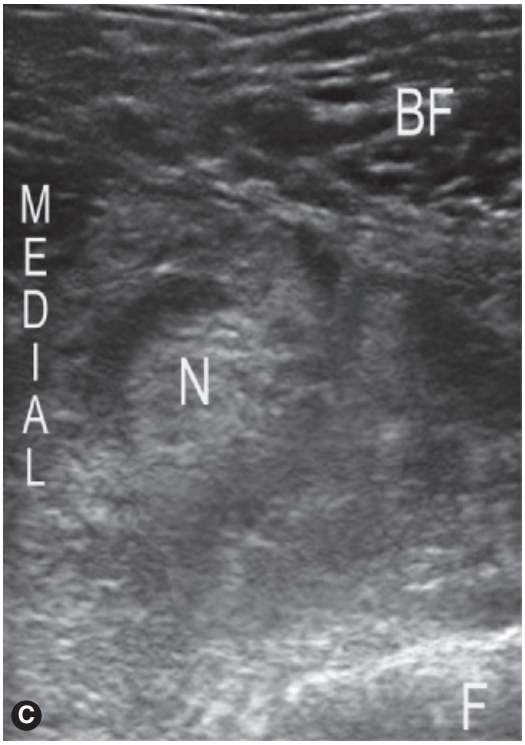
Approach and Technique

Ⓑ With the patient in prone position, place a high-frequency (HF) linear transducer oriented perpendicular to the long axis of the femur over the intertendinous junction.

Ⓒ Identify the sciatic nerve in short axis anterior and medial to the fascia of biceps femoris muscle, posterior and medial to the femur, and posterior to the popliteal vessels when visible. Trace the sciatic nerve proximally and distally from this point to reveal bifurcation of the sciatic nerve. Place a local anesthetic skin wheal lateral to the transducer on



A. The popliteal fossa is bordered laterally by the biceps femoris (BF) tendon and medially by the tendons of semimembranosus (SM) and semitendinosus (right popliteal fossa shown). The sciatic nerve is reliably located below the intertendinous junction identified by the "X." **B.** Orient the ultrasound transducer perpendicular to the long axis of the femur across the intertendinous junction to image the sciatic nerve in cross section. Insert the block needle lateral to the transducer and direct medially for in-plane needle guidance (left thigh shown).



C. Short-axis ultrasound image demonstrating the relevant cross-sectional anatomy for the popliteal sciatic nerve block. BF, biceps femoris muscle; F, femur; N, sciatic nerve.

the lateral thigh at a position corresponding to the depth of the sciatic nerve. Insert the block needle through the skin wheal using in-plane guidance in a medial direction toward the target nerve. After the needle tip passes through biceps femoris fascia and is positioned near the sciatic nerve, incremental aspiration and injection of local anesthetic solution should result in circumferential spread around the target.

Evaluation of Block

Motor Testing

Testing strength of plantar flexion and dorsiflexion of the foot tests blockade of the posterior tibial nerve and common peroneal nerve, respectively.

Sensory Testing

Decreased temperature and pinprick sensation over the lateral calf and dorsal/plantar surfaces of the foot confirms appropriate sciatic nerve block.

186 Lumbar Plexus (Psoas Compartment) Block

Jan Boublik • Jason G. Ramirez

The lumbar plexus is formed from the L1-L4 spinal nerve roots with inconsistent contributions from T12.[1,2] The plexus ultimately gives rise to the genitofemoral, iliohypogastric, ilioinguinal, femoral, obturator, and lateral femoral cutaneous (LFC) nerves. Of these, the femoral, obturator, and LFC nerves are most important for lower extremity surgery, providing the major innervation of the thigh, hip, and knee (Fig. 186-1).[1] The posterior lumbar plexus block (LPB), also known as the psoas compartment block, can be utilized to provide anesthesia and analgesia for surgical procedures of the hip, thigh, and knee and may be combined with blocks of the sacral plexus to produce anesthesia of the entire lower extremity.

1) **Benefits and indications**
   a) **Postoperative analgesia following total hip arthroplasty (THA)[1,3-6]**
      i) Benefits of continuous LPB for THA include earlier mobilization, decreased length of stay, decreased opioid use, decreased opioid-related side effects, improved pain control, and improved patient satisfaction.[3,6]
   b) **Postoperative analgesia for major knee and thigh procedures[4]**
   c) **May be combined with a parasacral block to provide surgical anesthesia for hip fracture surgery[1]**
2) **Risks and contraindications**
   a) **Absolute contraindications**
      i) Infection at the planned block site
      ii) Patient refusal
   b) **Relative contraindications**
      i) Preexisting peripheral neuropathy
      ii) Bacteremia
      iii) Previous spine surgery
      iv) Coagulopathy
         (1) This block is not recommended in patients taking medications affecting hemostasis.
         (2) The lumbar plexus is deep and lies in close proximity to the neuraxis. It is not amenable to compression in the event of vascular puncture.[1]
         (3) LPB is considered a deep plexus block. The American Society of Regional Anesthesia and Pain Medicine guidelines for neuraxial techniques should be applied for patients receiving antithrombotic or thrombolytic therapy.[7]
   c) **Risks and complications[8]**
      i) General risks
         (1) Nerve injury
         (2) Localized bleeding
         (3) Infection

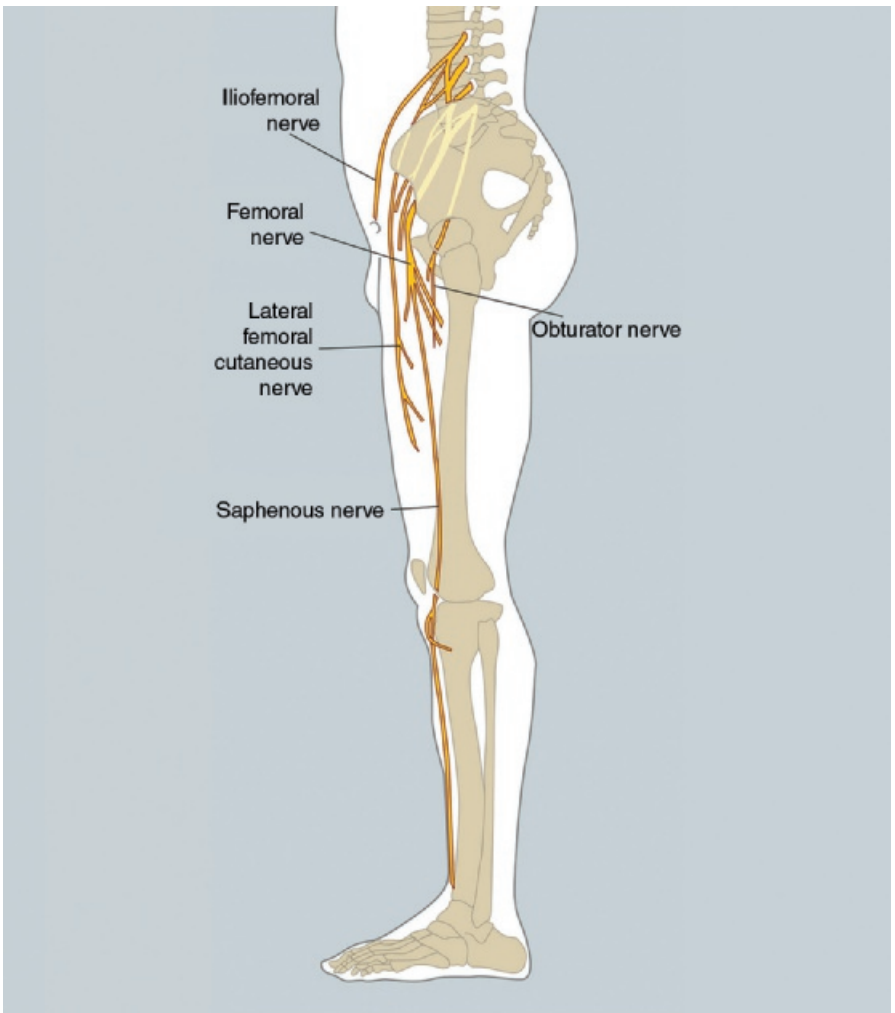


LPB is considered a deep plexus block. The American Society of Regional Anesthesia and Pain Medicine guidelines for neuraxial techniques should be applied for patients receiving antithrombotic or thrombolytic therapy.[7]

READ MORE

Regional anesthesia in the anticoagulated patient, Chapter 171.

FIGURE 186-1 Illustration Demonstrating the Main Distal Branches of the Lumbar Plexus That Contribute Significantly to Lower Extremity Innervation.



Reprinted with permission from Clark L. Anatomy of the lumbar and sacral plexus. In: Chelly JE, ed. *Peripheral Nerve Blocks: A Color Atlas*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009:100.

ii) Site-specific risks and potential complications
   (1) Intravascular injection with local anesthetic toxicity is a major concern when injecting large doses of local anesthetic solution into the highly vascular psoas muscle.
   (2) Diffusion or direct injection of local anesthetic into the neuraxis may result in epidural or subarachnoid block.[1,2]
      (a) Epidural spread occurs in up to 16% of LPBs and is attributed to retrograde spread of local anesthetic solution.

(i) Epidural spread can result in bilateral motor and sensory block and sympathectomy.

b) Subarachnoid injection can result in a high spinal block and total spinal anesthesia.

(3) Complications associated with deep needle placement may include renal hematoma, retroperitoneal hematoma, and intra-abdominal or intervertebral catheter placement.[1,2]

3) **Equipment**

a) Common equipment for all peripheral nerve block techniques include sterile skin cleansing solution, sterile gloves, a marking pen, and two 20-mL syringes of local anesthetic solution with extension tubing.

b) A nerve stimulator technique requires an electrical nerve stimulator with a compatible 4- to 6-in insulated needle and a grounding electrode. Using a needle of appropriate length based on the patient's body habitus may help reduce the likelihood of serious complications.

c) Ultrasound-guided regional anesthesia requires portable ultrasound equipment and a low-frequency large curvilinear transducer for LPB.

4) **Technique**

a) **Monitoring**

i) Peripheral nerve blocks should be placed in a location where the patient's noninvasive blood pressure, EKG, oxygen saturation, and end-tidal CO_2 may be monitored.

(1) Even when IV sedation is not planned, the potential hemodynamic changes and risks of complications associated with LPB necessitate close monitoring.

ii) Supplemental O_2 should be provided via a face mask or nasal cannulae, especially when IV sedation is administered.

b) **Positioning and preparation**

i) The patient should be placed in the lateral decubitus position with the operative side up.

ii) The hips should be flexed with the lower back pushed out toward the practitioner.

iii) IV sedation should be administered, but the patient must be able to follow commands and volunteer sensory information.

c) **Surface anatomy**

i) The LPB is routinely performed at the L4 vertebral level.[5]

ii) Draw a line marking the midline in a cephalad-caudad fashion along the spinous processes of the lumbar vertebrae.

iii) Next draw the intercrestal line between the tops of the iliac crests, intersecting the midline at a 90-degree angle.

iv) Mark a third line parallel to midline through the posterior superior iliac spine in a cephalad direction to intersect the intercrestal line. Dividing the distance between the two intersections into thirds, the site for needle insertion will be 1 cm cephalad from the junction of the middle and lateral thirds. This approximates the location of the L4 transverse process (Cognitive Aid: Panel A).[5]

(1) In many cases, this landmark strategy places the needle insertion site lateral to the transverse process.[9,10] Ultrasound imaging suggests that a medial adjustment of 0.75 cm optimizes the chance of needle contact with the lumbar transverse process.[10]



Local anesthetic systemic toxicity (LAST) is a risk with LPB. Careful choice of local anesthetic dose and additives, early recognition of symptoms, and preparation with resuscitative drugs and equipment may be lifesaving.

READ MORE
Local anesthetic systemic toxicity (LAST), Chapter 149.



Remain vigilant for signs and symptoms of epidural or subarachnoid spread of local anesthetic.



LPB is a deep plexus block and is not advised in patients at high risk for bleeding complications.



Ultrasound imaging suggests that a medial adjustment of 0.75 cm from the traditional landmark-based approach optimizes the chance of needle contact with the lumbar transverse process.[10]

d) **Stimulating lumbar plexus block**
   i) After sterile skin preparation and placement of a local anesthetic skin wheal, insert a 4- to 6-in simulating needle perpendicular to the skin (Cognitive Aid: Panel A).
   ii) The distance from skin to lumbar transverse process is highly variable and dependent on body mass index (BMI) and gender.[1,2] However, the distance from transverse process to the lumbar plexus anteriorly is independent of BMI or gender and is fairly constant at ~2-3 cm.[1,2,5,10]
   iii) Elicitation of a quadriceps contraction signifies correct needle tip placement in proximity to the lumbar plexus.
   iv) If bony contact with the L4 transverse process is established, withdraw the needle and walk caudally off the transverse process, then advance up to a maximum of 3 cm, seeking an evoked motor response of the quadriceps muscle.



Distance from skin to lumbar transverse process is highly variable. Distance from transverse process to lumbar plexus is fairly constant and independent of BMI or gender.

e) **Ultrasound-guided lumbar plexus block**
   i) Real-time ultrasound-guided techniques for the LPB have been described but remain challenging.[11,12] Anatomy can be difficult to visualize due to the depth of relevant structures and due to the acoustic shadowing effect from the transverse process.[13]
   ii) To date, most practitioners continue to rely on electrical nerve stimulation techniques for LPB in adults, even with ultrasound guidance.[14,15]
   iii) Prepuncture ultrasound scanning may help practitioners confirm the location and depth of the lumbar transverse processes prior to performing a stimulating LPB (Cognitive Aid: Panel C).[10]



Quadriceps contraction is the correct motor response to electrical stimulation for the lumbar plexus block.

f) **Alternative techniques**
   i) Lumbar plexus block is a technically complex block that is limited to patients without coagulopathy, who can assume a lateral position.
   ii) Fascia iliaca block is performed with the patient supine.[16] It reduces opioid consumption after THA[17] and may be noninferior to LPB for analgesia following hip arthroscopy.[18]
   iii) Anterior (transmuscular) quadratus lumborum block is an ultrasound-guided approach that provides similar analgesia to LPB and similar reductions in opioid consumption and length of stay following THA.[19]

5) **Evaluation of the block**
   a) **The quality of nerve blockade must be evaluated prior to surgical incision, even if intraoperative general anesthesia is planned.**
      i) Sensory and motor testing of the lumbar plexus can be accomplished using the "three Ps"—pull, pinch, and punt.[1]
         (1) "Pull" involves abducting the patient's leg and then asking the patient to move the leg voluntarily toward midline to evaluate the strength of the adductor muscles innervated by the *obturator* nerve.
         (2) The *lateral femoral cutaneous* nerve is a purely sensory nerve, so a "pinch" over the lateral thigh assesses anesthesia in this distribution.
         (3) "Punt" involves supporting the patient's leg under the knee and asking the patient to perform a kicking motion against resistance to test the quadriceps muscle innervated by the *femoral* nerve.



Sensory and motor testing of the lumbar plexus can be accomplished using the "three Ps"—pull, pinch, and punt.

6) **Postoperative management**
   a) Outpatients who undergo LPB should be discharged with a knee immobilizer and weight-bearing restriction.

b) Hospitalized patients with single-injection or continuous LPBs should have similar restrictions but may participate in physical therapy activities (ie, continuous passive motion and ambulation with assistance).
   i) Quadriceps weakness from an LPB and hip or knee surgery places the patient at high risk for falling if unassisted ambulation is initiated prior to nerve block resolution.
   ii) Ambulating patients with LPBs and perineural catheters is possible with the assistance of trained nursing staff and physical therapists.[6]



Patients who have undergone LPB should be considered high risk for falls and should not ambulate without proper assistance.

c) Patients experiencing epidural spread of local anesthetic may exhibit signs of sympathectomy and may require temporary supportive treatment including volume resuscitation and/or vasoactive medications.

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187 Lumbar Plexus Block

Jan Boublik • Jason G. Ramirez • Larry F. Chu

Patient Position: The patient should be placed in the lateral decubitus position with the operative side up. The hips should be flexed with the lower back pushed out toward the practitioner.

Needle Size: 4- to 6-in insulated needle

Volume: 20-40 mL local anesthetic

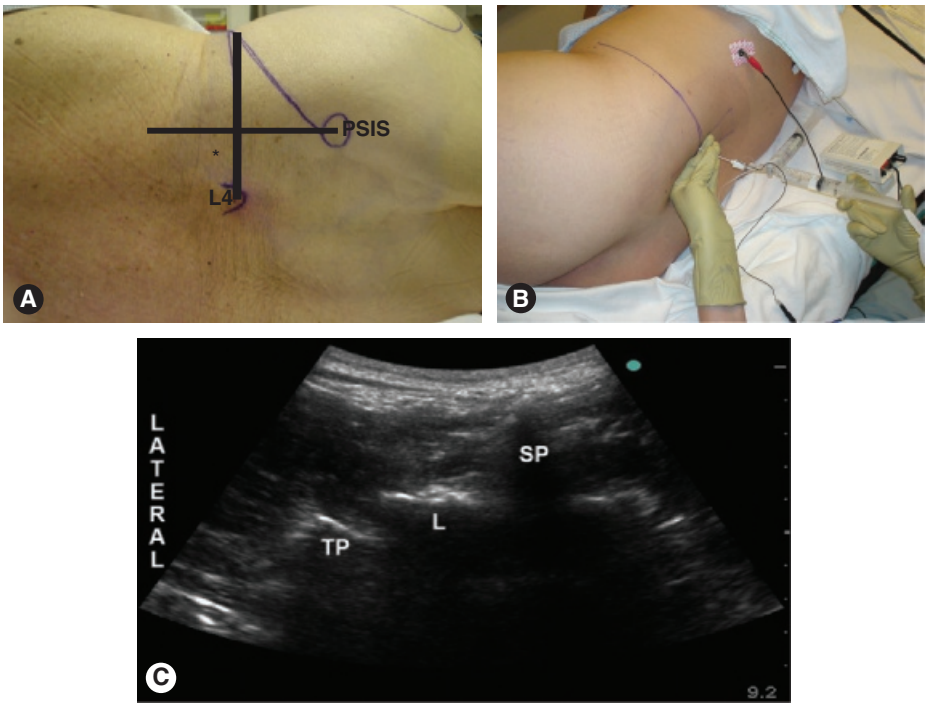
Anatomic Landmarks

Ⓐ The lumbar plexus block (LPB) is routinely performed at the L4 vertebral level. Draw a line marking the midline in a cephalad-caudad fashion along the spinous processes of the lumbar vertebrae. Next draw the intercrestal line between the tops of the iliac crests, intersecting the midline at a 90-degree angle. Mark a third line parallel to midline through the posterior superior iliac spine (PSIS) in a cephalad direction to intersect the intercrestal line. Dividing the distance between the two intersections into thirds, the site for needle insertion will be 1 cm cephalad from the junction of the middle and lateral thirds. This approximates the location of the L4 transverse process. In many cases, this landmark strategy places the needle insertion site lateral to the transverse process. Ultrasound imaging suggests that a medial adjustment of 0.75 cm optimizes the chance of needle contact with the lumbar transverse process.

Approach and Technique

Ⓑ After sterile skin preparation and placement of a local anesthetic skin wheal, insert a 4- to 6-in simulating needle perpendicular to the skin. If bony contact with the L4 transverse process is established, withdraw the needle and walk caudally off the transverse process, then advance up to a maximum of 3 cm, seeking an evoked motor response of the quadriceps muscle.

Ⓒ Prepuncture ultrasound scanning may help practitioners confirm the location and depth of the lumbar transverse processes prior to performing a stimulating LPB. The distance from skin to lumbar transverse process is highly variable and dependent on body mass index (BMI) and gender. However, the distance from transverse process to the lumbar plexus anteriorly is independent of BMI or gender and is fairly constant at ~2-3 cm. Elicitation of a quadriceps contraction signifies correct needle tip placement in proximity to the lumbar plexus.



A. Landmarks for the stimulating LPB include the intercrestal line approximately at the level of L4 and the posterior superior iliac spine (PSIS); asterisk marks the recommended site for needle insertion. **B.** Stimulating LPB technique: a 4- to 6-in insulated needle is inserted perpendicular to skin at a puncture site predetermined by surface anatomic landmarks. The intersection of the intercrestal line with the parasagittal line determines the site of introduction of the needle. **C.** Short axis ultrasound image at the level of the lumbar vertebra; SP, spinous process; L, lamina; TP, transverse process.

Evaluation of Block

The quality of nerve blockade must be evaluated prior to surgical incision, even if intraoperative general anesthesia is planned. Sensory and motor testing of the lumbar plexus can be accomplished using the "three Ps"—pull, pinch, and punt. "Pull" involves abducting the patient's leg and then asking the patient to move the leg voluntarily toward midline to evaluate the strength of the adductor muscles innervated by the *obturator* nerve. The *lateral femoral cutaneous* nerve is a purely sensory nerve, so a "pinch" over the lateral thigh assesses anesthesia in this distribution. "Punt" involves supporting the patient's leg under the knee and asking the patient to perform a kicking motion against resistance to test the quadriceps muscle innervated by the *femoral* nerve.

188 Femoral Nerve and Adductor Canal Blocks

Matthew T. Charous

The femoral nerve supplies the sensory and motor innervation of the anterior thigh and knee and then terminates as the saphenous nerve, which passes through the adductor canal and innervates the medial aspect of the leg. Techniques employing electrical stimulation, ultrasound guidance, or both have been described. Femoral and adductor canal perineural catheters provide prolonged analgesia following total knee arthroplasty and other major knee surgeries. Other potential benefits of femoral and adductor canal blocks in total knee arthroplasty include decreased bleeding and need for transfusion[1] and shorter time to achieve hospital discharge criteria.[1-3]

1) **Benefits and indications**
   a) Surgical anesthesia or postoperative analgesia of the anterior and medial thigh or the medial aspect of the leg below the knee
   b) The posterior thigh is supplied by the sacral plexus *via* the posterior cutaneous nerve of the thigh.
   c) Surgical anesthesia of the entire lower extremity requires blockade of the sciatic nerve in addition to the lumbar plexus branches—femoral, lateral femoral cutaneous (LFC), and obturator nerves.

READ MORE
Proximal sciatic nerve blocks, Chapter 182.

2) **Risks and contraindications**
   a) **Absolute contraindications**
      i) Local infection at the site of planned needle placement
   b) **Relative contraindications**
      i) Coagulopathy
      ii) Preexisting nerve injury
   c) **Risks and complications**
      i) General risks
         (1) Nerve injury
         (2) Localized bleeding
         (3) Infection
         (4) Intravascular injection with local anesthetic toxicity
      ii) Site-specific risks and potential complications
         (1) Injury to femoral vessels and subsequent hematoma



When the potential benefits outweigh the risks, femoral nerve block may be safely performed in the coagulopathic patient below the inguinal ligament since manual compression of vascular structures may be performed if necessary.

3) **Equipment**
   a) Common equipment for all peripheral nerve block techniques include sterile skin cleansing solution, sterile gloves, a marking pen, and two 20-mL syringes of local anesthetic solution with extension tubing.
   b) A nerve stimulator technique requires an electrical nerve stimulator with a compatible 1-2 in insulated needle and a grounding electrode.
   c) The fascia iliaca approach utilizes a Tuohy-tip needle with or without extension tubing attached to syringes of local anesthetic and is performed without nerve stimulation.[4]
   d) Ultrasound-guided regional anesthesia requires portable ultrasound equipment and a high-frequency linear transducer for femoral nerve block.

4) **Technique**

a) **Monitoring**

i) Peripheral nerve blocks should be placed in a location where the patient's noninvasive blood pressure, EKG, oxygen saturation, and end-tidal CO_2 may be monitored.

ii) Supplemental O_2 should be provided via a face mask or nasal cannulae, especially when IV sedation is administered.

b) **Positioning and preparation**

i) Position the patient supine with a small hip roll under the side to be blocked to flatten the inguinal crease.

(1) The leg should be straight and not internally or externally rotated.

ii) Apply monitors and supplemental O_2.

iii) IV sedation should be administered, but the patient must be able to follow commands and volunteer sensory information.

c) **Surface anatomy**

i) The important landmarks for this block include the inguinal crease and the femoral artery (Cognitive Aid: Panel A).

(1) An anatomic study has shown the femoral nerve to be widest and most superficial at the level of the inguinal crease (Cognitive Aid: Panel B) where it can be found immediately lateral to the artery 71% of the time.[6]

(2) If the femoral pulse is difficult to palpate, Doppler or ultrasound may be helpful.

ii) After identifying the femoral artery, mark the point 1 cm lateral to the artery along the inguinal crease.

d) **Stimulating femoral nerve block**

i) After sterile preparation and injection of a local anesthetic skin wheal, insert a 1-2 in insulated needle at a 45-degree angle, aiming cephalad at the point identified above (**Fig. 188-1**).

ii) Contraction of the quadriceps muscle and the patellofemoral ligament indicates an appropriate femoral nerve motor response.

(1) The ability to maintain the motor response at <0.5 mA of stimulating current confirms proper needle tip placement.

(2) After negative aspiration for blood, inject the total local anesthetic volume (30-40 mL) in 5-mL increments.

(3) A continuous peripheral nerve block catheter may be placed at this location to administer a postoperative perineural infusion of local anesthetic.

iii) The classic "3-in-1" block was originally described as a large-volume single-injection femoral nerve block with firm distal pressure to encourage cephalad spread of the anesthetic to the lumbar plexus to block the LFC and obturator nerves.

(1) Magnetic resonance imaging has since disproved this theoretical concept by demonstrating local anesthetic spread only in the lateral, caudad, and medial directions.[7]

(2) In a clinical study, the "3-in-1" block consistently produced anesthesia in the LFC and femoral nerve distributions but only achieved obturator nerve block in 4% of patients.[8]

e) **"Fascia iliaca" block technique**

i) Performed 1 cm caudad to the junction of the lateral and middle thirds of the inguinal ligament.



The traditional fascia iliaca block anesthetizes the femoral and LFC nerves and is performed without nerve stimulation or ultrasound, relying on anatomical landmarks for localization.



Newer approaches to the fascia iliaca block incorporate ultrasound guidance and necessitate appropriate sonographic interpretation of underlying musculature.

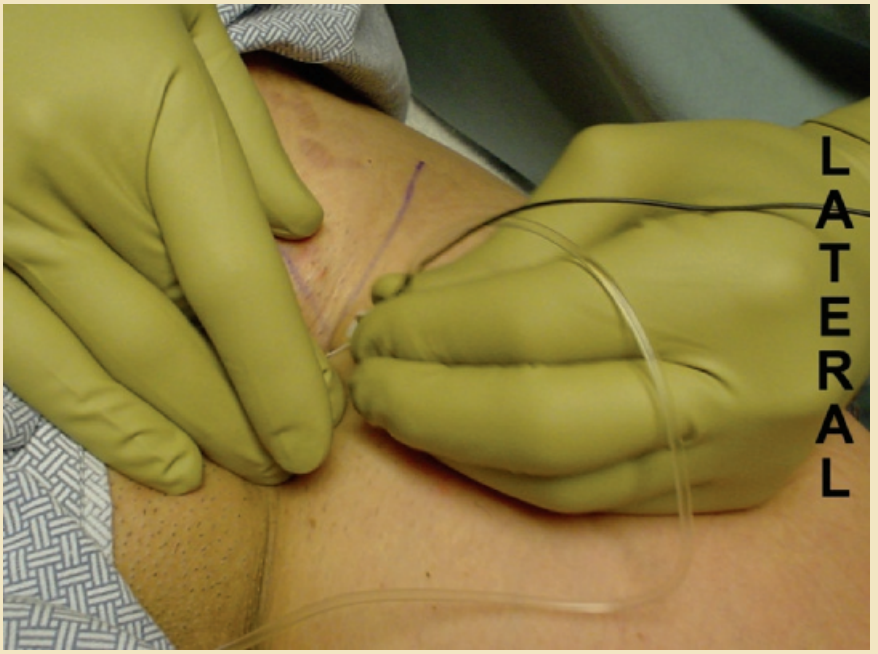


The femoral nerve is widest and most superficial at the level of the inguinal crease.



The adductor canal block allows for more preservation of quadriceps strength and is now typically favored over the femoral nerve block for analgesia following knee surgery.

FIGURE 188-1 Stimulating Femoral Nerve Block Technique



Insert a 1- to 2-in insulated needle lateral to the femoral artery at a 45-degree angle aiming cephalad at the level of the inguinal crease (left inguinal region shown).

(1) This technique does not require nerve stimulation or ultrasound guidance.
(2) May be used as adjunctive analgesia for hip fractures and surgery;[9] however, quadriceps weakness and consequent fall risk outweigh clinical benefit in ambulatory patients.[10]

ii) Insert a Tuohy-tip needle at a 45-degree angle aiming cephalad (Fig. 188-2) until two distinct losses of resistance are palpated as the needle traverses the fascia lata and fascia iliaca, respectively.

iii) After confirming negative aspiration for blood, inject the total volume of local anesthetic in 5-mL increments. This produces a very reliable block of the femoral and LFC nerves.[4]

The main target of the adductor canal block is the saphenous nerve, a sensory nerve; however, the nerve to vastus medialis, a motor nerve that travels in the adductor canal, is also affected.[5]

f) **Ultrasound-guided femoral nerve block**

i) With a high-frequency linear transducer positioned at the level of the inguinal crease and oriented parallel to the inguinal ligament (Cognitive Aid: Panel C), identify the femoral artery in cross section.
(1) If both the femoral and profunda femoris arteries are visible, move the transducer more cephalad until the two vessels merge to form the common femoral artery.
(2) At this level, the femoral nerve lies lateral to the femoral artery between the fascia iliaca and the iliacus muscle (Cognitive Aid: Panel D).

READ MORE
Overview of peripheral nerve blocks, Chapter 44

ii) Raise a local anesthetic skin wheal lateral to the ultrasound transducer after sterile skin preparation.

FIGURE 188-2 Fascia Iliaca Block Technique



Insert a Tuohy-tip needle 1 cm below the junction of the lateral and middle thirds of the inguinal ligament at a 45-degree angle aiming cephalad until the needle tip traverses the fascia lata and fascia iliaca (right inguinal region shown).

iii) Insert the block needle through this skin wheal using in-plane guidance in a lateral-to-medial direction toward the femoral nerve.[11]
iv) Once the needle tip has traversed the fascia iliaca lateral to the femoral nerve, incrementally inject local anesthetic until injectate spread surrounding the femoral nerve can be visually confirmed.

g) **Ultrasound-guided adductor canal block**
i) To perform a saphenous nerve block in either the femoral triangle or adductor canal, externally rotate the leg and follow the superficial femoral artery distally until the sartorius muscle can be identified overlying the artery (Cognitive Aid: Panel E).
ii) The proximal border of the adductor canal can be identified where the medial border of the Sartorius and adductor longus muscles come together (Cognitive Aid: Panel B).
iii) Deposit 10-20 mL of local anesthetic just lateral to the artery, deep to the subsartorial fascia, superficial to vastus medialis muscle.



Patients who have received a femoral nerve, fascia iliaca, or adductor canal block should be considered **high fall risk** and should not ambulate without proper assistance.

5) **Evaluation of the block**
a) The quality of nerve blockade must be evaluated prior to surgical incision, even if intraoperative general anesthesia is planned.
b) **Motor testing**
i) Strength of leg extension against resistance or gravity tests the quadriceps muscle innervated by the *femoral* nerve.
c) **Sensory testing**
i) Testing cold temperature and pinprick sensation over the anterior thigh assesses sensory anesthesia in the *femoral* nerve distribution.
ii) Similar testing over the lateral thigh assesses anesthesia in the *LFC* nerve distribution.
iii) Sensory testing over the medial malleolus assesses anesthesia of the *saphenous* nerve.



Exercise caution when approaching the femoral nerve from a far lateral location. Although rare, inadvertent trauma to the LFC nerve can occur as the needle is advanced medially.

6) **Postoperative management**
   a) Outpatients who undergo femoral nerve blockade should be discharged with a knee immobilizer and weight-bearing restriction. Patients with adductor canal blocks should be tested for quadriceps weakness prior to discharge.
   b) Hospitalized patients with single-injection or continuous femoral nerve blocks should have similar restrictions but may participate in physical therapy–directed activities (ie, continuous passive motion and ambulation with assistance).
      i) Quadriceps weakness from a femoral nerve block and knee surgery places the patient at high risk for falling if unassisted ambulation is initiated prior to nerve block resolution. More distal approaches such as the adductor canal block result in less quadriceps muscle weakness.[12]
      ii) Ambulation with femoral nerve blocks and perineural catheters is possible with the assistance of trained nursing staff and physical therapists.[2]

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189 US-Guided Femoral Nerve and Adductor Canal Blocks

Matthew T. Charous • Larry F. Chu

Patient Position: Position the patient supine with a small hip roll under the side to be blocked to flatten the inguinal crease. The leg should be straight and not internally or externally rotated.

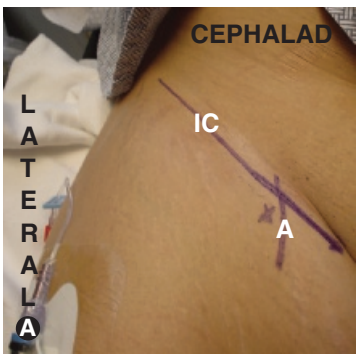
Needle Size: 22-17 gauge, 50- to 100-mm block needle

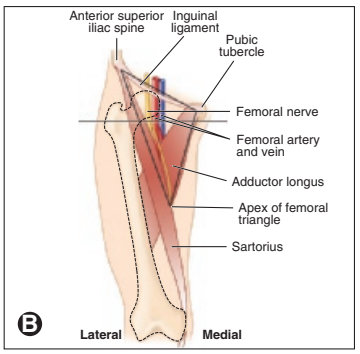
Volume: 20-40 mL local anesthetic

Anatomic Landmarks

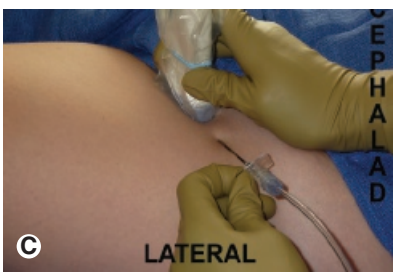
Ⓐ The important landmarks for this block include the inguinal crease and femoral artery (FA).

Ⓑ An anatomic study has shown the femoral nerve (FN) to be widest and most superficial at the level of the inguinal crease where it can be found immediately lateral to the artery 71% of the time.





A. Surface anatomic landmarks relevant to the FN block. IC, inguinal crease; A, femoral artery. An "X" is placed lateral to the FA to identify the site for needle insertion (left inguinal region shown). **B.** Illustration of the relationship of the FN, FA, and femoral vein below the inguinal ligament. Reproduced from Perlas A. Ultrasound guided femoral nerve block. In: Chelly JE, ed. *Peripheral Nerve Blocks: A Color Atlas*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009:295, with permission.

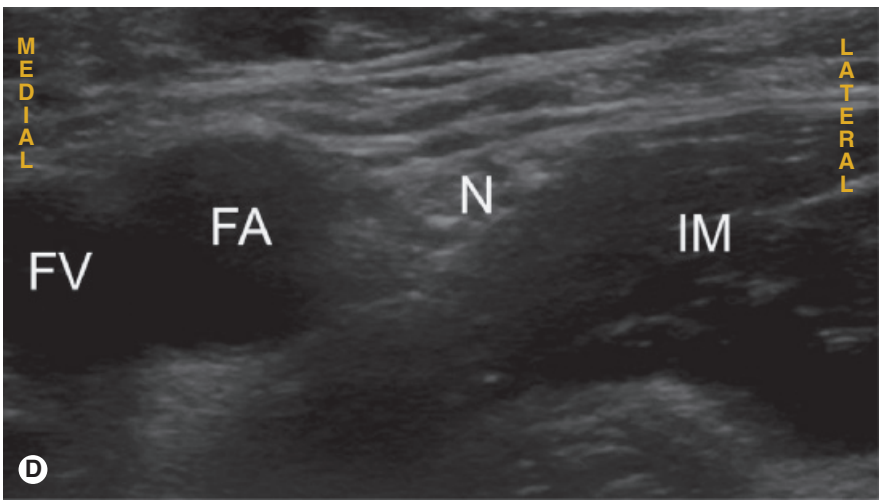


C. Place a high-frequency linear transducer over the FA pulsation at the level of the inguinal crease. Insert the block needle lateral to the transducer and direct medially for in-plane needle guidance (left inguinal region shown).

Approach and Technique

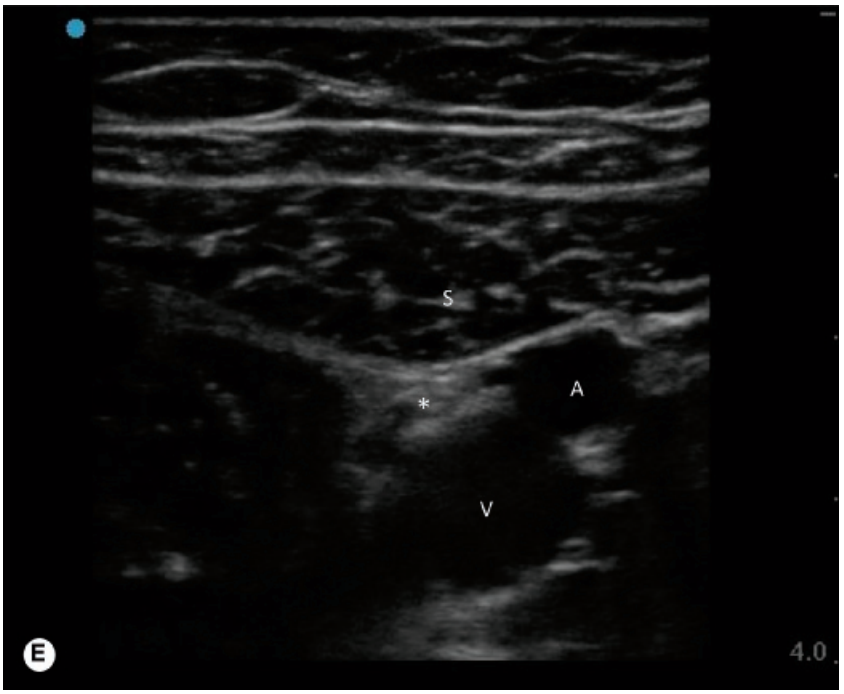
Ⓒ With a high-frequency linear transducer positioned at the level of the inguinal crease and oriented parallel to the inguinal ligament, the FA is easily identified.

Ⓓ If the femoral and profunda femoris arteries are both visible, move the transducer more cephalad until the two vessels merge to form the common FA. At this level, the FN lies lateral to the FA between the fascia iliaca and iliacus muscle. Insert the block needle through the skin wheal using in-plane guidance in a lateral-to-medial direction toward the FN. Once the needle tip has traversed the fascia iliaca lateral to the FN, inject local anesthetic incrementally until visual confirmation of injectate spread surrounding the FN is achieved.



D. Short-axis ultrasound image of the femoral vessels and FN at the inguinal crease. FV, femoral vein; FA, femoral artery; IM, iliacus muscle; N, femoral nerve.

Ⓔ To perform a saphenous nerve block in either the femoral triangle or adductor canal, externally rotate the leg and follow the superficial femoral artery distally until the sartorius muscle can be identified overlying the artery. The proximal border of the adductor canal can be identified where the medial border of the Sartorius and adductor longus muscles come together. Deposit 10-20 mL of local anesthetic just lateral to the artery, deep to the subsartorial fascia, superficial to vastus medialis muscle.



E. Ultrasound image of femoral triangle block. S, Sartorius muscle; A, superficial femoral artery; V, femoral vein.

Evaluation of Block

Motor Testing

Strength of leg extension against resistance or gravity tests the quadriceps muscle innervated by the *femoral* nerve. Adductor canal block will affect quadriceps strength only minimally.

Sensory Testing

Testing cold temperature and pinprick sensation over the anterior thigh assesses sensory anesthesia in the *femoral* nerve distribution. Similar testing over the lateral thigh assesses anesthesia in the *lateral femoral cutaneous* nerve distribution. Sensory testing over the medial malleolus assesses anesthesia of the *saphenous* nerve.

190 Ankle Block

Matthew T. Charous

The ankle block anesthetizes the five most distal branches of the lumbosacral plexus: the posterior tibial, sural, deep peroneal, superficial peroneal, and saphenous nerves.[1] An advantage of this technique for foot surgery is that motor function is essentially spared since the tendons that are responsible for motor function originate proximal to the site of local anesthetic injection. Therefore, the ankle block is regularly performed for patients undergoing ambulatory foot surgery.[2]

1) **Benefits and indications**
   a) Surgical anesthesia or postoperative analgesia for procedures on the toes or foot[1]
      i) Ankle blocks provide adequate surgical anesthesia for foot procedures of limited duration with an ankle tourniquet.
      ii) Prolonged foot surgeries or proximal tourniquet locations (eg, thigh or calf) will likely require an additional regional or general anesthetic.
   b) Selective blocks of individual branches may be used to supplement more proximal sciatic or saphenous nerve blocks.
2) **Risks and contraindications**
   a) **Absolute contraindications**
      i) Local infection at the site of planned needle insertion
      ii) In patients with localized infection of the lower limb, tissue acidosis may limit the amount of nonionized local anesthetic available to cross cell membranes.
   b) **Relative contraindications**
      i) Preexisting nerve injury
      ii) Coagulopathy
         (1) Ankle block may have advantages over deeper blocks in the patient with bleeding tendencies since compression can be easily applied.
   c) **Risks and complications**
      i) General risks
         (1) Nerve injury
         (2) Localized bleeding
         (3) Infection
         (4) Intravascular injection with local anesthetic toxicity
      ii) Site-specific risks and potential complications
         (1) Procedure-induced pain resulting from multiple needle insertions
         (2) Injection of local anesthetic into terminal arteries
            (a) Significant IV injection is rare due to division of local anesthetic volume into multiple sites of injection.
            (b) Even large volumes of local anesthetic solution (up to 30 mL) injected at the ankle result in relatively low serum levels.[3]
         (3) Epinephrine-free local anesthetic solutions are commonly used for ankle block to avoid vasoconstriction of the terminal arteries that supply blood flow to the foot and digits.

3) **Equipment**
   a) Common equipment for all peripheral nerve block techniques include sterile skin cleansing solution, sterile gloves, a marking pen, and up to 30 mL of local anesthetic solution with extension tubing.
   b) A nerve stimulator technique may be employed for the tibial nerve block and requires an electrical nerve stimulator with a compatible 1-in insulated needle and a grounding electrode.
   c) Ultrasound guidance for tibial nerve block at the ankle requires portable ultrasound equipment and a small high-frequency linear or curvilinear transducer.

4) **Technique**
   a) **Monitoring**
      i) Peripheral nerve blocks should be placed in a location where the patient's noninvasive blood pressure, EKG, oxygen saturation, and end-tidal CO_2 may be monitored.
      ii) Supplemental O_2 should be provided via a face mask or nasal cannulae, especially when IV sedation is administered.
   b) **Positioning and preparation**
      i) Position the patient supine (preferred) or prone with the operative foot elevated on a roll.
      ii) Apply monitors and supplemental O_2 prior to administering IV sedation.
   c) **Surface anatomy**
      i) First identify the most cephalad portions of the medial malleolus (MM) and lateral malleolus (LM), and the Achilles tendon posteriorly.
         (1) The sural nerve is located in the subcutaneous tissue between the LM and the Achilles tendon (Cognitive Aid: Panel A).
         (2) Between the MM and the Achilles tendon, the posterior tibial nerve is located immediately posterior to the tibial artery and is the largest of the five nerves innervating the foot (Cognitive Aid: Panel A).
      ii) Next have the patient dorsiflex the great toe to identify the extensor hallucis longus (EHL) tendon.
         (1) The tendon of tibialis anterior (TA) lies medial to the EHL, and the dorsalis pedis artery is typically palpable between these two tendons.
         (2) The deep peroneal nerve, which innervates the webspace between the first and second toes, lies deep to the extensor retinaculum between the two tendons and is closely associated with the dorsalis pedis artery (Cognitive Aid: Panel A).
      iii) Superficial branches of the saphenous nerve run anterior to the MM.
      iv) The superficial peroneal nerve runs anterior to the LM and innervates the dorsum of the foot (Cognitive Aid: Panel A).
   d) **Landmark-based ankle block**[5]
      i) After sterile skin preparation, externally rotate the leg.
      ii) While palpating the posterior tibial artery behind the MM, insert a 1-in 22-gauge B-bevel needle posterior to the pulse at a 45-degree angle toward the MM.
      iii) If bone is contacted without eliciting a paresthesia, inject 5 mL of anesthetic while slowly withdrawing the needle to block the posterior tibial nerve.



The posterior tibial nerve gives rise to three terminal branches: medial plantar, lateral plantar, and calcaneal nerves at the level of the MM. Therefore, complete blockade requires blockade at or proximal to this level.



The posterior tibial nerve is responsible for innervation of all bony structures of the foot.



If extended post-operative analgesia is desired, consider a more proximal approach (such as sciatic nerve block) and/or a perineural catheter.[4]



Avoid epinephrine in the distal extremities, such as the hand and foot, as vasoconstriction in these areas may compromise blood flow.



Ankle block will not provide analgesia for calf or thigh tourniquets.

(1) Alternatively, a 1-in insulated needle may be used with electrical stimulation to locate the posterior tibial nerve by eliciting plantar flexion of the toes at a stimulating current <0.5 mA.

iv) Next, internally rotate the leg to expose the posterior border of the LM and infiltrate 3-5 mL of local anesthetic subcutaneously between the LM and the Achilles tendon to anesthetize the sural nerve.

v) While palpating the EHL and TA tendons on the dorsum of the foot, insert a 1-in 22-gauge B-bevel needle between the two tendons and inject 3-5 mL of local anesthetic after bony contact and gentle aspiration.

vi) From this needle insertion site, inject local anesthetic subcutaneously toward the MM and the LM using a 1.5-in 25- or 27-gauge needle to anesthetize the saphenous and superficial peroneal nerves, respectively.

e) **Ultrasound-guided ankle block** (Cognitive Aid: Panels B, C, and D)

i) Tibial nerve block

(1) Compared to a landmark-based approach, ultrasound-guided tibial nerve block at the ankle results in a higher success rate.[6]

(2) Place the transducer cephalad to the MM and image the posterior tibial artery in short axis.

(3) The nerve will appear immediately posterior to the artery.

(4) Injection of 5 mL of local anesthetic around the nerve will result in reliable anesthesia.

ii) Sural nerve block

(1) The sural nerve travels in close proximity to the lesser saphenous vein posterior to the LM, and ultrasound guidance improves success rates compared to landmark-based techniques.[7]

(2) Using a small linear or curvilinear transducer, image the lesser saphenous vein in short axis.

(3) Applying a tourniquet proximally helps to distend the lesser saphenous vein and facilitates its identification.

(4) Insert a 1- to 1.5-in 22-gauge B-bevel needle in-plane and deposit up to 5 mL of local anesthetic solution circumferentially around the lesser saphenous vein.

iii) Deep peroneal nerve block

(1) Compared to landmark-based localization of the deep peroneal nerve, ultrasound guidance may speed the onset but does not improve the overall success or quality of the block.[8]

(2) Superficial to the tibial surface, the deep peroneal nerve can be visualized lateral to the anterior tibial artery (continuing as the dorsalis pedis artery distally).

(3) Applying the transducer at the level of the superior margin of the MM, image the deep peroneal nerve and anterior tibial artery in short axis.

(4) Insert a 1- to 1.5-in 22-gauge B-bevel needle in-plane and deposit up to 5 mL of local anesthetic circumferentially around the deep peroneal nerve or lateral to the anterior tibial artery if the nerve cannot be identified.



Ultrasound guidance improves success rates for sural and tibial nerve block at the ankle but not for deep peroneal nerve block.[6–8]

5) **Evaluation of the block**

a) The quality of nerve blockade must be evaluated prior to surgical incision, even if intraoperative general anesthesia is planned.

b) **Sensory testing**

i) Blockade of the deep and superficial peroneal nerves may be assessed by testing sensation in the webspace between the first and the second toes and over the dorsum of the foot.

ii) The plantar surface, medial aspect, and lateral aspect of the foot are innervated by the posterior tibial nerve, saphenous nerve, and sural nerve, respectively. The major innervation of the foot is from the posterior tibial nerve.

6) **Postoperative management**

a) All patients who undergo ankle block should be discharged with a hard walking shoe.

b) Patients may bear weight as tolerated and may safely ambulate with minimal assistance.

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191 US-Guided Ankle Block

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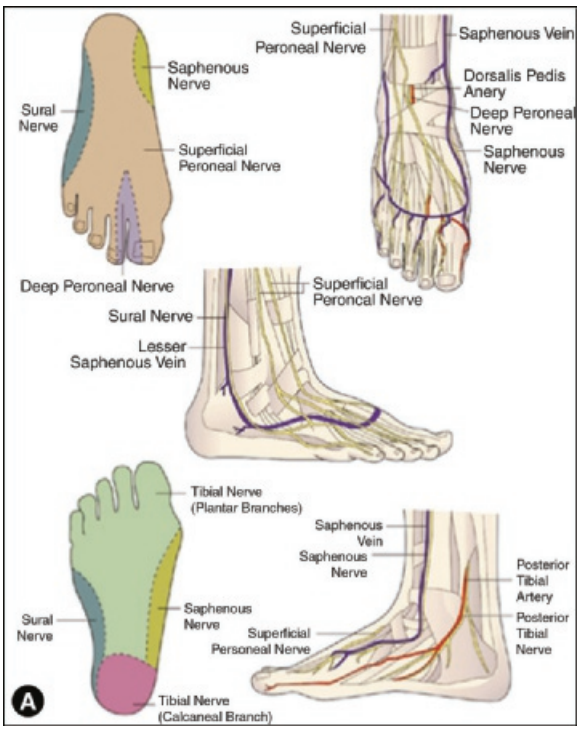
Patient Position: Position the patient supine (preferred) or prone with the operative foot elevated on a roll.

Needle Size: 22-gauge Tuohy-tip or B-bevel block needle

Volume: 5 mL per nerve

Anatomic Landmarks

Ⓐ First identify the most cephalad portions of the medial malleolus (MM) and lateral malleolus (LM), and the Achilles tendon posteriorly. The sural nerve is located in the subcutaneous tissue between the LM and the Achilles tendon. Have the patient dorsiflex the great toe to identify the extensor hallucis longus (EHL) tendon. The tendon of tibialis anterior (TA) lies medial to the EHL, and the dorsalis pedis artery is typically palpable between these two tendons. The deep peroneal nerve, which innervates the webspace between the first and second toes, lies deep to the extensor retinaculum between the two tendons and is closely associated with the dorsalis pedis artery.

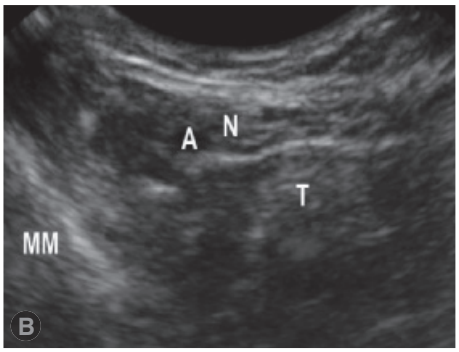


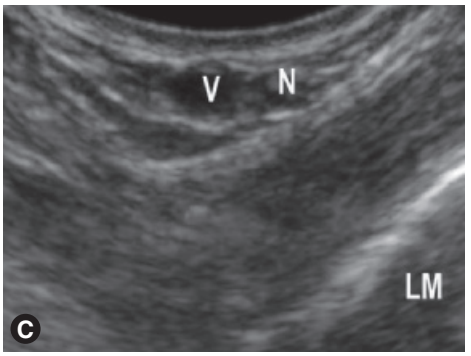
A. Anatomic landmarks. Reprinted with permission from Clanton TO, Loncarich DP. Ankle block. In: Chelly JE, ed. *Peripheral Nerve Blocks: A Color Atlas*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009:158.

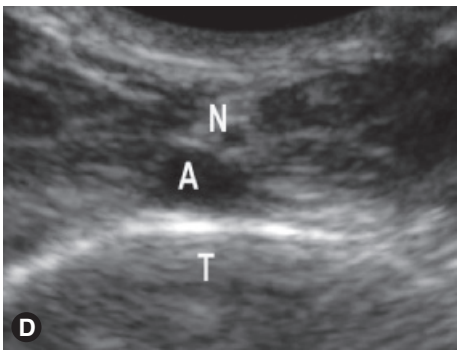
Approach and Technique

Ultrasound-Guided Tibial Nerve Block

Ⓑ Place the transducer cephalad to the MM and image the posterior tibial artery in short axis. The nerve will appear immediately posterior to the artery. Injection of 5 mL of local anesthetic around the nerve will result in reliable anesthesia.







B. Sonoanatomy relevant to the tibial nerve block. MM, medial malleolus; A, posterior tibial artery; N, nerve; T, flexor hallucis longus tendon. **C.** Sonoanatomy relevant to the sural nerve block. LM, lateral malleolus; V, lesser saphenous vein; N, nerve. **D.** Sonoanatomy relevant to the deep peroneal nerve block. T, tibia; A, anterior tibial artery; N, nerve.

Ultrasound-Guided Sural Nerve Block

Ⓒ Using a small linear or curvilinear transducer, image the lesser saphenous vein in short axis. Applying a tourniquet proximally helps to distend the lesser saphenous vein and facilitates its identification. Insert the block needle in-plane and deposit up to 5 mL of local anesthetic solution circumferentially around the lesser saphenous vein.

Ultrasound-Guided Deep Peroneal Nerve Block

Ⓓ Apply the transducer over the anterior tibia at the level of the superior margin of the malleoli. Superficial to the tibial surface, the deep peroneal nerve can be visualized lateral to the anterior tibial artery (continuing as the dorsalis pedis artery distally). Insert the block needle in-plane and deposit up to 5 mL of local anesthetic solution circumferentially around the deep peroneal nerve or lateral to the anterior tibial artery if the nerve cannot be identified.

Evaluation of Block

Blockade of the deep and superficial peroneal nerves may be assessed by testing sensation in the webspace between the first and second toes and over the dorsum of the foot. Testing sensation along lateral and plantar surfaces of the foot will assess anesthesia in the sural and tibial nerve distributions, respectively.

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Part C: Truncal Blocks

Transversus Abdominis Plane and Rectus Sheath Blocks

Matthew J. Charles • Justin W. Heil • Matthew T. Charous

Transversus abdominis plane (TAP) blocks anesthetize the somatic nerves that innervate the abdominal wall and may provide postoperative analgesia for abdominal and pelvic surgery.[1-4] TAP may be as effective as an epidural in some colorectal surgery,[5] and is an effective alternative to intrathecal opioids for cesarean delivery.[6] The thoracolumbar nerves supplying the abdomen arise from spinal nerves T7-L1 and course through the plane between the transversus abdominis (TA) muscle and the internal oblique muscle—the TAP.[3] By infiltrating the space between these muscles with local anesthetic solution, distal spinal nerves and peripheral branches of L1 (ilioinguinal and iliohypogastric nerves) may be anesthetized. However, the distance between the nerves is substantial, and coverage of the entire abdominal wall requires multiple injections.[7,8] TAP blocks may be used in combination with rectus sheath blocks or subcostal TAP blocks to cover a wider area. Although the TAP block may be performed using surface landmarks and a "double-loss-of-resistance" technique in the triangle of Petit, real-time ultrasound guidance yields better results.[9-11]

1) **Benefits and indications**
   a) Postoperative analgesia in the abdominal, pelvic, or inguinal regions
      i) A TAP catheter and continuous local anesthetic infusion extend the duration of analgesia and may have other benefits.[12]
   b) The surgical site and desired distribution of anesthesia dictate the exact location of the block.
      i) A single-injection posterior TAP block reliably anesthetizes the ipsilateral abdominal wall in the dermatomal distributions of T10-L1.[8] These are ideal for procedures below the umbilicus such as C-section or inguinal hernia repair.
      ii) Subcostal TAP blocks may be used for upper abdominal surgeries.
      iii) Midline incisions require bilateral blocks.
      iv) Extensive incisions spanning multiple dermatomes require multiple injections for adequate coverage.[7,8]
      v) Posterior TAP blocks may be combined with rectus sheath blocks when incisions extend above and below the umbilicus.[13]
2) **Risks and contraindications**
   a) **Absolute contraindications**
      i) Infection at the site of planned needle placement
   b) **Relative contraindications**
      i) Coagulopathy
      ii) Previous abdominal wall surgery may distort anatomy and make TAP block more difficult to perform with and without ultrasound guidance.



TAP blocks only anesthetize somatic nerves; therefore, patients may still experience visceral pain following intra-abdominal or pelvic procedures.



The distribution of a single-injection posterior TAP block is T10-L1 ipsilaterally. A subcostal TAP or rectus sheath block may be used in combination with a posterior TAP block for more complete coverage.

c) **Risks and complications**
   i) General risks
      (1) Nerve injury
      (2) Localized bleeding
      (3) Infection
      (4) Intravascular injection with local anesthetic toxicity
   ii) Site-specific risks and potential complications[14]
      (1) Peritoneal puncture
      (2) Bowel or organ perforation

3) **Equipment**
   a) Common equipment for all peripheral nerve block techniques include sterile skin cleansing solution, sterile gloves, a marking pen, and two 20-mL syringes of local anesthetic solution with extension tubing.
   b) Ultrasound-guided regional anesthesia requires portable ultrasound equipment and a high-frequency linear transducer or a large low-frequency curvilinear transducer (obese patients) for TAP block.



Rectus sheath blocks provide anesthesia at the midline and should be performed bilaterally.

4) **Technique**
   a) **Monitoring**
      i) Peripheral nerve blocks should be placed in a location where the patient's noninvasive blood pressure, EKG, oxygen saturation, and end-tidal CO_2 may be monitored.
      ii) Supplemental O_2 should be provided via a face mask or nasal cannulae, especially when IV sedation is administered.
   b) **Positioning and preparation**
      i) Position the patient supine or lateral with the operative side up.
         (1) The supine position is preferred when performing bilateral TAP blocks for postoperative pain.
         (2) The lateral decubitus position is helpful when placing a unilateral posterior TAP catheter preoperatively.
            (a) Space between costal margin and iliac crest expanded
            (b) Bowel displaced anteriorly, away from needle insertion site
            (c) Catheter site is away from the surgical field.
      ii) Apply monitors and supplemental O_2.
      iii) IV sedation may be administered, but the patient must be able to follow commands and volunteer sensory information.
   c) **Surface anatomy**
      i) The borders of the triangle of Petit serve as surface landmarks (Cognitive Aid: Panel A).
         (1) Iliac crest: inferior
         (2) External oblique muscle (EO): anterior
         (3) Latissimus dorsi muscle: posterior
      ii) For the ultrasound-guided technique, a line can be drawn along the costal margin (cephalad) and the iliac crest (caudad) on the operative side.
   d) **Landmark-based TAP block**
      i) Identify the triangle of Petit.
      ii) After sterile preparation, insert a blunt-tipped block needle (eg, 22-gauge Tuohy-tip needle) perpendicular to skin.
      iii) The needle will penetrate two layers of fascia; thus, two distinct "pops" or losses of resistance will be palpable.
         (1) The first loss of resistance indicates penetration of the EO fascia.
         (2) The second loss of resistance indicates penetration of the internal oblique (IO) fascia and proper placement of the needle tip in the TAP.

e) **Ultrasound-guided TAP block**

i) The patient may be positioned supine (bilateral and/or single-injection TAP blocks) or lateral (preoperative TAP catheter).

ii) Place a high-frequency linear transducer in a transverse orientation on the lateral abdominal wall, cephalad to the iliac crest, along the midaxillary line (Cognitive Aid: Panel B).

iii) Identify the EO, IO, and TA muscles.

iv) Needle placement in the TAP

(1) Posterior approach

(a) After sterile preparation, place a local anesthetic skin wheal posterior to the edge of the ultrasound transducer.

(b) Insert the block needle through this skin wheal using in-plane guidance and advance anteriorly through the EO and IO. A "pop" may be felt and observed under ultrasound.

(2) Anterior approach

(a) After sterile preparation, place a local anesthetic skin wheal anterior to the edge of the ultrasound transducer.

(b) Insert the block needle through this skin wheal and direct it posteriorly in-plane toward the TAP. A "pop" may be felt and observed under ultrasound.



The final location for local anesthetic deposition is in the plane between the IO and TA muscles.

v) Once the needle tip is properly placed between the IO and TA, inject local anesthetic incrementally until a discrete, elliptical fluid deposit can be visually confirmed (Cognitive Aid: Panel C).

(1) Intramuscular local anesthetic injection results in a more diffuse pattern with less clearly defined boundaries.

(2) If intramuscular injectate is detected, advance or withdraw the needle until the tip is positioned within the TAP.

(3) For an effective TAP block, a minimum of 15 mL of local anesthetic should be used.[15]



Preincisional blocks may result in better outcomes.[15]

f) **Ultrasound-guided Rectus Sheath Block**

i) Place a high-frequency linear transducer in a transverse orientation just lateral to the midpoint of the planned surgical incision and anterior to the midaxillary line.

ii) Scan from lateral to medial and visualize the external oblique, internal oblique, and transversus abdominis muscles as they terminate at the linea semilunaris. The anterior cutaneous nerves to the midline abdomen enter the rectus sheath at the lateral border and run in the plane between the rectus muscle and posterior rectus sheath.

iii) After sterile preparation, place a skin wheal just lateral to the ultrasound probe. Insert the block needle and advance in-plane until the tip of the needle passes through the posterior wall of the rectus muscle, but not through the posterior rectus sheath.



The duration of rectus sheath blocks may be less than the duration of TAP blocks, leading to earlier analgesic requirements for extensive midline incisions.[16]

iv) Inject local anesthetic incrementally until a discrete, elliptical fluid deposit is visually confirmed deep to the rectus abdominis muscle, optimally near the lateral border of the sheath.[17]

(1) If intramuscular injectate is detected, advance the needle until the tip is positioned within the rectus sheath.

(2) Repeat the procedure on the opposite side.

5) **Evaluation of the block**
   a) The quality of nerve blockade must be evaluated prior to surgical incision, even if intraoperative general anesthesia is planned.
   b) Sensory testing can be performed by assessing loss of sensation to cold or pinprick in the target dermatomes of the planned surgical procedure.
6) **Postoperative management**
   a) Since there is no significant loss of motor function or risk of pulmonary compromise, patients may be discharged when standard criteria are met.

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193 | US-Guided TAP and Rectus Sheath Blocks

US-Guided Posterior TAP Block

Justin W. Heil • Larry F. Chu • Matthew T. Charous

Patient Position: Supine (bilateral and/or single-injection TAP blocks) or lateral (preoperative TAP catheter).

Needle Size: 22-17 gauge Tuohy-tip or B-bevel block needle.

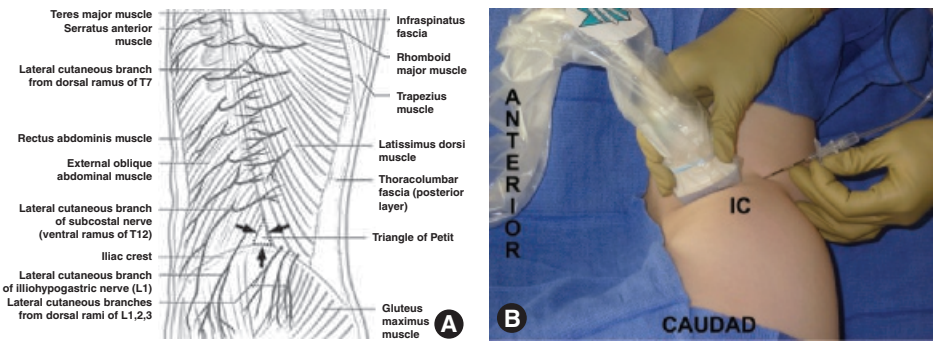
Volume: 20 mL local anesthetic per side.

Anatomic Landmarks

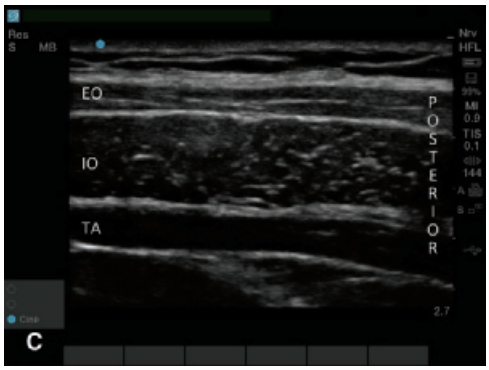
Ⓐ The borders of the triangle of Petit serve as surface landmarks: Iliac crest inferiorly, external oblique muscle (EO) anteriorly, and latissimus dorsi muscle posteriorly. A line can be drawn along the costal margin (cephalad) and the iliac crest (caudad) on the operative side.

Approach and Technique

Ⓑ Place a high-frequency linear transducer in a transverse orientation on the lateral abdominal wall, cephalad to the iliac crest, along the midaxillary line. Visualize the EO, IO, and TA muscles. Place a local anesthetic skin wheal at the site of planned needle insertion. Insert the block needle through this skin wheal using in-plane guidance and advance through the EO and IO. A "pop" may be felt and observed under ultrasound as the needle tip passes into the plane between the IO and TA.



A. Illustration demonstrating the borders of the triangle of Petit and other relevant anatomic landmarks for the TAP block. Reproduced from McDonnell JG, O'Donnell OD, Farrell T, et al. Transversus abdominis plane block: a cadaveric and radiological evaluation. *Reg Anesth Pain Med*. 2007;32:399-404, Fig. 1, pg. 400, with permission from BMJ Publishing Group Ltd. **B.** Position the ultrasound transducer cephalad to the iliac crest in a transverse (anteroposterior) orientation, slightly anterior to the mid-axillary line to image the abdominal wall layers in short axis. For preoperative TAP catheter insertion, position the patient lateral and insert the needle posterior to the transducer, directed anteriorly toward the target layer.



C. Short-axis ultrasound image demonstrating the relevant cross-sectional anatomy for the TAP block. EO, external oblique muscle; IO, internal oblique muscle; TA, transversus abdominis muscle.

- Once the needle tip is properly placed between the IO and TA, inject local anesthetic incrementally until a discrete, elliptical fluid deposit can be visually confirmed. If intramuscular injectate is detected, advance or withdraw the needle until the tip is positioned within the TAP.

Evaluation of Block

Sensory testing

Sensory testing can be performed by assessing loss of sensation to cold or pinprick in the target dermatomes of the planned surgical procedure. A single-injection posterior TAP block with 20 mL of local anesthetic should anesthetize the ipsilateral T10-L1 dermatomes.

US-Guided Rectus Sheath Block

Matthew J. Charles • Justin W. Heil

Patient Position: Position the patient supine for bilateral rectus sheath blocks.

Needle Size: 22-17 gauge Tuohy-tip or B-bevel block needle.

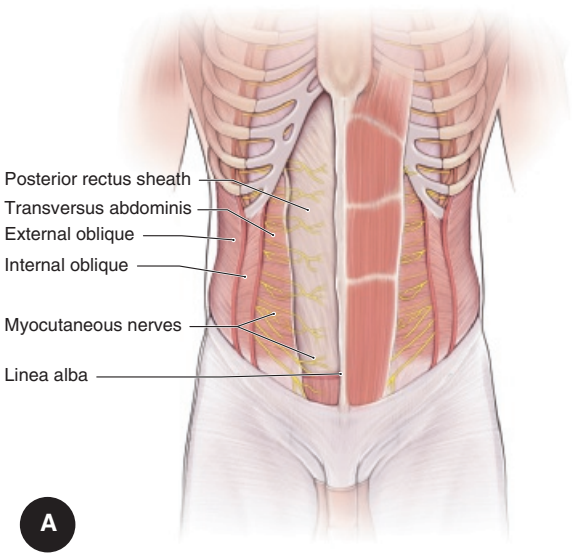
Volume: 10-15 mL local anesthetic per side.

Anatomic Landmarks

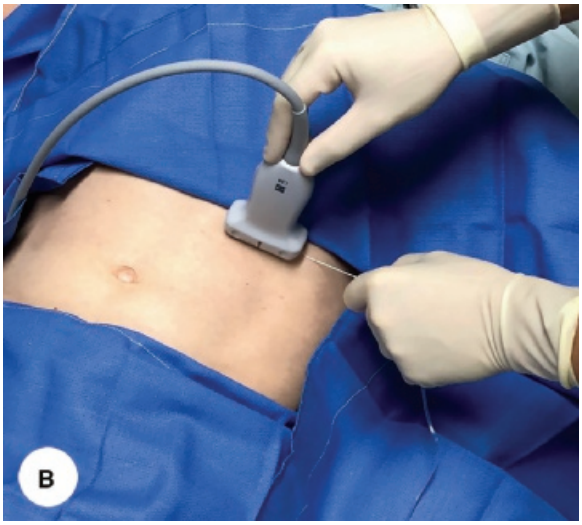
Ⓐ The lateral borders of the rectus abdominis (ie, linea semilunaris), if visible, serve as surface landmarks. The linea semilunaris runs from the ninth rib to the pubic tubercle. Injections should be performed near the site of the planned surgical incision.

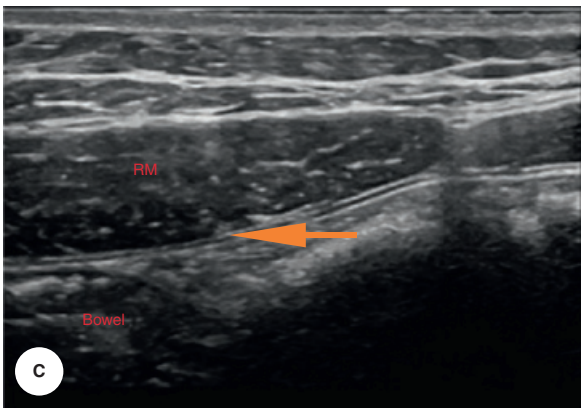
Approach and Technique

Ⓑ Place a high-frequency linear transducer in a transverse orientation just lateral to the midpoint of the planned surgical incision and anterior to the midaxillary line. Scan from lateral to medial and visualize the external oblique, internal oblique, and transversus abdominis muscles as they terminate at the linea semilunaris. The anterior cutaneous nerves to the midline abdomen enter the rectus sheath at the lateral border and run in the plane between the rectus muscle and posterior rectus sheath.



A. Anatomy of the anterolateral abdominal wall for ultrasound-guided rectus sheath block. Place the high-frequency linear ultrasound transducer in a transverse orientation superior to the umbilicus and visualize the lateral aspect of the rectus abdominis muscle and rectus sheath. Near the costal margin the transversus abdominis muscle lies deep to the rectus abdominis muscle; near the pubic symphysis, the posterior rectus sheath is absent, and the transversalis fascia lies deep to the rectus abdominis muscle epimysium. The rectus sheath block is performed in the posterior rectus sheath compartment between the lateral aspect of the rectus abdominis muscle and its deep investing layer of fascia, to target the terminal muscular branches and anterior cutaneous branches of the thoracoabdominal nerves before they ascend through the rectus abdominis muscle to their subcutaneous location.





B. Ultrasound positioning and needle placement for rectus sheath block. **C.** Ultrasound image of rectus muscle and posterior rectus sheath (*orange arrow*). **A, C.** Reprinted with permission from Horn JL, Nasiri M. Regional Blocks–A Brief Overview. In: Jaffe RA, Schmiesing CA, Golianu B, eds. *Anesthesiologist's Manual of Surgical Procedures*. 6th ed. Philadelphia, PA: Wolters Kluwer; 2020:1763-1799.

- Local anesthetic spread between rectus abdominis muscle (RAM) and posterior rectus sheath with transducer placed in transverse orientation above the umbilicus.

Evaluation of Block

Sensory testing

Sensory testing can be performed by checking for loss of sensation to cold or pinprick in the target dermatomes of the planned surgical procedure. A single-injection rectus sheath block with 10 mL of local anesthetic should anesthetize the ipsilateral terminal branches of the T9-T11 intercostal nerves.

194 Paravertebral Block

Justin W. Heil • Matthew J. Charles

Paravertebral block (PVB) can be performed at the thoracic or lumbar levels to provide anesthesia in specific dermatomal distributions, using landmark-based techniques[1–3] or ultrasound guidance.[4,5] The wedge-shaped paravertebral space contains spinal and sympathetic nerves as well as segmental vessels. In the thoracic region, it is bounded anteriorly by the parietal pleura, posteriorly by the superior costotransverse ligament, and medially by the posterolateral aspect of the vertebral column.[6] In the lumbar region, it is bounded anteriorly by the fascia transversalis over the psoas muscle, posteriorly by the ligaments of the lumbar spine, and medially by the vertebral column.[7] Multiple-level injections demonstrate higher efficacy in anesthetizing the desired region over larger volume single injection.[3] Paravertebral catheters may be inserted for continuous perineural infusion, can provide prolonged postoperative analgesia, and typically result in less hypotension than epidural catheters.[8,9] PVBs are easy blocks to learn and perform and effectively provide unilateral analgesia of the thorax and abdomen.

1) **Benefits and indications**
   a) **Surgical anesthesia or postoperative analgesia for breast surgery, thoracotomy, cholecystectomy, inguinal herniorrhaphy, and other thoracic and abdominal procedures**
      i) Select levels based on anticipated dermatomes to be included in planned surgical site.
      ii) Consider pectoral nerve block for surgical anesthesia of the breast; pectoral nerves originate from the brachial plexus, not from thoracic spinal nerves.
   b) **Bilateral PVBs may be performed to provide analgesia for bilateral breast surgery[10] or midline thoracic or abdominal procedures.**[11]
   c) **Relief of chronic painful conditions such as:**
      i) Complex regional pain syndrome
      ii) Neuropathic chest or abdominal pain (postsurgical or postherpetic)
      iii) Refractory angina pectoris
      iv) Relief of cancer pain
      v) Therapeutic control of hyperhidrosis
2) **Risks and contraindications**
   a) **Absolute contraindications**
      i) Local infection at the site of planned needle placement
   b) **Relative contraindications**
      i) Coagulopathy or anticoagulation therapy
      ii) Spine deformities that would increase the risk of pleural puncture
      iii) Local tumors
      iv) Preexisting pulmonary disease may increase morbidity from pneumothorax.
   c) **Risks and complications**
      i) General risks
         (1) Nerve injury
         (2) Localized bleeding
         (3) Infection
         (4) Intravascular injection with local anesthetic toxicity

ii) Site-specific risks and potential complications
(1) Pneumothorax
(2) Hypotension from local anesthetic-induced sympathectomy (may be pronounced for bilateral PVBs)
(3) Inadvertent intrathecal or epidural injection

3) **Equipment**
a) Common equipment for all peripheral nerve block techniques include sterile skin cleansing solution, sterile gloves, a marking pen, and two 20-mL syringes of local anesthetic solution with extension tubing.
b) A nerve stimulator technique requires an electrical nerve stimulator with a compatible insulated needle and a grounding electrode.
c) Ultrasound-guided regional anesthesia requires portable ultrasound equipment and a low-frequency large curvilinear transducer (preferred) or high-frequency linear transducer for PVB.

4) **Technique**
a) **Monitoring**
i) Peripheral nerve blocks should be placed in a location where the patient's noninvasive blood pressure, ECG, oxygen saturation, and end-tidal CO_2 may be monitored.
ii) Supplemental O_2 should be provided via a face mask or nasal cannulae, especially when IV sedation is administered.
b) **Positioning and preparation**
i) Position the patient sitting with the neck flexed. Placing a bedside table in front of the patient to rest the arms may provide more stability during the procedure.
ii) The patient may also be placed in the lateral decubitus or prone position if unable to maintain the sitting position. The prone position is preferred during the in-plane ultrasound-guided technique.
iii) Apply monitors and supplemental O_2.
iv) IV sedation may be administered, but the patient must be able to follow commands and volunteer sensory information.
c) **Surface anatomy**
i) The spinous processes (SPs) of the vertebrae representing the target dermatomal levels are the most important surface landmarks.
ii) Start by marking the SP one level above the desired target area, progressing caudally until all the relevant dermatomes are marked.
iii) Mark the point 2.5 cm lateral to each marked SP on the affected side (Cognitive Aid: Panel A).
(1) Each of these lateral marks should overlie the transverse process (TP) of the next caudal vertebrae in the thoracic region.
(2) These marks will be the sites for needle insertion when performing landmark-based PVB (**Table 194-1**).
d) **Landmark-based PVB**
i) Using sterile technique and following injection of a local anesthetic skin wheal at each planned insertion site, insert a 22-gauge Tuohy-tip needle or a short insulated block needle perpendicular to the skin at each marked point.
ii) Advance the needle until it contacts TP.
(1) The distance from skin to TP is ~2-4 cm (thoracic levels) or 4-6 cm (lumbar levels).
(2) This distance is highly variable and is affected by body habitus.[14]



Due to the angulation of the thoracic TPs and SPs, the mark 2.5 cm lateral to each SP should overlie the TP of the vertebra one level caudad.



Despite depth estimates, the exact distance from skin to TP is highly variable. Real-time ultrasound guidance is recommended, but ultrasound can also be used as a "scout" to estimate TP depth prior to employing landmark-based techniques.

TABLE 194-1 Recommended Levels for PVB by Surgical Procedure[7,12,13]

Surgical Procedure	Suggested Levels
Mastectomy with axillary node dissection	T1-T6
Breast biopsy or lumpectomy	T2-T5 (or planned dermatomes if needle localization is performed preoperatively)
Inguinal hernia	T11-L2
Lithotripsy	T8-L1
Thoracotomy or thoracoscopy	Variable depending on involved dermatomes
Cholecystectomy	T8-T10

(3) Ultrasound guidance is helpful in estimating depth to TP and confirming surface landmarks, even if not used for real-time guidance.[12,15]

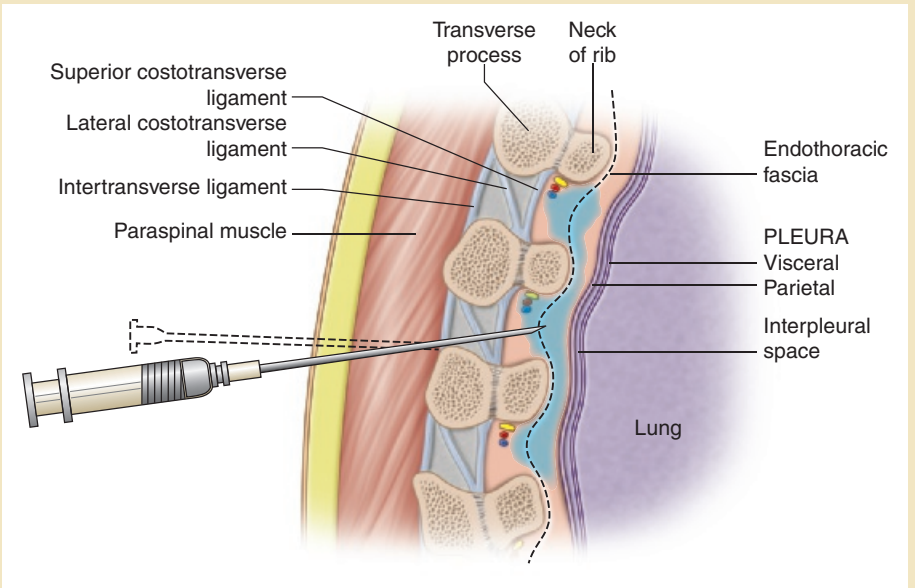
iii) Withdraw the needle and walk off the cephalad or caudad aspect of the TP (Fig. 194-1).

iv) Once the needle clears the TP, advance the needle up to 1 cm (thoracic levels) or 0.5 cm (lumbar levels) maximum.[7]

(1) If a tactile loss-of-resistance is identified when passing through the superior costotransverse ligament (thoracic level only), do not advance further.

(2) If employing a nerve stimulator technique, an intercostal (thoracic levels) or a unilateral abdominal wall (thoracic or lumbar levels) evoked motor response at <0.8 mA serves as the needle position end point.[12]

FIGURE 194-1 Illustrated Sagittal Section Through the Thoracic Paravertebral Region Demonstrating Needle Advancement into the Paravertebral Space and Relevant Anatomy.



Adapted with permission from Karmakar MK. Thoracic paravertebral block. *Anesthesiology*. 2001;95(3):771-780.

v) At each level, inject 4-5 mL of local anesthetic solution after negative aspiration for blood.

e) **Ultrasound-guided PVB**

i) Although varying approaches have been described,[4,5] no ultrasound-guided PVB technique is considered standard to date.

ii) When using a high-frequency linear transducer for out-of-plane needle guidance, position the patient sitting or in lateral decubitus, similar to the landmark-based techniques.

(1) Apply the transducer perpendicular to skin in a parasagittal plane ~2 cm lateral to the midline and oriented parallel to the spine.

(2) Identify the TP of the vertebral bodies cephalad and caudad to the target paravertebral space.

(3) Visualize the pleura anterior to the paravertebral space and note the depth.

(4) Insert the block needle perpendicular to skin next to the ultrasound transducer after injecting a local anesthetic skin wheal and advance the needle out-of-plane toward the paravertebral space (Cognitive Aid: Panel B).

(a) When the desired depth is achieved based on visualization of needle tip or tissue movement in the target area, or at a predetermined depth, inject 4-5 mL of local anesthetic solution after negative aspiration for blood.

(b) Injectate spread should be confirmed within the paravertebral space under ultrasound. Movement of the pleura anteriorly provides evidence for a properly placed injection.

(c) Repeat this process for each subsequent level.

iii) When performing in-plane needle guidance, position the patient sitting or prone.

(1) The large low-frequency curvilinear transducer is preferred because it enables the practitioner to visualize the needle at steeper angles.

(2) Apply the transducer perpendicular to skin in a parasagittal plane ~2 cm lateral to the midline and oriented parallel to the spine (Cognitive Aid: Panel C).

(3) Identify the TP of the vertebral bodies cephalad and caudad to the target paravertebral space and the pleura anteriorly (Cognitive Aid: Panel D).

(4) With the large curvilinear transducer, often more than one paravertebral space may be visualized. This allows the performance of more than one PVB by simply redirecting the needle cephalad or caudad using a single needle insertion.

(5) Place a local anesthetic skin wheal caudad to the ultrasound transducer and insert the block needle through the skin wheal, directed cephalad toward the paravertebral space.

(a) When the needle tip is visualized within the target paravertebral space, inject 4-5 mL of local anesthetic solution after negative aspiration for blood.



When performing landmark-based PVB, only one of three criteria (loss of resistance, predetermined depth, or evoked motor response) needs to be met before stopping needle advancement.



The pleura forms the anterior border of the paravertebral space, so use caution when advancing the needle past the TP to avoid pneumothorax.



The paravertebral space is easiest to visualize laterally, where it is closer to the skin and not shadowed by the transverse process, but performing the block more medially where the space is thicker may reduce the chance of pleural puncture.[16]



Although a transverse needle approach is commonly used, the rate of epidural spread may be higher since the needle is directed at the neural foramen.[8]

(b) Injectate spread should be confirmed within the paravertebral space under ultrasound.
(c) Repeat this process for each subsequent level.

5) **Evaluation of the block**
   a) The quality of nerve blockade must be evaluated prior to surgical incision, even if intraoperative general anesthesia is planned.
   b) Sensory testing can be performed by assessing for loss of sensation to cold or pinprick in the region of the surgical procedure.
6) **Postoperative management**
   a) Patients may be discharged as outpatients following PVB.
   b) Symptoms of respiratory compromise should prompt evaluation for pneumothorax, but routine postoperative chest x-ray in the absence of symptoms is not warranted.



If a patient presents with shortness of breath after PVB, the diagnosis is pneumothorax until proven otherwise.

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195 US-Guided Paravertebral Block

Justin W. Heil • Larry F. Chu • Matthew J. Charles

Patient Position: Position the patient sitting with the neck flexed. If the patient is unable to sit, the prone position is preferred.

Needle Size: 22-gauge, Tuohy-tip block needle

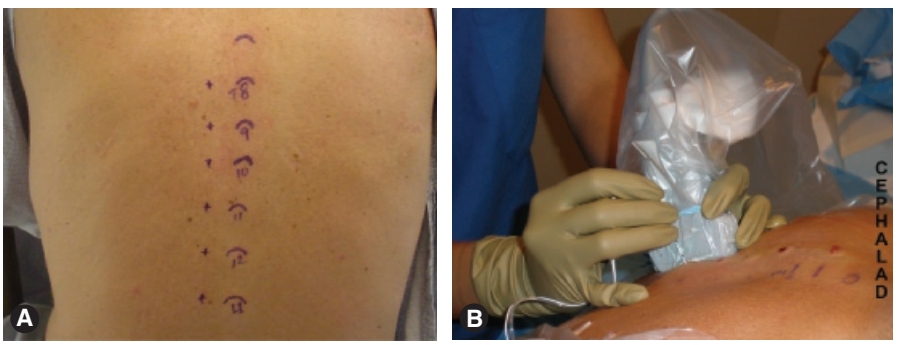
Volume: 5 mL local anesthetic per paravertebral space

Anatomic Landmarks

Ⓐ Mark a point 2.5 cm lateral to each spinous process.

Approach and Technique

Ⓑ Out-of-plane needle guidance: Apply transducer perpendicular to skin in a parasagittal plane 2 cm lateral to the midline and parallel to the spine. Insert the block needle perpendicular to the skin after injecting a local anesthetic skin wheal and advance the needle out-of-plane toward the paravertebral space. When the needle tip passes the superior costotransverse ligament (thoracic) or the desired depth is achieved (~1 cm past the transverse process for thoracic; 0.5 cm past the transverse process for lumbar), inject 5 mL of local anesthetic solution after negative aspiration for blood. Repeat process for each level.



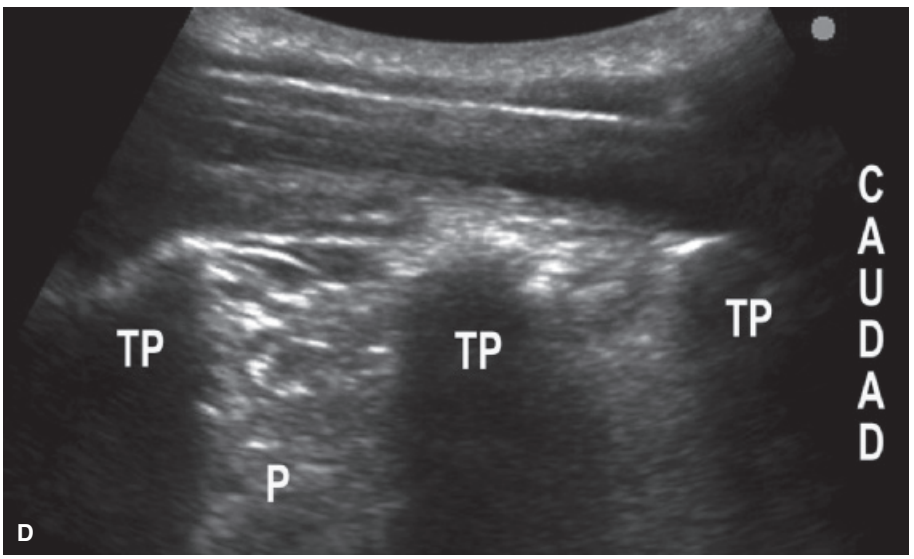
A. When performing out-of-plane thoracic paravertebral block, identify the spinous processes of the vertebrae representing the desired dermatomal levels of anesthesia. Mark the approximate sites for needle insertion 2.5 cm lateral to each spinous process over the transverse process of the vertebra one level caudad. **B.** Transducer positioning and needle insertion technique during out-of-plane ultrasound-guided paravertebral block.

Ⓒ In-plane needle guidance: Apply the transducer perpendicular to the skin in a parasagittal plane 2 cm lateral to the midline and parallel to the spine.



C. Transducer positioning and needle insertion technique during in-plane ultrasound-guided paravertebral block.

Ⓓ Identify the TP of the vertebral bodies cephalad and caudad to the target paravertebral space and the pleura anteriorly. Inject a local anesthetic skin wheal caudad to the ultrasound transducer, and insert the block needle through the wheal directed in-plane toward the paravertebral space. When the needle tip is visualized within the target space, inject 5 mL of local anesthetic solution after negative aspiration for blood. Repeat this process for each level.



D. Ultrasound image of the paravertebral space. TP, transverse process; P, pleura.

Evaluation of Block

The quality and extent of nerve blockade must be evaluated prior to surgical incision even if intraoperative general anesthesia is planned. Sensory testing can be performed by checking for loss of sensation to cold or pinprick in the distribution of the desired dermatomes.

196 Quadratus Lumborum Block

Ava Alamdari • Vanessa J. Loland

The quadratus lumborum block (QLB) is a plane block approached from the posterior abdominal wall and provides analgesia of the abdomen, hip, and femur. There are three potential injection sites, named by their relative positions to the quadratus lumborum muscle (QLM). The QLB is considered to be a safe regional technique with few known complications. Outpatients require careful consideration because of the potential for unintended lower extremity weakness.

1) **Benefits and Indications**
   a) Surgical anesthesia or postoperative analgesia of the abdomen,[1-6] hip,[7-9] and femur.[10]
   b) The lateral QLB, which anesthetizes the ilioinguinal and iliohypogastric nerves along with subcostal nerves, can cover dermatomes T10-L1.
   c) The anterior and posterior QLBs can cover dermatomes from T7-L1, but extension to the L2 and L3 dermatomes has been reported. This spread is likely through paravertebral planes.[3]
2) **Risks and Contraindications**
   a) **Absolute contraindications**
      i) Local infection at the site of planned needle insertion
   b) **Relative contraindications**
      i) Coagulopathy
      ii) Preexisting nerve injury
   c) **Risks and complications**
      i) General risks
         (1) Nerve injury
         (2) Localized bleeding
         (3) Infection
         (4) Intravascular injection with local anesthetic toxicity
      ii) Site-specific risks and potential complications
         (1) Unanticipated lower extremity weakness from possible spread of local anesthetic to the lumbar plexus, especially with the transmuscular QLB (QL3 block).[11-13] This can be especially problematic for outpatient cases.
         (2) Inadvertent puncture of intraabdominal or retroperitoneal structures, such as the kidney
      iii) Pros and cons of QLB are summarized in **Table 196-1**.
3) **Equipment**
   a) Common equipment for all peripheral nerve block techniques include sterile skin cleansing solution, sterile gloves, a marking pen, and two 20-mL syringes of local anesthetic solution with extension tubing.
   b) A 100 or 150 mm echogenic block needle or catheter kit
   c) Ultrasound-guided regional anesthesia requires portable ultrasound equipment and a low-frequency curved or high-frequency linear array transducer for QLB.

TABLE 196-1 Quadratus Lumborum Block Pros and Cons

Pros	Cons
• Potentially safer alternative to neuraxial techniques for patients with hemodynamic compromise or mild coagulopathy • Compared to the transversus abdominis plane (TAP) block, the target injection site is farther from the peritoneal cavity and visceral organs, and avoids vascular structures such as the deep circumflex iliac arteries • Can achieve larger dermatomal spread from T7-L3 in comparison to T10-L1 with the TAP block	• For postoperative patients or patients experiencing pain, the lateral or semilateral positioning for the QLB may be more challenging in comparison to the supine positioning for a TAP block • Neuraxial technique would provide a bilateral block with a single procedure • Potentially challenging in obese patients due to depth and/or ambiguity of structures on ultrasound imaging

4) **Technique**

a) **Monitoring**

i) Peripheral nerve blocks should be placed in a location where the patient's noninvasive blood pressure, EKG, oxygen saturation, and end-tidal CO_2 may be monitored.

ii) Supplemental O_2 should be provided via a face mask or nasal cannulae, especially when IV sedation is administered.

b) **Positioning and preparation**

i) Position the patient in either a lateral or semilateral position with a hip bump on the ipsilateral side.

ii) Apply monitors and supplemental O_2.

iii) IV sedation can be administered, but the patient must be able to follow commands and volunteer sensory information.

c) **Surface anatomy**

i) In the midaxillary line, identify iliac crest and costal margin; the transducer should be placed between these two landmarks.

d) **Ultrasound-guided quadratus lumborum block**

i) The QLB is performed with real-time ultrasound guidance.

ii) Place the ultrasound transducer in a transverse plane level with the area between the iliac crest and the costal margin. Begin scanning over the anterior abdomen (around the midclavicular line) to identify the external oblique, internal oblique, and transversus abdominis muscles arranged as three parallel structures.

iii) Scan posteriorly in the transverse plane, keeping the three muscles in view and maintaining the probe angle perpendicular to the body, until the transversus abdominis muscle tapers and the beginning of the quadratus lumborum muscle (QLM) appears.

iv) After sterile skin preparation, place a local anesthetic skin wheal anterior or posterior to the ultrasound transducer, depending on specific QLB trajectory preference.

v) Insert the block needle through the skin wheal and advance it toward the QLM. There are three potential injection sites around the QLM, all of which are performed with in-plane needle technique:

(1) Lateral QLB (QL1)[14]: Use a linear probe. Point the needle toward the lateral aspect of the QLM, where the transversus abdominis muscle tapers. Inject local anesthetic between the lateral border of the QLM and the transversalis muscle or fascia, ensuring spread along the anterior aspect of the QLM.

(2) Posterior QLB (QL2)[14]: Use a linear probe. Point the needle anterior-to-posterior toward the posterior aspect of the QLM, where the latissimus dorsi muscle can be seen superficial to the QLM. Deposit local anesthetic deep to the thoracolumbar fascia between the latissimus dorsi muscle and the QLM.

(3) Anterior/transmuscular QLB (QL3)[15]: Use a large curved array probe. Point the needle posterior-to-anterior through the QLM and inject local anesthetic between the QLM and the psoas major muscle.

5) **Evaluation of the block**
   a) **The quality of nerve blockade must be evaluated prior to surgical incision, even if intraoperative general anesthesia is planned.**
   b) **Motor testing**
      i) A motor block is not expected with a QLB; however, ipsilateral lower limb strength should be tested with resisted hip flexion and knee extension to evaluate the hip flexors and quadriceps strength, respectively.
   c) **Sensory testing**
      i) Can be tested with ice or pinprick to establish dermatomal distribution and spread
6) **Postoperative management**
   a) Evaluation of lower extremity strength to assess for unintended weakness.
   b) Patients may be discharged home without any special equipment, provided there is no motor weakness on examination.



Local anesthetic is thought to spread into the paravertebral space with a QLB[16] and is likely most pronounced with the QL2 or QL3 blocks.[14,15] These may therefore result in a broader block.



It is important to check for lower extremity weakness, especially for patients anticipating discharge, to avoid potential fall risk.

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197 US-Guided Quadratus Lumborum Block

Ava Alamdari • Vanessa J. Loland

Patient Position: Position the patient in either a lateral or semilateral position with a hip bump on the ipsilateral side.

Needle Size: 22-17 gauge, 100 to 150-mm block needle

Volume: 30 mL local anesthetic

Anatomic Landmarks

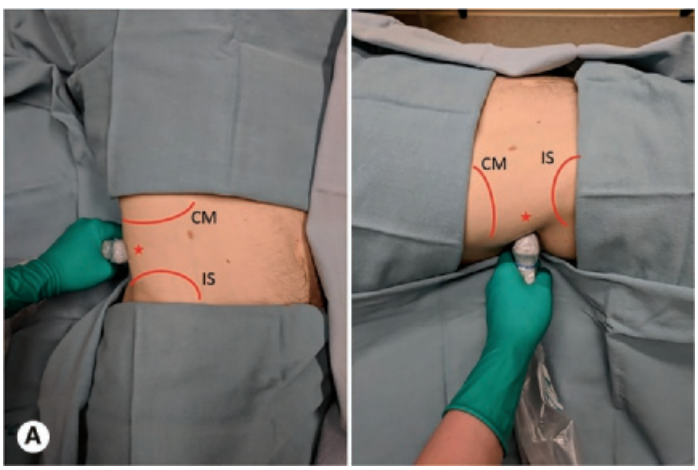
Ⓐ In the midaxillary line, identify iliac crest and costal margin; the transducer should be placed between these two landmarks.

Approach and Technique

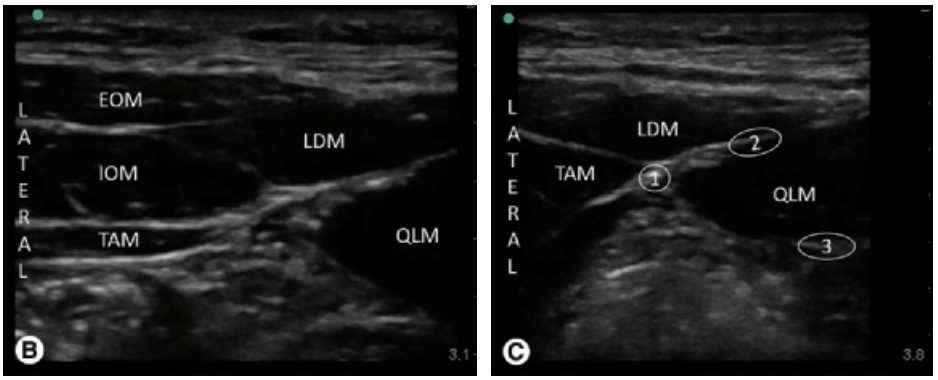
Ⓑ Scan posteriorly in the transverse plane, keeping the three muscles in view and maintaining the probe angle perpendicular to the body, until the transversus abdominis muscle tapers and the beginning of the quadratus lumborum muscle (QLM) appears.

Ⓒ After sterile skin preparation, place a local anesthetic skin wheal anterior or posterior to the ultrasound transducer, depending on specific QLB trajectory preference. Insert the block needle through the skin wheal and advance it toward the QLM. There are three potential injection sites around the QLM, all of which are performed with in-plane needle technique:

(1) Lateral QLB (QL1): Use a linear probe. Point the needle toward the lateral aspect of the QLM, where the transversus abdominis muscle tapers. Inject local anesthetic between the lateral border of the QLM and the transversalis muscle or fascia, ensuring spread along the anterior aspect of the QLM.



A. The patient is positioned here in a semilateral position with a hip bump to allow access to the posterior flank area. The transducer is placed between the costal margin (CM) and iliac spine (IS).



B. Ultrasound image showing the external oblique muscle (EOM), internal oblique muscle (IOM), and transversus abdominis muscle (TAM) arranged as three parallel structures. The quadratus lumborum muscle (QLM) appears as the TAM tapers, with the latissimus dorsi muscle (LDM) overlying it. **C.** Ultrasound image showing the three potential injection sites for QLB: lateral QLB (QL1), posterior QLB (QL2), and anterior/transmuscular (QL3)

(2) Posterior QLB (QL2): Use a linear probe. Point the needle anterior-to-posterior toward the posterior aspect of the QLM, where the latissimus dorsi muscle can be seen superficial to the QLM. Deposit local anesthetic deep to the thoracolumbar fascia between the latissimus dorsi muscle and the QLM.
(3) Anterior/transmuscular QLB (QL3): Use a large curved array probe. Point the needle posterior-to-anterior through the QLM and inject local anesthetic between the QLM and the psoas major muscle.

Evaluation of Block

Motor Testing

Motor loss is not expected but should be tested. Evaluate the strength of hip flexors using a resisted straight leg raise, and of quadriceps using resisted knee extension.

Sensory Testing

Using ice or pinprick, evaluate the dermatomal spread in a systematic manner. Begin testing in the mid/upper chest where sensation is intact and gradually descend until a change in sensation is appreciated to establish the upper limit of the block. Continue until normal sensation returns to establish the lower limit of the block.

198 Pecs Block

Matthew W. Lawrence • Vanessa J. Loland

The Pecs block is commonly used for surgery involving the breast and chest wall. As it was originally described, Pecs I involves injection of local anesthetic between the pectoralis major and minor muscles to block the medial and lateral pectoral nerves.[1] The Pecs II block is a modification of Pecs I that provides an additional injection deep to the pectoralis minor muscle.[2] This second injection blocks the lateral cutaneous branches of the intercostal nerves from T2-T6, along with the thoracodorsal and long thoracic nerves. Pecs blocks are primarily used for postoperative analgesia following breast surgery.[3-5] Additional indications will likely emerge with future study and refinement of fascial plane block techniques.

1) **Benefits and indications**
   a) Postoperative analgesia for surgery involving the breast and chest wall including placement of tissue expanders, subpectoral prostheses, mastectomy, lumpectomy with wide local excision, axillary dissection, and sentinel node biopsy
   b) Case reports support a role of Pecs I and II block for insertion of cardiac devices,[6] Port-a-caths, chest drains, as well as vascular surgery involving the upper arm where blockade of the intercostobrachial nerve is required.[7]



Pecs I block is particularly useful for relief of pain and muscle spasm associated with placement of tissue expanders and subpectoral prostheses.

2) **Risks and contraindications**
   a) **Absolute contraindications**
      i) Local infection at needle entry point
      ii) Patient refusal
   b) **Relative contraindications**
      i) Coagulopathy should be considered as it may predispose to hematoma formation, although strict adherence to guidelines for deep peripheral or neuraxial blocks is not required.
      ii) Pecs II block may hinder perioperative monitoring of the long thoracic and thoracodorsal nerves and should not be performed if such monitoring is required.
   c) **Risks and complications**
      i) General risks
         (1) Nerve injury
         (2) Localized bleeding
         (3) Infection
         (4) Intravascular injection with local anesthetic toxicity
      ii) Site-specific risks and potential complications
         (1) A study reviewing the complications associated with Pecs I and II block for 498 patients found hematoma to be the most frequent adverse event with an incidence of 1.6%. Pneumothorax was not observed among any of the 498 patients using an ultrasound-guided technique.[8]
      iii) Pros and cons of Pecs block are summarized in Table 198-1.



Hematoma is a potential complication of Pecs blocks, especially in patients receiving anticoagulant and antiplatelet medications.

TABLE 198-1 Pecs Block Pros and Cons vs Thoracic Epidural or Paravertebral Block

Pros	Cons
• Technically simple to perform • Less invasive and lower risk of complications compared to thoracic epidural and paravertebral techniques • Anesthetizes pectoral nerves in addition to lateral cutaneous branches of intercostal nerves • Does not require adherence to neuraxial anticoagulation guidelines • May be performed in awake or anesthetized patient • May be more suitable for ambulatory or short stay surgery • No sympathectomy • No risk of intrathecal spread of local anesthetic • Low risk of pneumothorax	• Requires additional injection (transversus thoracis plane) to anesthetize medial portion of chest supplied by anterior cutaneous branches of the intercostal nerves • Large volume of local anesthetic required when performing bilateral blocks • Continuous techniques not extensively studied

3) **Anatomy of the pectoral region**
   a) **Innervation of the breast and chest wall**
      i) Medial pectoral nerve (C8-T1) and lateral pectoral nerve (C5-C7)
         (1) Branches arise from the medial and lateral cords of the brachial plexus to supply sensory, motor, and sympathetic innervation to the pectoralis muscles.
         (2) The pectoral nerves course through the plane separating pectoralis major and minor. The medial pectoral nerve travels beneath pectoralis minor and penetrates the muscle more distally in a majority of patients. Otherwise, it is located along the lateral border of the pectoralis minor muscle.[9]
      ii) Thoracodorsal nerve (C6-C8)
         (1) Arises from the posterior cord of the brachial plexus
         (2) Innervates latissimus dorsi muscle
      iii) Long thoracic nerve (C5-C7)
         (1) Originates from the anterior rami of C5-C7 spinal nerve roots
         (2) Innervates serratus anterior muscle
      iv) Lateral intercostal cutaneous nerves (T2-T6)
         (1) The lateral branches of the intercostal nerves supply cutaneous sensation to the chest wall and axilla.
         (2) The lateral cutaneous branch of the second intercostal nerve forms the intercostobrachial nerve, which passes through the axilla to supply the skin of the upper medial and posterior aspect of the arm.
      v) Anterior branches of the intercostal cutaneous nerves
         (1) Innervate the medial cutaneous and mammary area
         (2) These branches are not blocked by Pecs II but may be anesthetized with a separate injection to the transversus thoracis plane (TTP).[10,11]

4) **Equipment**
   a) Common equipment for all peripheral nerve block techniques include sterile skin cleansing solution, sterile gloves, a marking pen, block needle, and two 20-mL syringes of local anesthetic solution with extension tubing.
   b) Ultrasound-guided regional anesthesia requires portable ultrasound equipment and a high-frequency linear (6-13 MHz) transducer for Pecs block.



Pecs I block provides anesthesia to the medial and lateral pectoral nerves but does not affect cutaneous sensation arising from the intercostal nerves.

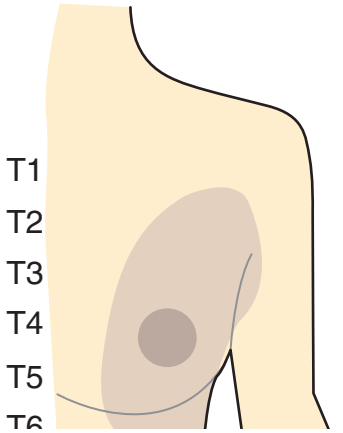
5) **Technique**
   a) **Monitoring**
      i) Peripheral nerve blocks should be placed in a location where the patient's noninvasive blood pressure, EKG, oxygen saturation, and end-tidal CO_2 may be monitored.
      ii) Supplemental O_2 should be provided via a face mask or nasal cannulae, especially when IV sedation is administered.
   b) **Positioning and preparation**
      i) Position the patient supine with the ipsilateral arm abducted.
      ii) Stand on the contralateral side or at the head of the bed.
   c) **Surface anatomy**
      i) The infraclavicular block site is useful for orientation purposes.
      ii) As with the infraclavicular block, begin scanning in the parasagittal plane medial to the coracoid process and inferior to the clavicle.
   d) **Ultrasound-guided Pecs block** (Cognitive aid, Chapter 199: Panels A-C)
      i) Relevant sonoanatomy: Pecs I
         (1) Pectoralis major is the most superficial muscle layer.
         (2) Pectoralis minor lies deep to pectoralis major. It has a fanlike distribution arising from the 3rd through 5th ribs and inserting on the coracoid process of the scapula.
         (3) The axillary artery and vein, and the cords of the brachial plexus are located deep to the pectoralis minor muscle.
         (4) The 2nd rib may be visualized deep and slightly caudal to the neurovascular bundle.
      ii) Relevant sonoanatomy: Pecs II
         (1) Translocate the probe caudally, bringing the 3rd and 4th ribs into view.
         (2) Rotate the probe to view the ribs in short axis. Slide the probe laterally until the serratus anterior muscle can be visualized deep to pectoralis minor.
            (a) The pectoral branch of the thoracoacromial artery may be identified in the plane between pectoralis major and minor. The lateral pectoral nerve frequently travels with this artery, making it a helpful sonographic landmark to guide injection within the plane.



The pectoral branch of the thoracoacromial artery lies in the plane between pectoralis major and minor. It is a useful landmark for the Pecs block.

      iii) Ultrasound-guided approach: Pecs I (Cognitive aid, Chapter 199: Panels A and B)
         (1) Pecs I may be performed at the infraclavicular site or more caudally at the level of the 3rd and 4th ribs.
         (2) If performing Pecs I in an awake patient, place a local anesthetic skin wheal prior to needle placement.
         (3) Advance the block needle in-plane to the fascial plane between pectoralis major and minor.
         (4) After negative aspiration for blood, incrementally inject 10 mL of local anesthetic to expand the fascial plane separating the pectoralis muscles.
      iv) Ultrasound-guided approach: Pecs II (Cognitive aid, Chapter 199: Panel C)
         (1) Pecs II is an extension of Pecs I and is performed at the level of the 3rd and 4th ribs.
         (2) After injecting 10 mL of local anesthetic between pectoralis major and minor as described above, advance the needle to the fascial plane between pectoralis minor and serratus anterior.
         (3) Incrementally inject 20 mL of local anesthetic with intermittent aspiration in the plane separating pectoralis minor and serratus anterior.

FIGURE 198-1 Dermatomal Distribution of Pecs II Block.



(4) Separation of the plane between pectoralis minor and serratus anterior may be technically challenging at its most anterior location. Local anesthetic injection for Pecs II can alternatively be placed deep, rather than superficial, to serratus anterior.
   a) Using the superficial aspect of the rib as a needle endpoint prevents entry into the intercostal space.

6) **Evaluation of the block**
   a) **Sensory testing**
      i) Dermatomal distribution of the Pecs II block (which includes the Pecs I injection) should be checked by loss of sensation to pinprick or cold from the parasternal area to the lateral aspect of the chest (**Fig. 198-1**).
      ii) The typical dermatomal distribution is from T2 to T4, possibly extending to T6.
      iii) Blockade of the intercostobrachial nerve (T2 intercostal nerve) produces sensory changes extending to the axilla and medial aspect of the upper arm.
      iv) The medial aspect of the chest supplied by anterior branches of the intercostal nerves will not be covered without additional injection to the TTP.

7) **Postoperative management**
   a) **Patients may be discharged when standard criteria are met.**

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199 US-Guided Pecs Block

Vanessa J. Loland • Matthew W. Lawrence

Patient Position: Position the patient supine with the ipsilateral arm abducted. Stand on the contralateral side or at the head of the bed. Begin scanning in the parasagittal plane medial to the coracoid process and inferior to the clavicle.

Needle Size: 22- to 17-gauge, 50- to 100-mm short bevel block needle.

Volume: 20-40 mL local anesthetic.

Anatomic Landmarks

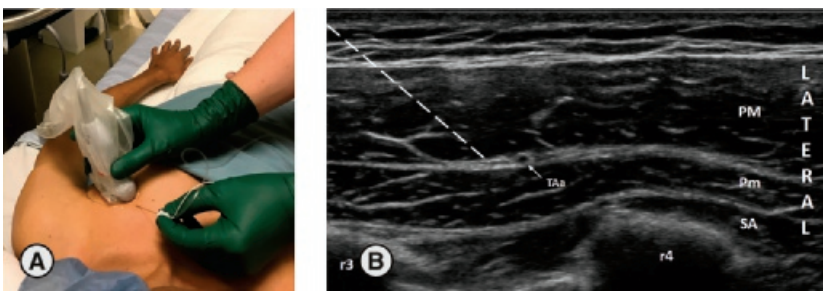
Ⓐ Pectoralis major is the most superficial muscle layer. Pectoralis minor lies deep to pectoralis major. It has a fanlike distribution arising from the 3rd through 5th ribs and inserting on the coracoid process of the scapula. The axillary artery and vein, and the cords of the brachial plexus are located deep to the pectoralis minor muscle. The 2nd rib may be visualized deep and slightly caudal to the neurovascular bundle.

Translocating the probe caudally brings the 3rd and 4th ribs into view. Rotate the probe to view the ribs in short axis. Slide the probe laterally until the serratus anterior muscle can be visualized deep to pectoralis minor. The pectoral branch of the thoracoacromial artery may be identified in the plane between pectoralis major and minor. The lateral pectoral nerve frequently travels with this artery, making it a helpful sonographic landmark to guide injection within the plane.

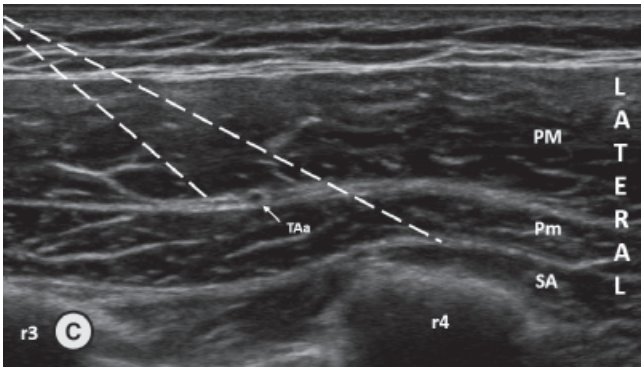
Approach and Technique

Ⓑ Pecs I may be performed at the infraclavicular site or more caudally at the level of the 3rd and 4th ribs. If performing Pecs I in an awake patient, place a local anesthetic skin wheal prior to needle placement. Advance the block needle in-plane to the fascial plane between pectoralis major and minor. After negative aspiration for blood, incrementally inject 10 mL of local anesthetic to expand the fascial plane separating the pectoralis muscles.

Ⓒ Pecs II is an extension of Pecs I and is performed at the level of the 3rd and 4th ribs. After injecting 10 mL of local anesthetic between pectoralis major and minor as described above, advance the needle to the fascial plane between pectoralis minor and serratus



A, B. Probe position and needle entry for Pecs block (**A**). Sonogram illustrating relevant anatomy for Pecs I and II block. (I) *dashed line* indicates the needle path and injection plane for Pecs I block (**B**).



C. The longer dashed line indicates the additional point of injection for Pecs II block (note: needle may be advanced along same path for both injections). PM, pectoralis major muscle; Pm, pectoralis minor; SA, serratus anterior; r3, 3rd rib; r4, 4th rib; TAa, pectoral branch of thoracoacromial artery.

anterior. Incrementally inject 20 mL of local anesthetic with intermittent aspiration in the plane separating pectoralis minor and serratus anterior. If separation of the plane between pectoralis minor and serratus anterior is technically challenging at this location, local anesthetic injection can alternatively be placed deep, rather than superficial, to serratus anterior. Using the superficial aspect of the rib as a needle endpoint prevents entry into the intercostal space.

Evaluation of Block

Sensory testing

Dermatomal distribution of the Pecs II block (which includes the Pecs I injection) should be checked by loss of sensation to pinprick or cold from the parasternal area to the lateral aspect of the chest. The typical dermatomal distribution is from T2 to T4, possibly extending to T6. Blockade of the intercostobrachial nerve (T2 intercostal nerve) produces sensory changes extending to the axilla and medial aspect of the upper arm.

200 Part D: Neuraxial Block
Lumbar Neuraxial Ultrasound

Mastoora Nasiri • M. Kwesi Kwofie

Preprocedural ultrasound of the lumbar spine may substantially increase the success, ease of placement, and potentially the safety of lumbar neuraxial anesthesia and analgesia. Improvements are most beneficial in patients who are obese, elderly, and have surgical or anatomic spinal deformities[1]; these populations are often both the most technically challenging and potentially likely to benefit from avoidance of general anesthesia.

1) **Indications and benefits**
   a) **Indications**
      i) All lumbar neuraxial procedures may benefit from preprocedural ultrasound scanning.
   b) **Benefits**
      i) Increased first-pass success, decreased anatomical failure, fewer needle passes, and the ability to predict the most patent space[2]
         (1) This may increase safety by decreasing trauma to nearby structures.
         (2) This may increase comfort for patients.
      ii) Increased accuracy in the identification of vertebral levels compared to palpation
      iii) Identification of neuraxial midline in patients with impalpable landmarks
      iv) Estimation of depth to epidural/intrathecal space[2]
      v) The greatest benefits have been demonstrated in patients with predicted difficult anatomy (ie, obesity, previous lumbar surgery, advanced age, or scoliosis).[1]
2) **Limitations**
   a) Ultrasound assessment of the lumbar spine can be challenging for learners. Review of the bony anatomy and practice on a wide variety of patients will increase success.
   b) Accurate placement of skin markings requires practice and care to be reliable and effective.
   c) Real-time needle guidance for neuraxial procedures can be very challenging, even for the experienced practitioner, and is not a focus of this chapter.[3]
   d) Preprocedural scanning of the thoracic spine has been well described. The close proximity or even overlapping of adjacent lamina, as well as the highly sloped nature of the spinous processes, can make routine visualization of the spinal canal through the interlaminar space with ultrasound either difficult or impossible. The clinical utility of scanning the thoracic spine has not been validated in studies.[4]
3) **Equipment**
   a) The preprocedural ultrasound examination requires only a curvilinear, low-frequency transducer, ultrasound gel, and a marking pen.



The use of preprocedural ultrasound allows for objective assessment of

1. Location of the midline
2. Intervertebral level
3. Most patent level
4. Depth to the spinal canal
5. Lateral rotation due to scoliosis



Preprocedural ultrasound can also be used in the preassessment clinic to predict if a neuraxial procedure is likely to be difficult or easy in cases with known difficult anatomy or in cases with technical difficultly during previous lumbar procedures.[5]

i) A low-frequency, curvilinear transducer (~2-5 MHz) allows for increased penetration and a wide field of view.
ii) An initial depth of 9-11cm is appropriate depending on the body habitus. Adjustment of depth, focus, and gain may be required.

b) Appropriate equipment for aseptic technique and the planned neuraxial procedure are required.

4) **Technique**

a) **Monitoring**

i) An ultrasound examination alone does not require any monitoring. However, if a subsequent percutaneous neuraxial procedure is planned, then the patient should be in an appropriate anesthetizing location with ASA standard monitors.

b) **Positioning**

i) Position the patient sitting or in the lateral decubitus position with forward flexion of the spine.
ii) If applicable, apply monitors and provide sedation and supplemental O_2.

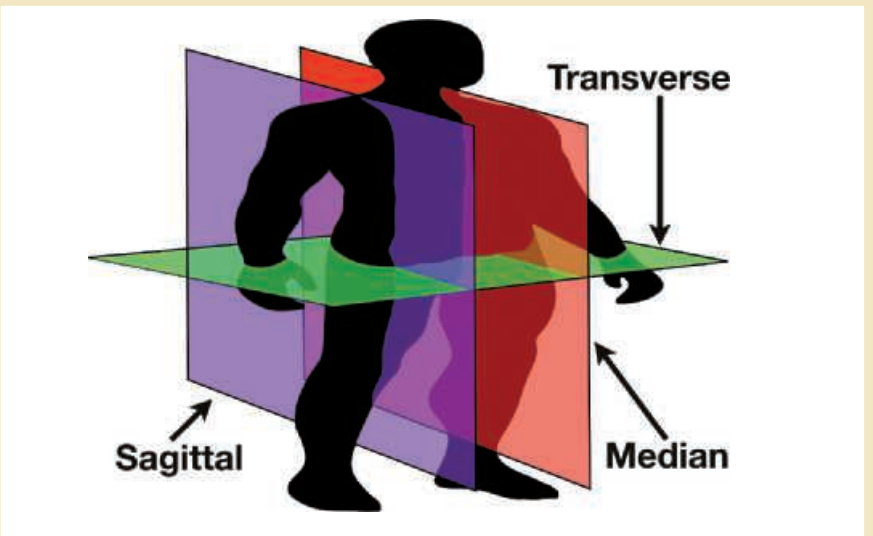
c) **Ultrasound background and features**

i) Sonographic planes of view (**Fig. 200-1**)
(1) The transverse plane is any plane that creates a slice through a standing person horizontally.
(2) The median plane is the single plane that creates a slice through a standing body vertically exactly through the midline.
(3) Any slice that creates a plane parallel to the median plane is a sagittal plane (or a paramedian plane).



There is some animal evidence that large amounts of ultrasound gel may cause neuroinflammatory reactions within the subarachnoid space, and therefore, its use should be avoided within the sterile prepped area.[6] Gel can be used preprocedurally and removed from the skin before skin preparation. Sterile saline or water can be used as a nontoxic coupling agent in most cases if ultrasound is needed within a sterile prepped area.

FIGURE 200-1 Sonographic Planes: The Most Important Reference Planes of the Body for Ultrasound of the Spine Are Transverse, Median, and Sagittal.



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(4) A plane that is tilted medially or laterally relative to a sagittal plane is known as a sagittal oblique plane (or a paramedian oblique plane).

ii) Sonographic features and elements

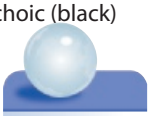
(1) Bone: a shallow hyperechoic (white) rim with dense deep hypoechoic (black) acoustic shadowing. Important bony elements include the spinous process, articular process, transverse process, and lamina.

(2) Muscle and connective tissue (ligaments, fascia): mixed hypo- and hyperechoic elements with no acoustic shadowing. Common muscular and connective tissue elements visualized include erector spinae muscle, psoas muscle, and interspinous ligament.

(3) Posterior complex: a hyperechoic line, which represents the sonographic fusion of the ligamentum flavum and posterior dura. These individual elements are typically not seen individually.

(4) Anterior complex: a hyperechoic line, which represents the sonographic fusion of the anterior dura, posterior longitudinal ligament, and posterior vertebral body. These individual elements are typically not seen individually.

(5) The space between the posterior complex and the anterior complex represents the location of the dural sac.



The most important structure to identify during a scan of the lumbar spine is the anterior complex. If the sound waves can pass through the interlaminar/interspinous space, then the needle should be able to do the same. A relatively larger, brighter anterior complex may be indicative of a more patent space when compared with adjacent levels.

d) **Standard views**

i) Transverse scans

(1) Transverse spinous process view (**Fig. 200-2A**): With the transducer in the transverse plane, the midline is identified by visualizing the erector spinae muscles on either side of the spinous process. Translation of the transducer caudal and cephalad over the spinous process reveals the hyperechoic rim of the posterior aspect of the spinous process and a dense acoustic shadow, which hides the deeper structures from view.

(2) Transverse interspinous view (**Fig. 200-2B**): From the transverse spinous process view, translate cephalad or caudal until the deep acoustic shadowing is lost, and the anterior complex is visualized. Cephalad tilt may improve this visualization. Identifiable structures within the critical view may include erector spinae muscles, interspinous ligament, articular processes, transverse processes, the posterior complex, and the anterior complex.

(3) Transverse scan to measure depth to ligamentum flavum: If a posterior complex is visualized in the transverse interspinous view, then the calipers can be used to measure the depth from the skin to the posterior complex. This represents an objective estimate of the depth from the skin to the ligamentum flavum and dural sac. Care should be taken, as compression with the ultrasound may lead to underestimation of the needle distance.

(4) Transverse scan to rule out scoliosis: From the transverse interspinous view, place the midline M-mode marker on the screen. Orient the transducer so that the M-mode line

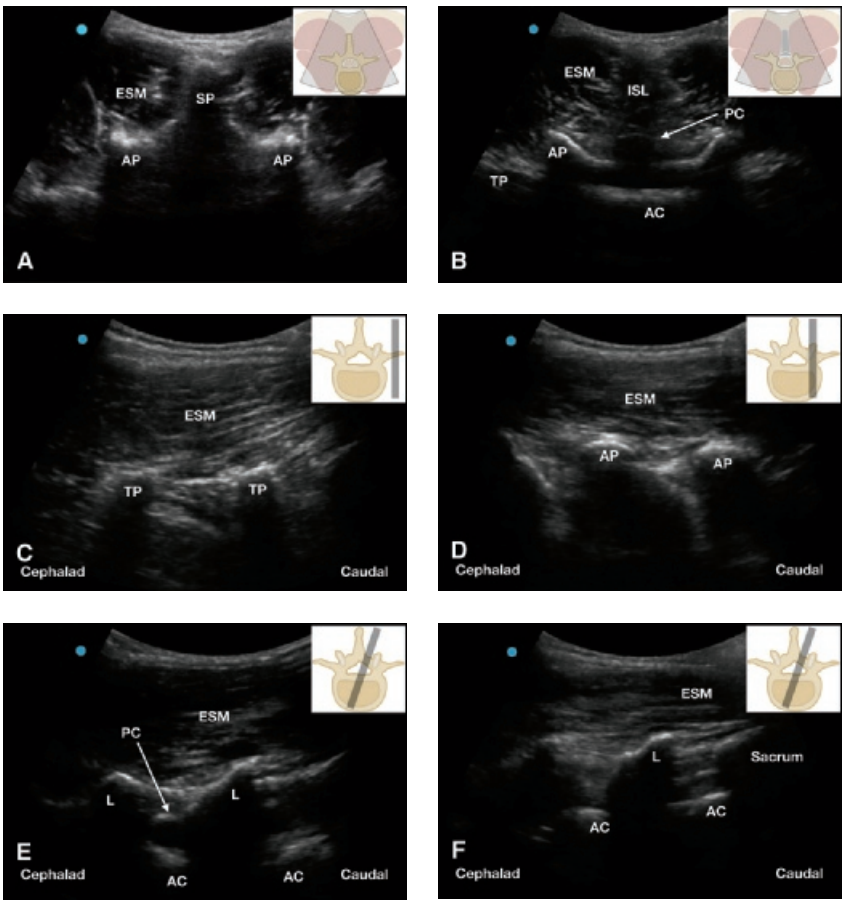


If the depth with compression from the transducer to the posterior aspect of the spinous process is >4 cm, then there is a higher likelihood that a longer needle will be required.



An initial sagittal oblique scan may be misleading in the setting of unrecognized scoliosis or when the location of the midline is unknown. It is best to start with a quick transverse scan to identify and mark the location of the midline and to identify any lumbar rotation due to scoliosis before proceeding to a sagittal oblique scan.

FIGURE 200-2 **A.** Transverse spinous process view (ESM, erector spinae muscle; SP, spinous process; AP, articular process). **B.** Transverse interspinous view (ESM, erector spinae muscle; ISL, interspinous ligament; TP, transverse process; AP, articular process; PC, posterior complex; AC, anterior complex). **C.** Sagittal transverse process view (ESM, erector spinae muscle; TP, transverse process). **D.** Sagittal articular process view (ESM, erector spinae muscle; AP, articular process). **E.** Sagittal oblique view (ESM, erector spinae muscle; L, lamina; PC, posterior complex; AC, anterior complex). **F.** Sagittal oblique view with sacrum (ESM, erector spinae muscle; L, lamina; AC, anterior complex).



Figures A-F, reproduced with permission from Kwesi Kwofie Educational Series.

passes through the most posterior aspect of the interspinous ligament, the most anterior portion of the interspinous ligament, and if possible, through the middle of the anterior complex. Then, directly visualize the orientation of the transducer of the patient's back. If it is symmetrical, then there is no lateral rotation of the spine at that level. If the transducer is tilted obliquely to the left or right, then this is the angle of the spinal rotation.

To accurately reproduce the location of the midline, the M-mode marker can be used and placed through the midline structures. With the M-mode midline marker passing through the spinous process and bisecting the anterior complex, any lateral tilt of the ultrasound transducer represents the angle of lateral rotation due to scoliosis.

ii) Paramedian/sagittal scans

(1) Sagittal transverse process view (**Fig. 200-2C**): With the transducer in the sagittal orientation 3-5 cm lateral to midline, there is a bright superficial rim with a dense acoustic shadow deep into the ultrasound field. The origin of the acoustic shadow and bright rim represents the posterior aspect of the transverse process. The erector spinae muscle is visible superficial to the origin of the acoustic shadow and the psoas muscle is visible deep to the origin of the acoustic shadow.

(2) Sagittal articular process view (**Fig. 200-2D**): From the sagittal transverse process view, slide the transducer medially but still lateral to the midline. The bony rim will become slightly more superficial and the deep psoas muscle will be lost to acoustic shadowing. The hump-shaped bright rims may have an overlapping appearance. These represent the articular processes.

(3) Sagittal oblique view (**Fig. 200-2E**): From the parasagittal articular process view, with the transducer about 2-4 cm lateral to midline, the transducer is tilted inward toward the spinal canal. The hyperechoic rim will become less steep and slanted going deep from caudal to cephalad and has the appearance of a "saw-tooth" pattern. This is characteristic of the laminae. In the interlaminar spaces, there may be hyperechoic elements that interrupt the acoustic shadowing. The most important and prominent is the anterior complex (a fusion complex of the anterior dura, posterior longitudinal ligament and the vertebral body), about 2 cm deep to the lamina. The posterior complex (a fusion complex of the posterior dura and ligamentum flavum) may be visualized as a thin hyperechoic line, shallow within the interlaminar spaces.[7]

If an anterior complex is visualized in the sagittal oblique scan but not visualized in a transverse interspinous scan; this is suggestive that a paramedian needle approach might be technically easier than a midline approach.

(4) Identification of vertebral level (**Fig. 200-2F**): The sagittal oblique scan is first obtained at the level of the iliac crest. Then, the transducer is translated caudally while noting the "saw-tooth" appearance of the lamina. The "saw-tooth" appearance will be interrupted caudally by a continuous, hyperechoic, bony shelf with acoustic shadowing; this is the sacrum. The first gap between this hyperechoic rim and the most caudal lamina ("saw-tooth") represents the L5/S1 interspace. Counting the interlaminar spaces can identify successive levels (**Fig. 200-3**).

FIGURE 200-3 A Step-by-Step Method to Lumbar Neuraxial Ultrasound.

1. **Optimize the patient's procedural position** including lumbar flexion.
2. **Complete aTransverse Scan** to identify the midline and torule out scoliosis.
3. **Mark the patient's mid line** with ask in marker.
4. **Obtain a Sagittal Oblique Scan** at the level of the iliaccrest and then move caudally to identify the sacrum. From the sacrum count up to identify the levels cephalad.
5. In the Sagittal Oblique Scan **identify the levels that have the largest anterior complex** and mark the level at the midline of the transducer with a skin marker.
6. **Obtain a Transverse Scan** throughout the lumbar spine focusing particularly on the previously marked levels and look for an anterior complex.
7. If an anterior complexis identified, the level should be noted with ask in mark, and the depth from the skin to the posterior complex can be recorded to predict needle depth.**The inter section between them id line and the level of the marked anterior complex represents the target**. The needle should be introduced at that level or slightly caudal.



Using a palpation technique to assess spinal level may be off by two or more levels in 27% of patients.[8] Lumbarization or sacralization of S1 or L5, respectively, has been reported in 4%-21% of patients and may not be determined with ultrasound.[9] This might cause the anatomic level to be off by one level in those patients.

The L5-S1 space is often the largest interlaminar space and is often widely patent when other spaces are severely narrowed.

Remember that only the cephalad and caudal translational position of the transducer can be marked with skin markings. Any angle of transducer tilting (cephalocaudal or lateral) must be remembered by the operator during the preprocedural scan and may need to be reproduced during the needling procedure.

The ultrasound examination and skin markings should be done before sterile preparation to decrease risk of contamination.

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201 Transesophageal Echocardiography Views§

Benjamin A. Abrams • Nathaen Weitzel

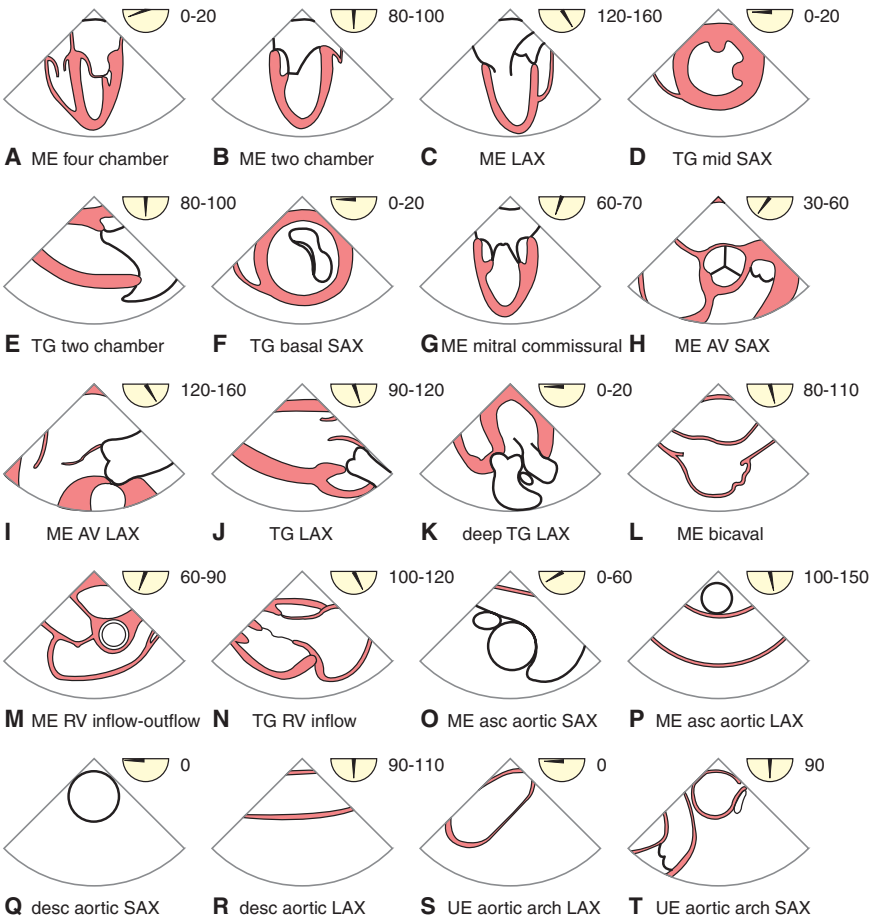
Intraoperative transesophageal echocardiography (TEE) is widely used in modern operating rooms as it provides valuable real-time information to the surgical team. The TEE allows intraoperative decisions to be made based on data collected throughout the procedure instead of waiting until after the case to evaluate surgical outcomes. Outcomes have been shown to improve secondary to intraoperative TEE evaluation specifically in cardiac valvular surgery,[1] which has promoted the routine use in most cardiac cases today. The anesthesiologist performs the TEE at most institutions intraoperatively and may perform examinations in emergency situations in noncardiac operating rooms or locations such as the intensive care unit. Therefore, it is useful for all anesthesiologists to have basic knowledge of the TEE and the intraoperative examination.

READ MORE
ASE/SCA 20 view exam overview, Chapter 207

1) **TEE examination basics**: A TEE examination is performed by placing the TEE probe into the esophagus.
   a) **Placement:** consider placing an orogastric tube first to suction the gastric contents; then, use a lubricant jelly and a mouth/bite guard when placing the probe.
   b) **Relative contraindications:** patient history of mediastinal radiation, dysphagia, recent upper gastrointestinal surgery or bleeding. Known esophageal pathology is also a relative contraindication including stricture, varices, tumor, diverticulum, or esophagitis.
2) **TEE views**

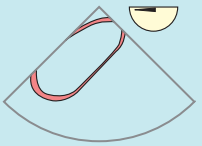
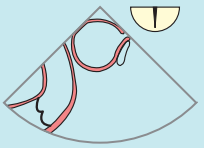
   The Perioperative TEE examination was originally described as 20 views adapted from Shanewiseet al.[2] (**Fig. 201-1**) in 1999. These have been the standard views used by anesthesiologists to evaluate the heart in the operating room. Over the past 20 years, TEE use has expanded dramatically, and now there are different levels of exams used depending on the certification of the user. In 2013 the American Society of Echocardiography, in partnership with the Society of Cardiovascular Anesthesiologists, published guidelines for separate basic and advanced perioperative TEE exams.[3,4] The basic TEE exam is now described as a streamlined TEE study that users can do in urgent situations when time is critical, or for those practitioners who are certified with Basic TEE certification; and consists of 11 views.3 The Advanced Exam was described as well with 8 additional views on top of the previous standard 20, that focus largely on the right side of the heart, and tricuspid valve. The original 20 views that have been in place since 1999, are described in this chapter as well as how to obtain the view. The views are discussed as they are found cephalad to caudad in the esophagus. There are markings of the depth in centimeters on the TEE probe. The depth is usually measured at the patient's incisors.

FIGURE 201-1 Twenty Views of the Recommended Comprehensive TEE Examination.

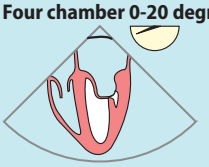
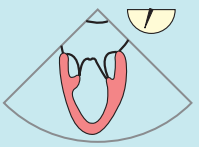
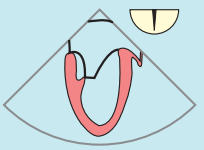
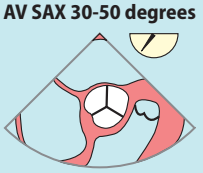


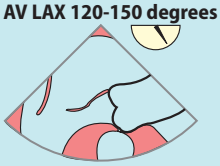
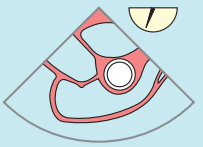
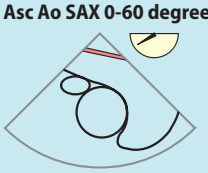
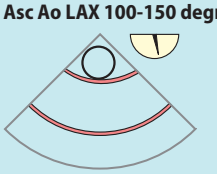
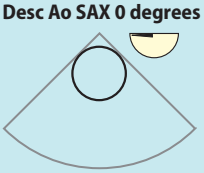
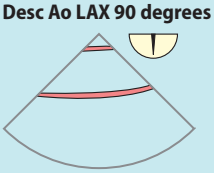
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Upper Esophageal 20-25 cm

Aortic arch LAX 0 degree 	The long axis (LAX) view of the aortic arch. It is typically found after imaging the descending thoracic aorta. The probe is withdrawn and the thoracic aorta is followed superiorly until the arch is in view. This view is useful to look for aortic pathology including atherosclerotic plaques, mobile plaques, calcification, and dissections.
Aortic arch SAX 90 degrees 	The short axis (SAX) view of the aortic arch. It is found similarly to the above view of the arch in LAX. Once the LAX is in view, rotate the angle to 90 degrees to obtain the SAX view. The aortic arch is seen along with the subclavian artery. The pulmonary valve and main pulmonary artery are seen in LAX. The aorta is evaluated for pathology in this view similarly to the LAX aortic arch view above.

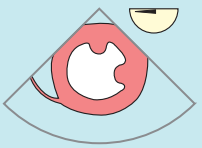
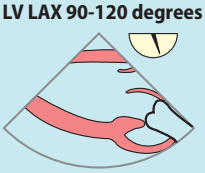
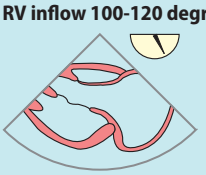
Mid Esophageal 30-40 cm

Four chamber 0-20 degrees 	The four chamber view is often considered the "home base" view of the TEE examination. To obtain this view once the four chambers are visualized, retroflex the probe slightly and rotate the angle to 0-20 degrees. The four chamber view is used to evaluate the ventricular size, wall motion, masses, and systolic function of each chamber. Atrial size and structure can be examined along with evaluation of the tricuspid (TV) and mitral valve (MV).
Mitral commissural 60-80 degrees 	The commissural view of the MV. The leaflets visualized from left to right: **P3, A2, P1**. To obtain this view from the four chamber view above, rotate the angle to 60-80 degrees. Utilize this view to evaluate the MV leaflets, their motion, and to measure the commissural distance/annular dilatation.
Two chamber 80-100 degrees 	The two chamber view of the left atrium and left ventricle is obtained by rotating the angle to 80-100 degrees until the right side of the heart disappears. Utilize this view to evaluate the MV leaflets, and the anterior and inferior walls of the left ventricle. The left atrium is also examined in this view for evidence of thrombus.
LV LAX 120-160 degrees 	The midesophageal LAX view is obtained by rotating the angle to 120-160 degrees in the ME plane. The left ventricle outflow tract (LVOT) should come into view. The aortic valve and proximal ascending aorta are seen in this view. Utilize this view to evaluate for systolic anterior motion of the MV and to evaluate the anteroseptal and inferolateral walls of the left ventricle.
AV SAX 30-50 degrees 	The aortic valve SAX view (AV SAX) is obtained in the ME plane usually at 30-50 degrees. Adjust the probe depth until the AV is centered in screen. In this view, all three cusps of the AV can be visualized. The noncoronary cusp is adjacent to the interatrial septum, the right coronary cusp is anterior, and the remaining cusp is left. The right and left coronary arteries can often be visualized superior to their corresponding cusp. Utilize this view to evaluate the aortic valve cusps for prolapse, calcification, restriction, and aortic insufficiency.

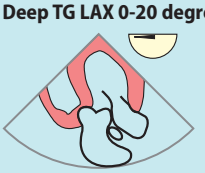
AV LAX 120-150 degrees 	The aortic valve LAX view (AV LAX) can be obtained by starting from the AV SAX view and rotating the angle to 120-150 degrees. Utilize this view to examine the aortic valve movement, prolapse, restriction, calcification, and for aortic insufficiency using color flow Doppler. In addition, this view is used to measure the annulus, sinus, and ascending aorta.
RV inflow-outflow 60-90 degrees 	This is the right ventricle (RV) inflow-outflow view. To obtain this view, start at the four chamber view and rotate the probe to the right until the TV is in the center of the screen. Then rotate the angle to 60-90 degrees to see the pulmonic valve (PV) and the main pulmonary artery. The RV outflow tract is well visualized. Utilize this view to evaluate the TV and PV.
Bicaval 80-110 degrees 	To obtain the midesophageal bicaval view, start at the four chamber view and **rotate** the probe to the right, centering on the right atrium (RA). Then increase the angle to 80-110 degrees. Utilize this view to evaluate the RA, the inferior vena cava (left), and the superior vena cava (right). This view is also useful to evaluate for atrial septal defects (ASDs). A bubble study can be performed while in this view to look for the ASD.
Asc Ao SAX 0-60 degrees 	To obtain the midesophageal ascending aortic SAX view (Asc Ao SAX), locate the ascending aorta in the center of the screen, and advance or withdraw the probe to examine different levels of the aorta. The main PA and right PA and the SVC are also visualized here.
Asc Ao LAX 100-150 degrees 	The midesophageal ascending aortic LAX view (Asc Ao LAX) is obtained by adjusting the multiplane angle to 100-150 degrees from the Asc Ao SAX view. Utilize this view to examine the aorta in LAX as well as the right pulmonary artery in SAX.
Desc Ao SAX 0 degrees 	To obtain the descending aortic SAX view (Desc Ao SAX), begin with the four chamber view and turn the probe left by ~90 degrees until the aorta comes into view. The length of the thoracic aorta can be examined by advancing and withdrawing the probe.
Desc Ao LAX 90 degrees 	To obtain the descending aortic LAX view (Desc Ao LAX), start with the Desc Ao SAX view and adjust the multiplane angle to 90 degrees. Utilize this view to complement the SAX evaluation of the descending aorta.

(Continued)

Transgastric 40-45 cm

TG basal MV SAX 0-20 degrees 	The transgastric views are obtained by advancing the probe into the stomach and strongly anteflexing the tip. The basal MV SAX view (basal MV SAX) is located superior/proximal to the mid SAX view and corresponds to the basal segments of the left ventricle. Utilize this view to evaluate the MV.
TG mid SAX 0-20 degrees 	The transgastric mid SAX view (TG mid SAX) is obtained by advancing the probe from the ME plane or TG basal view until the papillary muscles are visualized. Anteflexion is typically required. Utilize this view to evaluate global ventricular function, monitor regional wall motion abnormalities, and assess volume status (preload) of the LV.
TG two chamber 80-100 degrees 	This is the transgastric two chamber view. To obtain this view from the TG mid SAX view, adjust the multiplane angle to between 80 and 100 degrees. Utilize this view to evaluate the MV, chordae tendineae, MV leaflets, along with the anterior (bottom) and inferior (top) left ventricular walls.
LV LAX 90-120 degrees 	This is the transgastric left ventricular LAX view. To obtain this view from the above image, further advance the multiplane angle to 120 degrees. Utilize this view to evaluate the AV and LVOT, especially to take measure gradients through the AV.
RV inflow 100-120 degrees 	This is the RV inflow view. To obtain this view, rotate the probe to the right from the mid SAX view. Utilize this view to examine the RA, RV, TV, and papillary muscle.

Deep Transgastric 45-50 cm

Deep TG LAX 0-20 degrees 	This is the deep transgastric LAX (TG LAX) view. To obtain this view, advance the probe in the neutral position deep into the stomach and maximally anteflex the probe. Then slowly withdraw the probe until it contacts the stomach wall. Utilize this view to interrogate the aortic valve and LVOT, again like the **LV LAX**, a useful view for the measurement of aortic valve gradients.

The 20 views above are important for every anesthesiologist to understand and be able to recognize cardiac structures. TEE is an excellent tool to evaluate cardiac function and pathology in the OR as well outside the OR.

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202 TEE Evaluation of the Aortic Valve§

Benjamin A. Abrams • Nathaen Weitzel

Aortic valve (AV) replacement is a common surgical procedure in the cardiac operating room. A complete transesophageal echocardiographic (TEE) examination is important to determine etiology of AV disease as well as severity. An overview of the basic TEE evaluation techniques for both regurgitant and stenotic lesions is presented.

1) **TEE evaluation of the AV**: The real-time evaluation of the heart prior to and following bypass is an invaluable tool in the operating room both to the surgeon and the anesthesiologist.

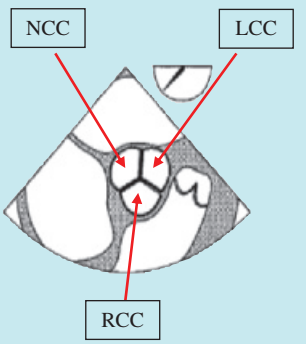
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Evaluation of aortic valve, Chapter 210

   a) **Basic anatomy**: the AV is a semilunar valve with three cusps. The cusps are named for the coronary artery that exits superior to them. Therefore, there is a right coronary cusp that sits anteriorly, a noncoronary cusp (NCC), which is closest to the interatrial septum and a left coronary cusp. Key anatomic features associated with the AV are the left ventricular outflow tract (LVOT) and the aortic root.
      i) The LVOT is the outflow region of the left ventricle (LV) immediately inferior to the valve.
      ii) The aortic root refers to the AV annulus, cusps, sinuses of Valsalva, coronary artery ostia, sinotubular junction, and proximal ascending aorta (**Fig. 202-1**).

2) **TEE views of the AV**

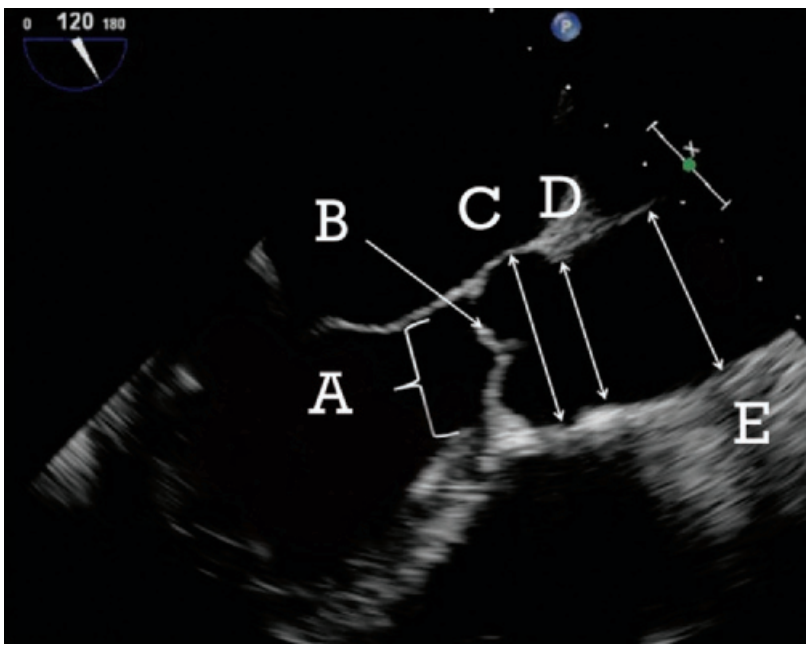
The TEE examination for the AV uses the following four views from the ASE/SCA 20 TEE views.[1]

a) **Midesophageal AV short axis view**—This cross-section view of the AV allows the echocardiographer to view all three cusps of the valve at one time. In this view, the cusp adjacent to the atrial septum is the NCC, the most anterior cusp is the right coronary cusp, and the remaining cusp is the left coronary cusp. This view can be used to measure the area of the AV using planimetry. Color Doppler can be used here to evaluate aortic insufficiency or prosthetic ring abnormalities following AV replacement.



b) **Midesophageal AV long axis view**—This view is useful to assess aortic regurgitation (AR) using color flow Doppler, along with evidence of turbulent flow seen in aortic stenosis. It is also used to assess the aortic root measuring the diameters of the AV annulus, sinuses of Valsalva, sinotubular junction, and the proximal ascending aorta.	
c) **Transgastric long axis view**—Utilize this view to obtain Doppler quantification of blood flow through the LVOT and AV.	
d) **Deep transgastric view**—Like the transgastric LAX view, this image is used to obtain Doppler quantification of flow velocities in the LVOT, AV, and can be useful in measuring elevated gradients for patients with LVOT obstruction (HOCM patients, or patients with systolic anterior motion of the mitral valve). Also, a good view for measuring pressure half-time of the AR Doppler tracing. Finally, this view can provide good imaging in patients with prosthetic AV or MV where there are significant artifacts at the ME levels, but the deep transgastric approach avoids this.	

FIGURE 202-1 Long Axis View of LVOT



ME LAX view showing the LVOT **(A)**, aortic valve **(B)**, sinus of Valsalva **(C)**, sinotubular junction **(D)**, and ascending aorta **(E)**.

TABLE 202-1 Aortic Regurgitation[2]

	Mild	Moderate	Severe
Qualitative			
Angiographic data	1+	2+	3-4+
Color Doppler jet width	Central jet, width <25% of LVOT	Greater than mild, but no signs of severe AR	Central jet, width >65% LVOT
Doppler vena contracta width (cm)	<0.3	0.3-0.6	>0.6
Quantitative (cath or echo)			
Regurgitant volume (mL/beat)	<30	30-59	≥60
RF (%)	<30	30-49	≥50
ROA (cm^2)	<0.1	0.1-0.29	≥0.3
Pressure half-time (ms)	>500	200-500	<200
Additional criteria			
LV size			Increased
Aortic diastolic flow reversal			Holodiastolic flow reversal

Sources: Adapted from Bonow RO, Carabello BA, Chatterjee K, et al. ACC/AHA 2006 Guidelines for the management of patients with valvular heart disease: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2006;114(5):e84-e231; Perrino A, Reeves S. *Transesophageal Echocardiography*. Philadelphia, PA: Lippincott Williams & Wilkins; 2008.

3) Aortic regurgitation

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Quantitative echocardiography, Chapter 204

AR describes an AV that is incompetent and allows reversal of blood flow into the LV during diastole. For assessment and quantification of AR, see **Table 202-1**.

a) **Qualitative measures**: the Color Doppler jet width and vena contracta represent the main tools in assessing the severity of AI.

b) **Quantitative assessment**: a quantitative assessment of the valve is undertaken by calculating the regurgitant volume, regurgitant fraction (RF), and regurgitant orifice area (ROA).

i) The regurgitant volume is the difference between the LV stroke volume and the right ventricular (RV) stroke volume.

$$RV_{AV} = SV_{LV} - SV_{RV}$$

The LV stroke volume is calculated by multiplying the cross-sectional area of the LVOT by the velocity time integral (VTI) of LVOT flow. The LVOT VTI can be obtained using pulsed wave Doppler at the LVOT (**Fig. 202-2**).

Calculate the RV stroke volume similarly by multiplying the cross-sectional area of the pulmonary out flow tract by the VTI measured in the proximal pulmonary artery. This technique is time consuming and requires multiple measurements leading to possible error and thus is used infrequently.

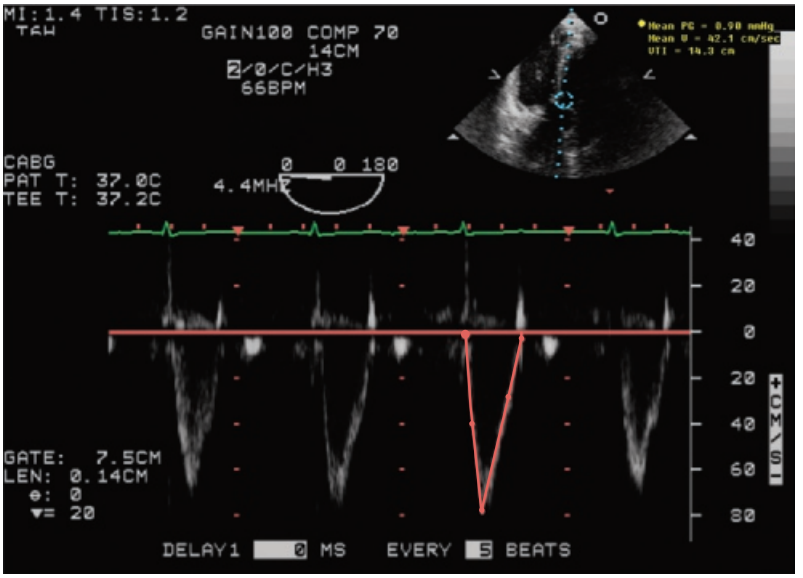
ii) The regurgitant fraction (RF) is the regurgitant volume divide by the LV stroke volume.

$$RF = RV_{AV} / SV_{LV}$$

iii) The pressure half-time is defined as the time interval between the maximal regurgitant pressure gradient and the time when the gradient is half of the maximum (**Fig. 202-3**). To obtain this measurement, use either the transgastric long axis view or the deep transgastric view. A pressure half-time <200 ms indicates severe AR.

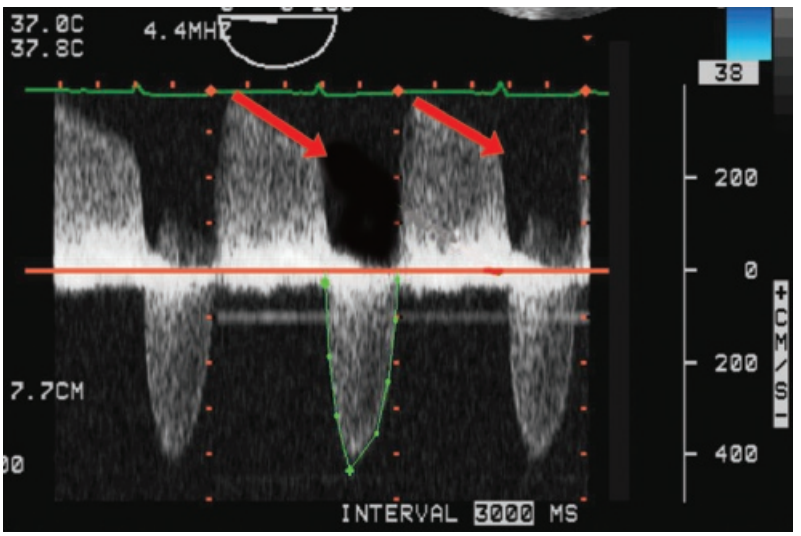
iv) Aortic flow reversal: measurement of aortic flow in the descending aorta is an adjunct measure of AR severity. Presence of holodiastolic flow reversal in the descending aorta confirms severe AR.

FIGURE 202-2 Pulsed Wave Doppler Evaluation of AV



This is the deep transgastric view with pulse wave Doppler across the AV. A VTI can be measured in this view by outlining the Doppler flow. The VTI is used to calculate the stroke volume by multiplying it by the LVOT area.

FIGURE 202-3 Continuous Wave Doppler Evaluation of AV



This is a deep transgastric view of the AV with continuous wave Doppler across the valve. There is AR in the patient as seen by the reversal of flow during diastole. The *arrows* represent the pressure half-time measurement, with an average value of 375 ms, indicating moderate aortic insufficiency.

TABLE 202-2 Aortic Stenosis

Indicator	Mild	Moderate	Severe
Peak jet velocity (m/s)	<3.0	3.0-4.0	>4.0
Mean gradient (mm Hg)	<25	25-40	>40
Valve area (cm^2)	>1.5	1.0-1.5	<1.0
Valve area index (cm^2/m^2)			<0.6

Sources: Adapted from Bonow RO, Carabello BA, Chatterjee K, et al. ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: executive summary. *Circulation*. 2006;114; Perrino A, Reeves S. *Transesophageal Echocardiography*. Philadelphia, PA: Lippincott Williams & Wilkins; 2008.

4) **Aortic valve stenosis (AS)**

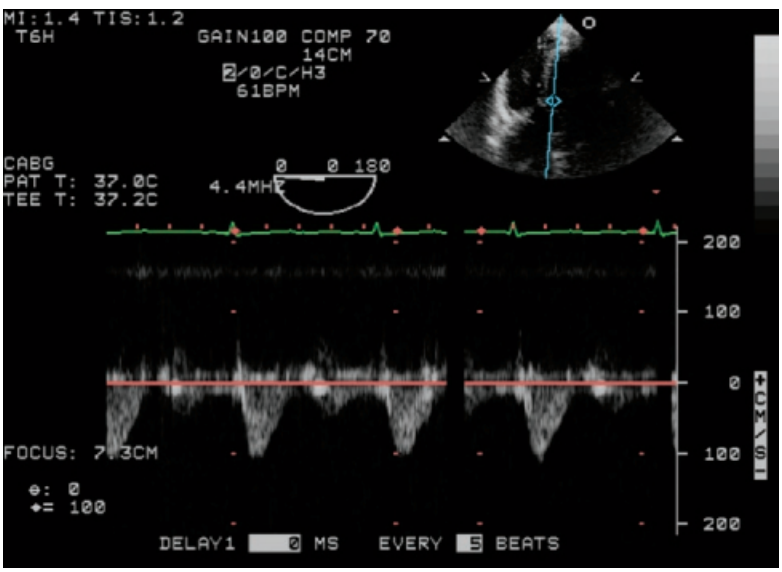
AS is the narrowing of the AV orifice, typically due to calcification/sclerosis of the valve leaflets. The normal area of an AV in an adult is 2.6-3.5 cm^2. Severe AS is defined as a valve area <1.0 cm^2 or a mean gradient >40 mm Hg. **Table 202-2** describes the criteria for grading aortic stenosis.[3]

a) **Quantitative measures**

i) Bernoulli equation: to calculate the above values and estimate the severity of aortic stenosis, use the modified Bernoulli equation to measure the gradient across the valve and the continuity equation to measure the AV area. To obtain the peak velocity, place the continuous wave Doppler beam parallel to the flow of blood across the AV in the deep transgastric view (**Fig. 202-4**). With this velocity, use the modified Bernoulli equation to calculate the mean gradient.

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FIGURE 202-4 Deep Transgastric View of the AV Using Continuous Wave Doppler.



This CW Doppler across the AV is used to measure a peak velocity. The peak velocity measurement is then used to calculate a peak pressure by the equation: Peak gradient (mm Hg) = 4 (peak velocity)^2. A high gradient indicates a stenotic valve.

$$\text{Peak gradient (mm Hg)} = 4\ (\text{aortic peak velocity})^2$$
$$\text{Mean gradient (mm Hg)} = 4\ (\text{mean velocity})^2$$
$$= 2.4\ (\text{Vmax})^2$$

ii) The continuity equation

LVOT stroke volume = AV stroke volume
Stroke volume = CSA × VTI

The equation rearranges to:

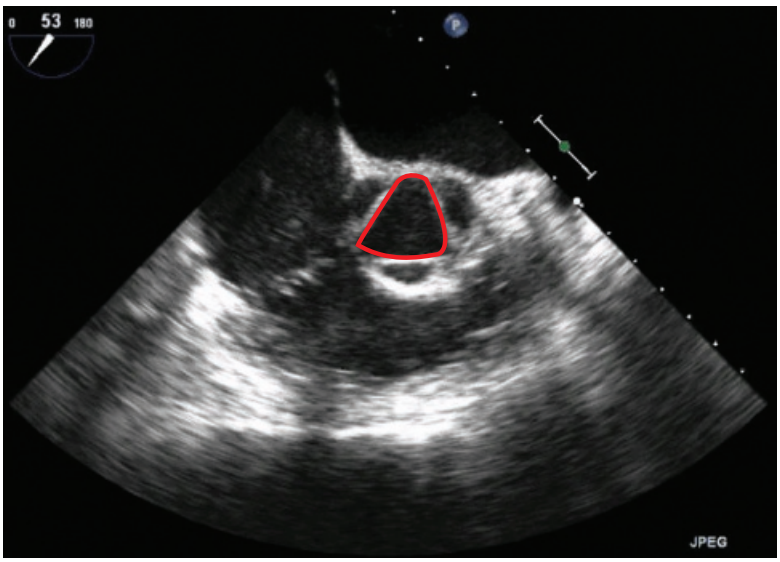
$$VTI_{LVOT} \times area_{LVOT} = VTI_{AV} \times area_{AV}$$
$$Area_{AV} = VTI_{LVOT} \times area_{LVOT} \div VTI_{AV}$$

The LVOT velocity is obtained from the deep transgastric view; however, use the pulsed wave Doppler placed directly in the LVOT to obtain this Doppler tracing.

iii) Planimetry

The area of the valve can also be measured using planimetry in systole by measuring the narrowest orifice of the valve. Planimetric techniques are not considered as accurate as the continuity equation in severely calcified and sclerotic valves as significant shadowing can result in errors in measurement. Despite this, planimetry can provide a rapid estimate of valve area (**Fig. 202-5**).

FIGURE 202-5 Planimetry of AV in the Short Axis



The short axis view of the AV is used to estimate valve area using planimetry. The valve area is outlined using software on the tee machine. This is a quick estimate of area most useful in normal valves. Calcified and stenotic valve area is difficult to measure with this method.

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203 TEE Evaluation of the Mitral Valve§

Benjamin A. Abrams • Nathaen Weitzel

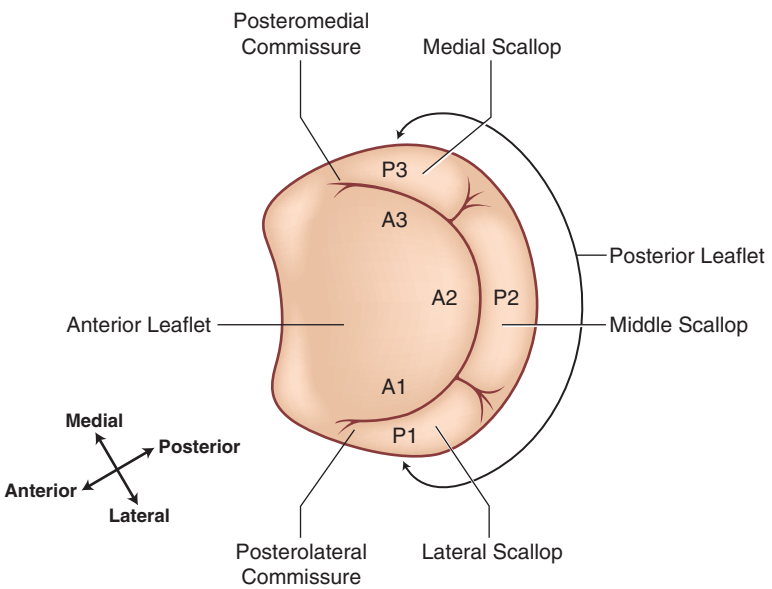
Mitral valve surgery remains a mainstay in the cardiothoracic surgical arena. New interventional approaches are also in practice and development for mitral valve repair. These procedures require the anesthesiologist to have a solid understanding of the mitral valve anatomy both for physiologic management as well as TEE interpretation.

Read More
Evaluation of the mitral valve, Chapter 209

1) Anatomy

The mitral valve separates the left atrium from the left ventricle. Two leaflets make up the mitral valve—the anterior and posterior leaflets. The anterior leaflet makes up ~55% of the surface area of the valve, while the posterior leaflet makes up ~45%. The leaflets meet at the anterolateral and posteromedial commissures. The valve is surrounded by a fibromuscular ring called the mitral annulus (**Fig. 203-1**).

FIGURE 203-1 Schematic of the Mitral Valve Illustrating the Anterior and Posterior Leaflets. This illustration uses the widely accepted Carpentier classification system.

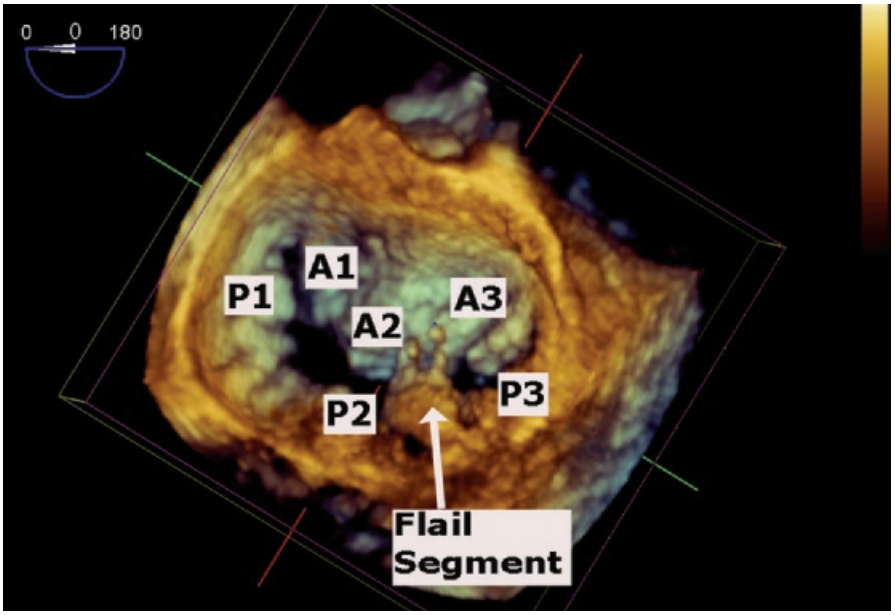


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2) **Nomenclature:** the Carpentier system used to describe the mitral anatomy[2]
   a) The posterior leaflet is composed of three scallops: P1, P2, and P3. P1 is closest to the atrial appendage (**Fig. 203-2**).
   b) The anterior leaflet is not scalloped but is named with a corresponding numerical system for easy identification: A1, A2, and A3.
3) **TEE views of mitral valve**
   To image the mitral valve, use the following 6 midesophageal and transgastric TEE views.[1]

ME four chamber—Utilize this view to evaluate MV with and without color flow Doppler. In this view, P2 is on the right and A2 is on the left.	
ME mitral commissural—Utilize this view to evaluate MV annular size. This is a good view to evaluate restriction, calcification, and specific leaflet motion. In this view, A2 is in the middle, P1 is on the right, and P3 is on the left.	
ME Two chamber—Utilize this view to evaluate MV with and without color flow Doppler. In this view, P3 is on the left and A3/A2 are on the right.	
ME long axis—Utilize this view to evaluate the MV, and look for systolic anterior leaflet of the motion of the valve. In this view, P2 is on the left and A2 is on the right.	
TG Basal short axis—Utilize this view to evaluate the MV. This view can be used to calculate the MV area. In this view, the posteromedial commissure is in the upper left of the display, and the anterolateral commissure is in the lower right. The posterior leaflet is on the right side, and the anterior leaflet is to the left.	
TG two chamber—Utilize this view to evaluate the mitral valve attachments to the papillary muscles and chordae tendineae.	

FIGURE 203-2 3D Image of the Mitral Valve in the "Surgeons View" Orientation.



3D magnified image of the mitral valve looking down on the atrial surface of the mitral valve. Note that in this image, there is a flail segment at the P2/P3 junction.

4) **Mitral valve regurgitation (MR)**
MR is a condition where blood can flow back into the left atrium during systole due to coaptation defects in the mitral apparatus.
   a) **Classification**—a classification system by Carpentier describes the leaflet motion in relation to the regurgitation.[2]
      i) Type 1 lesion: MR is present—with normal leaflet motion. The most common cause for this is annular dilatation. Other causes include mitral valve clefts, perforations, or aneurysms. In this type of lesion, the jet is often centrally directed.
      ii) Type 2 lesion: MR with excessive leaflet motion. The jet in these cases is often directed away from the involved leaflet. Examples of this include:
         (1) Billowing—part of the mitral leaflet projects above the annulus in systole
         (2) Prolapse—the leaflet tip is above the level of the mitral annulus
         (3) Flail—the leaflet tip is free flowing in the left atrium in systole, usually with chordal rupture
      iii) Type 3 lesion: MR with restricted leaflet motion
         (1) Type 3a—structural restriction—leaflet motion affected in systole and diastole
         (2) Type 3b—functional restriction—leaflet motion affected in systole
5) **Evaluation**
   a) **Qualitative**
      i) Color flow Doppler allows a visual estimation of the severity of regurgitation based on the size of the regurgitant jet. (See **Table 203-1** for the severity grading in MV disease.)

TABLE 203-1 MR Classification

Indicator	Mild	Moderate	Severe
Qualitative			
Angiographic grade	1+	2+	3-4+
Color Doppler jet area	small, central, narrow; usually brief	variable	large central jet (>50% of LA) or eccentric wall hugging jet
Doppler vena contracta width (cm)	<0.3	0.3-0.69	≥0.7
Quantitative (cath or echo)			
Regurgitant volume (mL/beat)	<30	30-44, 45-59	≥60
RF (%)	<30	30-39, 40-49	≥50
ROA (cm^2)	<0.10	0.20-0.29, 0.3-0.39	≥0.4
Additional essential criteria			
Left atrial size	normal	normal or mildly enlarged	Enlarged
Left ventricular size	normal	normal or mildly enlarged	Enlarged

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ii) The vena contracta is measured at the narrowest portion of the regurgitant jet (**Fig. 203-3**).

b) **Quantitative**: a quantitative assessment of the mitral valve is undertaken by calculating the regurgitant volume, regurgitant fraction (RF), and regurgitant orifice area (ROA).

i) To calculate the regurgitant volume, first calculate the stroke volume across the left ventricular outflow tract (LVOT).

$$\textbf{LVOT SV} = (\textbf{VTI}_{\textbf{LVOT}} \times \textbf{area}_{\textbf{LVOT}})$$

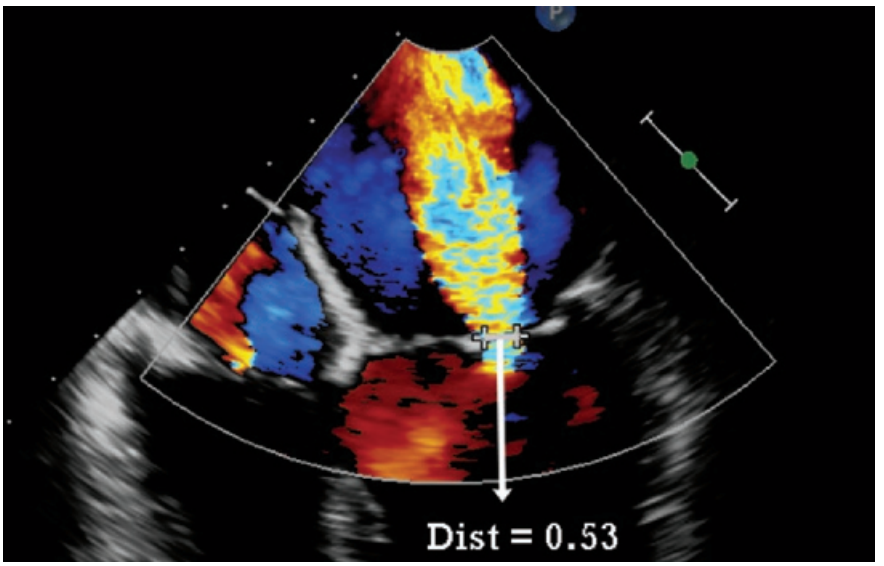
Subtract this value from the stroke volume measured at either the mitral valve in diastole $(\textbf{VTI}_{\textbf{MV}} \times \textbf{Area}_{\textbf{MV}})$ or the pulmonary artery.

ii) The RF is the percentage of left ventricular volume that flows back into the left atrium in systole. To calculate this percentage, the regurgitant volume is divided by the overall stroke volume.

iii) Calculation of the effective regurgitant orifice area (EROA) is done using the PISA method. This method is explained in detail in Quantitative Echocardiography, Chapter 204. The equation is:

$$\text{EROA} = \frac{2\pi r^2 \times \text{Nyquist limit}}{\text{MR velocity}}$$

FIGURE 203-3 Measurement of Vena Contracta



Measurement of vena contracta utilizing the ME four chamber view. The diameter of the regurgitant jet is measured using the caliper function of the TEE machine. The diameter here (0.53 cm) is consistent with severe mitral regurgitation.

6) **Mitral valve stenosis**—describes the condition of a narrowed mitral valve orifice. The normal MV area is 4-6 cm^2. A valve area <2 cm^2 causes an increase in transvalvular pressure; however, the clinical presentation of MS and significant increase in the transvalvular pressure is not seen until the valve area is <1.4 cm^2 **(Table 203-2)**.

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   a) **Gradient—calculate the mean pressure gradient across the mitral valve with the modified Bernoulli equation**

$$\text{Pressure gradient(mm Hg)} = 4V^2$$

V = instantaneous velocity

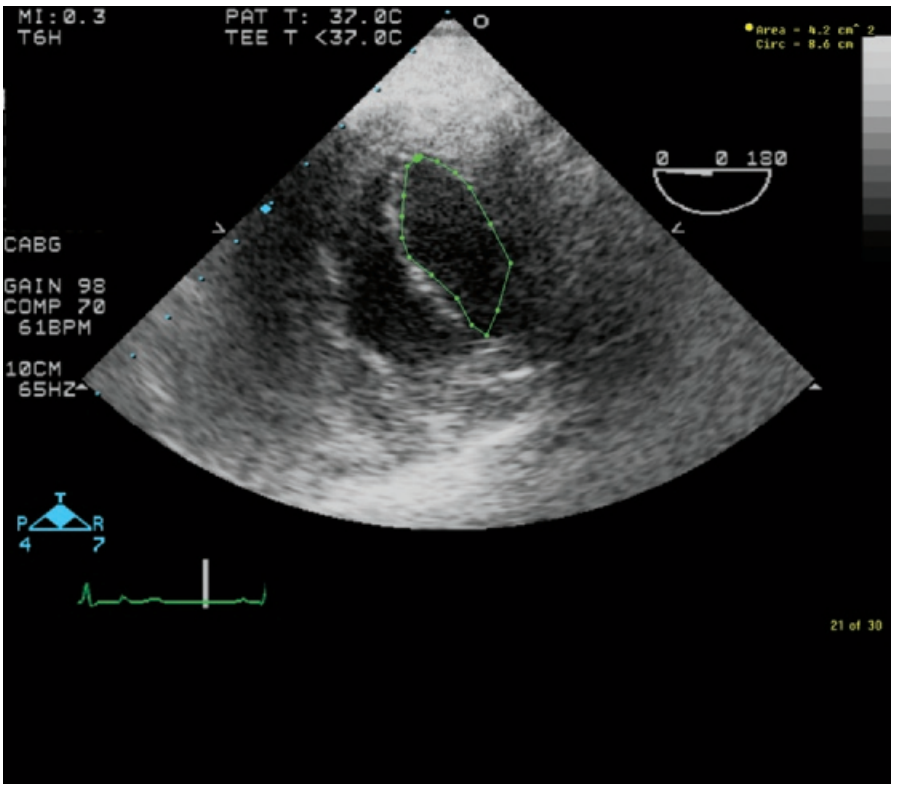
   b) The velocity measurement is obtained using continuous wave Doppler across the mitral valve.

TABLE 203-2 Mitral Stenosis

Indicator	Mild	Moderate	Severe
Mean gradient (mm Hg)	<5	5-10	>10
Pulmonary artery systolic pressure (mm Hg)	<30	30-50	>50
Valve area (cm^2)	>1.5	1.0-1.5	<1.0

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FIGURE 203-4 Planimetry Measurement of MV Area.



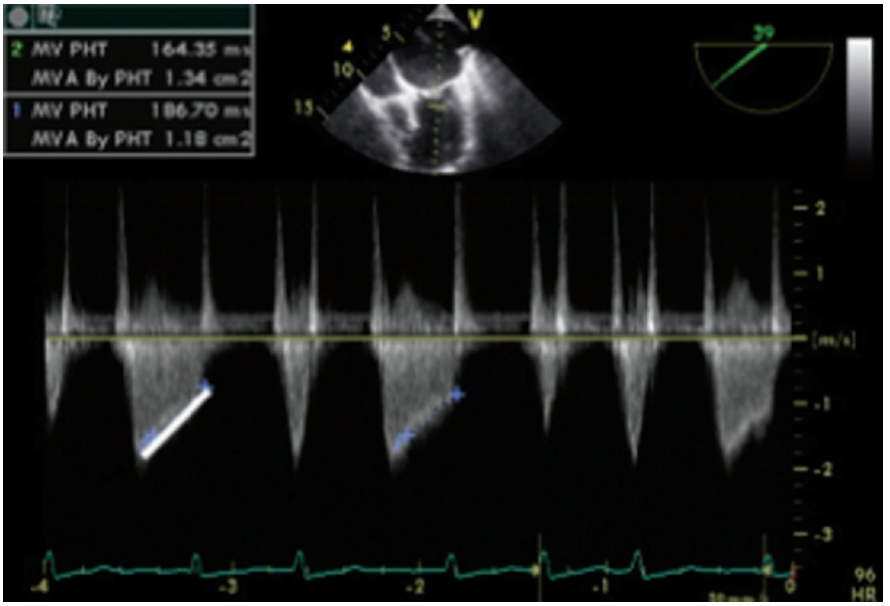
Planimetry measurement of the mitral valve area is accomplished with the transgastric basal view. The valve opening is traced using the measurement software embedded in the TEE machine.

c) **Valve area**: the valve area is obtained by either measuring the valve opening in diastole using planimetry or by the pressure half-time technique.
   i) Planimetry involves tracing the valve opening in the TG basal view during diastole—giving an estimate of the open valve area. Image quality is the main limitation with this technique (**Fig. 203-4**).
   ii) Pressure half-time: to use the pressure half-time technique, the transmitral flow velocity is measured using continuous wave Doppler (**Fig. 203-5**). The time it takes to decrease by a factor of the square root of 2 is calculated. A normal value for pressure half-time is <60 ms. Severe mitral stenosis will have a pressure half-time >220 ms.
   To calculate the valve area from the pressure half-time, use the following formula:

$$\textbf{MV area (cm}^2\textbf{)} = \textbf{220/pressure half} - \textbf{time (ms)}$$

   iii) Alternatives: the continuity equation and PISA method can also be used to calculate the valve area. The basic equations are below.

FIGURE 203-5 Midesophageal Four Chamber View With Continuous Wave Doppler to Measure Pressure Half-Time.



The pressure half-time is measured using continuous wave Doppler through the valve. The *white line* is the slope of the decrease of the velocity in diastole. The pressure half-time is calculated by the TEE machine and can be used to calculate the valve area using the formula: MV area (cm^2) = 220/pressure half-time (ms).

$$\text{Continuity equation}: \text{MVA} = \frac{(\text{LVOT}_{\text{AREA}} \times \text{LVOT}_{\text{VTI}})}{\text{MV}_{\text{VTI}}}$$

$$\text{PISA}: \text{MVA} = 2\pi r^2 \times \frac{\alpha}{180} \times \frac{V_a}{V_p}$$

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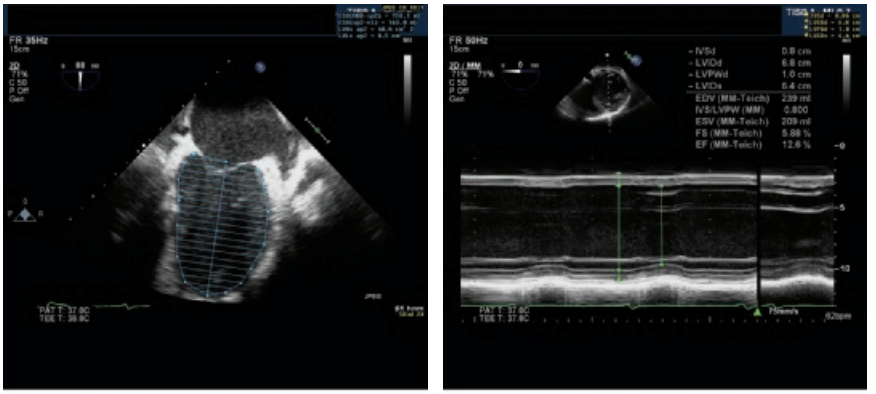
204 Quantitative Echocardiography

Tamas Seres

Transesophageal echocardiography (TEE) is an important monitoring and diagnostic modality in the field of cardiac anesthesia. Due to the reliability and safety of TEE, its application is extending into other areas of anesthesiology as well. This chapter provides an overview of TEE evaluation of systolic and diastolic function along with tools to evaluate pressure gradients (PGs) and valve areas in patients with stenotic or regurgitant valves.

1) **General concepts—myocardial function**
   One of the most common indications for a TEE examination in the OR is to evaluate left ventricular (LV) systolic function. It is the most important part of the intraoperative examination in cardiac anesthesia before and after cardiopulmonary bypass. The TEE measurements provide parameters to evaluate the LV systolic function in patients with chronic or acute heart failure (HF).
   a) **Myocardial remodeling**: Myocardial remodeling is manifested clinically by changes in cardiac size, shape, and function in response to increased load or myocardial injury. The cause of pathologic myocardial remodeling include:
      i) Pressure overload (eg, aortic stenosis [AS], hypertension)
         (1) Concentric myocardial hypertrophy—normal heart volumes and ejection fraction (EF)
      ii) Volume overload (eg, valvular regurgitation)
         (1) Eccentric myocardial hypertrophy—large LV end-diastolic volume (LVEDV), LV end-systolic volume (LVESV), ↓ EF, along with normal or altered stroke volume (SV) and cardiac output (CO)
      iii) Altered myocardial contractility
         (1) Myocardial infarction, inflammatory myocardial disease, idiopathic dilated cardiomyopathy
         (2) Characterized by ↓ SV, ↑ LVESV and LVEDV, and ↓ EF. The SV may be normal depending on the stage of the remodeling process and the treatment of the heart disease.[1]
2) **Systolic function of the left ventricle**
   Principle: A heart with normal systolic function generates appropriate CO with low filling pressures for appropriate oxygen delivery to the body to cover the oxygen consumption at rest or during exercise.[2]
   a) **TEE image planes:** For the evaluation of the LV volumes and systolic function, the following TEE image planes are considered[3,4]:
      i) ME 4-chamber view, ME 2-chamber view, ME long axis (LAX) view
      ii) Transgastric (TG) views are used for diameter, area change, and direct Doppler measurements.
      iii) TG mid short axis (SAX) view, deep TG view, TG LAX
   b) **Evaluation of systolic function includes the measurement of**
      i) LVEDV/LVESV
         Principle: As the LV contracts, it shortens in the LAX/SAX, and there is a twisting effect where the base and the apex rotate around the LAX of the left ventricle. Based on the complex pattern of the LV systolic contraction, the ME LV views are the most

FIGURE 204-1 Examples for Systolic Function Determined by Simpson Method and M-mode.



A. LVEDV and LVESV were measured in 4- and 2-chamber views by tracing the endocardial border. The LV was divided to discs, and the volume was calculated by the summation of the volumes of each disc. **B.** The LVIDd and LVIDs were measured in the same patient. Fractional shortening (FS) can be calculated: (LVIDd − LVIDs)/LVIDd × 100 (%). The EDV and ESV were computed by the Teichholz method. This patient has a dilated heart with severe systolic dysfunction.

appropriate.[5] LVEDV and LVESV are measured by using the method of discs (MOD) or Simpson method by using two ME views. The Simpson method slices the LV into several discs and measures the volume of each disc. Aberrations in LV shape will not introduce as much error into the result compared to other standard measurements.[6]
Image planes: ME 4-chamber and 2-chamber views
Measurement: The measurement is performed by tracing the endocardial border of the LV in 4-chamber and 2-chamber views in end-diastole and end-systole. After tracing the endocardial border under the option of MOD, the computer automatically calculates the volumes (**Fig. 204-1, Panel A**).
Normal values can be seen in **Table 204-1**.

ii) LVEDV and LVESV measurement by using M-mode images
Principle: The measurement of LV internal diameter at end-diastole (LVIDd) and end-systole (LVIDs) can be used for mathematical assumption of the LV volume using the Teichholz method.
Image planes: TG SAX mid-papillary view
Measurement: The diameter can be measured by caliper under the M-mode measurement option, and the volume will be calculated by the computer of the echo machine using the Teichholz method:

$$\mathbf{V = [7/(2.4 + LVID)] \times LVID^3}$$

Comment: The measurement of LVEDV, LVESV, and EF with the Teichholz method requires absence of regional wall motion abnormalities and normal shape of the LV (see **Fig. 204-1**).[7]

iii) Ejection fraction
Principle: EF can be calculated quantitatively by using the volumes measured by the Simpson or Teichholz methods (see **Fig. 204-1, Panel B**).

$$\mathbf{EF = (EDV - ESV)/EDV \times 100}$$

Normal values: 55%-75%

TABLE 204-1 LV Systolic Function

	Men		Women
LVEDV (mL)	62-170		55-101
LVESV (mL)	14-76		13-60
LVIDd (cm)	4.2-5.9		3.9-5.3
LVIDs (cm)	2.6-4.0		2.3-3.5
SV (mL)		70-100	
CO (L/min)		4-8	
CI (L/min/m^2)		2.5-4.2	
EF (%)		55-70	
FS (%)		28-44	
FAC (%)		40-75	
Wall thickness Posterior wall (cm) Septum (cm)		 0.6-1.1 0.6-1.1	
Contractility (d*P*/d*t*) (mm Hg):		>1200 (normal) <1000 (abnormal)	

Comment: The advantage of EF is the uniformity among patients. While the patient's size, body habitus, and sex may be different, EF represents systolic function that is comparable between patients and comparable in a single patient under different conditions. Preload, afterload, and contractility can affect the EF. An increase in preload or contractility as well as a decrease in afterload will increase the EF.

iv) SV, CO, cardiac index (CI)

(1) Volumetric measurement

Principle: SV can be measured by using the volume data of the Simpson or Teichholz methods (see **Fig. 204-1**). SV = EDV − ESV

Normal values: 70-100 mL

(2) Doppler measurement

Principle: There is a direct way to measure SV by using pulsed wave Doppler (PWD) in LV outflow track (LVOT). The SV measurement is based on the concept that the blood is flowing through the LVOT cross-sectional area (CSA_{LVOT}) with changing velocity during systole. The time integral of the velocity curve (VTI) or the area under the curve is the distance that the blood travels during systole through the CSA_{LVOT}. The SV is the product of the $CSA_{LVOT} \times VTI_{LVOT}$. The velocity curve should be obtained at the level of the measurement of the CSA to get exact result.

Image plane: ME LAX view, deep TG view, or TG LAX view **(Fig. 204-2, Panel A and B)**

Measurement: The calculation of CSA_{LVOT} is based on the measurement of the diameter (*D*) of the LVOT in the ME LAX view (**Fig. 204-2, Panel A**). Assuming that the LVOT area is a circle, the area is:

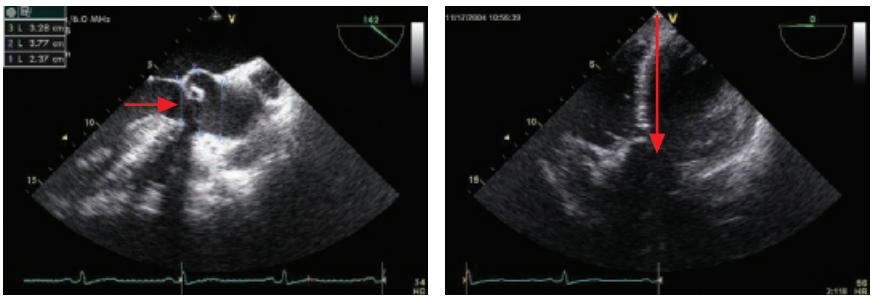
$$\mathbf{CSA_{LVOT} = r^2 \times \pi = (D/2)^2 \times \pi = D^2 \times \pi/4 = D^2 \times 0.785}$$

The SV is calculated as follows:

$$\mathbf{SV = CSA_{LVOT} \times VTI_{LVOT}}$$ (**Fig. 204-3, Panel A**).[8]

Comment: The SV derived from volumetric measurements can be different from Doppler SV measurement in the LVOT. For example, in a patient with mitral regurgitation (MR) or ventricular septal defect, the volumetric SV is

FIGURE 204-2 Typical Views for CSA_{LVOT} and Doppler Measurement.



A B

A. The LVOT diameter is measured on the LV LAX view, and the CSA is calculated using the equation for area of circle. LVOT diameter = 2.37 cm
$CSA_{LVOT} = 2.37^2 \times 0.785 = 4.4\ cm^2$.
B. The deep TG view is seen for Doppler measurements. The *arrow* shows the site of the LVOT diameter. The *arrow* localizes the cursor of the pulse wave Doppler at the level where the diameter was measured.

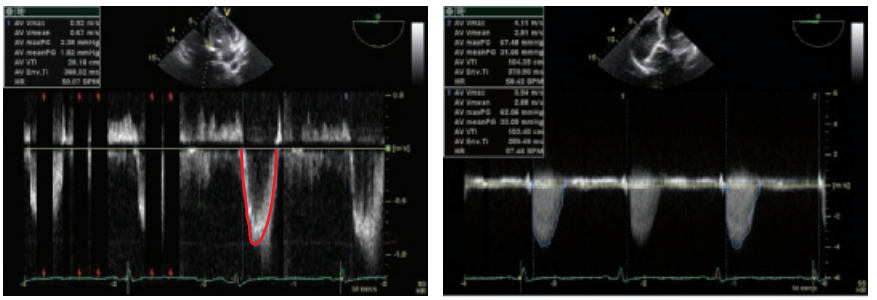
bigger than the Doppler SV. The difference is the regurgitant or the shunt volume, respectively.

(3) **CO and CI**

Principle: The measured SV is multiplied with the heart rate (HR) to obtain the CO:

$$\mathbf{CO = SV \times HR}$$

FIGURE 204-3 Evaluation of AS Using the Continuity Equation.



A B

A. PWD is used to measure the flow and SV in the LVOT. The $CSA_{LVOT} = 4.4\ cm^2$ from **Figure 204-2**. $V_{LVOT} = 92$ cm/s, $VTI_{LVOT} = 26$ cm
$Flow_{LVOT} = CSA_{LVOT} \times V_{LVOT} = 405$ mL/s
$SV = CSA_{LVOT} \times VTI_{LVOT} = 4.4 \times 26 = 114$ mL.
B. CWD is used to measure the flow and SV at the AS. The AS cross-sectional area (CSA_{AS}) can be calculated by the flow or SV continuity equations. $V_{AS} = 402$ cm/s, $VTI_{AS} = 104$ cm
Calculation of the AS area (CSA_{AS}) with flow continuity: $CSA_{LVOT} \times V_{LVOT} = CSA_{AS} \times V_{AS}$
$CSA_{AS} = CSA_{LVOT} \times V_{LVOT}/V_{AS} = 405/402 = 1\ cm^2$
Calculation of the AS area with SV continuity: $CSA_{LVOT} \times VTI_{LVOT} = CSA_{AS} \times VTI_{AS}$
$CSA_{AS} = CSA_{LVOT} \times VTI_{LVOT}/VTI_{AS} = 114/104 = 1.1\ cm^2$
The severity of AS can be evaluated by the peak and mean PG measured by CWD.
$PG_{peak} = 64$ mm Hg and $PG_{mean} = 32$ mm Hg represent moderate AS.

CI: CO/body surface area
Normal values: 2.5-4.2 L/min/m^2

v) Evaluation of regional wall motion abnormality (RWMA)

Principle: The LV is divided to 16 segments to describe the wall motion of different areas supplied by different coronary arteries. There are basal, mid, and apical segments. There are six segments in the basal and mid level (anterior, anteroseptal, septal, inferior, posterior, lateral) and four segments at apical level (anterior, septal, inferior, lateral).

Image planes: To evaluate the different segments, multiple views should be used: ME 4-chamber, ME 2-chamber, ME LAX views. TG SAX views are frequently used to evaluate the SAX movement of the segments.

Measurement of wall motion: During systole, the myocardial wall thickens and the endocardial wall moves toward the center of the heart (wall motion). In current clinical practice, analysis of LV segmental function is based on a qualitative visual assessment of the motion and thickening of a segment during systole. The recommended qualitative grading scale for wall motion is:

(1) Normal (>30% movement of the endocardium to the center of the LV)
(2) Mildly hypokinetic (10%-30% movement of the endocardium)
(3) Severely hypokinetic (<10% movement of the endocardium)
(4) Akinetic (no movement of the endocardium)
(5) Dyskinetic (moves paradoxically during systole).

Comments: RWMA represents segmental systolic dysfunction and alters long-term morbidity and mortality in ischemic heart disease.[9] The causes of RWMA include ischemia, infarction, hibernation, stunning, bundle-branch block, pacemaker, or artifact. Myocardial ischemia can be detected early by monitoring wall motion in intraoperative setting.[3]

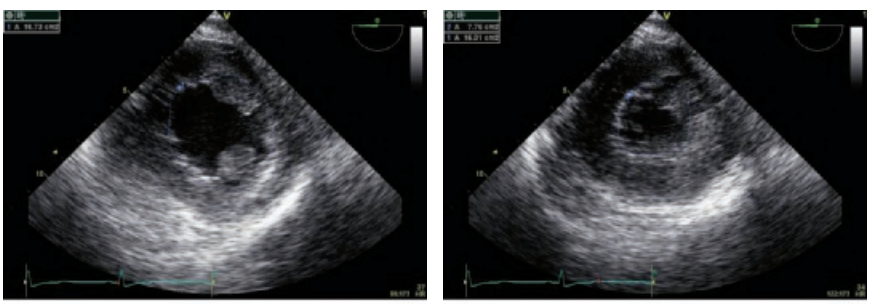
vi) Additional measurements

(1) LV SAX area measurement

Principle: More than 80% of the SV is due to the shortening of the SAX. Therefore, the change in area during systole in LV SAX view at mid-papillary level may represent the systolic volume change of the LV.

Image planes: TG mid SAX

FIGURE 204-4 LV SAX Area Measurement.



A. The area of the LV during diastole (EDA). **B.** The area of the LV during systole (ESA). FAC = (EDA − ESA)/EDA × 100 (%).

Measurement: The LV areas are measured by manual planimetry of the area of the LV at end-diastole (EDA) and end-systole (ESA) excluding the papillary muscles (**Fig. 204-4, Panels A and B**).
Normal values: EDA: 9.5-22 cm^2, ESA: 4-11.6 cm^2
Comments: Changes of ESA may reflect the changes in contractility (increased contractility decreases ESA) or afterload (increased afterload increases ESA). Qualitatively, if the papillary muscles are seen "kissing" or making contact during systole, then hypovolemia (most frequent), low peripheral resistance, or increased contractility can be present.[10]

(2) Fractional area change (FAC)
Principle: The area change during systole compared to the EDA is the FAC, which correlates well with EF (see **Fig. 204-4**).

$$\mathbf{FAC = (EDA - ESA)/EDA \times 100}$$

Normal values: 40%-75%
Comment: Although the value of FAC is generally used for off-line quantitative analysis, a qualitative assessment of ESA, EDA, and FAC is important in evaluating the etiology of hemodynamic instability.[3]

(3) LV internal diameter measurement with M-mode and fractional shortening (FS)
Principle: LVIDd and LVIDs can be measured in either 2D or M-mode images. Internal diameters can be obtained easily, and they are characteristic in different remodeling conditions.
Image planes: TG mid SAX view
Measurement: Fractional shortening
After obtaining the TG mid SAX view, the diameter of the LV can be measured in diastole (LVIDd) or in systole (LVIDs) by using a caliper in the M-mode option of the echo software (see **Fig. 204-1, Panel B**). The percentage change in the LVID during systole compared to end-diastole can be used to evaluate LV systolic function.

$$\mathbf{FS = (LVIDd - LVIDs)/LVIDd \times 100}$$

Normal values: Table 204-1
Comments: The papillary muscles should be excluded from the measurement. The LVIDd and LVIDs can be used to evaluate eccentric remodeling in mitral or aortic valve regurgitation[11] (see **Fig. 204-1, Panel B**). FS has limited value in evaluating systolic function because it measures changes only in a narrow segment of the LV in one dimension.[3]

3) **Diastolic function**
   a) **Principle**: The optimal performance of the left ventricle depends on its ability to cycle between two states:
      i) A compliant chamber in diastole that allows the left ventricle to fill with low left atrial (LA) pressure
      ii) A stiff chamber with rapidly rising pressure in systole that ejects the SV at arterial pressures.
      iii) The SV must increase in response to demand, such as exercise, without much increase in LA pressure. Elevated filling pressures are the main physiologic consequence of diastolic dysfunction.
      iv) Filling pressures are considered elevated when the mean pulmonary capillary wedge pressure (PCWP) is >12 mm Hg or when the LVEDP is >16 mm Hg.
      v) Impaired LV relaxation and/or poor LV compliance may lead to elevated filling pressures with or without the clinical symptoms of HF.

vi) Diastolic HF represents patients with abnormal diastolic function and normal EF. The staging and functional classes of diastolic HF are the same as it was described for systolic HF.[2]

b) **The phases of diastole**

i) Isovolumic relaxation time (IVRT): The time from the aortic valve closure to mitral valve opening. During isovolumic relaxation, the volume of the left ventricle is constant but the pressure falls. The period of IVRT is an active, ATP burning phase. The mitral valve will open when the LAP exceeds the LV pressure. If the LA pressure is elevated, the IVRT will be shortened. Conversely, if the relaxation of the left ventricle is decreased, then the IVRT will be increased.

ii) Rapid filling phase: The rapid filling phase accounts for most of the filling of the left ventricle (80%). The factors that account for the amount of filling during this phase are the LV suction, pressure difference between the left atrium and the left ventricle, the LV compliance, and the left atrial volume.

iii) Diastasis: During diastasis, the LA-LV pressure difference is small. Filling of the left ventricle (5%) is mostly due to pulmonary venous flow with the LA acting as a conduit. LV compliance is the main determinant of filling during diastasis.

iv) Atrial contraction: Factors determining the filling of the left ventricle (15%-20%) from an atrial contraction are ventricular compliance, pericardial restraint, atrial contractility, and electrical synchrony (PR interval).[2,12]

c) **Evaluation of diastolic function with TEE**

i) Mitral Doppler inflow velocity

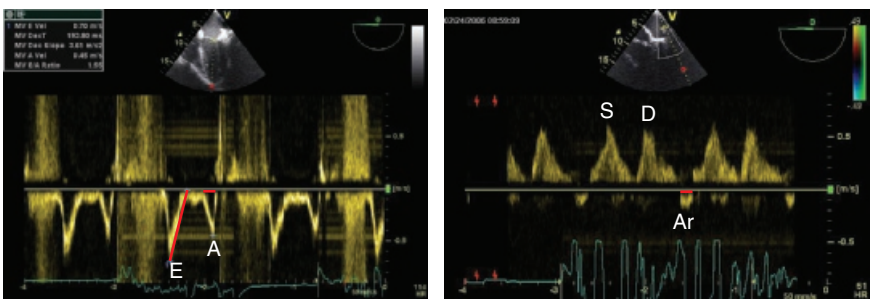
Principle: Pulsed wave Doppler is used to visualize the flow through the mitral valve. The early diastolic wave of the mitral inflow is the E wave. In normal patients, the LV relaxation and untwisting generate negative pressure in the LV at the time of the opening of the MV—generating the E wave. The second wave or A wave is generated by the atrial contraction.

The velocity of E and A, their ratio, deceleration time (DT) of E, and duration of A are used to characterize the diastolic function (**Fig. 204-5, Panel A).**

Image plane: ME 4-chamber view

Measurements: The PWD cursor is positioned at the tip of the mitral leaflets to gain the best possible velocity curve. Maximum velocity of the E and A wave is measured by using the velocity scale. DT of the E wave can be measured by using

FIGURE 204-5 Evaluation of Diastolic Function With PWD at the MV and Pulmonary Vein.



A B

A. Mitral valve inflow was obtained with PWD. The early diastolic wave (E) and the atrial kick (A) can be differentiated. The *red lines* represent DT measurement at the E wave and A duration at the A wave. **B.** Pulmonary vein flow was obtained with PWD. The S wave represents the flow during systole, the D wave is the early diastolic flow, and the Ar is a backflow into the pulmonary veins during the atrial contraction. The *red line* represents the duration of Ar.

TABLE 204-2 LV Diastolic Function

	Normal	Grade I	Grade II	Grade III
Decreased relaxation		+	+	+
Increased LVEDP			+	++
Decreased compliance			+	++
NYHA functional class	I	I-II	II-III	III-IV
Mitral inflow				
E/A ratio	0.8-1.9	<0.8	0.8-1.9	≥2
DT (ms)	160-200	>200	160-200	<160
IVRT (ms)	60-100	>100	60-100	<60
Pulmonary vein flow				
S/D	≥1	>1	<1	<<1
Ar (m/s)	<0.35	<0.35	≥0.35	≥0.35
Ar − A (ms)	<0	<0	≥30	≥30
Tissue Doppler velocity of the MV annulus				
E′ septal (cm/s)	≥7	<7	<7	<7
E′ lateral (cm/s)	≥10	<10	<10	<10
E/E′ average ratio	<14	<14	>14	>14
Flow propagation velocity				
*V*p (cm/s)	>50	<50	<50	<50

the slope measurement tool, which calculates the time from the maximum velocity to zero velocity. The duration of the A wave can be measured by time measurement options (see **Fig. 204-5, Panel A**).

Comments: Decreased relaxation accompanies decreased E wave, E/A ratio, and increased A wave. Increased LA pressure increases the PG during early diastole resulting in high E wave and E/A ratio. Increased LV end-diastolic pressure causes decreased A-wave velocity and duration (**Table 204-2**).

ii) Pulmonary vein flow

Principle: Pulsed wave Doppler is used to measure the velocity of the flow through the pulmonary vein. The wave during LV systole is the S wave, which has two components. The S1 wave represents the relaxation of the LA after atrial contraction. The S2 wave is more prominent than S1 and represents the flow from the pulmonary vein into the LA during LV systole due to the movement of the closed MV toward the LV apex. The D wave represents the flow from the pulmonary vein into the LA during early diastole after the opening of the MV. The A reversal (Ar) is a flow from the LA into the pulmonary vein during the atrial contraction (see **Fig. 204-5, Panel B**).

Image plane: ME 2-chamber view focusing on the area of left atrial appendage and the left upper pulmonary vein running close to it

Measurement: The PWD cursor is placed into the pulmonary vein, and the velocity curve is registered. The velocities of S2, D, and Ar waves; the S/D ratio; as well as the duration of the Ar wave are measured and used to characterize the diastolic function in addition to the parameters of the mitral inflow.

Comments: Increased LA pressure decreases the S-wave velocity. Relaxation abnormality decreases the velocity of the D wave. Increased LV end-diastolic pressure may increase the velocity and duration of Ar (see **Table 204-2, Panel B**).

iii) Tissue Doppler velocity measurement

Principle: By eliminating high-velocity/low-amplitude signals in color Doppler, the low-velocity/high-amplitude movements of the myocardium during systole and diastole can be visualized and measured. The MV annulus moves toward the apex during systole (S wave) and away from the apex during early and late diastole. The

early diastolic annular velocity is expressed as E′ and the late diastolic velocity as A′. The peak velocity of these diastolic movements and their ratio are measured for characterization of the diastolic function parallel with the flow evaluation through the MV and pulmonary vein. For the assessment of global LV diastolic function, it is recommended to acquire and measure tissue Doppler signals at least at the septal and lateral sides of the mitral annulus and their average (E′ average), given the influence of regional function on these velocities and time intervals (**Fig. 204-6, Panel A**).

Image plane: ME 4-chamber view using tissue Doppler image

Measurement: For obtaining the velocity curve of the MV annulus, the PWD cursor is placed on the septal or lateral edge of the MV annulus in tissue Doppler mode.

Comments: Tissue Doppler parameters are important for full evaluation of diastolic function and LV filling pressures. Low E′ (<8 cm/s) represents impaired relaxation. The MV inflow E/tissue Doppler E′ average ratio <8 is usually associated with normal LV filling pressures, whereas a ratio >13 is associated with increased filling pressures (see **Table 204-2**).

iv) Color M-mode flow propagation velocity

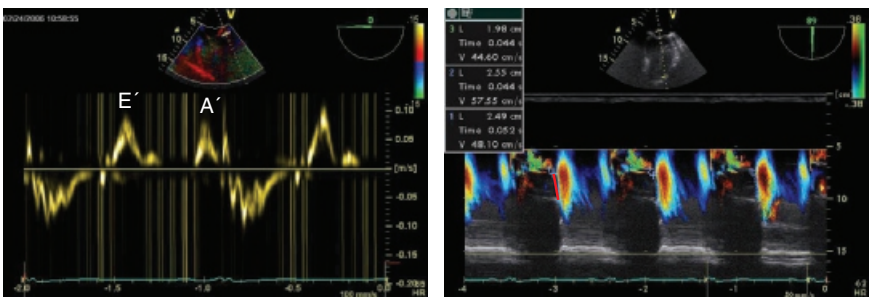
Principle: The most widely used approach for measuring mitral-to-apical flow propagation is the slope method. Acquisition is performed in the apical 4-chamber view, using color flow imaging. The M-mode scan line is placed through the center of the LV color inflow from the mitral valve to the apex. Flow propagation velocity (V_p) is expressed as the slope (cm/s) of the first aliasing velocity during early filling, measured from the mitral valve plane to 4 cm distally into the LV cavity. $V_p > 50$ cm/s is considered normal.

Image plane: ME 4-chamber view with optimal MV opening

Measurement. The edge of the aliasing velocity, as it described above, is traced by the slope function, and the V_p can be obtained (see **Fig. 204-6, Panel B**).

Comment: In patients with depressed EF, V_p can provide useful information for the prediction of LV filling pressures. The E/V_p ratio >2.5 predicts PCWP > 15 mm Hg with reasonable accuracy.

FIGURE 204-6 Evaluation of Diastolic Function With Tissue Doppler and Color M-Mode Method.



A. Tissue Doppler modality was used, and the PWD cursor was placed on the lateral part of the mitral valve annulus. The annulus is moving toward the apex during systole (first negative wave) and away from the apex during early diastole (E′) and late diastole or atrial kick (A′). The E/E′ ratio is depending on the LA pressure. **B.** Color Doppler on the MVA and M-mode is directed through the color flow. The *red line* is at the flow propagation during early diastole. The measurement is performed at the *yellow-blue* transition line using the slope measurement option on the computer.

d) **Evaluation of the severity of LV diastolic dysfunction:** Three grades of diastolic dysfunction are identified by Doppler echocardiography:
   i) Grade I—impaired relaxation
      (1) In patients with mild diastolic dysfunction, the mitral E/A ratio is <0.8, DT is >200 ms, IVRT is ≥100 ms, predominant systolic flow is seen in pulmonary venous flow (S/D > 1), annular E′ septal is <8 cm/s and E′ lateral is <10 cm/s, and the E/E′ average ratio is <8 (see **Table 204-2**). Importantly, even in asymptomatic patients, grade I diastolic dysfunction was associated with a fivefold higher 3- to 5-year mortality in comparison with subjects with normal diastolic function.
      (2) Because the majority of subjects aged >60 years without a history of cardiovascular disease have E/A ratios <1 and DTs >200 ms, such values in the absence of further indicators of cardiovascular disease (eg, LV hypertrophy) can be considered normal for age.
      (3) Reduced mitral E/A ratio in the presence of normal annular tissue Doppler velocities can be seen in volume-depleted normal subjects, so an E/A ratio <0.8 should not be universally used to infer the presence of grade I diastolic dysfunction.
   ii) Grade II—pseudonormal
      (1) In patients with moderate diastolic dysfunction (pseudonormal pattern), the mitral E/A ratio is 0.8-1.9 (pseudonormal) and decreases by >50% during the Valsalva maneuver. DT is 160-200 ms and IVRT is 60-100 ms. E′ septal is <8 cm/s and E′ lateral is <10 cm, and the E/E′ average ratio is 9->14. Other supporting data include an Ar velocity >35 cm/s and an S/D ratio <1 (see **Table 204-2**).
      (2) Grade II diastolic dysfunction represents impaired myocardial relaxation with mild to moderate elevation of LV filling pressures due to moderately decreased LV compliance. These patients may have symptoms of HF with NYHA II-III functional status.
   iii) Grade III—restrictive
      (1) Severe diastolic dysfunction or restrictive LV filling occurs with an E/A ratio >2, DT < 160 ms, IVRT < 60 ms, E/E′ average ratio >14, S/D << 1, Ar duration − A duration (Ar − A) >30 ms (see **Table 204-2**).
      (2) LV filling may revert to pseudonormal or impaired relaxation pattern with successful therapy in some patients (grade IIIa), whereas in others, LV filling remains restrictive (grade IIIb). The later predicts a high risk for cardiac morbidity and mortality. Patients with grade III diastolic function may have symptoms of HF with NYHA III-IV functional status.
      (3) Grade IIIb dysfunction should not be determined by a single examination and requires serial studies after treatment is optimized. LA volume is increased in grades II and III of diastolic dysfunction but can be within normal limits in grade I.

e) **Hemodynamic measurements using the diastolic parameters**

f) Elevated LV filling pressures are important characteristics of clinically significant systolic and diastolic dysfunction. The increased filling pressures represent elevated preload. A clinical scenario of elevated preload with small SV and EF suggests significant systolic dysfunction. However, elevated preload with normal SV and EF may represent significant diastolic dysfunction. Elevated LV filling pressures in the OR might suggest high risk for pulmonary edema in cases with significant fluid shifts and volume changes (**Table 204-3**).[13] This table reflects the newest recommendations for evaluation of LA pressure. Besides E' septal and lateral velocities and E/E' ratio it considers the tricuspid regurgitation (TR) velocity and LA volume index also. Velocity of TR reflects pulmonary artery pressure, which increases with increasing LA pressure. LA volume index representing the effect of long lasting diastolic dysfunction with increased LA pressure.[14] If TR velocity or LA volume index is not available the additional echo parameters like S/D ratio can be used for evaluating patients with diastolic dysfunction.

TABLE 204-3 Increased Filling Pressures Suggested by Diastolic Parameters

Patient with depressed EF and LA pressure >12 mm Hg
1. E/A ratio ≥2 and DT < 160 ms
2. E/A ratio 0.8-1.9: E/E′ average >14 TR velocity >2.8 m/s, LA volume index >34 ml/m^2
E/V_p ratio ≥2.5
S/D < 1
Ar − A ≥30 ms
Patient with normal EF and LA pressure >12 mm Hg
1. E/E′ average >14
2. E′ septal velocity <7 cm/s or E′ lateral velocity <10 cm/s
3. TR velocity >2.8 m/s
4. LA volume index >34 ml/m^2

4) **Doppler measurements of pressure gradients (PG) and valve areas**

a) **Measurement of PGs**

Principle: The PG through a stenotic valve needs to be evaluated to determine the severity of the valve disease. Based on the physical principle of flow continuity in a closed system, wherever the area of the flow is decreased, the velocity is increased. The velocity is highest at the point where the area is the smallest. In the case of AS or mitral stenosis (MS), the velocity of the flow is highest at the site of the stenosis. Measurement of the peak velocity allows calculating the peak PG at the site of the stenosis by using the simplified Bernoulli equation.

$$\textbf{Peak PG} = 4 \times \textbf{V}^2$$

V = the peak velocity of the flow through the stenotic valve

Image planes: Deep TG or TG LAX view for AS and ME 4-chamber view for MS[8]

Measurement: Continuous wave Doppler (CWD) aligned to the blood flow through the stenotic valve is used to measure the highest velocity in the direction of the flow. After obtaining the velocity curve, the curve is traced and the peak and mean PG are determined by the computer of the echo machine. In clinical practice, the mean PG gradient is used to determine the severity of the stenosis (see **Fig. 204-3, Panel B**).

b) **Evaluation of LV contractility by measuring PGs in MR**

Principle: Contractility is described as the change of pressure in the LV over time (dP/dt). The highest dP/dt can be measured during the isovolumic contraction. The isovolumic contraction becomes visible for TEE measurement in patients with MR. The MR jet can be used for the measurement of contractility using CWD.

Image plane: ME 4-chamber view

Measurement: A velocity measurement, early in the flow acceleration phase of systolic ejection, is used to calculate an early pressure, using the simplified Bernoulli equation (PG = $4 \times V^2$). Another velocity, later in the flow acceleration phase, is used to calculate another pressure. The difference in pressures is the dP part of the dP/dt value. The difference in time between the last and first velocity measurements make the dt part of the formula. The typical values used are the time difference, in seconds, between the velocity values of 3 and 1 m/s. The pressures are $4 \times 3^2 = 36$ mm Hg and $4 \times 1^2 = 4$ mm Hg with a difference of 32 mm Hg divided by the seconds between the velocity measurements (**Fig. 204-7**).

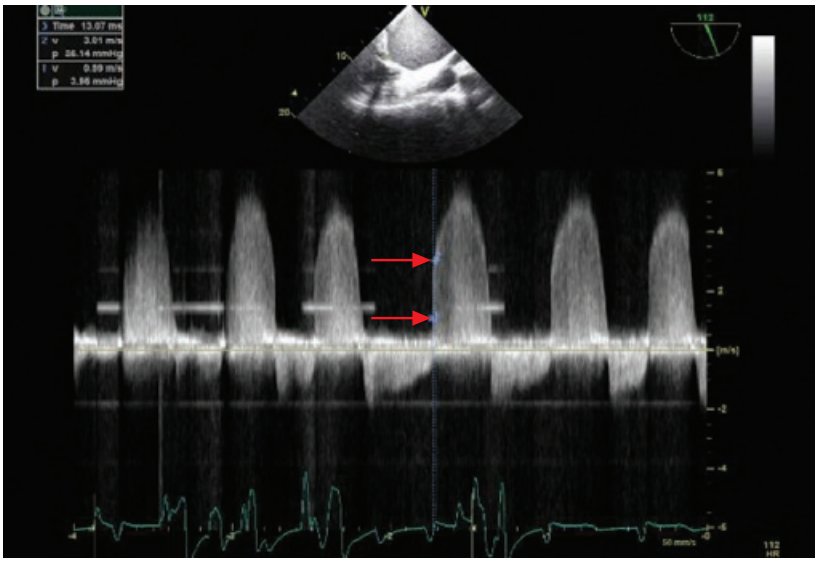
Normal value: 1200 mm Hg/s, abnormal value is <1000 mm Hg/s

Comment: One study found that $dP/dt < 600$ mm Hg/s identified a high-risk group with a reduced event-free survival after myocardial infarction.[14]

c) **Measurement of MS area with pressure half-time (PHT)**

Principle: PHT is the time required for the gradient between the left atrium and the left ventricle to fall to one half of its initial value. Based on catheterization data, a PHT

FIGURE 204-7 Measurement of LV Contractility Using CWD Image of the MR Jet.



CWD measures the velocity change through the MR orifice. The PG at 1 and 3 m/s was calculated by using the simplified Bernoulli equation ($4 \times V^2$). The pressure difference and the time difference were determined between the two points (*red arrows*), and the contractility was computed by using the d*P*/d*t* formula. Contractility = 32 mm Hg/0.13 s = 2461 mm Hg/s.

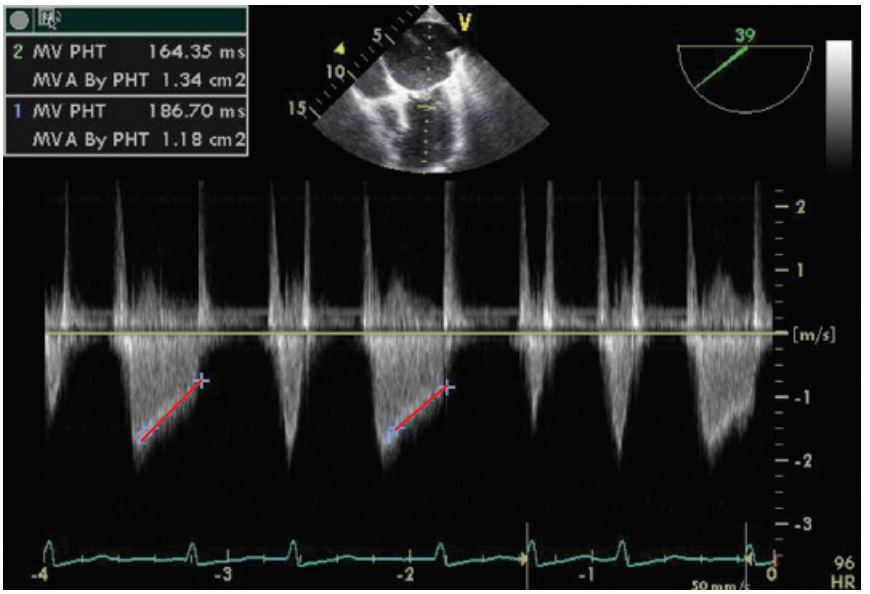
of 220 ms is equivalent to a mitral valve area (MVA) of 1 cm^2. MVA can be calculated by obtaining PHT from the MS velocity curve:

MVA = 220/PHT

Image planes: ME 4-chamber view
Measurement: CWD aligned trough the MS. The PHT is measured on the slope between the E and A wave with the PHT option (**Fig. 204-8**).
Comments: In case of MS and MR, the MVA will be overestimated because of the increased flow and decreased PHT during the diastole.[8]

d) **Measurement of the AS area with the continuity equation**
Principle: Continuity equation is an important tool to evaluate areas with unknown size such as areas of AS, MS, or regurgitant areas of aortic insufficiency or MR. The severity of the AS can be graded by the mean PG and area of the stenosis. The area measurement is especially important when the patient has low CO because the PG generated through the aortic valve is low in this situation and underestimates the severity of the AS.
The continuity equation is based on assuming a constant flow of fluid through a conduit. Flow (cm^3/s) in a conduit is the product of CSA of the conduit (cm^2) and the velocity (V) of the fluid (cm/s). If there is a stenosis in the conduit, the velocity of fluid will increase at the site of stenosis to keep the continuity of flow. In case of AS, the flow in the LVOT is the same as the flow at the AS. The flow in the LVOT can be calculated by determining CSA_{LVOT} and multiplying it with the V_{LVOT} (see **Figs. 204-2** and **204-3**). The flow at the AS cannot be calculated because the CSA_{AS} is unknown. V_{AS} can be

FIGURE 204-8 Measurement of MS Area With PHT.



The flow through MS was visualized by CWD. The *red line* represents the slope of the decrease of velocity during diastole. The PHT is calculated by the computer of the echo machine. MVA = 220/PHT − 220/186.7 = 1.2 cm^2.

measured by CWD as the highest velocity in the direction of the flow through the CSA_{AS} (see **Fig. 204-3, Panel B**). From the flow continuity, CSA_{AS} can be calculated[8]:

$$\mathbf{CSA_{LVOT} \times V_{LVOT} = CSA_{AS} \times V_{AS}}$$
$$\mathbf{CSA_{AS} = CSA_{LVOT} \times V_{LVOT}/V_{AS}}$$

The SV continuity states that the SV is the same in the LVOT as at the AS. SV is defined as the product of CSA and VTI. The VTI values in the LVOT and at the AS can be used to express CSA_{AS}:

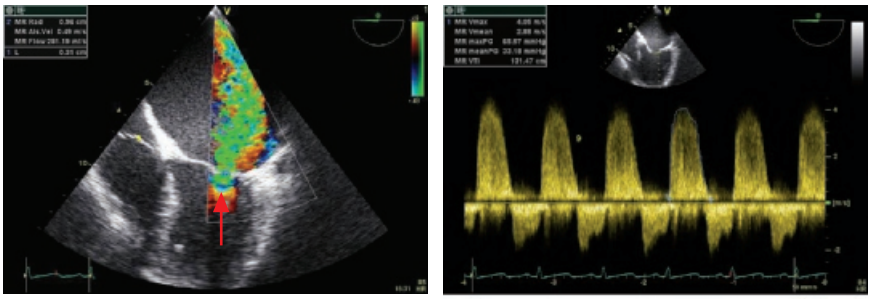
$$\mathbf{CSA_{AS} = CSA_{LVOT} \times VTI_{LVOT}/VTI_{AS}}$$

Comments: Although the method is mostly used for AS area measurement, the MS area can also be measured by this method. In this case, the LVOT flow will be compared to the flow through MS. The peak velocity of MS can be measured by CWD. The MVA can be calculated as follows:

$$\mathbf{CSA_{LVOT} \times V_{LVOT} = MVA \times V_{MS}}$$
$$\mathbf{MVA = CSA_{LVOT} \times V_{LVOT}/V_{MS}}$$

e) **Proximal isovelocity surface area (PISA) technique**
Principle: The area of MR, called effective regurgitant orifice area (EROA), can be calculated as a reliable parameter for grading the severity of the MR. The measurement is based on the fact that as blood flows toward a regurgitant orifice, the blood flow velocity increases with the formation of multiple concentric isovelocity shells. Using

FIGURE 204-9 Calculation of the EROA in MR.



A B

A. Color Doppler image of the MR. The tip of the *arrow* is positioned at the transition of color from *yellow* to *blue* (PISA).
The area of PISA = $2 \times \pi \times r^2$. The velocity at PISA is 49 cm/s, which is the peak velocity on the color scale ($V_{aliasing}$). The flow at PISA = $2 \times \pi \times 0.96^2 \times 49 = 284$ mL/s. **B.** The velocity curve of the MR obtained by CWD. The peak velocity of MR and the VTI of the MR can be measured. $V_{MR} = 405$ cm/s, $VTI_{MR} = 131$ cm.
The $Flow_{MR} = EROA \times V_{MR} = Flow_{PISA}$
$EROA = Flow_{PISA}/V_{MR} = 284/405 = 0.7$ cm^2
$RV = EROA \times VTI_{MR} = 0.7 \times 131 = 92$ mL

color Doppler, the isovelocity shell with the aliasing velocity can be identified. The aliasing velocity is the peak velocity on the color Doppler velocity scale.
In MR, using the 4-chamber view, the regurgitant flow is toward the transducer (red color). Close to the regurgitant orifice, the velocity of the flow is high (yellow color). At the shell surface where the velocity is higher than the maximum velocity on the color scale, the color turns blue from the yellow. This shell surface represents the PISA where the velocity equals to the maximum velocity on the color scale or aliasing velocity. This shell is a hemisphere, and its area can be calculated from the radius (*r*), which is the distance between the PISA and the orifice (**Figs. 204-9, Panel A**).

$$\mathbf{PISA = 2 \times r^2 \times \pi}$$

The flow through the PISA can be calculated because the aliasing velocity can be identified as the maximum velocity on the color scale.

$$\mathbf{Flow_{PISA} = PISA \times V_{aliasing}}$$

Using the continuity equation, the flow through the PISA should be the same as the flow through the EROA. The flow through the EROA can be derived from the product of EROA and the maximum velocity of the MR (V_{MR}) (see **Fig. 204-9, Panel B**):

$$\mathbf{Flow_{EROA} = EROA \times V_{MR}}$$
$$\mathbf{Flow_{PISA} = Flow_{EROA}}$$
$$\mathbf{PISA \times V_{aliasing} = EROA \times V_{MR}}$$
$$\mathbf{EROA = PISA \times V_{aliasing}/V_{MR}}$$ [8]

Image plane: ME 4-chamber view
Measurement: At first, the PISA radius is measured. The aliasing velocity is determined from the color scale. The second step is the measurement of the maximum velocity of MR using CWD. The MR velocity curve is traced, and the maximum velocity (V_{MR}) as well as the VTI_{MR} is measured (see **Fig. 204-9, Panel A and B**).

EROA is calculated as it was described above. Knowing the EROA and the VTI_{MR} of the regurgitant flow, the regurgitant volume (RV) can be calculated:

$$\mathbf{RV = EROA \times VTI_{MR}}$$

Regurgitant fraction (RF) expresses the percentage of the RV of the volumetric SV. Volumetric SV can be calculated with the Simpson or the Teichholz method.

$$\mathbf{RF = RV/SV \times 100}$$

PISA measurement value in severe MR: EROA > 0.4 cm^2

$$\mathbf{RV > 60\ mL}$$
$$\mathbf{RV > 60\ \%}$$

Comment: Measurement of EROA is a relatively volume-independent measurement of the severity of MR.

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205 Part A: Atlas of Transesophageal Echocardiography Cognitive Aids

Normal Values

Larry F. Chu • Nathaen Weitzel

Normal

Aortic annulus size	1.8-2.3 cm
Mitral annulus size	3.0-3.5 cm
Aortic VTI	18-25 cm
Mitral VTI	10-13 cm

Mitral valve

Normal area	4.0-6.0 cm^2
Mild stenosis	1.5-2.5 cm^2
Moderate stenosis	1.0-1.5 cm^2
Severe stenosis	<1.0 cm^2

Mitral valve mean gradient

Mild MS	<5 mm Hg
Moderate MS	5-10 mm Hg
Severe MS	>10 mm Hg

Mitral regurgitation (MR)

	Mild	Moderate	Severe[a]
Vena contracta (cm)	<0.3	0.3-0.7	>0.7
Color Doppler jet area (cm^2)	<4.0	4-8	>8.0[b]

[a]ERO > 0.4 cm^2, radius >1.0 cm, color Nyquist = 40 cm/s, MR peak velocity = 500 cm/s.
[b]or wall hugging jet.

Aortic valve

Normal area	2.5-4.5 cm^2
Mild stenosis	>1.5 cm^2
Moderate stenosis	1.0-1.5 cm^2
Severe stenosis	<1.0 cm^2

Aortic stenosis

	Mild	Moderate	Severe
Peak jet velocity (m/s)	<3.0	3-4	>4.0
Mean gradient (mm Hg)	<25	25-40	>40

Aortic regurgitation

	Mild	Moderate	Severe
Vena contracta (cm)	<0.3	0.3-0.6	>0.6
PHT (ms)	>500	<500-200	<200
Decel rate (m/s^2)	<2.0	2.0-3.5	>3.5

CW Doppler evaluation: holosystolic flow reversal in descending aorta.

Decel rate, deceleration rate; ERO, effective regurgitant orifice; PHT, pressure half time.

AR jet width and LVOT ratio

Mild AR	<25%
Moderate AR	25%-65%
Severe	>65%

Aortic and mitral regurgitant fraction

Mild	<30%
Moderate	30%-50%
Severe	>50%

Pulmonary hypertension

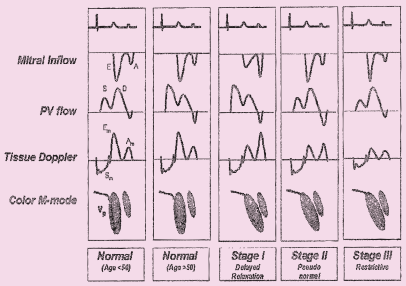
Mild PA systolic pressure	30-40 mm Hg
Moderate PA systolic pressure	40-70 mm Hg
Severe PA systolic pressure	>70 mm Hg
Severe mean PA pressure	>40 mm Hg
Normal d*P*/d*t*	>1200 mm Hg/s (<27 ms)
Borderline	1000-1200 mm Hg/s (27-32 ms)
Abnormal	<1000 mm Hg/s (>32 ms)

Normal LV systolic function parameters

Stroke volume	70-100 mL
Cardiac output	4-8 L/min
Fractional shortening	28%-44%
Fractional area change	40%-75%
Ejection fraction	55%-70%
Mild dysfunction	40%-55%
Moderate dysfunction	26%-39%
Severe dysfunction	<25%

LV hypertrophy (TG SAX)

End-diastole	>1.2 cm
LV wall thickness	



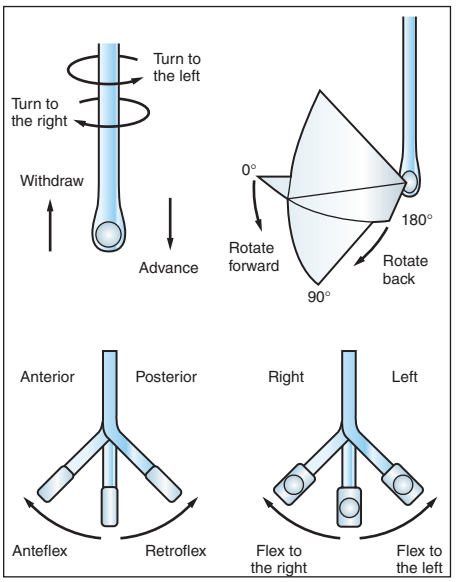
Patterns of diastolic dysfunction by TEE.

206 Performing a TEE Examination

Benjamin A. Abrams

Once you have properly set up your TEE according to manufacturer's guidelines, you may perform a TEE examination.

1. Place an OG tube and suction air out of the stomach.
2. Place ~10 cc of gel into the mouth.
3. Jaw lift and gently intubate the esophagus with the TEE probe, it should pass easily. **If you feel resistance, stop and reevaluate the positioning.** Occasionally direct laryngoscopy with a MAC 3 to visualize the esophagus assists in probe placement.
4. Start by advancing the probe to 35 cm at the incisors and then take a look for the 4 chamber view (0 degree) and evaluate LV/RV size and function. Evaluate the mitral (MV) and tricuspid valves. If desired, a 2 chamber (90 degrees) view can examine the LV apex for pathology.
5. Pull the probe out ever so slightly until the 5 chamber view is visualized (0 degree). Examine the MV and aortic valve (AV) at various angles and in color (see AV and MV pages).
6. Advance the probe to 40-45 cm and obtain the transgastric LV mid-papillary short axis view (0 degree) to assess LV filling and function and SWMA (segmental wall motion abnormalities). To obtain the transgastric view, you will need to anteflex the probe (push down on the wheel with your thumb so that turns clockwise).
7. The deep transgastric long axis view (see 20 views card) which is useful to assess the AV by Doppler (this view can be challenging to obtain).
8. Evaluate the aorta, beginning with the descending and moving up all the way to the arch. Note abnormalities such as dissection and plaques. The surgeon will occasionally ask you to look for their bypass cannulas in the aorta.

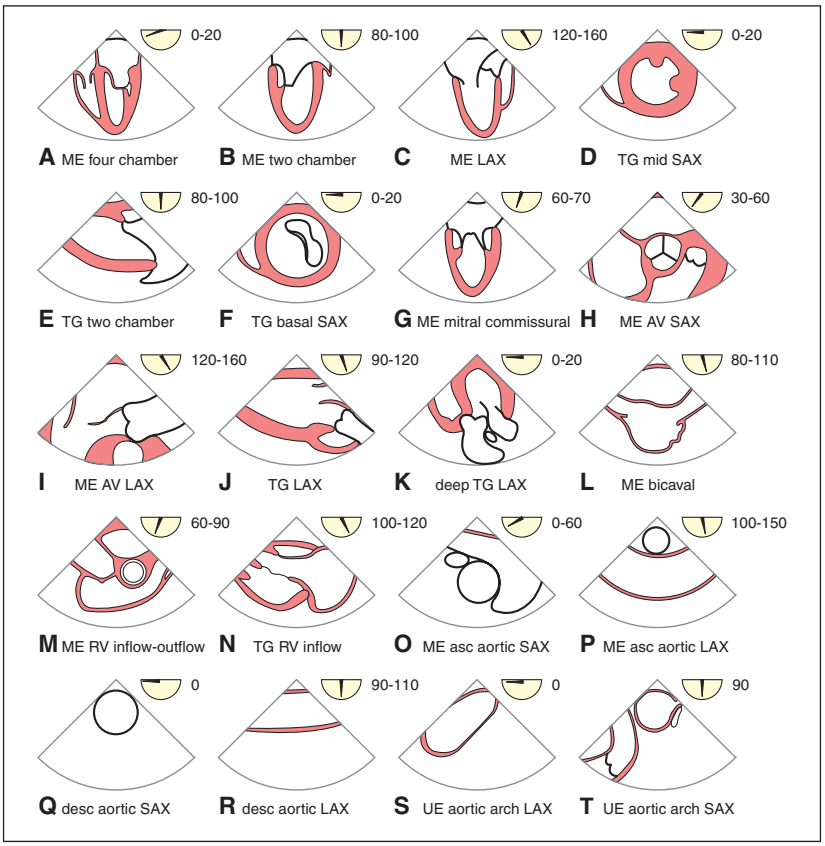


CAVEAT: This is an abbreviated examination. The ASE/SCA guidelines describe a 20 view TEE examination. The key is to be systematic so important pathology and information is not missed during the ETT examination.

DID YOU KNOW? The contraindications for TEE include UGI bleed, perforation, diverticulum, tumor, stricture, varices, and recent gastric or esophageal surgery.

207 ASE/SCA 20 View Exam Overview

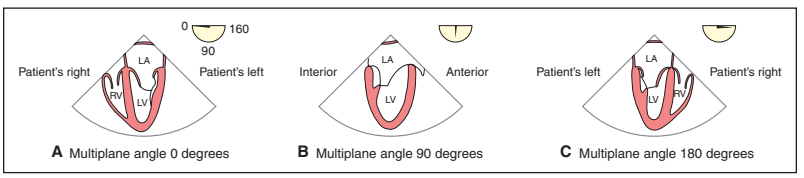
Benjamin A. Abrams • Nathaen Weitzel • Shanthala Keshavacharya • Larry F. Chu



ASE/SCA 20 View Comprehensive TEE Examination

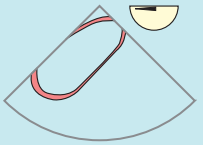
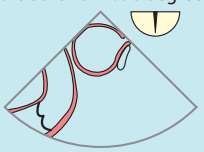
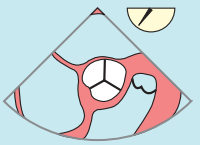
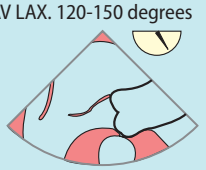
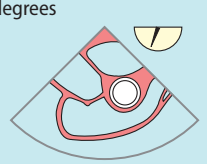
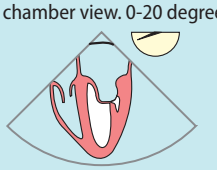
Journal of the American Society of Echocardiography, 12(10):887-900.

20 cross-sectional views composing the recommended comprehensive transesophageal echocardiographic examination. Approximate multiplane angle is indicated by the icon adjacent to each view. ME, mid esophageal (30-40 cm at incisors); LAX, long axis, TG, transgastric (40-45 cm at incisors); SAX, short axis; AV, aortic value; RV, right ventricle, asc, ascending; desc, descending; UE, upper esophageal (20-25 cm at incisors).



ASE/SCA 20 View Exam Card 1/4

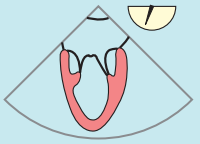
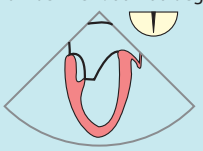
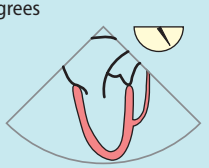
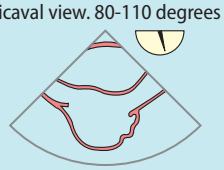
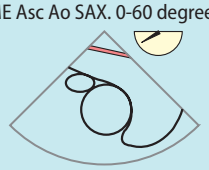
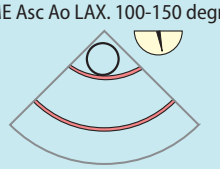
Guided ASE/SCA 20 View TEE Examination (1/4)

20	A. Upper Esophageal (20-25 cm)	How to Obtain View	Comments
	1. Aortic arch LAX. 0 degree 	Obtained at the highest level. Can also be imaged by withdrawing probe after imaging the descending thoracic aorta evaluation.	Look for evidence of atherosclerosis, calcification, mobile plaques, and dissection flaps. PWD for flow reversal in AI.
	2. Aortic arch SAX. 90 degrees 	As above. Slightly retroflex the probe and move side to side to evaluate arch vessels.	As above. Examine PV, MPA. Spectral Doppler of PV, parallel intercept angle. Look for L brachiocephalic vein adjacent to L subclavian artery.
30	B. Mid esophageal (30-40 cm)	How to obtain view	Comments
	3. AV SAX. 30-50 degrees 	Advance the probe from the above view to obtain the classic "Mercedes-Benz" sign at 30-50 degrees.	Highest and most anterior valve. RCC is anterior, NCC is adjacent to IAS, the remainder is LCC. Look for R & L coronary arteries. CFD for AI, any calcification, vegetation, prolapse, restriction of leaflet motion or dissection.
	4. AV LAX. 120-150 degrees 	Move the probe slightly to open up the Ao and LVOT, with valve cusps opening parallel to Ao wall.	Look for valve thickening, opening motion, prolapse, vegetation, dissection. CFD for AI. Measure annulus, sinus, STJ, and ascending Ao.
	5. RV inflow-outflow view. 60-80 degrees 	From the aortic valve view rotate forward to 60-80 degrees.	Evaluate TV, PV with and without CFD. Spectral (CW) Doppler of TR jet for RVSP. Look for PA catheter, pacing wires.
	6. 4 chamber view. 0-20 degrees 	Advance the probe slightly from the aortic image. Retroflex and angle 0-20 degrees to minimize LVOT and to open the LV apex to prevent foreshortening.	Evaluate all the chambers for size, wall motion, thrombi (in LAA and LV apex), and thickening. Evaluate TV, MV with and without CFD, mitral inflow spectral Doppler. Evaluate septal motion and thickening.

AI, aortic insufficiency; **Ao**, aorta; **CFD**, color flow Doppler; **CW**, color wave; **IAS**, interatrial septum; **LAA**, left atrial appendage; **LCC**, left coronary cusp; **LVOT**, left ventricular outflow tract; **MPA**, main pulmonary artery; **NCC**, noncoronary cusp; **PA**, pulmonary artery; **PV**, pulmonary vein; **PWD**, pulse wave Doppler; **RCC**, right coronary cusp; **RVSP**, right ventricular systolic pressure; **STJ**, sinotubular junction; **TV**, tricuspid valve.

ASE/SCA 20 VIEW EXAM CARD 2/4

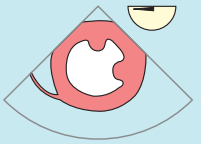
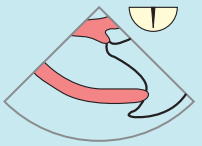
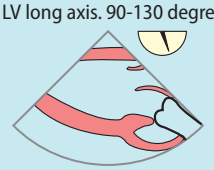
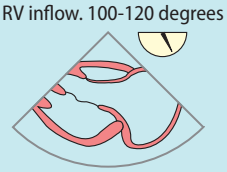
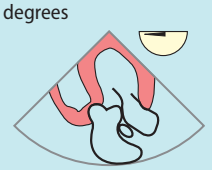
Guided ASE/SCA 20 View TEE Examination (2/4)

30	B. Mid Esophageal (30-40 cm)	How to Obtain View	Comments
	7. Mitral commissural view. 60-80 degrees 	Rotate the angle forward to 60-80 degrees to get "trap door" view.	Measure the commissural distance for annular dilation. Look for restriction, calcification, redundancy of leaflet motion, CFD. See MV card.
	8. 2-chamber view. 80-100 degrees 	Increase the angle further to obtain the 2 chamber view.	Evaluate MV, CFD, look for regional wall motion, thickening in anterior and inferior wall, also examine the LAA for evidence of thrombus. PWD: A LAA velocity >0.5 m/s indicates good atrial contractility and less likelihood of thrombus or low flow state. PWD LUPV.
	9. LV long axis view. 120-160 degrees 	Increase the angle further from above image.	Examine the LV anteroseptal, posterior wall thickening, and wall motion. Look for SAM.
	10. Bicaval view. 80-110 degrees 	From 4-chamber view, rotate the probe to the right in order to center the RA. Increase the multiplane angle to 80-110 degrees.	Examine RA, RAA, IAS, SVC, IVC. CFD for ASD, PFO. Perform bubble study, look for lines, aid in femoral cannulation.
	11. ME Asc Ao SAX. 0-60 degrees 	From AV SAX pull the probe back to evaluate great vessels.	Can be used to image Asc Ao, MPA, RPA, LPA, and SVC. Look for PAC, saddle embolus in MPA and bifurcation, Ao dissection, plaque.
	12. ME Asc Ao LAX. 100-150 degrees 	As above, increase the angle 100-150 degrees.	Can be used to view the asc Ao in long axis and can image RPA in SAX.

Ao, aorta; **Asc**, ascending; **ASD**, atrial septal defect; **CFD**, color flow Doppler; **IAS**, interatrial septum; **IVC**, inferior vena cava; **LAA**, left atrial appendage; **LUPV**, left upper pulmonary vein; **MV**, mitral valve; **MPA**, main pulmonary artery; **PA**, pulmonary artery; **PAC**, pulmonary artery catheter; **PFO**, patent foramen ovale; **PV**, pulmonary vein; **PWD**, pulse wave Doppler; **RA**, right atrium; **RAA**, right atrial appendage; **RPA**, right pulmonary artery; **RVSP**, right ventricular systolic pressure; **SAM**, systolic anterior motion; **SAX**, short axis; **SVC**, superior vena cava; **TV**, tricuspid valve.

ASE/SCA 20 View Exam Card 3/4

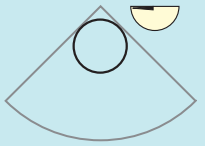
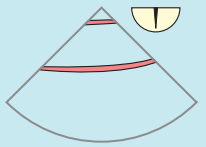
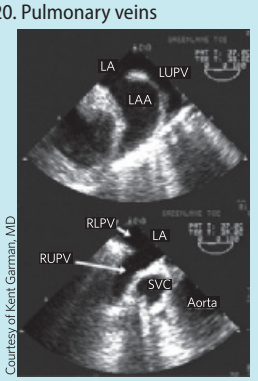
Guided ASE/SCA 20 View TEE Examination (3/4)

40	C. Trans Gastric Views (40-45 cm)	How to Obtain View	Comments
	13. Basal MV SAX. 0-20 degrees 	Advance the probe gently to the stomach and anteflex the tip. Pull the probe back until the MV is seen.	Evaluate the MV, trace the MV area. CFD to localize the leaflet pathology.
	14. Mid papillary SAX. 0-20 degrees 	Advance the probe to image at the level of the papillary muscle.	Assess global ventricular function, regional WMA all RCA, LAD, LCX distributions. Evaluate preload. Obtain cine loop for comparison. M Mode for LV dimension.
	15. Trans gastric 2 chamber. 80-100 degrees 	Increase the angle to 90 degrees from the above image.	Evaluate the mitral apparatus, that is, papillary muscle, chordae, leaflet, anterior and inferior basal ventricular wall motion. Look for restriction or redundancy of chordae.
	16. LV long axis. 90-130 degrees 	Further advance the angle from the above image.	Provides reasonable image for spectral Doppler interrogation of AV and LVOT.
	17. RV inflow. 100-120 degrees 	Rotate the probe to right form mid papillary view to center the RV and increase the angle.	Examine right atrium, right ventricle, tricuspid valve, and papillary muscle. Further rotation of the angle can sometimes reveal RVOT and PV.
	18. Deep trans gastric view. 0-20 degrees 	Advance the probe deep into stomach and maximally anteflex and slowly withdraw the probe. This view is hard to obtain and probe needs manipulation in terms of advance, pull, rotate, etc.	This view aligns the probe almost parallel to the heart to facilitate spectral Doppler interrogation of AV and LVOT.

AV, aortic valve; **CFD**, color flow Doppler; **LCA**, left coronary artery; **Lcx**, left circumflex; **LVOT**, left ventricular outflow tract; **MV**, mitral valve; **PV**, pulmonary vein; **RCA**, right coronary artery; **RV**, right ventricle; **RVOT**, right ventricular outflow tract; **WMA**, wall motion abnormality.

ASE/SCA 20 View Exam Card 4/4

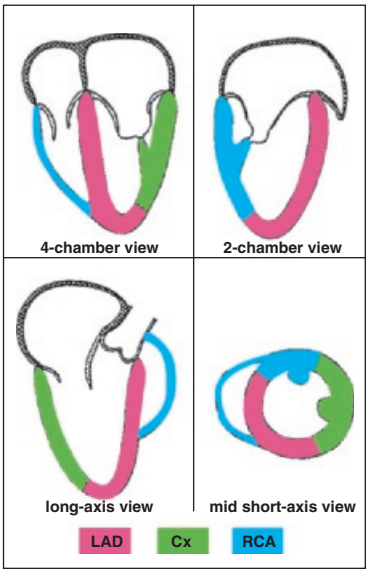
Guided ASE/SCA 20 View TEE Examination (4/4)

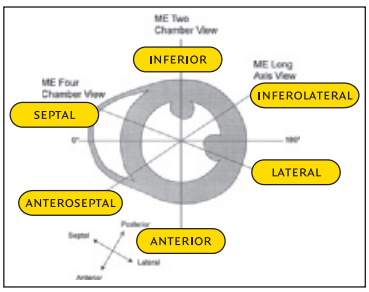
40	C. Trans Gastric Views (40-45 cm)	How to Obtain View	Comments
	19. Evaluation of Desc Thoracic 0 degree  A	For complete examination of the aorta, rotate the probe to the left until the descending thoracic aorta is visualized. Reduce the image depth and increase the transducer frequency. Withdraw the probe slowly to image the entire length up to the aortic arch. Rotate the probe as required to keep the image in the center.	Image in 0 and 90 degrees. Look for dissection, false and true lumen plaques, aneurysm or thrombus. PWD for flow reversal in AI.
	90-130 degrees  B		
	20. Pulmonary veins  Courtesy of Kent Garman, MD	Begin with 4-chamber view. LUPV—Adj to LAA at 0 and 90 degrees. LLPV—turn probe further left and advance 1-2 cm. RUPV—turn probe to right from LAA at 90 degrees. RLPV—advance 1-2 cm from above.	Confirm the pulmonary vein with CFD. Do PWD to evaluate diastolic dysfunction and assess the severity of MR. Localize source air during de-airing maneuver.
	21. Coronary sinus 0-20 degrees 	Obtain 4-chamber view. Rotate the probe to center TV. Advance 1-2 cm and retroflex.	Assist with placement and confirmation of retrograde cardioplegia catheter. Can also be imaged in SAX 2-chamber view, transgastric basal view and in cross section in bicaval view.

Ao, aorta; **AV**, aortic valve; **CFD**, color flow Doppler; **CWD**, continuous wave Doppler; **IAS**, inter atrial septum; **LAA**, left atrial appendage; **LAX**, long axis view; **LCC**, left coronary cusp; **LVOT**, left ventricular outflow tract; **NCC**, noncoronary cusp; **PV**, pulmonary vein, right, left, upper, lower; **PWD**, pulse wave Doppler; **RCC**, right coronary cusp; **RVOT**, right ventricular outflow tract; **SAX**, short axis view; **WMA**, wall motion abnormality.

208 Evaluation of Myocardial Perfusion

Larry F. Chu • Nathaen Weitzel





Once you have mastered obtaining the different TEE views, you can use your skills to assist the surgeon in identifying areas of myocardial ischemia.

This can be done by recording loops during the prebypass period and comparing contractility and wall motion abnormalities to the postbypass heart.

By looking for segmental wall motion abnormalities, you can help localize areas of ischemia to specific vessels.

The mid short axis view (see Chapter 207) may be best to visualize LV function. However, changes that occur in the apex will not be visualized.

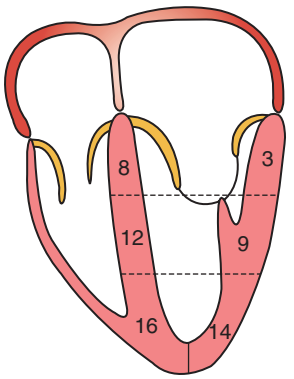
You should also familiarize yourself with the orientation of the heart in the mid short axis view. The mnemonic "SALI" describes the main four segments-septal, anterior, lateral, and inferior. The remaining two segments are "sandwiched in between—anteroseptal and inferolateral." You should know what these are and how to identify them on your TEE examination.

Qualitative grading of wall motion is

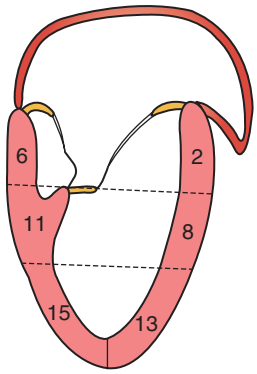
1 = Normal (>30% thickening)
2 = Mildly hypokinetic (10%-30%)
3 = Severely hypokinetic (<10%)
4 = Akinetic (does not thicken)
5 = Dyskinetic (paradoxical movement)

DID YOU KNOW? TEE experts divide the left ventricle into 16 segments by examining 5 cross-sectional views (see "Left Ventricle 16 Segment Model").

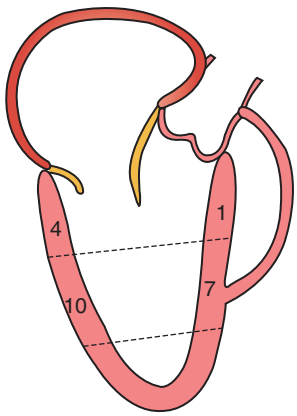
Left Ventricle 16 Segment Model



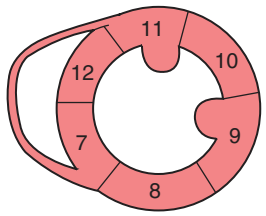
A 4 Chamber view



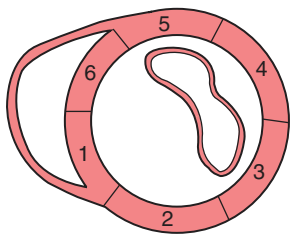
B 2 Chamber view



C Long axis view



D Mid short axis view



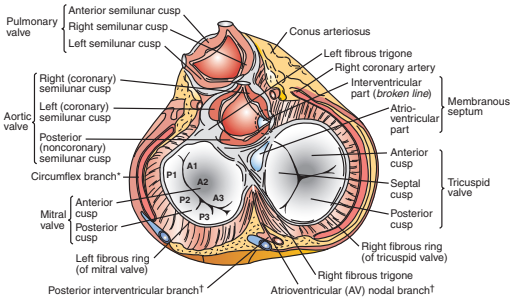
E Basal short axis view

Basal Segments	Mid Segments	Apical Segments
1 = Basal anteroseptal	7 = Mid anteroseptal	13 = Apical anterior
2 = Basal anterior	8 = Mid anterior	14 = Apical lateral
3 = Basal lateral	9 = Mid lateral	15 = Apical inferior
4 = Basal posterior	10 = Mid posterior	16 = Apical septal
5 = Basal inferior	11 = Mid inferior	
6 = Basal septal	12= Mid septal	

Data from *Journal of the American Society of Echocardiography* 1999;12(10):884-900.

209 Evaluation of the Mitral Valve

Benjamin A. Abrams • Nathaen Weitzel • Larry F. Chu



The anatomy displayed above and on following page will help to orient you to the valve structures seen in each 2-D TEE view. Begin by inserting the probe to 30-35 cm for the ME 4-chamber view and identify the anterior and posterior mitral leaflets. The anterior leaflet will be on the left and the posterior leaflet on the right. Move through the 5 ME views to fully evaluate leaflet anatomy and function. As the multiplane angle is increased beyond 90 degrees, note that the posterior leaflet will be to the left of your screen, and the anterior to the right. The ME LAX view (120 degrees) is a key view looking at the coaptation of A2/P2 for most patients.

Once oriented, assess the overall structure and function of the valve. This includes looking for and describing prolapsing or flail segments, stenotic or restricted leaflet motion (CW Doppler through the valve), and degree of annular dilation. Color Doppler is paramount in evaluation of MR and aids in locating the site of the lesions. Surgeons want to know the degree of MR, whether it is central or eccentric, which segment is affected (ie, P2 flail), mitral valve dimensions (commissural view, ME LAX view), and degree of annular calcification.

Echocardiographic Methods to Evaluate Mitral Regurgitation

Method	Mild	Moderate	Severe
Vena contracta (cm)	<0.3	0.3-0.7	>0.7
Color doppler jet area	Small, central, narrow; usual brief	Variable	Large central jet area (>50% of LA) or eccentric wall-hugging jet
Pulmonary vein flow characteristics	S wave > D wave	S wave < D wave	Systolic flow reversal
Estimation of the mitral regurgitant fraction	20%-30%	Subgroups: 30-39%; 40-49%	>50%
Estimation mitral effective regurgitant orifice by the PISA method	<20 mm^2	Subgroups: 30-39%; 40-49%	>40 mm^2
Secondary 2-D changes: left atrial enlargement	Mild	Moderate	Severe
Peak E-wave velocity			>1.2 m/s

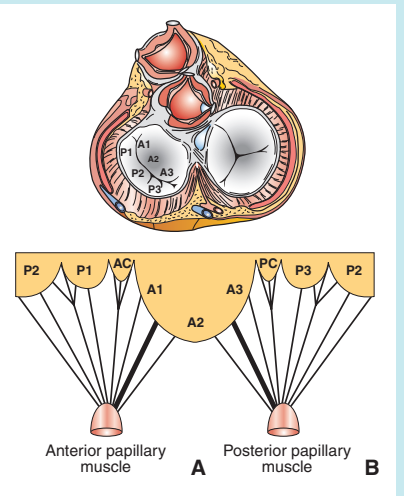
Assessment of mitral stenosis severity

	Normal Values	Mild	Moderate	Severe
Valve area (cm^2)	4-6	1.5-2.0	1.0-1.5	<1.0
Mean gradient (mm Hg)	0-4	5	5-10	>10
Pressure half-time (ms)	70-100	100-150	150-220	>220

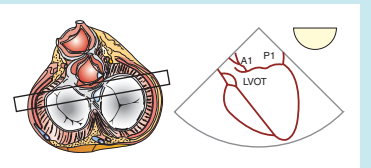
Calculation of mitral valve area

$$\text{MVA}\,(\text{cm}_2) = \frac{220}{\text{Pressure half-time}} = \frac{760}{\text{Deceleration time}}$$

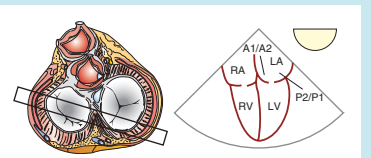
LVOT (area) × LVOT (TVI) = MV (area) × MV (TVI) {continuity equation}
PISA (area) × PISA (velocity) = MV (area) × MV (velocity) {PISA method}



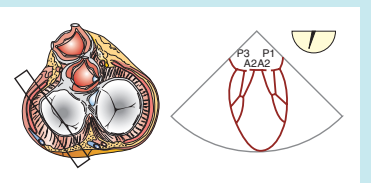
Midesophageal 3 chamber view



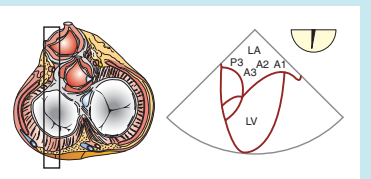
Midesophageal 4 chamber view



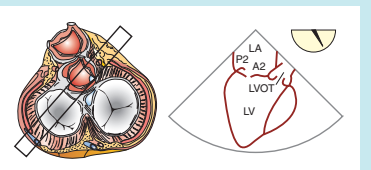
Midesophageal commissural view



Midesophageal 2 chamber view



Midesophageal long axis view (longitudinal)



Mitral Valve ASE/ SCA Terminology (per Carpentier)

Chordal relationships: Anterior leaflet divided into A1, A2, A3.

Posterior leaflet divided into P1 (anterolateral scallop), P2 (middle scallop), P3 (posteromedial scallop).

Commissural clefts not named anterior or posterior.

Chordae arising from the anterior papillary muscle attach to A1, AC, P1, and lateral half of P2, A2.

Chordae arising from the posterior papillary muscle attach to A3, PC, P3, and medial half of P2, A2.

Carpentier Terminology

Column A illustrates the cross section the midesophageal views make through the mitral valve leaflets. Anatomical diagram is drawn viewing mitral and tricuspid leaflets from their atrial side. Column B shows schematic of TEE view seen. Section transversed by TEE plane may vary from patient to patient, however general relationship remains.

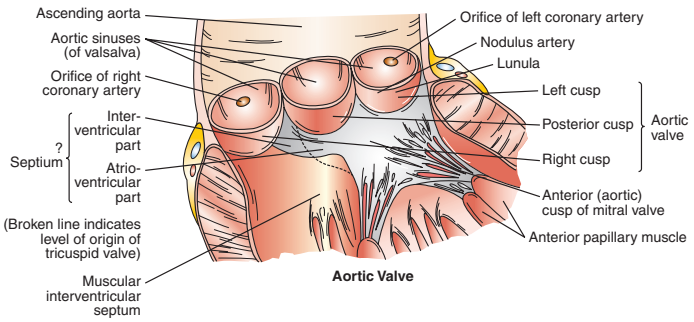
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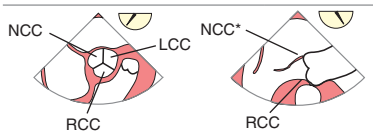
210 Evaluation of the Aortic Valve

Benjamin A. Abrams • Nathaen Weitzel • Larry F. Chu



Aortic Valve

Echocardiographic Views of the Aortic Valve



Left: Mid-esophageal short-axis view of aortic valve leaflets. **Right:** Mid-esophageal long-axis view of the aortic valve leaflets. NCC, noncoronary cusp; LCC, left coronary cusp; RCC, right coronary cusp. *Can be either NCC or LCC

Echocardiographic Methods to Evaluate Aortic Valve

Method	Mild	Moderate	Severe
QUALITATIVE METHODS TO ASSESS AORTIC REGURGITATION			
Angiographic data	1+	2+	3-4+
Color Doppler jet width	Central jet width <25% LVOT	Greater than mild no signs severe AR	Central jet width >65% LVOT
Doppler vena contracta width (cm)	<0.3	0.3-0.6	>0.6
QUANTITATIVE METHOD (CATH OR ECHO) TO ASSESS AORTIC REGURGITATION			
Regurgitant volume (mL/beat)	<30	Subgroups: 30-44; 45-59	≥60
Regurgitant fraction (%)	<30	Subgroups: 30-39; 40-49	≥50
Regurgitant orifice area (cm^2)	<0.1	Subgroups: 0.10-0.19; 0.20-0.29	≥0.3
Pressure half-time (ms)	>500	200-500	<200
ADDITIONAL CRITERIA TO ASSESS AORTIC REGURGITATION			
Left ventricular size			Increased
Aortic diastolic flow reversal			Holodiastolic flow reversal
ASSESSMENT OF AORTIC STENOSIS			
Peak jet velocity (m/s)	<2.6-2.9	3.0-4.0	>4.0
Mean gradient (mm Hg)	<20	20-40	>40
Valve area (cm^2)	>1.5	1.0-1.5	<1.0
Valve area index (cm^2/m^2)	>0.85	0.6-0.85	<0.6

The AV lends itself to high-quality TEE imaging due to its anatomic location. The valve is oriented such that slight anterior flexion of the probe is typically required to obtain ideal imaging. Evaluation should be carried out utilizing the ME AV SAX and LAX views, and the deep transgastric LAX or transgastric LAX view. Look for the degree of calcification along with any flail/prolapsing segments of the leaflets. Additionally, look for possibility of congenital lesions such as a bicuspid valve. Color Doppler, along with CW Doppler evaluation are key in evaluation of regurgitant flow along with elements of stenosis. Heavy calcification should be reported to the surgeons, along with degree of mitral annular calcification as this can make an AVR more difficult.

Assessment of the AV annular size, along with dimensions of the aortic root are helpful in aortic valve surgery. Degree of atheromatous disease in the ascending aorta should be assessed as well. Finally, look for evidence of dissection in all cases, and if found assess the proximity to the coronary arteries and aortic valve leaflets.

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Baumgartner H, Hung J, Bermejo J, et al. Recommendations on the echocardiographic assessment of aortic valve stenosis: a focused update from the European Association of Cardiovascular Imaging and the American Society of Echocardiography. *Eur Heart J Cardiovasc Imaging*. 2017;18(3):254-275. doi: 10.1093/ehjci/jew335.

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211 Local Anesthetics§

Lynn K. Gerber • Dan Hoang

	Max Adult Dosing WITHOUT Epi	Max Adult Dosing WITH Epi	pKa	Onset	Duration	Miscellaneous Facts
ESTERS—Metabolized by Pseudocholinesterase						
Chloroprocaine (Nesacaine)	7 mg/kg	11 mg/kg	9.1	**Infiltration/epidural**: 6-12 min	**Infiltration/epidural**: 30-60 min (prolonged with epi)	Administer over 30 min. Observe for symptoms of toxicity (ie, hypotension, slurred speech, drowsiness), and adjust rate accordingly.
Procaine (Novocaine)	10 mg/kg	15 mg/kg	8.9	**Infiltration/spinal**: 2-5 min **Epidural**: 5-25 min	**Infiltration**: 30-60 min (w/epi 30-90 min) **Epidural/spinal**: 30-90 min (prolonged with epi)	Metabolized to PABA. Caution if sunblock allergy.
Tetracaine (Pontocaine)	3 mg/kg	3 mg/kg	8.5	**Infiltration**: 15 min **Spinal**: <10 min	**Infiltration**: 2-3 h **Spinal**: 1.25-3 h	Prolongs the effect of succinylcholine; metabolite (PABA) inhibits the action of sulfonamides and aminosalicylic acid.
AMIDES—Metabolized through Hepatic Metabolism						
Bupivacaine (Marcaine)	2.5 mg/kg	2.5-3 mg/kg	8.1	**Infiltration**: 2-10 min **Epidural**: 4-17 min **Spinal**: <1 min	**Infiltration/epidural/spinal**: 3-6 h **PNB**: 4-12 h	Most cardiotoxic; levobupivacaine (L-isomer) less cardiotoxic.
Lidocaine (Xylocaine)	4-5 mg/kg	7 mg/kg	7.9	**IV**: (antiarrhythmic effects) 45-90 s **Tracheal**: 1-2 min **Infiltration**: 0.5-1.0 min **Epidural**: 5-15 min	**IV**: (antiarrhythmic effects) 10-20 min **Tracheal**: 30-50 min **Infiltration**: 0.5-1.0 h (w/epi 2-6 h) **Epidural**: 1-2 h	Intrathecal administration associated with transient neurologic symptoms (TNS) and cauda equina syndrome.

(Continued)

	Max Adult Dosing WITHOUT Epi	Max Adult Dosing WITH Epi	pKa	Onset	Duration	Miscellaneous Facts
Mepivacaine (Carbocaine, Polocaine)	4 mg/kg	7 mg/kg	7.6	**Infiltration**: 3-5 min **Epidural**: 5-15 min	**Infiltration**: 45-90 min (w/epi 2-6 h) **Epidural**: 1-3 h, prolonged with epi **PNB**: 2-6 h	Metabolized slower in newborn and fetus.
Prilocaine (Citanest)	8 mg/kg	8 mg/kg	7.9	**Infiltration**: 1-2 min **Epidural**: 5-15 min **Peak Effect**: Infiltration/epidural <30 min **IV**: 15-20 min	**Infiltration**: 0.5-1.5 h (w/epi 2-6 h) **Epidural**: 1-3 h	Metabolite can cause methemoglobinemia at high doses >500 mg (including EMLA-benzocaine/prilocaine). No more than 500 mg prilocaine should be administered within a 2-h period in healthy adults.
Ropivacaine (Naropin)	3 mg/kg (max 200 mg)	3 mg/kg	8.1	**Infiltration**: 1-15 min **Epidural**: 10-20 min **Spinal**: <2 min	**Infiltration/epidural**: 2-6 h **PNB**: 5-8 h	Preferentially blocks sensory fibers over motor fibers.

Shortcut for determining if local anesthetic is an amide or ester: If it has an "i" in the letters before the "-caine" it is an amide. If not, it is an ester.
Treatment of toxicity: 20% lipid emulsion for significant toxicity in unstable patients or persistent toxicity. Seizures may be terminated with benzodiazepines. Establish airway and consider intubation. If cardiac arrest, follow ACLS guidelines and note, lidocaine should not be used as an antiarrhythmic.

READ MORE
Local anesthetic toxicity cognitive aid, Chapter 149

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212 Neuromuscular Blocking Agents[§]

Lynn K. Gerber • Dan Hoang

	Adult Dosing	Onset	Duration	Elimination Half-life	Primary Elimination
Atracurium[a,b] (nondepolarizing)	*Intubating*: **IV**: 0.3-0.5 mg/kg *Maintenance*: **IV**: 0.1-0.2 mg/kg *Infusion*: 2-15 µg/kg/min	<3 min	20-35 min	Normal: 21 min ESRD: 18-25 min ESLD: 20-25 min	90% nonenzymatic degradation in plasma via Hoffman elimination and hydrolysis 10% renal
Cisatracurium[a,b] (nondepolarizing)	*Intubating*: **IV**: 0.15-0.2 mg/kg *Maintenance*: **IV**: 0.02-0.1 mg/kg *Infusion*: 1-2 µg/kg/min	1.5-2 min	20-35 min	Normal: 22-30 min ESRD: 25-34 min ESLD: 21 min	100% nonenzymatic degradation in plasma via Hoffman elimination and hydrolysis
Mivacurium[a,b] (nondepolarizing)	*Intubating*: **IV**: 0.15-0.25 mg/kg over 15-30 s *Maintenance*: **IV**: 0.01-0.1 mg/kg *Infusion*: 1-15 µg/kg/min	<2 min	6-16 min	Normal: 1-3 min	100% hydrolysis via plasma cholinesterase
Pancuronium[a,b,c] (nondepolarizing)	*Intubating*: **IV**: 0.04-0.1 mg/kg *Maintenance*: **IV**: 0.01-0.05 mg/kg *Infusion*: 1-15 µg/kg/min	1-3 min	40-65 min	Normal: 1.5-2 h ESRD: 4-18 h ESLD: 3-4 h	80% renal 10% hepatic 10% biliary
Rocuronium[a,b] (nondepolarizing)	*Intubating*: **IV**: 0.6 mg/kg (RSI 1.2 mg/kg) *Maintenance*: **IV**: 0.06-0.6 mg/kg *Infusion*: 5-15 µg/kg/min	45-90 s	15-150 min	Normal: 90 min ESRD: 1.5-3.5 h ESLD: 2-6 h	10%-25% renal 10%-20% hepatic 50%-70% biliary
Vecuronium[a,b] (nondepolarizing)	*Intubating*: **IV**: 0.1 mg/kg *Maintenance*: **IV**: 0.01-0.015 mg/kg *Infusion*: 1-2 µg/kg/min	<3 min	25-30 min	Normal: 1-2 h ESRD: 1.5-3 h ESLD: 1-3 h	15%-25% renal 20%-30% hepatic 40%-75% biliary

(Continued)

	Adult Dosing	Onset	Duration	Elimination Half-life	Primary Elimination
Succinylcholine[d] (depolarizing)	*For skeletal muscle relaxation*: **IV**: 0.7-1 mg/kg **Deep IM**: 2.5-4 mg/kg, maximum 150 mg *To relieve laryngospasm*: **IV**: 0.1-0.5 mg/kg, start at the low end, for example, 10-20 mg for 70 kg adult	**IV**: 30-60 s **IM**: 2-3 min	**IV**: 4-6 min **IM**: 10-30 min	N/A	Metabolized by plasma cholinesterase *Maximum total dose (to avoid prolonged phase II block)*: <5 mg/kg

Adverse effects:
[a]Nondepolarizing agents: Because of their steroid-like structure, they have been implicated in causing critical illness myopathy. In addition, nondepolarizing drugs may be an underappreciated source of allergic reactions during anesthesia.
[b]Interactions of nondepolarizing agents: A large number of drugs interact with the nondepolarizers, specifically causing prolonged effect. Volatile inhalational anesthetics, aminoglycoside antibiotics, local anesthetics, antiarrhythmics, diuretics, magnesium, lithium, and cyclosporine may all prolong NMB. In addition, myasthenia gravis, hypothermia, female gender, and hyperkalemia can potentiate the effects of these drugs. Hypokalemia, burns, and spinal cord injuries can all cause resistance to these agents.
[c]Atropine-like drug; may see tachycardia
[d]Succinylcholine: Administration of succinylcholine will lead to a 0.5-1 mEq/dL rise in serum potassium concentrations and should not be used in patients who have preexisting elevated serum potassium. This rise can be exaggerated in patients who have altered numbers of ACh receptors. Though not an exhaustive list, this includes patients with spinal cord injury, burns, and genetic myopathies. It is also a trigger for malignant hyperthermia. Activation of muscarinic ACh receptors may cause bradycardia, an important consideration in the pediatric population that has rate-dependent cardiac output.
IV, intravenous; IM, intramuscular; ESRD, end-stage renal disease; ESLD, end-stage liver disease.

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213 Anticholinesterases and Sugammadex§

Lynn K. Gerber • Dan Hoang

	Adult Dosing	Duration	Chemical Structure	Site of Action
Edrophonium	*NMB reversal*: **IV**: 0.5-1.0 mg/kg. Max dose 40 mg. Co-administer with atropine or glycopyrrolate[b] *Diagnose myasthenia gravis*: 2-3 mg every 60-90 s	**IV**: 5-20 min **IM**: 10-40 min	Quaternary amine	Neuromuscular junction
Neostigmine	*NMB reversal*: **IV**: 0.02-0.05 mg/kg, max dose 5 mg. Co-administer with atropine or glycopyrrolate[b]	**IV**: 40-60 min **IM/PO**: 2-4 h	Quaternary amine	
Physostigmine	*Anticholinergic reversal*: **IV/IM**: 0.5-2.0 mg (10-30 µg/kg) at a rate of ≤1 mg/min	**IV/IM**: 30 min to 5 h	Tertiary amine[a]	
Pyridostigmine	*NMB Reversal*: **IV**: 0.25 mg/kg (max dose 30 mg). Co-administer with atropine or glycopyrrolate[b] *Myasthenic crisis*: About 1/30th of patient's PO dose	**IV**: 90 min **PO**: 3-6 h **IM**: 2-4 h	Quaternary amine	
Sugammadex	*NMB reversal*: **IV**: 2.0 mg/kg for moderate blockade (2 or more twitches on TOF stimulation), 4.0 mg/kg for deep blockade (1-2 post-tetanic counts), 16.0 mg/kg for immediate reversal after 1.2 mg/kg of rocuronium	**IV**: 100-150 min	Gamma-cyclodextrin	Encapsulates rocuronium in a 1:1 ratio; binds vecuronium and pancuronium with less affinity

[a]Tertiary amines penetrate the blood-brain barrier.

[b]Typical dose of atropine IV is 0.015 mg/kg and glycopyrrolate IV is 0.01 mg/kg.

SUGGESTED READINGS

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214 Benzodiazepines[§]

Lynn K. Gerber • Dan Hoang

	Adult Dosing	Peak Effect	Duration	Elimination Half-life[a]	Pain on Injection	Clinically Active Metabolites
Diazepam	*Sedation/ premedication*: **IV**: 0.05-0.2 mg/kg **PO**: 2-10 mg *Induction*: **IV**: 0.3-0.5 mg/kg *Muscle spasm*: **IV**: 5-10 mg	**IV**: 3-4 min **PO**: 60 min	**IV**: 15 min to 1 h **PO**: 2-6 h	30 h	++	Many, contributing to long elimination half-life
Lorazepam	*Sedation/ premedication*: **IV**: 1-4 mg (0.02-0.08 mg/kg) *Sedation*: **PO**: 2-3 mg, for elderly 1-2 mg **IM**: 1-4 mg (0.05 mg/kg) undiluted injectate (max 4 mg)	**IV**: 15-20 min **PO**: 120 min	**IV/IM/PO**: 6-10 h	15 h	++	None
Midazolam	*Sedation/ premedication*: **IV**: 0.5-5.0 mg (0.025-0.1 mg/kg). Typically 2.0 mg IV for normal healthy adult **PO**: 20-40 mg **IM**: 2.5-10.0 mg (0.05-0.2 mg/kg) *Induction*: **IV**: 50-350 µg/kg	**IV**: 3-5 min **IM**: 15-30 min **PO**: 30 min **Intranasal**: 10 min **PR**: 20-30 min	**IV/IM**: 15-80 min **PO/PR**: 2-6 h	2 h	0	Few

	Adult Dosing	Peak Effect	Duration	Elimination Half-life[a]	Pain on Injection	Clinically Active Metabolites
Flumazenil (antagonist)	**IV**: for adults 4-20 µg/kg For reversal of sedation from benzodiazepines: 0.2 mg IV over 15 s, may be repeated at 1-min intervals (max total dose: 1 mg)	5-10 min	45-90 min	1 h Note risk of resedation due to short half-life	+	None

Synergistic respiratory depression occurs with concomitant narcotic usage, consider decreasing dose in this situation.

[a]Elimination: All the above benzodiazepines are highly protein bound, rely on the liver for initial biotransformation, and are renally cleared. Peak effect, duration, and half-life can vary based on patient characteristics. Consider decreasing dose/frequency in liver or kidney disease. Lorazepam may be preferable for use in renal failure because of inactive metabolites.

0, none; +, moderate; ++, severe.

SUGGESTED READINGS

Hemmings HC, Egan TD. *Pharmacology and Physiology for Anesthesia*. 2nd ed. Philadelphia, PA: Elsevier; 2019.
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215 Opioids§

Lynn K. Gerber • Dan Hoang

	Potency (Relative to Morphine IV)	Adult Dosing	Peak Effect	Duration	Elimination Half-Life	Primary Elimination	Interactions/ Side Effects/ Toxicities
Alfentanil (IV)	15-30	*Analgesia with spont vent*: **IV**: 3-10 µg/kg *Induction*: **IV**: 100-300 µg/kg *Analgesia ventilated*: **IV bolus**: 10-100 µg/kg **Infusion**: 0.05-1.25 µg/kg/min *For retrobulbar/peribulbar block*: **IV**: 50-100 µg/kg	**IV**: 1-2 min **IM**: <15 min **Epidural**: 30 min	**IV**: 10-15 min **IM**: 10-60 min	1.4-1.5 h	Hepatic, CYP3A4	Chest wall rigidity, bradycardia
Fentanyl (IV)	50-100	*Induction*: **IV bolus**: 1-2 (up to 20) µg/kg *Analgesia*: **IV/IM**: 0.5-5 µg/kg **PCA**: 25-100 µg every 5-30 min **Infusion**: 0.5-5 µg/kg/h *Epidural*: **Bolus**: 50-100 µg **Infusion**: 10-50 µg/h	**IV**: 5-15 min **IM**: <15 min **Epidural/ spinal**: <30 min	**IV**: 30-60 min **IM**: 1-2 h **Epidural/ spinal**: 1-2 h	**IV**: 2-4 h	Hepatic, pulmonary	Chest wall rigidity, bradycardia, pruritus, nausea, constipation
Hydromorphone (IV)	5-7	*Analgesia*: **IV**: 0.5-2 mg every 2-3 h **IM/SC/PO**: 2-4 mg every 4-6 h **PCA**: 0.2-1 mg every 10-15 min **Spinal**: 0.1-0.2 mg *Epidural*: **Bolus**: 0.4-2 mg (lower dose for thoracic epidurals) **Infusion**: 0.15-0.3 mg/h	**IV**: 5-20 min **IM/SC/PO**: 30-60 min **Epidural**: 30 min	**IV**: 2-4 h **IM/SC/PO**: 4-6 h **Rectal**: 6-8 h **Epidural**: 10-16 h	For immediate release formulations: 1-3 h	Hepatic	Nausea, constipation, myoclonus

(Continued)

	Potency (Relative to Morphine IV)	Adult Dosing	Peak Effect	Duration	Elimination Half-Life	Primary Elimination	Interactions/ Side Effects/ Toxicities
Meperidine (IV)	0.1	*Analgesic*: **PO/IM/SC**: 50-150 mg **IV**: 25-100 mg *Epidural*: **Bolus**: 50-100 mg **Infusion**: 10-20 mg/h *Spinal*: **Bolus**: 10-50 mg **Infusion**: 5-10 mg/h *Shivering/rigors*: **IV**: 12.5-25 mg	**PO**: <1 h **IV**: 5-20 min **IM**: 30-50 min **Epidural/ spinal**: 30 min	**PO/IV/IM**: 2-4 h **Epidural/ spinal**: 0.5-3 h	2.5-4 h	Hepatic	Normeperidine accumulation may result in seizures (especially in renally insufficient patients); can act as a local anesthetic; can act as anticholinergic, possible severe reaction with MAO inhibitors; nausea
Morphine (IV)	1	*Analgesic*: **IV**: 2.5-15 mg **Infusion**: 0.5-10 mg/h **IM/SC**: 2.5-20 mg **PO**: 10-30 mg every 4 h **PO, extended release**: 30-60 mg every 8-12 h **Rectal**: 10-20 mg every 4 h *Epidural*: **Bolus**: 2-5 mg **Infusion**: 0.1-1 mg/h *Spinal*: 0.1-1 mg	**IV**: 5-20 min **IM**: 30-60 min **SC**: 50-90 min **PO**: 30-60 min **PO (slow release)**: 1-4 h **Rectal**: 20-60 min **Epidural/ spinal**: 90 min	**IV/IM/SC/PO**: 4-6 h **PO (slow release)**: 6-12 h **Epidural/ spinal**: 6-24 h	2-4 h	Hepatic, renal	Histamine release; morphine-6-glucuronide is an active metabolite and can accumulate in renally insufficient patients; morphine-3-glucuronide can lower seizure threshold and can accumulate in renally insufficient patients; pruritus, nausea, constipation, myoclonus

Remifentanil (IV)	50-100	*Anesthesia induction*: **IV bolus**: 1 µg/kg over 30-60 s *Anesthesia maintenance*: **Infusion**: 0.03-0.25 µg/kg/min **Supplemental IV bolus**: 0.5-1 µg/kg *Analgesia/MAC*: **IV bolus**: 0.6-2.0 µg/kg over 30-60 s, then infusion 0.03-0.1 µg/kg/min	**IV**: 1-2 min	**IV**: 5-10 min	3-10 min	Plasma and tissue esterases	Chest wall rigidity, bradycardia; no metabolic interaction with other esterase-hydrolyzed drugs; remifentanil clearance is reduced by ~20% during hypothermia
Sufentanil (IV)	250-500	*Induction*: **IV bolus**: 0.5-1 µg/kg *Analgesia* **IV/IM**: 0.1-0.5 µg/kg **Infusion**: 0.1-0.5 µg/kg/h *Epidural*: **Bolus**: 10-50 µg **Infusion**: 5-30 µg/h *Spinal*: 1-10 µg	**IV**: 3-5 min **Epidural/ spinal**: <30 min	**IV**: 20-45 min **IM**: 2-4 h **Epidural/ spinal**: 2-4 h	2.2-4.6 h	Hepatic and small intestine	Chest wall rigidity, bradycardia, constipation
Codeine (PO)	0.25-0.5	**PO**: 15-120 mg every 4-6 h	1-1.5 h	4-6 h	2.5-3.5 h	Hepatic, CYP2D6	Pruritus, nausea, constipation
Hydrocodone (PO)	1.5	**PO**: 2.5-10 mg every 4-6 h	1.5 h	4-6 h	3-6 h	Hepatic	Pruritus, nausea, constipation
Methadone (PO)	2-5	**IV/PO**: 2.5-10 mg every 8-12 h	**IV**: 5-20 min **IM**: 30-60 min	**IV/IM**: 4-6 h **PO**: 22-48 h	8-59 h (increased with repeated doses)	Hepatic	Pruritus, nausea, prolonged QT interval, possible severe reaction with MAO inhibitors; slow half-life may lead to overdose when first starting long-term treatment

(Continued)

	Potency (Relative to Morphine IV)	Adult Dosing	Peak Effect	Duration	Elimination Half-Life	Primary Elimination	Interactions/ Side Effects/ Toxicities
Oxycodone (PO)	1	**PO**: 5-30 mg every 4 h	0.5-1 h	3-6 h	2-5 h	Hepatic	Pruritus, nausea, constipation
Propoxyphene (PO)	0.1-0.2	**PO**: 65-100 mg every 4 h	2-2.5 h	4-6 h	6-12 h	Hepatic	Pruritus, nausea, constipation
Buprenorphine (mixed agonist-antagonist)		*Acute pain*: **IV**: 0.3-0.6 mg every 6 h *Chronic pain*: **PO**: 75-900 µg every 12 h	**IV**: 10-15 min **PO**: 2.5-3 h	**IV**: 2-3 h **PO**: 20-30 h	**IV**: 2-3 h **PO**: 20-30 h	Hepatic	Pruritus, nausea, constipation
Nalbuphine (mixed agonist-antagonist)		*Pain*: **IV**: 10 mg every 3-6 h *Pruritus*: **IV**: 2.5-5 mg	2-3 min	3-6 h	6 h	Hepatic	Nausea, constipation

Note: Most opioids will cause respiratory depression. IV, intravenous; IM, intramuscular; PCA, patient-controlled analgesia; PO, per oral; SC, subcutaneous.

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216 Opioid Antagonists[§]

Lynn K. Gerber • Dan Hoang

	Route/Adult Dosing	Onset	Duration	Elimination Half-life	Site of Clearance
Naloxone[a,b]	*Postop opioid reversal*: **IV**: 0.1-0.2 mg[a] *Intoxication/respiratory arrest*: **IV**: 0.4-2 mg **IM/SC**: 0.4-2 mg every 2-3 min **Nasal**: 4 mg (1 spray) every 2-3 min	**IV**: 1-2 min **IM/SC/Nasal**: 2-5 min	30-120 min, depending on route	0.5-1.5 h	Hepatic
Naltrexone[a]	*Prophylaxis/treatment of opioid addiction or ethanol abuse*: **PO**: 12.5-50.0 mg daily *Opiate cessation*: **PO**: 12.5-25.0 mg daily initially, then 50 mg daily for maintenance	**PO**: 15-30 min (peak ~60 min)	24-72 h (dose dependent)	4 h	Hepatic
Methylnaltrexone	*Opioid-induced constipation*: **PO**: 450 mg **SC**: 12 mg	**PO**: 1.5 h **SC**: 30 min	8-15 h	8 h	Renal
Alvimopan	*Postop opioid-induced constipation*: **PO**: 12 mg	2 h	10-17 h	10-17 h	GI

[a]Used for complete or partial reversal of opioid drug effects, including respiratory depression and management of known or suspected opioid overdose.
[b]Dose every 2-3 minutes until desired response (adequate ventilation/alertness). Repeat dosing may be needed depending on type, dose, and timing of the last opioid administered. Titrate to assure adequate ventilation and minimize withdrawal symptoms. Duration of action may be shorter than opioid being reversed—monitor for recurrence.

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217 NSAIDs§

Lynn K. Gerber • Dan Hoang

	Mechanism of Action	Adult Dosing	Peak Effect	Duration	Elimination Half-life	Primary Metabolism
Aspirin	Irreversibly acetylates the COX-1 and COX-2 enzymes, which causes decreased synthesis of prostaglandin precursors	*For analgesic purposes*: 500-1000 mg every 4-6 h, max 4 g/d *For antiplatelet purposes*: 81-325 mg daily Monitor serum concentration and/or clinical signs of toxicity (tinnitus, nausea, presyncope, etc.)	2 h	4-6 h	Parent drug: 15-20 min Salicylates: 3-10 h (dose dependent)	Hepatic
Celecoxib	Inhibits synthesis of prostaglandins by decreasing mainly COX-2 (and not COX-1) enzyme activity	**PO**: 100-200 mg once or twice daily	3 h	12-24 h	~11 h (fasted)	Hepatic
Ibuprofen	Reversibly inhibits COX-1 and COX-2, with mechanisms including inhibition of chemotaxis, altering lymphocyte activity, inhibiting neutrophil aggregation, or decreasing proinflammatory cytokine levels	*For analgesic purposes*: **IV/PO**: 400-800 mg every 6 h	1-2 h	4-6 h	2-4 h	Hepatic

(Continued)

	Mechanism of Action	Adult Dosing	Peak Effect	Duration	Elimination Half-life	Primary Metabolism
Indomethacin	Reversibly inhibits COX-1 and COX-2	**PO**: 25-50 mg, 2-3 daily *Maximum total:* daily 200 mg	2 h	4-6 h	4.5 h	Hepatic
Ketorolac[a]	Reversibly inhibits COX-1 and COX-2; may be considered more of a peripherally acting analgesic; useful as a non-narcotic adjunct since ~30 mg of parenteral toradol is equivalent to 10-mg parenteral morphine	*Single dose*: **IV**: 15-30 mg; **IM**: 60 mg *Multiple dose*: **IV/IM**: 15-30 mg every 6 h. Not to exceed 120 mg/d **PO**: 10-20 mg initially and then 10 mg every 4-6 h. Not to exceed 40 mg/d. Duration of treatment should not exceed 5 d	**IV/IM/PO**: 1-3 h	**IV/IM/PO**: 3.5-8 h	2-6 h	Hepatic
Naproxen	Reversibly inhibits COX-1 and COX-2	*For analgesic purposes*: **PO**: 500 mg initially, followed by 250 mg every 6-8 h *Maximum total*: 1250 mg/d	1-4 h	*Analgesic*: 7 h *Anti-inflammatory*: 12 h	12-17 h	Hepatic

General NSAID side effects: May cause fluid retention, kidney or liver failure, antiplatelet aggregation leading to increased risk of bleeding; may cause gastric mucosal damage with ulceration and bleeding. COX-2 specific NSAIDS are believed to cause less damage to gastric mucosa and less antiplatelet effects versus COX-1 agents. However, studies have shown an increased risk for thrombosis, myocardial infarction, and stroke.
[a]Avoid in patients with bleeding or platelet problems.
IV, intravenous; IM, intramuscular; PO, per oral.

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218 IV Induction Agents[§]

Lynn K. Gerber • Dan Hoang

	Mechanism of Action	Adult Dosing	Elimination Half-life	Elimination	Interactions/Toxicities
Propofol (2,6-diisopropylphenol)	Allosteric binding to $GABA_A$, increasing duration of Cl^- channel opening	*Induction*: 1.0-2.5 mg/kg *Maintenance*: 100-200 µg/kg/min *Sedation*: **IV bolus**: 25-50 mg **Infusion**: 25-75 µg/kg/min	Biphasic: Initial: 40 min Terminal: 4-7 h	*Metabolism*: Hepatic, extrahepatic (pulmonary) *Excretion*: Renal	Circulatory depression, pancreatitis, propofol infusion syndrome (characterized by dysrhythmia, heart failure, hyperkalemia, lipemia, metabolic acidosis, and/or rhabdomyolysis or myoglobinuria with subsequent renal failure). Potentiates CNS and respiratory depressant effects of narcotics, sedative hypnotics, and volatile anesthetics; pulmonary extraction decreased and plasma levels increased with concomitant administration of alfentanil, fentanyl, or halothane
Etomidate (*R*-1-ethyl-1-[a-methylbenzyl] imidazole-5-carboxylate)	Carboxylated imidazole Modulates or activates $GABA_A$ receptors by potentiating GABA-induced chloride currents	*Induction*: **IV**: 0.2-0.3 mg/kg **Infusion**: 0.25-1 mg/min	2.6 h (terminal)	*Metabolism*: Hepatic *Excretion*: Renal	Cardiovascular and CNS depressant effects potentiated by other sedatives, narcotics, and volatile anesthetics; venous pain and myoclonus on rapid injection. Inhibits the activity of 11-β-hydroxylase causing adrenal suppression which persists for 6-8 h, up to 24 h in critically ill patients
Ketamine	Phencyclidine derivative that acts as a noncompetitive antagonist at the glutamatergic *N*-methyl-D-aspartate (NMDA) receptor	*Induction*: **IV**: 1.0-2.5 mg/kg **IM/PR**: 5-10 mg/kg *Sedation/analgesia*: **IV**: 0.5-1.0 mg/kg **IM/PR**: 2.5-5 mg/kg **PO**: 5-6 mg/kg *Infusion*: 15-80 µg/kg/min *Epidural/caudal*: 0.5 mg/kg	Alpha: 10-15 min Beta: 2.5 h	*Metabolism*: Hepatic *Excretion*: Primarily renal	May cause emergence delirium. Decreased requirement for volatile anesthetics. HTN, arrhythmias, or MI with concomitant use of sympathomimetics. Enhancement of depolarizing and nondepolarizing NMB

Methohexital (1-methyl-5-allyl-5-[1-methyl-2-pentanyl] barbituric acid)	Allosteric enhancement of duration of $GABA_A$ receptor opening. At higher doses, may also be GABA-mimetic. Also depresses the transmission of excitatory neurotransmitters in the CNS, including acetylcholine and glutamate	*Induction*: **IV**: 1-3 mg/kg **IM**: 7-10 mg/kg *Sedation*: **IV**: 0.25-1 mg/kg *Infusion*: 50-150 µg/kg/min (0.2% solution) **PR**: 20-30 mg/kg	4 h	*Metabolism*: Hepatic *Excretion*: Renal	Circulatory depression, thrombophlebitis (rare), seizure (typically in patients w/hx seizures). Potentiates CNS and respiratory depressant effects of narcotics, sedative hypnotics, alcohol, and volatile anesthetics. Can precipitate if given with NMB
Thiopental (5-ethyl-5-[1-methylbutyl]-2-thiobarbituric acid)	Allosteric enhancement of duration of $GABA_A$ receptor opening. At higher doses, may also be GABA-mimetic. Also depresses transmission of excitatory neurotransmitters in the CNS, including acetylcholine and glutamate	*Induction*: **IV**: 3-5 mg/kg *Anesthesia supplementation*: **IV**: 0.5-1 mg/kg	3-11.5 h	*Metabolism*: Hepatic *Excretion*: Primarily renal	Myocardial dysfunction; hemolytic anemia (rare), radial nerve palsy (rare), renal failure (rare). Potentiates CNS and respiratory depressant effects of narcotics, sedative hypnotics, alcohol, and volatile anesthetics; arterial or extravascular injection can produce necrosis and gangrene; can form precipitates when injected with muscle relaxants. Interferes with porphyrin metabolism: administration of the drug may result in elevated concentrations of porphyrins in serum and/or urine, which may exacerbate acute intermittent porphyria

	Protein Binding	Volume of Distribution	Time to Onset	Peak Effect	Duration of Action
Etomidate	76%	2-4.5 L/kg	15-45 s	1 min	3-12 min
Ketamine	12%	2-3 L/kg	**IV**: 45-60 s	1 min	5-20 min
Methohexital	85%	1.9-2.2 L/kg	**IV**: <30 s **PR**: 5-15 min	**IV**: 45 s **PR**: 5-10 min	**IV**: 5-10 min **PR**: 30-90 min
Propofol	97%-99%	2-10 L/kg	15-45 s	1 min	5-10 min
Thiopental	72%-86%	1.6-2.5 L/kg	**IV**: <30 s	**IV**: 30-40 s	**IV**: 5-15 min

IV, intravenous; IM, intramuscular; PO, per oral; PR, per rectum.

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219 Inhaled Anesthetics§

Lynn K. Gerber • Dan Hoang

	Desflurane	Isoflurane	Nitrous Oxide	Sevoflurane
1 MAC	6%	1.2%	105%	2%
Vapor pressure (mm Hg at 20°C)	681	240	Gas at room temperature	160
Blood/gas partition coefficient	0.42	1.4	0.47	0.65
CNS effects	↑ CBF ↓↓ $CMRO_2$ ↑ ICP	↑ CBF ↓↓ $CMRO_2$ ↑ ICP	↑ CBF ↑ $CMRO_2$ ↑ ICP	↑ CBF ↓↓ $CMRO_2$ ↑ ICP
Cardiovascular effects	↓ BP ↑ or N/C HR ↓ SVR ↓ or N/C CO Rapid ↑ associated with transient elevations in HR, BP, and catechols	↓ BP ↑ HR ↓ SVR ↓ or N/C CO Rapid ↑ associated with transient elevations in HR, BP, and catechols	N/C BP N/C HR N/C SVR N/C CO Increases PVR due to constriction of pulmonary smooth muscle	↓ BP N/C HR ↓ SVR ↓ or N/C CO May prolong QT interval
Pulmonary effects	↓ TV ↑ RR Depresses ventilatory response to ↑ $PaCO_2$	↓ TV ↑ RR Depresses ventilatory response to ↑ $PaCO_2$ Modest bronchodilation	↓ TV ↑ RR Depresses hypoxic ventilatory drive	↓ TV ↑ RR Depresses ventilatory response to ↑$PaCO_2$ Mild bronchodilation
Hepatic effects	↓ Hepatic blood flow	↓ Hepatic blood flow	↓ Hepatic blood flow	N/C Hepatic blood flow
Renal effects	↓ Renal blood flow ↓ GFR ↓ UOP	↓↓ Renal blood flow ↓↓ GFR ↓↓ UOP	↓↓ Renal blood flow ↓↓ GFR ↓↓ UOP	↓ Renal blood flow ↓ GFR ↓ UOP
Metabolism	<0.1% absorbed anesthetic metabolized with pulmonary, hepatic, renal elimination	0.2% absorbed anesthetic metabolized with pulmonary, hepatic, renal elimination	0.004% absorbed anesthetic metabolized with pulmonary, renal, GI elimination	5% absorbed anesthetic metabolized with pulmonary, hepatic, renal elimination

(Continued)

	Desflurane	Isoflurane	Nitrous Oxide	Sevoflurane
Considerations against use	MH trigger; pungency can cause airway irritation, breath-holding, coughing, and laryngospasm More potent greenhouse gas/ contributor to carbon footprint over isoflurane and sevoflurane	MH trigger	Diffuses into air-containing cavities. Beware in conditions where air embolism, pneumothorax, acute intestinal obstruction, intracranial air, intraocular air bubbles, tympanic membrane perforation may exist. Avoid use in patients with preexisting pulmonary HTN or right-sided CHF due to increased PVR	MH trigger
Additional pearls	Ultra-short duration with quick wake-up times Desflurane emergence associated with delirium in some pediatric patients Special vaporizer needed given high vapor pressure	Theoretical possibility of coronary steal via dilation of normal coronary arteries	Increased incidence of PONV Addition of N_2O decreases anesthetic requirements of other agents. Mild analgesic properties	Accumulation of compound A seen in low-flow anesthesia, dry barium hydroxide (Baralyme) absorbent but no known harm demonstrated in humans. Nonpungent features mixed with rapid increase in alveolar concentration make ideal for inhalational induction in pediatric and adult patients

↑, increase; ↓, decrease; ↓↓, large decrease; BP, blood pressure; CBF, cerebral blood flow; CHF, congestive heart failure; $CMRO_2$, cerebral metabolic rate of oxygen; CO, cardiac output; GFR, glomerular filtration rate; GI, gastrointestinal; HR, heart rate; HTN, hypertension; ICP, intracranial pressure; N/C, no change; $PaCO_2$, arterial CO_2 partial pressure; RR, respiratory rate; SVR, systemic vascular resistance; TV, tidal volume; UOP, urine output; PVR, pulmonary vascular resistance; PONV, postoperative nausea and vomiting; MH, malignant hyperthermia

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220 Adrenergic Agonists[§]

Lynn K. Gerber • Dan Hoang

	Mechanism of Action	Adult Dosing	Onset	Peak Effect	Duration	Elimination Half-Life	Primary Metabolism
Dobutamine	Direct β_1 agonist with mild α_1- and β_2-agonist effects	**Infusion**: 2-20 µg/kg/min	1-2 min	1-10 min	<10 min	2 min	Hepatic
Dopamine	Both direct and indirect agonist; stimulates α_1 and β_1 receptors	**Infusion**: 1-20 µg/kg/min	2-4 min	2-10 min	<10 min	2 min	Hepatic
Ephedrine	Indirect agonist with some direct agonist effects; stimulates α_1- and β-adrenergic receptors	*For hypotension*: **IV**: 5-20 mg (100-200 µg/kg) **IM/SC**: 25-50 mg	**IV**: 10-20 seconds **IM**: 10-20 min	**IV**: 2-5 min **IM**: <10 min	**IV/IM**: 10-60 min	2.5-3.6 h	Hepatic, renal
Epinephrine	Directly activates α- and β-adrenergic receptors; β effects predominate at therapeutic parenteral doses, and α-adrenergic effects predominate at higher doses	*Cardiac arrest*: **IV bolus**: 1 mg or 0.02 mg/kg every 3-5 min, followed by a 20-mL flush of IV fluid *Inotropic support*: **Infusion**: 0.1-1 µg/kg/min *Anaphylaxis*: **IM/SC**: 0.1-5 mg *Test dose in regional anesthesia*: 10-15 µg	**IV**: 30-60 s **SC**: 6-15 min **Intratracheal**: 5-15 s **Inhalation**: 3-5 min	**IV**: within 3 min	**IV**: 5-10 min **Intratracheal**: 15-25 min **Inhalation/SC**: 1-3 h		Enzymatic degradation (hepatic, renal, and GI tract)

Isoproterenol	Directly activates β_1 and β_2 receptors	*Arrhythmias/resuscitation*: **IM/SC**: 0.2 mg **IV push**: 0.02-0.06 mg **Infusion**: 0.02-0.15 µg/kg/min **Sublingual**: 10 mg then 5-50 mg as needed *Bronchospasm*: **Metered dose inhaler**: 120-262 µg every 3-4 h	**IV**: immediate **Inhalation**: 2-5 min **Sublingual/SC**: 15-30 min	**IV**: 1 min	**IV**: 1-5 min **Inhalation**: 0.5-2 h **Sublingual/SC**: 1-2 h	2.5-5 min	Hepatic
Norepinephrine	Directly activates α- and β_1-adrenergic receptors	**Infusion**: 0.02-0.4 µg/kg/min	<1 min	1-2 min	2-10 min		Enzymatic degradation, pulmonary
Phenylephrine	Directly activates α-adrenergic receptors with minimal β activation	*Hypotension*: **IM/SC**: 2-5 mg **IV**: 50-100 µg **Infusion**: 0.15-4.0 µg/kg/min *Shunt reversal*: **IV**: 1-2 µg/kg	**IV**: <1 min **IM/SC**: 10-15 min	**IV**: <1 min	**IV**: 15-20 min **IM/SC**: 0.5-2 h		Hepatic

IV, intravenous; IM, intramuscular; SC, subcutaneous; PO, per oral.

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APPENDIX OF ANESTHESIA DRUG INFORMATION

221 Adrenergic Antagonists[§]

Lynn K. Gerber • Dan Hoang

	Mechanism of Action	Adult Dosing	Onset	Peak Effect	Duration	Elimination Half-Life	Primary Elimination
Atenolol	β_1 selective antagonist	*Antihypertensive/antianginal*: **PO**: 50-200 mg once daily *Acute MI*: **IV**: 5 mg over 5 min, followed by another 5 mg intravenous injection 10 min later *Alcohol withdrawal/migraine prophylaxis*: **PO**: 50-100 mg once daily	**IV**: <5 min **PO**: <1 h	**IV**: 5 min **PO**: 2-4 h	**IV**: up to 12 h **PO**: >24 h	6-7 h	Little to no biotransformation; majority is excreted from the kidneys as unchanged drug
Bisoprolol	β_1 selective antagonist	**PO**: 2.5-20 mg once daily	1-2 h	2-4 h	>24 h	9-12 h	Hepatic (significant first-pass effect)
Carvedilol	α_1, β_1, and β_2 antagonist	**PO**: 6.25-25 mg twice daily	1-2 h	1-2 h	12-24 h	7-10 h	Hepatic (significant first-pass effect)
Esmolol	β_1 selective antagonist	*Antihypertensive*: **IV**: 0.5-2 mg/kg, repeated every 5 min if necessary **Infusion**: 50-300 µg/kg/min *Supraventricular tachyarrhythmias*: **IV**: 500 µg/kg, given over 1 min **Infusion**: 50-200 µg/kg/min	1-2 min	5 min	10-20 min	10 min	Esterases in the cytosol of red blood cells
Labetalol	Predominant β_1 and β_2 antagonist, some a1 (alpha-1) antagonist activity	**IV bolus**: 0.25 mg/kg slowly over 2 min **Infusion**: 0.5-2 mg/min, maximum 1-4 mg/kg **PO**: 100-400 mg twice daily *Maximum total dose*: **IV**: 300 mg	**IV**: 2-5 min **PO**: 20 min to 2 h	**IV**: 5-15 min **PO**: 1-4 h	**IV**: 2-4 h **PO**: 8-24 h	**IV**: 5.5 h **PO**: 6-8 h	Hepatic; urine and feces

(*Continued*)

Metoprolol	β_1 selective antagonist but can inhibit β_2-receptors in high doses	*Antihypertensive*: **IV**: 1-5 mg every 2 to 5 min up to 15 mg **PO**: 50-400 mg daily *Acute MI*: **IV**: 15 mg over 6 min in three divided doses **PO**: 50 mg every 6 h, beginning 15 min after the last IV dose for 48 h **PO**: 100 mg twice daily *Migraine prophylaxis*: **PO**: 50-100 mg twice daily	**IV**: <1 min **PO**: <15 min	**IV**: 20 min **PO**: 1.5-4 h	**IV**: 5-8 h **PO**: 10-20 h	3-8 h	Hepatic (significant first-pass effect)
Phenoxybenzamine	Irreversible, nonselective alpha-receptor antagonist	**PO**: 10 mg—dose is increased to titrate to effect	2 h	4-6 h	14-48 h	24 h	Hepatic
Propranolol	Nonselective β blockade	*Antihypertensive*: **PO**: 20-40 mg twice daily, usually ≤320 mg daily **IV**: 10-30 μg/kg every 2 min (max 6-10 mg) **IV bolus**: Usual dose is 1-3 mg administered slowly (1 mg/min) under careful monitoring. If necessary, a second dose may be given after 2 min. Thereafter, additional drug should not be given for at least 4 h *Maximum total dose*: ≤5-10 mg or 0.1 mg/kg	**IV**: <2 min **PO**: <30 min	**IV**: within 1 min **PO**: variable (immediate release: 1-4 h; extended-release formulations: ~6-14 h)	**IV**: 1-6 h **PO**: 6-12 h	3-6 h (for immediate release formulation)	Hepatic, pulmonary

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222 Corticosteroids§

Lynn K. Gerber • Dan Hoang

Drug Name (Approximate mg of Equivalent Dose)	Mechanism of Action	Adult Dosing	Peak Effect	Duration	Elimination Half-Life	Primary Elimination	Glucocorticoid, Mineralocorticoid Potency (Relative to Cortisol)
Hydrocortisone (cortisol) (20)	Anti-inflammatory and mineralocorticoid activity; binds specific intracellular cytoplasmic receptors in target tissues (a mechanism of all steroidal agents). The receptor-hormone complex then acts as a transcription factor in the nucleus, turning certain genes on or off	*For inflammatory diseases*: **IV/IM**: 20-300 mg every 2-10 h *Steroid replacement*: **IV**: 50-100 mg preoperatively, intraoperatively, and postoperatively	*For anti-inflammatory effects*: **IV/IM**: <1 h	*For anti-inflammatory effects/HPA suppression*: 30-36 h	Biological: 8-12 h	Hepatic	1, 2
Dexamethasone (0.75)	Similar to hydrocortisone but with negligible mineralocorticoid activity; synthetic long-acting glucocorticoid with potent anti-inflammatory effects	*For inflammatory diseases*: **IA/IV/IM/IT**: 4-16 mg/day *Cerebral edema or raised ICP*: **IV**: 10-50 mg, then **IV/IM/PO**: 4 mg q6h *Airway swelling*: **IV**: 10-25 mg q6h *For bronchospasm*: (**inhalation**): 300 µg (3 inhalations) 3-4 times daily	*For anti-inflammatory effects*: **IV/IM**: 12-24 h	36-54 h	1.8-3.5 h	Hepatic	25-30, 0

(Continued)

Drug Name (Approximate mg of Equivalent Dose)	Mechanism of Action	Adult Dosing	Peak Effect	Duration	Elimination Half-Life	Primary Elimination	Glucocorticoid, Mineralocorticoid Potency (Relative to Cortisol)
Fludrocortisone (–)	Similar to hydrocortisone but has prolonged effect on electrolyte balance and carbohydrate metabolism; produces significant sodium retention, increased potassium excretion from renal distal tubules, and rise in blood pressure	**PO**: 0.05-0.2 mg/d	1.7 h	24 h	Plasma: 30-35 min Biological: 18-36 h	Hepatic	10, 125
Prednisone (5)	Similar to hydrocortisone; intermediate acting	**PO**: 5-60 mg/d	1-2 h	12-36 h	~3.5 h	Hepatic	4, 1

General corticosteroid side effects: Osteoporosis, impaired wound healing and increased risk of infection, increased appetite, hypertension, edema, peptic ulcers, euphoria, and psychoses. Systemic corticosteroids used for <7 d, even at high doses, are not likely to cause adverse side effects, nor are inhaled corticosteroids.
IA, intra-articular; ICP, intracranial pressure; IV, intravenous; IM, intramuscular; IT, intratissue; PO, per oral.

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223 Antibiotics§

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	Surgery Type and Likely Pathogens	Adult Dosing	Administration Dosing	Excretion	Class	Comments
Ampicillin	Colorectal, appendectomy *Proteus mirabilis*, *Escherichia coli*, and *Streptococcus*	**IV**: 2 g	**Redose**: 2 h	Hepatic metabolism; renal excretion	Penicillinase-susceptible with activity against gram-negative bacilli	Associated with skin rash, allergic reactions
Cefazolin	At-risk surgical procedures (cardiac, noncardiac thoracic, colorectal, esophageal and gastroduodenal, biliary tract, general surgery, pancreatic, vascular, genitourinary, neurosurgery, head and neck, vaginal or abdominal hysterectomy)	**IV**: 2 g If >120 kg, 3 g	**Intraop**: Redose every 3-4 h **Postop**: every 8 h	Renal excretion	1st-generation cephalosporins	Allergic reactions Toxicity: prolonged thrombin time, neutropenia
Cefoxitin **Cefotetan** **Cefuroxime**	Colorectal, appendectomy Colorectal Cardiac, noncardiac thoracic, pancreatic, vascular	**IV**: 1-2 g **IV**: 1-2 g **IV**: 1.5 g	**Redose**: 6-8 h **Redose**: 12 h **Redose**: 8 h, max 6-9 g/d	Renal excretion	2nd-generation cephalosporins	Allergic reactions Toxicity: prolonged thrombin time, neutropenia
Ceftriaxone	Good CNS penetration for neurosurgical procedures. To treat meningitis or epiglottitis (*Haemophilus influenzae*)	**IV**: 1-2 g	**Redose**: 24 h	Renal excretion	3rd-generation cephalosporins	Allergic reactions Toxicity: prolonged thrombin time, neutropenia
Ciprofloxacin	Colorectal, esophageal and gastroduodenal, biliary tract, appendectomy, genitourinary To treat upper and lower respiratory tract infections, bone and joint infections, and most strains of *Mycobacterium tuberculosis*	**IV**: 400 mg **PO**: 500 mg	**Redose**: 6-8 h	Hepatic metabolism (active metabolites); renal excretion	Fluoroquinolones	Allergic reactions Toxicity: prolongation of QTc interval, tendinopathy, photosensitivity, teratogenicity, GI irritation

Clindamycin	Cardiac, noncardiac thoracic, colorectal, esophageal and gastroduodenal surgery, biliary tract, appendectomy, neurosurgery, head and neck	**IV**: 900 mg	**Infusion time**: 60 min **Redose**: 6 h	Hepatic metabolism (active metabolites); renal excretion	Lincomycin	Allergic reactions Pseudomembranous colitis can be a complication; produces prejunctional and postjunctional effects at the neuromuscular junction; maternal toxicity; peripheral neuropathy
Gentamicin	Colorectal, esophageal and gastroduodenal, biliary tract, appendectomy, head and neck. Treats *Pseudomonas aeruginosa*	**IV**: 5 mg/kg	**Infusion time**: 60 min **Redose**: 6-8 h	Renal excretion	Aminoglycoside	Allergic reactions Ototoxicity, nephrotoxicity
Metronidazole	Colorectal, appendectomy	**IV**: 500 mg	**Infusion time**: 60 min **Redose**: 6-8 h	Hepatic metabolism; renal excretion	Antiprotozoal, bactericidal (forms toxic metabolites in the bacterial cell)	Allergic reactions Toxicity: seizures, peripheral neuropathy, teratogenicity
Piperacillin/tazobactam	Broad spectrum for sepsis or skin/soft tissue infections, good for pseudomonas coverage	**IV**: 3.375 g (4.5 g for nosocomial pneumonia)	**Redose**: 2-3 h	Hepatic metabolism; renal excretion	Antipseudomonal penicillin, β-lactamase inhibitor	Does not cover MRSA, VRE, atypicals; extended use >10 d may cause neutropenia
Vancomycin	Cardiac and orthopedic surgery Treats MRSA, *Staphylococcus epidermidis* Good alternative for penicillin-allergic patients	**IV**: 1-2 g	**Infusion time**: 60 min **Redose**: 8 h	Renal excretion	Bactericidal glycopeptide that impairs cell wall synthesis of gram-positive bacteria	Infusion-related flushing due to histamine release Allergic reactions Toxicity: neutropenia, ototoxicity

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	Mechanism of Action	Adult Dosing	Elimination Half-Life	Site of Metabolism	Interactions/Toxicities
Dexamethasone	Glucocorticoid, unknown mechanism for PONV	*Antiemetic before surgical incision*: **IV**: 4-8 mg	1.8-3.5 h Biological half-life: 36-54 h	Hepatic	Primary adrenocortical insufficiency. Clearance enhanced by phenytoin, phenobarbital, rifampin, ephedrine; increases requirements of insulin; increases risk of GI bleeding with concomitant NSAID use
Droperidol	Dopamine receptor antagonist at the chemoreceptor trigger zone	*Antiemetic*: **IV/IM**: 0.625-1.25 mg	2.3 h	Hepatic	Respiratory depression, neuroleptic malignant syndrome. May cause sedation, extrapyramidal signs, anticholinergic effects. May intensify hypotensive effects of vasodilators **Black box warning**: May cause QT prolongation and torsade de pointes. Use with caution in patients with cardiac disease, electrolyte disturbances
Metoclopramide	Blocks dopamine D_2 receptors centrally and GI peripherally; enhances GI motility and accelerates gastric emptying; increases lower esophageal sphincter tone	**IV/IM**: 10-20 mg every 4-6 h as needed **PO**: 10 mg	5-6 h	Renal	Neuroleptic malignant syndrome. May cause extrapyramidal symptoms; antagonized by anticholinergics and opioids; potentiated by sedatives, opioids, tranquilizers, and hypnotics **Black box warning**: May cause tardive dyskinesia, which is often irreversible
Ondansetron	Serotonin 5-HT_3 receptor antagonist, targets vagal nerve terminals and chemoreceptor trigger zone of the area postrema	*Antiemetic before end of surgery*: **IV/IM**: 4 mg **PO**: 4-8 mg	3-6 h	Hepatic	Cardiac dysrhythmia, anaphylaxis, bronchospasm Toxicities potentiated by hepatic disease; use with caution in patients with risk factors for QT prolongation and patients allergic to other 5-HT_3 receptor antagonists
Prochlorperazine	Dopaminergic blockade in the chemoreceptor trigger zone of the medulla	**IV**: 2.5-10 mg every 3-4 h (maximum 40/d) **IM**: 5-10 mg every 3-4 h (maximum 40 mg/d) **PO**: 5-10 mg 3-4 times/d (maximum 40 mg/d) **PR**: 25 mg twice daily	For IV and oral single dose: 6-10 h For oral repeated dosing: 14-22 h	Hepatic	Prolonged QT interval, torsades de pointes, agranulocytosis, cholestatic jaundice syndrome May potentiate or increase levels of alcohol, opioid analgesics, anticholinergics, beta-blockers, and CNS depressants; hypersensitivity may result in jaundice and/or extrapyramidal symptoms **Black box warning**: Increased risk of death seen in elderly patients being treated with antipsychotics for dementia-related psychosis

Promethazine	Blocks dopaminergic receptors in the brain. Histamine blocking agent at H_1 receptors; antimuscarinic activity may be responsible for antiemetic properties	**IV/IM/PO/PR**: 12.5-25 mg every 4-6 h	9-16 h	Hepatic	Injection site reaction, agranulocytosis, jaundice, neuroleptic malignant syndrome. Potentiates CNS, respiratory and circulatory depressant effects of alcohol, volatile anesthetics, and sedative hypnotics. May increase levels/effects of anticholinergics; serotonin modulators **Black box warning**: Contraindicated in children <2 y. Extravasation can cause serious tissue injury
Propofol	Mechanism of action in regard to antiemesis is currently not well understood	*Antiemetic*: **IV**: 10-20 mg	Biphasic: Initial: 40 min Terminal: 4-7 h	Hepatic	May cause sedation, hypotension, respiratory depression, or anaphylaxis; potentiates CNS and circulatory depressant effects of narcotics, sedative hypnotics, and volatile anesthetics
Scopolamine	Competitive antagonist at muscarinic receptor; weak peripheral muscarinic cholinergic effect; its tertiary amine structure allows it to readily cross blood-brain barrier to exert effects on the CNS	*Scopolamine base*: **Transdermal patch**: 1.5 mg, apply to postauricular skin **SC**: 0.6-1 mg	9.5 h	Hepatic	Alteration in heart rate, drug-induced psychosis Central anticholinergic syndrome. Potentiates sedative effects of narcotics, benzodiazepines, anticholinergics, antihistamines, and volatile anesthetics; may cause amnesia, dry mouth, and vertigo; use with caution in patients with narrow-angle glaucoma

IV, intravenous; IM, intramuscular; PO, per oral; PR, per rectum; SC, subcutaneous.

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225 Hematologics§

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Disclaimer: Anticoagulant and antiplatelet medications should not be discontinued without first taking into account the patient's clinical situation. Abrupt discontinuation of these medications may be life threatening or may lead to serious morbidity.

	Effect Site	Adult Dosing	Peak Effect	Half-Life	Stop Before Surgery	Metabolism	Adverse Reactions	Usage
Anticoagulant Drugs								
Argatroban	Thrombin	**IV** (*infusion*): start at 2 µg/kg/min, target PTT 1.5-3 times normal or <100 s	**IV**: steady state 1-3 h	50 min	4-6 h	Hepatic elimination	Hemorrhage	Alternative to heparin in patients that have heparin-induced thrombocytopenia
Aspirin[a]	COX, irreversible inhibition of platelet activation and aggregation	81-325 mg daily **ACS**: 325 mg (chewed)	**PO**: 1-2 h	6 h	7 d	Hepatic elimination	Anaphylaxis, angioedema, GI bleed, hemorrhage, anion gap metabolic acidosis, Reye syndrome	ACS, stroke
Clopidogrel	ADP blocking IIb/IIIa activity and inhibition of platelet aggregation	**PO**: 75-150 mg qd	**PO**: 45 min	7 h	7-10 d	Hepatic	Neutropenia, hepatic dysfunction, hemorrhage	CAD, PVD, ischemic stroke, prevention of thrombosis
Dabigatran	Direct thrombin inhibitor	**PO**: 150 mg bid	**PO**: 1 h	12-17 h	3-5 d	Hepatic	Hemorrhage, allergy, anaphylaxis	Nonvalvular a-fib, VTE treatment, or prophylaxis
Eptifibatide	Blocking IIb/IIIa activity and inhibition of platelet aggregation	**IV**: Load with 180 µg/kg then 2 µg/kg/min up to 96 h for **PCI**, stop within 24 h postprocedure	**IV**: 5 min	2.5 h	4 h	Renal	Hemorrhage, anaphylaxis, hypotension	ACS, PCI

Heparin	Binds to antithrombin III to inhibit factor Xa and thrombin	**IV flush**: 10-100 units *Cardiopulmonary bypass*: **IV**: 350-450 units/kg *Low-dose thrombosis prophylaxis*: **SC**: 5000 units 2 h before surgery then every 8-12 h *Full-dose continuous IV therapy*: **Loading IV**: 5000 units then infusion 20 000-40 000 units over 24 h	**SQ**: 2-4 h	1-3 h	4-6 h	Hepatic elimination	Allergy, hemorrhage, thrombocytopenia, decreased antithrombin concentrations	Prophylaxis and treatment against VTE, treatment of ACS, ischemic stroke
Low molecular weight heparin (LMWH) (enoxaparin)	Binds to antithrombin to inhibit factor Xa only	*VTE prophylaxis* 30 mg SQ q12h; *treatment for VTE* 1-1.5 mg/kg SQ q12h, *MI* 1 mg/kg SQ q12h *Bridge to warfarin*: 1-1.5 mg/kg SQ q12h until INR in desired range	**SQ**: 3 h	3 h	12 h if VTE prophylaxis dose, 24 h if full anticoagulation dose	Hepatic elimination	Allergy, hemorrhage, thrombocytopenia	Monitor by factor Xa level, VTE prophylaxis, VTE prophylaxis and treatment, MI
Ticagrelor	P2Y12 receptor antagonist; blocks ADP	**PO**: Loading dose 180 mg, then 90 mg bid	2 h	7-9 h	3-5 d	Hepatic	Hemorrhage, dyspnea, ventricular pause	ACS, antiplatelet therapy
Warfarin	Vitamin K-dependent clotting factors (II, VII, IX, X, C, S)	**Initial**: 10-15 mg **Daily**: **PO** 2-10 mg	1-5 d	24-60 h	≥5 d	Renal and hepatic elimination	Hemorrhage, skin necrosis	Prophylaxis and treatment of VTE disease including PE, DVT, ischemic stroke, dysrhythmia (a-fib, a-flutter)

(Continued)

Hemostatic drugs							
DDAVP (desmopressin)	Kidney, endothelium	**IV**: 0.3 µg/kg over 30 min	**IV**: 90–120 min	3 h (up to 9 h in renal failure)	Renal	Volume overload, hyponatremia, thrombosis	Hemophilia, vWD,[b] diabetes insipidus, platelet dysfunction
FEIBA	Factor II, VII, IX, X	100–200 units/kg/d IV div q6-12h Max: 200 units/kg/d Info: dose, frequency, duration varies by site/ severity of bleeding, severity of deficiency	10–15 min	Factor dependent	Factor dependent	Anaphylaxis, anaphylactoid, thrombosis, DIC, MI	Pts with factor VIII inhibitors (also hemophilia A or B)
Protamine	Binds to heparin (heparin antagonist)	**Slow IV**: 1 mg neutralizes 100 units of heparin. Dose is determined by dose of heparin given and time since last dose	<5 min	Duration of action (unknown half-life): 2 h; dependent on body temperature	Hepatic elimination	Hypo- and hypertension, bradycardia, pulmonary HTN, dyspnea, bronchospasm, anaphylactoid, anaphylaxis, histamine release, thrombocytopenia	Heparin antagonism

Recombinant factor VIIa	Activates factor IX and X	**IV**: 15-90 µg/kg depending on situation (slow IV bolus)	10-15 min	2-3 h	Hepatic	Anaphylaxis, thrombosis, hypertension, hemarthrosis, fever, MI, supraventricular tachycardia, blood coagulation disorder, disseminated intravascular coagulation, hemorrhage, acute renal failure, VTE	Bleeding, uncontrolled hemorrhage, long CPB duration, hemophilia A or B. FDA-labeled indications: acquired factor VIII deficiency disease-bleeding; bleeding-factor VII deficiency; factor VII/VIII deficiency-postoperative hemorrhage; prophylaxis Bleeding-hemophilia, with inhibitors to factor VIII or factor IX. Hemophilia, with inhibitors to factor VIII or factor IX—postoperative hemorrhage; prophylaxis

[a]Aspirin: Half-life elimination; parent drug: 15-20 min; salicylates (dose dependent): 3 h at lower doses (300-600 mg), 5-h (after 1 g), 10 h with higher doses.
[b]vWD type I patients are usually responsive, vWD type II patients may have variable responses, vWD type III patients are usually not responsive. Consult a hematologist to address your individual patient needs.
ACS, acute coronary syndrome; a-fib, atrial fibrillation; CAD, coronary artery disease; COX, cyclooxygenase; CPB, cardiopulmonary bypass; DVT, deep vein thrombosis; HTN, hypertension; INR, international normalized ratio; MI, myocardial infarction; Pt, patient; Pts, patients; PVD, peripheral vascular disease; PCI, percutaneous coronary intervention; PE, pulmonary embolism; qday, daily; VTE, venous thromboembolism; vWD, von Willebrand disease

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APPENDIX OF ANESTHESIA DRUG INFORMATION

226 Anesthesia for the Patient with Opioid Use Disorder

T. Kyle Harrison • Larry Chu

The rising number of people with opioid use disorder in the United States has led to increased use of medication-assisted treatment (MAT) of opioid use disorder. These patients require special perioperative considerations when presenting for surgery, including perioperative management of MAT medications and multimodal anesthesia and analgesia to optimize intraoperative and postoperative pain management.

1) **Introduction**
   a) Epidemiology—The United States is facing an opioid crisis where 63000 drug overdose deaths in 2016 represented double the mortality than traffic accidents or gun violence.[1]
   b) By conservative estimates, 2.5 million people in this country are addicted to opioids (prescription and illicit), and more than 11 million people in the United States are misusing prescription opioids.[2]
2) **Medication-assisted treatment of opioid use disorder**
   The FDA has approved three classes of medications for the treatment of opioid use disorder/addiction. They include the following:
   a) Opioid agonist treatment
      Multiple placebo-controlled trials across continents and decades demonstrate the effectiveness of opioid agonist treatment (methadone and buprenorphine) in opioid use disorder.[3,4]
      i) Methadone is a long-acting opioid that helps decrease physiologic cravings that drive drug-seeking behaviors (generic oral and injectable forms, Dolophine, or Methadose). Given as a daily oral dose, but also available in IV form.
      ii) Buprenorphine is a long-acting opioid that has a high binding affinity at the mu receptor but only partially activates it compared with other opioids. Prescription for buprenorphine for MAT requires a special prescribing registration (DEA-X), but any provider with DEA license may prescribe for pain. Doses range from 0.075 mg up to 24 mg. Low-dose forms are commonly used to treat non–opioid dependent patients with pain.
      iii) Buprenorphine and naloxone is a combination medication that is thought to decrease the ability for abuse of buprenorphine. The addition of naloxone will precipitate withdrawal if injected intravenously.
   b) Opioid antagonist treatment
      i) Naltrexone—A deterrent to opioid abuse, it blocks opioid receptor binding and may also reset the reward pathway in the brain through an opponent processing mechanism (generic tablets, ReVia, or Vivitrol long-acting injectable form).
3) **Management**
   a) Preoperative
      i) Methadone—Patients should be instructed to continue their usual methadone maintenance therapy including the day of surgery.

(1) These patients will have analgesic tolerance to opioids and require higher than normal doses of opioids to achieve similar analgesic levels compared to nontolerant patients.
(2) The alpha elimination of methadone is associated with analgesia and is 8 hours, so consider dividing the oral methadone dose into three divided doses to improve pain control postoperatively.
(3) If the patient cannot take po and conversion to IV is needed, the dose should be reduced by ½-⅔ and given in divided doses every 6-8 hours. Conversion of large po doses can be difficult, and consultation with a pharmacist or methadone prescriber may be warranted.



Conversion of large po doses of methadone can be difficult. Consult a pharmacist or methadone prescriber for assistance.

ii) Buprenorphine—Optimal use in the perioperative setting has not been studied. Still, there is some evidence to suggest that buprenorphine-maintained patients can achieve adequate perioperative pain control with opioids and multimodal analgesia (**Table 226-1**).[5,6]
(1) Patients should be instructed to continue their usual buprenorphine dose on the day of surgery.
(2) Multimodal analgesia should be used intraoperatively.
iii) Naltrexone—Patients should be instructed to stop oral naltrexone 2-3 days before surgery since it has a half-life of 10 hours. This should be done in close coordination with the prescribing physician.
(1) Less evidence is available for XR-NXT, the injectable form of naltrexone. Complete lack of analgesia to opioids in the first 2 weeks of treatment should be expected, but successful pain management has been reported starting in the 4th week of treatment.

b) Intraoperative
i) Multimodal analgesia is an important component of anesthesia for patients on MAT. Therapies that constitute MAT are summarized in **Table 226-1**.
ii) Opioids are an important component, but care should be taken to consider increased dosing in opioid-tolerant patients.



Selection of high-affinity mu-opioid binding opioids such as sufentanil may be more effective in patients on buprenorphine.

c) Postoperative
Postoperative planning for patients on MAT is critical to achieve effective postoperative pain management and transition back to effective MAT. Plans for perioperative management of MAT including postoperative planning are summarized in **Table 226-2**.
i) Methadone—Multimodal analgesia should be continued in the postoperative period. Documenting the methadone prescriber is important so that proper postoperative follow-up at discharge can be arranged.
(1) Avoid up-titration of methadone perioperatively due to its long half-life.
(2) If a patient's methadone dose has been interrupted for more than 5 days, restarting at discharge should be done in consultation with a provider who is experienced in methadone maintenance induction.
ii) Buprenorphine—Multimodal analgesia should be continued in the postoperative period. Despite partial activation of the opioid receptor, buprenorphine still provides analgesia but has a ceiling effect on respiratory depression, conferring significantly less risk of respiratory compromise and overdose compared with opioids, such as morphine and fentanyl.
(1) Patients who continued buprenorphine perioperatively can be up-dosed postoperatively to target pain control if needed.

TABLE 226-1 Multimodal Analgesia

Mode of Analgesia	Recommended Treatment Guideline	Comments
Opioids	• Still important component; doses needed to be increased in opioid tolerant patients.	• Consider opioids with higher mu-binding like sufentanil for patients on buprenorphine.
Regional or Neuraxial Anesthesia	• Single injection spinal (morphine or hydromorphone) ± local anesthetic • Thoracic epidural • Transversus abdominis plane (TAP) • Continuous peripheral nerve block	• Regional or neuraxial anesthesia is vital to anesthetic management in opioid-tolerant patients.
Ketamine	• 0.5 mg/kg IV prior to incision • 0.25 mg/kg IV/h infusion	• NMDA receptor antagonism helps decrease wound hyperalgesia, decreases postoperative opioid use, and improves postoperative pain control.
Lidocaine	• 0-1.5 mg/kg IV bolus • 1.5-3.0 mg/kg/h IV infusion	• No data for use >24 h • Treat lidocaine toxicity with 20% lipid emulsion.
Magnesium	• 30-50 mg/kg slow IV bolus • 10 mg/kg/h IV infusion	• May contribute to intraoperative hypotension
Acetaminophen	• Oral and IV administration have similar efficacy. • 1000 mg IV or po, 650 mg PR. Not to exceed 4 g/d (2 g/d in adults with liver disease, moderate alcohol intake).	• Combining with NSAIDs confers additional analgesia over either drug alone.
Gabapentinoids	• Gabapentin 300 mg po or • Pregabalin 50 mg po	• When gabapentin is added to methadone or buprenorphine MAT, withdrawal symptoms are reduced.
Nonsteroidal anti-inflammatory drugs	• Ketorolac 15-30 mg IV • Ibuprofen 400 mg po	• Caution should be used in patients with cardiovascular disease, renal insufficiency, and gastrointestinal bleeding.
Steroids	• Dexamethasone 0.11-0.2 mg/kg IV	• Infusing over 10 min in 50 mL can reduce perineal pain.
Dexmedetomidine	• 1 mcg/kg IV loading over 10 min • 0.2-0.7 mcg/kg/h IV maintenance	• Can cause hypotension and bradycardia
Esmolol	• 0.15-0.3 mg/kg/min IV	• Intraoperative use decreases intraoperative and postoperative opioid consumption.
Psychological	• 15 min mindfulness session or hypnosis	• Has been shown to decrease postoperative pain by 30% in 1/3 of patients reporting severe pain

(2) Patients who have stopped buprenorphine postoperatively and are still taking additional opioids potentially could be restarted on buprenorphine by using a daily microdose escalation known as the Bernese method without precipitating withdrawal. Once a sufficient dose of buprenorphine is reached, patients can discontinue their additional postoperative opioids.

TABLE 226-2 Perioperative Considerations for Medication-Assisted Treatment of Opioid Use Disorder

Drug	Preoperative	Day of Surgery	Postoperative
Methadone	• Continue daily dose. • Document methadone provider's contact information.	• Patient should receive usual dose. • If unable to take po, convert to IV. • Plan for multimodal analgesia.	• Continue daily dose but consider tid dosing. • Do not up-titrate methadone for pain control. • Continue multimodal pain management. • Coordinate follow-up with methadone provider early in postoperative period. • Discharge home with the lowest dose and shortest duration of opioid needed.
Buprenorphine	• Continue daily dose. • Document buprenorphine provider's contact information.	• Patient should receive usual dose. • Plan for multimodal analgesia.	• Continue daily dose but consider tid dosing. • Buprenorphine can be up-titrated for pain control. • Continue multimodal pain management. • Coordinate follow-up with buprenorphine provider early in postoperative period. • Discharge home with the lowest dose and shortest duration of opioid needed.
Naltrexone	• Oral—stop 2-3 d before surgery. • XR-NXT—stop 30 d before surgery.	• Confirm last dose >48 h (oral) >30 d (XR-NXT) • Plan for multimodal analgesia.	• Continue multimodal pain management. • Patient may be significantly less sensitive to opioids. • Discharge home with the lowest dose and shortest duration of opioid needed. • Resume after patient has been off opioids for 7 d. • Patients that have been on naltrexone and have stopped preoperatively maybe very sensitive to the respiratory depressant effects of opioids. Patients that have continued naltrexone perioperatively will have a variable response to opioids and may require very high doses of opioids for analgesia.

(a) The patient's buprenorphine prescriber should manage this induction process and should be consulted early in the postoperative recovery process.

iii) Naltrexone—Multimodal analgesia should be continued in the postoperative period. Close monitoring may be required if patients receive opioids postoperatively. This is because variable responses to opioids have been observed and levels 6-20 times typical doses of opioids have been needed to achieve adequate analgesia.

(1) Patients are to be free of opioids before restarting naltrexone to avoid acute withdrawal.
(2) FDA-approved prescribing information indicates a period of 7-10 days of abstinence from opioids prior to induction of naltrexone.
(3) Patients that have been on naltrexone and have stopped preoperatively maybe very sensitive to the respiratory depressant effects of opioids. Patients that have continued naltrexone perioperatively will have a variable response to opioids and may require very high doses of opioids for analgesia.

CHAPTER SUMMARY FOR ANESTHESIA FOR THE OPIOID-ADDICTED PATIENT[7]

Methadone	Long-acting opioid Continue up to day of surgery, usual dose. Consider dividing daily dose into tid dosing for better pain control. If need to convert po to IV dosing, reduce ½-⅔ and give in divided doses every 6-8 h.
Buprenorphine **Buprenorphine and naloxone**	Take last dose the day of surgery. Multimodal anesthesia and analgesia Consider opioids with high mu opioid receptor binding, sufentanil > hydromorphone > oxymorphone > fentanyl.
Naltrexone	Stop oral form 2-3 d before surgery. The injectable form is long acting; consider opioid-free anesthesia if dosed within 2-3 wk. In 4th week, consider opioids postoperatively, but 6-20 times usual dose may be needed and the response is variable.
Intraoperative	
Multimodal anesthesia	Consider multimodal anesthesia using variety of agents including regional/neuraxial anesthesia, dexamethasone, dexmedetomidine, ketamine, lidocaine infusion, esmolol, NSAIDs, or magnesium.
Postoperative	
	Mindfulness relaxation, lidocaine infusion, and nonopioid analgesia can be helpful adjuncts to opioids for postoperative analgesia.

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227 Perioperative Considerations in a Patient Under Investigation or Patient With COVID-19

Amy Price • Larry F. Chu

Coronavirus disease 2019 (COVID-19) is a highly contagious respiratory disease that is spread person-to-person. Anesthesiologists should understand and implement safe clinical practice for patients with or suspected of having COVID-19. Suspicion for those who carry the virus and implementing safe clinical practice can help prevent community spread and protect both patients and health care providers themselves from illness.

1) **Etiology**
   a) **Definition: COVID-19 is a disease caused by novel coronavirus, known as SARS-CoV-2.**
      i) A series of acute atypical respiratory syndromes were originally observed in Wuhan, Hubei Province, China.
      ii) The disease caused by this virus was termed COVID-19.
      iii) The virus is transmittable between humans and has caused a pandemic worldwide.
   b) **COVID-19: "CO" stands for "corona," "VI" for "virus," and "D" for disease and 19 indicates the year it was discovered.**
   c) SARS-CoV-2 is the virus that causes COVID-19. SARS is the acronym for severe acute respiratory syndrome.
      i) It is a positive-sense, single-stranded, RNA enveloped virus belonging to the coronavirus family.
      ii) The 2 refers to a similar SARS virus that caused an outbreak in 2002.
2) **Pathophysiology of COVID-19**
   a) **Mechanism of invasion into host cells:** occurs through the spike or corona of the virus, which attaches at the surface of the host cell and then fuses viral and host cell membranes to initiate infection[1]
   b) **Causes of mortality:** Mortality estimates vary between 1% and 7% with those older than 65 or with underlying medical conditions considered at higher risk for severe illness.
      i) The leading cause of mortality is respiratory collapse followed closely by multi-organ failure.
   c) **Symptoms:** COVID-19 causes a syndrome of symptoms including cough, shortness of breath, fevers, chills, myalgias, muscle pain, sore throat, and sudden loss of taste or smell.
      i) Symptoms usually appear 2-14 days after exposure to the virus.
   d) **Diagnosis of COVID-19**: is confirmed when SARS-CoV-2 is detected in respiratory secretions[1]
      i) RNA or molecular tests are used confirm current viral infection and are detected using nasopharyngeal swabs. Sputum and bronchoalveolar lavage fluid can be tested when pneumonia or lung infection is present.
      ii) Antibody tests are used to identify if a person has been exposed to SARS-CoV-2 and developed immunity against SARS-CoV-2 infection. It takes 1-3 weeks to develop antibodies after exposure.



The mortality of patients with COVID-19 varies between 1% and 7%, with those older than 65 or underlying medication conditions having a higher risk for severe disease.

iii) Antigen tests are fast and very specific for the virus, but they are not as sensitive as molecular PCR tests and have more false positives.
iv) CT scans based on CORADS classification 1-6 are used to confirm COVID-19.

e) **MIS-C:** New reports of pediatric patients presenting with multisystem inflammatory syndrome (MIS-C) associated with COVID-19 are being seen.
i) The condition was first reported by clinicians in the United Kingdom.
ii) Kawasaki disease–like symptoms were reported in children who were previously healthy.
iii) The children had tested positive for current or recent infection by SARS-CoV-2.
iv) Presentation includes: persistent fever and a variety of symptoms including
(1) Elevated inflammatory markers
(2) Multiorgan involvement
(3) Hypotension
(4) Not all cases had respiratory symptoms.

f) **Classification System**: **Table 227-1** outlines the classification of COVID-19 patients[1]

3) **Treatment Considerations for COVID-19**: At the time of submission of this chapter there is no specific medication approved for the treatment of COVID-19. Current approaches toward treatment include repurposing existing FDA-approved medications as well as developing new medications such as a vaccine.

a) **Antiviral medications:** The RNA-dependent polymerase inhibitor remdesivir has shown some initial promise in leading to quicker recovery from COVID-19 in patients with advanced disease. Additional randomized placebo-controlled clinical trials are forthcoming.[2]

b) **Convalescent plasma transfusion:** Convalescent plasma collected from people who have recovered from COVID-19 contains antibodies to SARS-CoV-2. The FDA has issued guidance to clinicians for use of convalescent plasma as an investigational treatment.[3]

4) **Anesthesiology and COVID-19**

a) **The role of the anesthesiologist in the care of COVID-19 patients**
i) At the outset of the COVID-19 outbreak in New York City, one-third of patients hospitalized required invasive mechanical ventilation.
ii) Respiratory failure and need for vasopressor support are just some of the indications for the involvement of anesthesiologists and critical care physicians in the care of patients with COVID-19.[4]

b) Anesthesiologists should be prepared to manage COVID-19 patients in respiratory distress and multiorgan failure.

TABLE 227-1 Classification of COVID-19 Patients by Symptoms

Asymptomatic	These patients have no symptoms but test positive by nucleic acid test for SARS-CoV-2, the virus that causes COVID-19.
Mild	The patient exhibits symptoms of upper airway tract infection (URI) or gastrointestinal tract (GI) symptoms. These can include fever, fatigue, sore throat, myalgias, rhinorrhea, sneezing, nausea, vomiting, abdominal pain, or diarrhea.
Moderate	Pneumonia without hypoxemia. A CT examination of the chest may show bilateral and peripheral ground-glass and consolidative pulmonary opacities.
Severe	Pneumonia with hypoxemia
Critical	Acute respiratory distress syndrome (ARDS), and may have other signs of multiorgan failure including shock, encephalopathy, myocardial infarction, heart failure, coagulopathy or acute renal insufficiency/failure

c) The perioperative and critical care management of COVID-19 patients and those suspected to carry COVID-19 can place patients and health care professionals at increased risk for transmission of the SARS-CoV-2 virus.[5]



The perioperative and critical care management of COVID-19 and PUI can place patients and health care professionals at increased risk for transmission of the SARS-CoV-2 virus.

5) **Transporting Patients With COVID-19 or Patient Under Investigation PUI**
   a) **Indications**
      i) Need to transport for procedures or studies that are essential for care
      ii) Minimize transport for nonessential purposes.
   b) **Preparation**
      i) Consult your hospital infection control expert for advice or procedures.
      ii) Collect appropriate personal protective equipment (PPE).
      iii) Ensure for enough personnel to help with the transport.[4]
      iv) Recommendations intubated patients: A HEPA filter should be placed between the bag-valve-mask device and the endotracheal tube.[6]
      v) Nonintubated patients: A surgical mask should be worn by the patient.
      vi) The transporting team should include one person who dons appropriate PPE (per CDC guidance) that is in physical contact with the patient.
      vii) Ideally one member of the transportation team should not wear gown or gloves and can interact with the hospital environment such as doors and elevator switches.
      viii) According to the American Society of Anesthesiologists (ASA), health care professionals transporting a patient should not routinely wear gowns and gloves unless they are in direct contact with the patient or contaminated equipment.
      ix) All members of the medical team must wear medical protective masks during transport regardless of whether the patient is intubated or not.
6) **Preoperative care of Patients With COVID-19 or PUI**
   a) **Anesthesia plan**
      i) Consideration should generally be given to methods of anesthesia that minimize airborne and droplet transmission of the virus during the surgical procedure.
      ii) General anesthesia is recommended to reduce transmission. Consider minimizing fresh gas flows during preoxygenation, and utilizing rapid-sequence intubation technique to reduce exposure to any airway droplets or aerosols.
      iii) Spinal/Combined spinal-epidural is recommended for parturients presenting for cesarean delivery. Care should be taken to have the patient wear a mask throughout the surgery.
   b) The patient should be brought directly to the operating room and should not be directed to the regular preoperative holding area.
7) **Intraoperative care of COVID-19 or patients under investigation**
   a) **Laryngoscopy and intubation:** Methods should be undertaken to minimize potential exposure to SARS-CoV-2 virus in the airway. This can be done through distancing, time, and minimizing particle generation.
      i) Distancing: Use of video laryngoscopy techniques increases the distance between the anesthesiologists and the patient airway, ostensibly reducing viral load exposure.
      ii) Time: Use of rapid-sequence induction rapidly establishes intubating conditions, reducing the time needed for intubation.
         (1) Use of video laryngoscopy may also reduce the time and increase success rate of first-attempt intubation.

iii) Particle/droplet generation: Use of rapid-sequence induction eliminates the need for mask ventilation and potential droplet generation from the airway.
(1) Minimizing fresh gas flow during preoxygenation may also help minimize airway droplet generation.
(2) Use of lidocaine 1.5 mg/kg IV may suppress airway reflexes and reduce coughing that could generate aerosol or airway droplets during induction and intubation.[7]
(3) Avoid awake fiber-optic intubation as this method of intubation can generate respiratory droplets and aerosols.

b) **Reduce cross-contamination of work environment**
i) Use of double gloves during intubation allows soiled gloves to be immediately removed after intubation and reduces the chance of cross-contamination of workplace surfaces.
ii) Covering of anesthesia carts and work surfaces with plastic sheets during induction and intubation can also help reduce contamination.
iii) After removing PPE, avoid touching your face or hair and perform hand hygiene with WHO-approved hand sanitizer.
(1) Use an alcohol-based hand sanitizer with at least 60%-95% alcohol or hand washing with soap and water for at least 20 seconds.
(2) Extreme caution should be used in removing contaminated PPE to avoid self-inoculation with the virus.
(3) The help of an independent observer during doffing to assist the anesthesiologist in observing for possible self-contamination and providing advanced warning may be warranted.[5]



Avoid awake fiber-optic intubation in COVID-19 patients as this method of intubation can generate respiratory droplets and aerosols that carry the SARS-CoV-2 virus.

iv) Ideally, a closed-suction system should be used for suction of the airway.
v) Ensure that the anesthesia machine has proper HEPA filters.
(1) A HEPA filter should be placed between the Y-piece of the anesthesia circuit and the airway (eg, mask, endotracheal tube).
(2) The CO_2 sampling line should also be filtered by the HEPA filter.
(3) All gas exiting the anesthesia machine and sampling system should be scavenged outside and not be allowed to reenter the operating room.

c) **On extubation, airway secretions can be minimized by covering the patient's mouth and nose with two layers of moistened gauze.**
i) Use nasal cannula or facemask with low-flow oxygen.

d) **Obstetric anesthesia considerations for the patient with COVID-19 or PUI**
i) COVID-19 is not a contraindication to neuraxial anesthesia.
ii) There are insufficient data to determine the risk of inhaled nitrous oxide on aerosolization or generation of respiratory droplets. It is currently not recommended.
iii) Implement COVID-19 screening for all parturients.
iv) Cesarean section considerations include:
(1) Anesthesia providers should don droplet, contact, and airborne precautions (N95 or powered air purifying respirator [PAPR]).
(2) The patient should don a surgical mask at all times.
(3) Carboprost should be avoided in COVID-19 patients as they may be prone to bronchospasm.



The help of an independent observer during doffing to assist the anesthesiologist in observing for possible self-contamination and providing advanced warning may be warranted.

8) **Postoperative care of COVID-19 or person under investigation for COVID-19 (PUI)**
   a) **Extubation and recovery:** Patients should be recovered from anesthesia in the operating room or in a negative-pressure room such as an isolated room in the ICU.
      i) The patient should not be recovered in the standard PACU areas in order to limit transmission of the virus.



Carboprost should be avoided in COVID-19 patients since they are prone to bronchospasm.

9) **PPE Guidelines (Table 227-2)**
   a) **Indications:** For aerosol generating procedures (eg, tracheal intubation, noninvasive ventilation, tracheostomy, cardiopulmonary resuscitation, manual ventilation before intubation and bronchoscopy) clinicians should use respirators, eye protection, gloves, and gowns; aprons are needed when gowns are not fluid resistant. Minimum recommended PPE for direct patient care are gowns, gloves, medical mask, and eye protection such as goggles or a face shield.
   b) **Recommendations:** A joint statement by The American Society of Anesthesiologists (ASA) and others recommend anesthesia professionals should use properly fitted N95 masks or PAPRs. For those who are not N95 fit-tested, have facial hair, or fail N95 fit-testing, PAPRs should be used if possible. Surgical face masks protect against COVID-19 droplet transmission but do not protect against aerosolized small particles. When possible with infected patients, source control is recommended.

TABLE 227-2 How to Don and Doff PPE (PAPR or N95)

Donning
1. Put on scrubs and head covering.
2. Disinfect hands.
3. Put on N95 mask.
4. Put on inner gloves.
5. Put on coveralls.
6. Put on eye protection (goggles, face shield).
7. Put on foot protection (shoe cover).
8. Put on isolation gown.
9. Put on PAPR.
10. Put on outer gloves.
11. Put on PAPR hood.
12. Put on outer apron, if used.
13. Test the fit of PPE components.
Doffing
1. Remove foot protection (shoe cover).
2. Remove outer gloves.
3. Disinfect hands.
4. Remove protective clothing.
5. Disinfect hands.
6. Remove eye protection (goggles, face shield).
7. Disinfect hands.
8. Remove mask.
9. Disinfect hands.
10. Remove head covering.
11. Disinfect hands.
12. Remove inner gloves.
13. Disinfect hands.
14. Inspect your body for contamination.
15. Change to personal clothes.

10) **Disaster resource allocation**
   a) Reuse of single use PPE: Not recommended unless there are no alternatives. Infection risk to patients and health care professionals is increased and multi-use of single use PPE has not been endorsed as without risk. Unvalidated decontamination methods, use across multiple patients, extended wear, and increased donning and doffing multiply risk for self-inoculation and cross contamination.
      i) N95 respirators: The reprocessing of respirators requires that devices are effectively decontaminated, maintain filtration efficacy, and maintain structural integrity to preserve mask-fit. We recommend that reprocessed masks be returned to the original wearer to avoid cross contamination and infection by other pathogens and to reduce sensitivities to other contaminants contained in the mask.[8]
      ii) Surgical masks: These might be reused in lower risk conditions where decontamination is effective and filtration efficiency is maintained bearing in mind that ~40% of patients may be asymptomatic and they should never be used in conditions where there is the possibility of aerosolization.
      iii) Placing multiple patients on a single mechanical ventilator: The Society for Critical Care Medicine (SCCM), ASA, Anesthesia Patient Safety Foundation (APSF), and other leading medical societies have issued a joint statement on ventilating multiple patients on a single mechanical ventilator.
         (1) Indications: A shortage of mechanical ventilators during a global pandemic requiring respiratory support for large numbers of patients
         (2) Recommendations: The SCCM, ASA, APSF, and other leading medical societies that issued the joint statement on ventilating multiple patients on a single mechanical ventilator do not recommend placing multiple patients on a single mechanical ventilator for the following reasons:
            (a) It cannot be done safely with current equipment.
            (b) The respiratory pathophysiology of COVID-19 ARDS is complex, requiring individualized and careful ventilation.
               (i) Even in ideal circumstances, ARDS can be associated with 40%-60% mortality.



Placing multiple patients on a single mechanical ventilator is not recommended as it cannot be safely done with current equipment.

   b) **Purposing anesthesia machines as an ICU ventilator**
      i) The FDA does not provide approved labeling for the use of anesthesia ventilators for long-term ventilatory support.[9]
      ii) The ASA and APSF have provided guidance for the use of anesthesia ventilators in the event of a shortage of ICU ventilators. Among their recommendations:
         (1) An anesthesia professional should be available at all times to manage the anesthesia machine as an ICU ventilator.
         (2) An anesthesia professional should ideally round once an hour on all machines used as ventilators.
         (3) Anesthesia machines are not protected against nonauthorized users changing settings or turning off the machines. Appropriate warning signs should be posted.[10]
         (4) Consider use of anesthesia machines as ICU ventilators for non-COVID patients so their rebreathing circuits do not become inadvertent transmission vectors of SARS-CoV-2.
         (5) Use of heat and moisture exchange filters (HMEF) at the end of the endotracheal tube connection to the breathing circuit and on the expiratory hose is recommended.

11) **Management of medical staff after exposure to COVID-19**
   a) **Assessment:** After an exposure to a COVID-19 patient or their contamination, an anesthesiologist should report to their supervisor and/or occupational health immediately.
      i) Exposure risk should be assessed according to WHO guidelines.[11]

b) **Recommendations:**
   i) Health care workers who are assessed by WHO guidelines and found to be and high risk should:
      (1) Stop all health care interactions with patients for 14 days after the last exposure to a confirmed COVID-19 patient
      (2) Obtain testing for COVID-19
      (3) Self-quarantine for 14 days
   ii) Health care workers who are assessed by WHO guidelines and found to be a low risk should:
      (1) Self-monitor temperature and respiratory symptoms for 14 days after the last exposure to a confirmed COVID-19 patient
      (2) Notify their workplace immediately if they develop symptoms
      (3) Follow strict contact and respiratory droplet restriction measures

12) **Cancelling or rescheduling elective surgeries during COVID-19 or global pandemic**
   a) **The ASA and APSF have issued joint guidance on nonurgent care during the COVID-19 pandemic.**[12] Some of their recommendations include
      i) Reducing nonurgent surgical, diagnostic, and interventional procedures in areas with increasing COVID-19 incidences
      ii) Rescheduling of non–time critical procedures to a date when community transmission of SARS-CoV-2 is no longer taking place
   b) **The Centers for Medicare Services (CMS) recommends that Tier 3 (high acuity treatment or service) not be postponed.**
      i) Tier 3: Lack of in-person treatment would lead to patient harm.
      ii) Consider triage to appropriate facility of care as necessary.

13) **Restarting elective cases**
   a) **The ASA, American College of Surgeons (ACS), Associate of Perioperative Nurses, and American Hospital Associate have issued a joint statement on resuming elective surgery after the COVID-19 pandemic.**[12] Some of their recommendations include the following:
      i) There should be a sustained reduction in the incidence of COVID-19 in the hospital region for at least 14 days prior to resuming elective surgery.
      ii) COVID-19 testing within a hospital should protect staff and patients whenever possible.
      iii) Facilities should have adequate amounts of PPE and medical surgical supplies for the number and type of procedures.
      iv) Facilities should adopt policies that address care issues specific for COVID-19 across the perioperative journey, including postponement of surgery.

CHAPTER SUMMARY FOR SAFE ANESTHESIOLOGY PRACTICE IN THE PATIENT WITH OR SUSPECTED OF COVID-19 DURING A GLOBAL PANDEMIC	
Overview	• COVID-19 is a highly-contagious disease caused by novel coronavirus, known as SARS-CoV-2.
Etiology	• COVID-19 causes a syndrome of symptoms including cough, shortness of breath, fevers, chills myalgias, muscle pain, sore throat, and sudden loss of taste or smell.
Pathophysiology	• Mortality estimates vary between 1% and 7% with those older than 65 or with underlying medical conditions considered at higher risk for severe illness.
Preoperative strategies	• Patients should be taken directly to the OR.

Intraoperative strategies	• General anesthesia is recommended to reduce transmission. • Neuraxial anesthesia is not contraindicated for c-section. • Rapid-sequence induction with video laryngoscopy may reduce transmission risk.
Postoperative strategies	• Low-flow oxygen by nasal cannula or face mask • Patients should not go to regular PACU, but recover in OR or a negative pressure room or ICU room.

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Practical Clinical Drug Dosing

Karim T. Rafaat

THE CLINICAL DRUG DOSING AND INFORMATION CARD CAN BE DETACHED FROM THIS BOOK FOR EASY REFERENCE

Doses on this card may differ slightly from those listed in the appendix.

This reflects the Author's practical approach to clinical drug dosing. The author recognizes that a wide range of valid drug doses exist and that each patient's individual needs may differ. Clinical judgement must be used in determining the proper drug dosing for each patient.

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STEROIDS		GLUCO	MINERAL
DEXAMETHASONE	**EXTUB**: 0.25 mg/kg/dose q6 IV, **CROUP**: 0.6 mg/kg × 1 IV, **MENINGITIS**: 0.6 mg/kg/d q6 × 4 d IV	25	0
FLUDROCORTISONE	0.005-0.2 mg/d IV	0	14
HYDROCORTISONE	**SHOCK**: 2-4 mg/kg/d (max 200 mg/d) Adults: 50 mg IV q6	1	1
METHYLPREDNISOLONE	**ASTHMA**: 2 mg/kg then 1 mg/kg q6 IV	4	0.8

ANTIARRHYTHMICS	
ADENOSINE	0.1 mg/kg then 0.2 mg/kg fast IV (**ADULTS**: 6 mg, then 12 mg IV)
AMIODARONE	5 mg/kg (mr × 1) then 7-21 mg/kg/d IV(**ADULTS**: 150-300 mg IV then 1 mg/h)
LIDOCAINE	0.5-1 mg/kg IV, **PREINTUB**: 1.5 mg/kg IV (max 3 mg/kg) ***(okviaETT)***
PROCAINAMIDE	5-10 mg/kg IV then 40-80 µg/kg/min IV (max 100 mg, then 6 mg/min)
ALBUTEROL	0.15 mg/kg/dose INH (2.5-5 mg) **CONT**: 0.3-0.45 mg/kg/h INH (10-15 mg/h)

MISCELLANEOUS	
DANTROLENE	2.5 mg/kg q10 IV, titrated to effect, then 1 mg/kg IV q6 × 1-2 d
DIPHENHYDRAMINE	1-2 mg/kg IV q6 (**ADULTS**: 25-50 mg IV)
PROSTAGLANDIN E_1	0.05-0.2 µg/kg/min IV
RACEMIC EPI	0.05 mL/kg/dose IV in 3 cc NS

OPIOID CONVERSION	
	1 MG MORPHINE EQUALS
FENTANYL	0.01 mg IV
HYDROMORPHONE	0.15 mg IV, 0.75 mg po
METHADONE	1 mg IV, 1.5 mg po
MEPERIDINE	10 mg IV
OXYCODONE	2-3 mg po

MAXIMUM LOCAL ANESTHETIC DOSE		
	WITHOUT EPI	WITH EPI
BUPIVICAINE	3 mg/kg	4 mg/kg
CHLOROPROCAINE	8 mg/kg	10 mg/kg
LIDOCAINE	5 mg/kg	7 mg/kg
ROPIVICAINE	2.3 mg/kg	3 mg/kg

CHILD'S ESTIMATED WEIGHT (kg) = (Age × 2) + 8 (up to 9 y old) **PEDS ETT SIZE** = Age/4 + 4
ETT SIZE BY WEIGHT <1000 g: 2.5, 1000-2000 g: 3.0, 2000-3000 g: 3.5, >3000 g: 4.0
ETT DEPTH = 3 × ETT size or Age +10 (adults: M 23 cm; F 20 cm)
LMA SIZE = **1** 0-5 kg; **1.5** 5-10 kg; **2** 10-20 kg; **2.5** 20-30 kg; **3** 30-50 kg; **4** 50-70 kg; **5** 70-100 kg

1 MAC						
	NEO	1-6 MO	6-12 MO	12-24 MO	CHILD	ADULT
DESFLURANE	9.2%	9.4%	9.9%	8.7%	7.9%	6.3%
ISOFLURANE	1.6%	1.9%	1.8%	1.6%	1.6%	1.2%
N_2O					109%	109%
SEVOFLURANE	3.2%	3.2%	2.5%	2.5%	2.5%	2%

q, every; h, hours; IV, intravenous; INH, inhaled; po, oral; PR, rectal; mg, milligram; kg, kilogram; IM, intramuscular; g, gram; cc, cubic centimeters; mr, may repeat; D25W, dextrose 25% in water

BENZODIAZEPINES/BARBITURATES/SEDATIVES	
DEXMEDETOMIDINE	**SED**: 0.1-0.7 mg/kg/h IV, **PREMED/DELERIUM**: 0.25-0.5 µg/kg IV or 0.5 µg/kg/NARE
DIAZEPAM	**SED**: 0.05-0.2 mg/kg IV, **STATUS**: 0.5 mg/kg PR
ETOMIDATE	**IND**: 0.2-0.3 mg/kg IV
FLUMAZENIL	**REVERSAL**: 0.01 mg/kg IV (titrate in 0.2 mg increments in adults)
KETAMINE	**PREMED**: 5-6 mg/kg po, 0.1-0.5 mg/kg IV, **IND**: 2-3 mg/kg IV, 3-5 mg/kg IM, **PAIN**: 0.1-0.5 mg/kg IV, **IM COCKTAIL**: ketamine 2-3 mg/kg IV, versed 0.1 mg/kg IV, glyco 0.01 mg/kg IV
LORAZEPAM	**SED**: 0.01-0.1 mg/kg IV (2 mg), 0.1 mg/kg IM (4 mg)
MIDAZOLAM	**PREMED**: 0.4-0.8 mg/kg po, **SED**: 0.1-0.2 mg/kg IM, 0.05-0.15 mg/kg IV, **STATUS**: 0.1-0.2 mg/kg IV
PROPOFOL	**IND**: 2-3 mg/kg IV (**ADULTS**: 1-2.5 mg/kg IV), **INF**: 50-200 µg/kg/min IV

OPIOIDS AND ANALGESICS	
ACETAMINOPHEN	40 mg/kg PR then 15 mg/kg po, PR q6h (max ~90 mg/kg/d) 15 mg/kg IV q6
ALFENTANIL	**PAIN**: 5-10 µg/kg IV (250-500 µg), **IND**: 10-50 µg/kg IV
FENTANYL	**IND**: 2-5 µg/kg IV, **PAIN**: 0.5-2 µg/kg IV
HYDROMORPHONE	**PAIN**: 0.015 mg/kg IV (0.2-1 mg)
IBUPROFEN	10 mg/kg po q6h (800-1000 mg)
KETOROLAC	0.5 mg/kg q6h IV (max 15 mg <50 kg, 30 mg >50 kg)
MEPERIDINE	**SHIVERING**: 0.1-1 mg/kg IV
MORPHINE	**PAIN**: 0.02-0.1 mg/kg IV
NALOXONE	**FULL**: 0.1 mg/kg IV (0.4-0.8 mg), **PARTIAL**: 0.01 mg/kg IV (0.04 mg)
REMIFENTANIL	**RSI**: 3-4 µg/kg IV, **INF**: 0.1-0.3 µg/kg/min IV
SUFENTANIL	**IND**: 0.3-1 µg/kg IV, **INF**: 0.3 µg/kg/h IV (dc 40 min pre end)

NEUROMUSCULAR BLOCKING AGENTS (INTUBATING DOSE)		
CISATRACURIUM	0.15-0.2 mg/kg IV	
ROCURONIUM	**CHILDREN + ADULTS**: 0.6 mg/kg IV, **RSI**: 1.2 mg/kg IV, 1.8-2 mg/kg IM	
SUCCINYLCHOLINE	**NEO**: 2-3 mg/kg IV, **KIDS/ADULTS**: 1-2 mg/kg IV, 4-5 mg/kg IM	
VECURONIUM	0.1 mg/kg IV	**SUX FOR LARYNGOSPASMSM**: 1/10th dose

FFP, fresh frozen plasma; DDAVP, desmopressin; IND, induction; INF, infusion; PRBC, packed red blood cells; RSI, rapid sequence induction; SED, sedation; STATUS, status epilepticus; T3, triiodothyronine

NMB REVERSAL/ANTICHOLINERGIC

SUGAMMADEX	**REVERSE RSI**: 16 mg/kg **0 TWITCH TOF**: 4 mg/kg **2 TWITCH TOF**: 2 mg/kg	Use only for reversal of Vecuronium and Rocuronium
ATROPINE	0.02 mg/kg IV (min: 0.1 mg) ***(2× dose via ETT)***	
NEOSTIGMINE	0.07 mg/kg IV	ratio: 1 mg
GLYCOPYRROLATE	5-10 µg/kg IV, 0.01 mg IM	0.2 mg
PHYSOSTIGMINE	0.01-0.03 mg/kg IV, then 0.03 mg/kg/h for cholinergic syndrome	

INOTROPES AND VASOPRESSORS

	CHILDREN	ADULTS
DOPAMINE	2-10 µg/kg/min IV	
DOBUTAMINE	2-10 µg/kg/min IV	
EPINEPHRINE *Can use in ETT (2× dose)*	0.1-1 µg/kg/min IV, **HYPOTENSION**: 1 µg/kg IV **CODE**: 10 µg/kg IV **SC**: 0.01 mg/kg (max 0.3 mg)	**INF**: 0.01-0.3 µg/kg/min IV **CODE**: 0.5-1 mg IV
EPHEDRINE	0.1 mg/kg IV	2.5-10 mg IV
GLUCAGON	50 µg/kg IV then 5 µg/kg/min IV	
ISOPROTERENOL	0.1-1 µg/kg/min IV	1-5 µg/min IV
METHYLENE BLUE	1-2 mg/kg over 15 min IV, then 0.5-2 µg/kg/h IV (potential for pulmonary hypertension)	
MILRINONE	**LOAD**: 50-75 µg/kg IV **INF**: 0.1-2 µg/kg/min IV	**LOAD**: 50-75 µg/kg IV **INF**: 0.375-0.75 µg/kg/min IV
NEOSYNEPHRINE	**INF**: 0.1-1 µg/kg/min IV **BOLUS**: 5-20 µg/kg IV	
NESIRITIDE	0.005-0.02 µg/kg/min IV	
NOREPINEPHRINE	0.05-2 µg/kg/min IV	0.01-0.3 µg/kg/min IV
OCTREOTIDE	**INF**: 1 µg/kg/h IV	
TERBUTALINE	0.4-2 µg/kg/min IV	
VASOPRESSIN	**SHOCK**: 0.02-0.12 units/kg/h **CODE**: 0.4 units/kg	0.01-0.04 units/min IV **CODE**: 40 units IV push × 1

BLOOD, ETC.

CRYOPRECIPITITATE	1-2 units/kg IV	↑fibrinogen by 60-100
DDAVP	0.3 µg/kg over 30 min IV	
FACTOR VIIA	100 µg/kg/dose IV (start with 1/4-1/2 dose)	
FFP	10-15 mL/kg IV	↑factor by 15%-20%
K-CENTRA	**INR**: 2-4 4-6 >6	25 units/kg (max 2500) 35 units/kg (max 3500) 50 units/kg (max 5000)
PLATELETS	5-10 mL/kg IV	↑platelets by 50-100k
PRBC	10-15 mL/kg IV	↑Hgb by 2-3 g/dL
PROTAMINE	1 mg IV neutralizes 100 units heparin	
TXA	Hyperfibrinolysis/Massive Transfusion - 10mg/kg (adults 1g) over 10 min	

ANTIHYPERTENSIVES

CLONIDINE	5-10 µg/kg/d q6-8 po	0.5-2 µg/kg IV ($T_{1/2}$ of 9 h)
ESMOLOL	**INF**: 50-300 µg/kg/min IV **ADULT BOLUS**: 10-20 mg IV titrated to effect	
FENOLDOPAM	0.05-0.2 µg/kg/min IV	
HYDRALAZINE	0.1-0.2 mg/kg q20min IV, then q4-6h (**ADULTS**: 5-20 mg IV) to effect	
LABETALOL	0.1-0.3 mg/kg/dose IV (**ADULTS**: 10-20 mg IV) to effect	
METOPROLOL	**ADULTS**: 5-10 mg in 1 mg increments IV	
NICARDIPINE	**INF**: 0.5-5 µg/kg/min IV (**ADULTS**: 2-5 mg/h IV), **BOLUS**: 1-2 µg/kg IV (**ADULTS**: 250-500 µg IV)	
NIFEDIPINE	5-15 µg/kg IV	
NITROGLYCERIN	**INF**: 0.1-1 µg/kg/min IV, **BOLUS**: 0.1-0.2 µg/kg IV (**ADULTS**: 10-20 µg IV)	
NITROPRUSSIDE	**INF**: 0.1-2 µg/kg/min IV, **BOLUS**: 01-0.2 µg/kg IV (**ADULTS**:25-50 µg IV)	

FLUIDS/DIURETICS

3% NACL	5-10 cc/kg IV (5 cc/kg ↑[Na] by ~4 mEq/L)
BUMETANIDE	0.015-0.1 mg/kg/dose IV (~1/40th furosemide dose,1/10th for bad kidneys)
CHLOROTHIAZIDE	2-4 mg/kg/d q6-12h IV (**ADULTS**: 250-500 mg q8-12h IV)
FUROSEMIDE	0.5-2 mg/kg q6-12h IV (**ADULTS**: 10-40 mg q6-12h IV)
MANNITOL	0.25-1.0 g/kg IV

ELECTROLYTES

BICARBONATE	1-2 mEq/kg/dose IV or 1/3rd base deficit × wt (kg)
CALCIUM CHLORIDE	10-20 mg/kg IV
CALCIUM GLUCONATE	100 mg/kg IV (max 4 g/dose)
DEXTROSE	2-4 cc/kg D25W IV
INSULIN AND GLUCOSE	Glucose 0.5-1.0 g/kg with insulin 0.1 units/kg IV
MAGNESIUM SULFATE	25-50 mg/kg IV (max 2 g), may use 50-70 mg/kg for status asthmatic
POTASSIUM	0.5-1 mEq/kg IV over 1-2 h
SODIUM POLYSTYRENE SULFONATE	(Kayexalate) 1 g/kg/dose with 5 cc 20% sorbital PR

ANTIEPILEPTICS (NOT BENZODIAZEPINES)

FOSPHENYTOIN	Dosed like phenytoin (max: 1.5 g), May be given IM
LEVETIRACETAM	50 mg/kg/d q12 IV (**ADULTS**: 500 mg IV q12)
PHENOBARB	**LOAD**: 15-20 mg/kg IV, then 5 mg/kg/d q12 IV
PHENYTOIN	**LOAD**: 15-20 mg/kg IV, MAINTENANCE: 5 mg/kg/d q12 IV

cc, cubic centimeters; D25W, dextrose 25% in water; DI, diabetes insipidus; g, gram; h, hours; IM, intramuscular,; IV, intravenous; kg, kilogram; mg, milligram; po, oral; PR, rectal; q, every