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Evidence Supporting Conservative Scar Management Interventions Following Burn Injury: A Review Article

Zoë Edger-Lacoursière, MSc[1,2] Mengyue Zhu, B.Med[3,4] Stéphanie Jean, MD, MSc, FRCPC[2,5,6,7] Elisabeth Marois-Pagé, MD[2] and Bernadette Nedelec, BSc, PhD[1,2,8,*]

Abstract

Conservative management for hypertrophic scars (HSc) and scar contractures is of utmost importance to optimally reintegrate burn survivors into society. Many conservative treatment interventions have been described in the literature for the management of HSc. Recent advancements in the literature pertaining to postburn scarring and HSc formation have advanced our understanding of the mechanisms that support or refute the use of common rehabilitation treatment modalities after burn injury. This is particularly relevant for recent advancements in the fields of mechanotransduction and neurogenic inflammation, resulting in the need for rehabilitation clinicians to reflect upon commonly employed treatment interventions.

The aim of this review article is to summarize and clinically apply the evidence that supports or refutes the use of common conservative treatment interventions for scar management employed after burn injury. The following treatments are discussed, and mechanotransduction and neurogenic inflammation concepts are highlighted: (1) edema management (compression, positioning/elevation, pumping exercises, retrograde massage, and manual edema mobilization); (2) pressure therapy (including custom fabricated pressure garments, inserts, face masks, and other low-load long-duration orthotic devices); (3) gels or gel sheets; (4) combined pressure therapy and gels; (5) serial casting; (6) scar massage; and (7) passive stretching.

This review supports the following statements: (1) Compression for edema reduction should be initiated 48–72 hours postinjury and continued for wounds that require longer than 21 days to heal until scar maturation; (2) Elevation, pumping exercises, and retrograde massage/MEM should be used in combination with other edema management techniques; (3) Custom-fabricated pressure garments should be applied once the edema is stabilized and adequate healing has occurred. Garments should be monitored on a regular basis to ensure that optional pressure, >15 mm Hg, is maintained, adding inserts when necessary. The wearing time should be >16 hours/day; (4) Gels for postburn scar management should extend beyond the scar; (5) Serial casting should be applied when contractures interfere with function; (6) Forceful scar massage should be avoided early in the wound healing process or when the scar is inflamed or breaks down; and (7) Other treatment modalities should be prioritized over passive stretching for scar management.

Key words: burn scar management; rehabilitation; hypertrophic scar; mechanotransduction; neurogenic inflammation

INTRODUCTION

Medical advancements in the past decade have allowed for substantial progress in the management of critically burned patients, which has resulted in increased survival rates.[1] Despite these advances, morbidity from a burn injury is still one of the highest for traumatic injuries. It is also one of the most common types of trauma worldwide following traffic accidents, falls,

[1]*School of Physical and Occupational Therapy, McGill University, Montréal, Québec, Canada;* [2]*Hôpital de réadaptation Villa Medica, Montréal, QC, Canada;* [3]*Department of Orthopedics, Zhongnan Hospital of Wuhan University, Wuhan, China;* [4]*Institute of Burns, Wuhan Third Hospital (Tongren Hospital of Wuhan University), Wuhan, China;* [5]*Centre Hospitalier de l'Université de Montréal (CHUM), Montréal, QC, Canada;* [6]*Institut de Réadaptation Gingras-Lindsay de Montréal (IRGLM), Montréal, QC, Canada;* [7]*Département de médecine physique et réadaptation, Université de Montréal, Montréal, QC, Canada;* [8]*Centre de recherche du Centre Hospitalier de l'Université de Montréal (CRCHUM), Montréal, QC, Canada*

[*]*Corresponding author: Bernadette Nedelec, (bernadette.nedelec@mcgill.ca)*

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and interpersonal violence.[2,3] One of the leading causes of morbidity following burn injury is the development of hypertrophic scars (HSc), which is a common occurrence ranging from 33% to 91% of burn survivors depending on various patient and injury characteristics.[4–8] HSc can develop after any deep dermal or full thickness injury and are described as being thick, red, and rigid scars that are less esthetic, have the potential to limit movement when close to joints, and can cause pain or pruritus.[6,9–12] However, it is important to distinguish a HSc from a keloid since the pathophysiology and response to treatment vary substantially.[13] HSc after burn injury are especially concerning since they are associated with the development of scar contractures, persistent functional limitations, psychological distress, decreased quality of life,[14] and delayed reintegration into society.[11,15] The development of scar contractures is also a common complication after a burn injury, ranging from 38% to 54% at the time of discharge from acute care.[16] Both HSc and scar contractures are associated with functional limitations and increased need for reconstructive surgery, which increases the overall recovery time for patients and the costs to the healthcare system.[17] Thus, conservative management for HSc and scar contractures is of utmost importance to optimally reintegrate burn survivors into society.

Many conservative treatment options have been described in the literature for the management of HSc: the use of gels,[18–20] pressure therapy,[21–24] and scar massage.[25,26] On the other hand, the use of a splint (orthosis),[18,27–29] serial casting[27,30–34] and passive stretching[29,35–38] have been described as conservative interventions to manage scar contractures after burn injury, but in doing so have a direct impact on scar management as well. Although some treatment options have sufficient evidence to support their use, others are still being investigated or are lacking sufficient evidence, as thoroughly discussed below.

The objective of this review article is to summarize and clinically apply the evidence that supports or refutes the use of common conservative treatment modalities for scar management employed after burn injury. Mechanotransduction and neurogenic inflammation will be discussed, followed by a description of how these concepts apply to the following rehabilitation treatments: (1) edema management (compression, positioning/elevation, pumping exercises, retrograde massage, and MEM); (2) pressure therapy (including custom-fabricated pressure garments, inserts, face masks, and other low-load long-duration orthotic devices); (3) gels or gel sheets; (4) combined pressure therapy and gels; (5) serial casting; (6) scar massage; and (7) passive stretching.

BASIC SCIENCE PRINCIPLES TO CONSIDER IN BURN SCAR MANAGEMENT

Risk factors for the development of HSc following burn injury are well described in the literature. Firstly, when comparing the cellular and biological events that occur during the wound healing phases of nonpathological scar and HSc, it is clear that sustained inflammation and prolonged wound closure time play a critical role.[39,40] Although increased systemic inflammation is inevitable following a major burn injury, when it is combined with local inflammation at the wound site, this increases the overall impact of the inflammatory phase of wound healing, which can translate to longer wound closure times. In fact, burn survivors are much more likely to develop HSc when their wounds take more than 21 days (3 weeks) to close.[4,41,42] Furthermore, the presence of an infection also increases the magnitude and length of the inflammatory phase.[9] Thus, factors that increase the risk of an infection, higher total body surface area burned or genetic predispositions,[43] could increase the risk of HSc formation. In addition, burn survivors often undergo many surgical procedures for debridement and grafting, which inevitably prolong the inflammatory phase by reactivating the various wound healing cascades. In fact, the number of surgical procedures has been described as a predictor to HSc formation.[5]

Finally, in recent years, evidence on the impact of mechanical forces in wound healing on HSc development has grown rapidly.[9] Thus, this topic will be thoroughly reviewed in the following sections of this manuscript.

MECHANOTRANSDUCTION

HSc typically forms in areas that experience high mechanical stress such as the axilla, neck, and sternum.[5,10,44–46] Furthermore, HSc are less likely to form in older individuals who sustain a burn injury since aging skin has reduced mechanical tension.[47–50] Recent studies have been investigating the underlying mechanisms of mechanotransduction[51] and its possible implication in HSc formation. Mechanotransduction is the process by which cells translate mechanical stimuli such as shear or stretching forces into biochemical responses.[46,52] Cells perceive mechanical signals through cytoskeletal modules, which are made of cell-cell and cell-matrix proteins and/or transmembrane mechanosensitive structures.[51–53] When the mechanical signaling of the cells is altered or distorted, due to high mechanical forces, this sets off a cascade of profibrotic signals, which leads to an increase in cell proliferation, motility, survival, contraction, and/or collagen production.[51,54–56] Increased tensile forces and increased production of proinflammatory cytokines in inflammatory skin disorders have also been shown to shift the behavior of keratinocytes and fibroblasts towards a more proliferative and immature state.[55,57] In fact, some mechanotransduction signaling pathways have been shown to play crucial roles in wound healing and HSc formation due to increased skin stretch in both human and animal models.[46,54,58]

Mechanical off-loading

Unsurprisingly, since studies demonstrated that mechanical loading applied to a wound increased HSc formation, other studies focused on what happens when a wound is protected from mechanical forces (mechanical off-loading). One study by Gurtner et al. investigated the effect of a stress-shielding polymer applied to incisional wounds created in red Duroc pigs.[59] They found that incisional wounds treated with a stress-shielding polymer produced a significantly reduced scar area, by 6 and 9-fold, compared to a control incision and stress-elevated incision. In humans, the application of a tension-reducing tape on a surgical incision for 12 weeks, in a randomized control trial (RCT), reduced the risk of

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developing a HSc by 13.6 times compared to the control group.[60] In addition, 2 RCTs investigated the effect of a silicone sheet-based polymer with tension-relieving properties on half of an incisional wound following abdominoplasty and for individuals undergoing scar revision surgery.[61,62] Participants in both studies had a significantly improved scar appearance, using subjective scar evaluation scores, on the half where the tension-relieving polymer was applied. However, it is important to point out that in all of the human studies discussed above, the participants were recovering from surgical wounds, not burn injuries.

Scar force properties

In addition, studies have investigated the impact of the nature of the mechanical forces on wound healing. A number of in vitro studies have investigated the effect of cyclic strain vs static strain on cultured human fibroblasts,[63–66] where cyclic strain refers to cycles of "on" and "off" force application (ex: passive stretching) and static strain refers to the same amount of strain being applied over time (ex: static orthosis or cast). In all of these studies, cyclic strain increased fibroblast proliferation, cytokine production, type 1 collagen production, and extracellular matrix accumulation. An in vivo study by Chin et al. investigated the effects of static vs cyclical stretching forces on mice skin.[67] They reported that epidermal growth factor, transforming growth factor-β1, and nerve growth factor, which are all profibrotic, demonstrated greater expression in cyclically stretched skin when compared to static stretch. Although this has not yet been investigated in humans, these studies suggest that cyclic strain could potentially increase the risk of HSc formation. However, in vitro human fibroblast studies and studies performed in mice may not be generalizable to the in vivo application of cyclical stretching in humans; therefore, further research is required.

NEUROGENIC INFLAMMATION

Neurogenic inflammation is an inflammatory process that can be activated by increased mechanical stress, which is why it is linked to the concept of mechanotransduction.[68] Skin stretching and itching can activate the release of neuropeptides, which causes a neuroinflammatory response: increase cell proliferation, inflammatory cytokine, and platelet-derived growth factor production. This neurogenic inflammation perpetuates pruritus and exacerbates extracellular matrix accumulation.[9] Neuropeptide activity in the skin is clinically associated with the presence of erythema, edema, and patient-reported pruritus,[9,68–70] which often coexist with HSc following burn injury.[71] One of the neuropeptides that increases inflammation, keratinocyte proliferation and fibrogenesis is substance P.[9] Conversely, when innervation is impaired or lacking, and therefore the production of substance P is reduced, wound healing and scar formation are reduced.[72] Substance P has also been shown to modulate matrix metalloprotease and fibroblast activity, which may promote HSc formation. Interestingly, elevated substance P levels were found in HSc following burn injury.[73] Although no study has clearly demonstrated that neurogenic inflammation contributes to HSc formation, studies suggest that the presence of sensory fibers and their mechanical activation may play an important role in fibrosis.[9,68,74] For example, in Chin et al.'s study on static vs cyclical stretching forces on mices skin, discussed above,[67] neuropeptides in the epidermis and dermis significantly increased after cyclical stretching of the skin, which was not the case for continuous stretching. Although further studies are needed to investigate this in human subjects, this suggests that cyclic stretching of the skin could increase neurogenic inflammation.

CONSERVATIVE TREATMENT FOR SCAR MANAGEMENT FOLLOWING BURN INJURY

Edema management

Burn injury results in both local and generalized edema that effects injured and noninjured tissue, including organs. The extent of the fluid shift depends on the depth and surface area of the burn as well as the fluid resuscitation provided.[18,75–77] The immediate impact of edema is reduced joint and soft tissue mobility and increased pain. Moreover, prolonged edema may result in reduced strength, function, and quality of life.[77] In addition, the elucidation of the mechanotransduction signaling pathways has provided an explanation of how edema increases the mechanical force at a cellular level, leading to an increase in scarring and therefore suboptimal esthetic and functional outcomes.[78]

In order to reduce edema and the negative associated complications, a number of conservative treatment techniques are employed[18,77] including compression garments or wraps,[79–83] elevation or positioning,[84] movement and pumping exercise,[85] electrical stimulation,[86] and retrograde massage or MEM.[87]

Compression garments or wraps help to reinforce the interstitial hydrostatic pressure, thereby facilitating the return of fluid to the venous and lymphatic systems and providing a counterpressure to working muscles.[79,80,85] Investigations with burn survivors have demonstrated that adhesive compressive wraps were more effective than an off-the-shelf glove or standard gauze in reducing edema; however, both were associated with a reduction in pain and an increase in active range of motion (ROM) and function.[18] Furthermore, Sharp et al.'s best evidence statement on the use of pressure garments for HSc management reported that compression should be initiated as soon as possible.[81] Arguably, when treating burn survivors, pressure therapy should be delayed for the first 48–72 hours postinjury due to the dynamic fluid extravasation to avoid excessive pressure and potential tissue damage, but once the fluid shift has peaked, compression should be initiated.[77] Consistent with the mechanotransduction literature, the edema reduction found with the application of compressive wraps was associated with a reduction in scar thickness, measured by ultrasound, at four weeks; however, the difference was no longer significant at 4 months.[80] Thus, compression therapy in the acute phase to reduce edema should be considered one of the first lines of scar management or prevention.

In addition, elevation allows gravity to assist with the drainage of edema from the distal limb.[85] This has been demonstrated in normal individuals[84]; however, the use of elevation to reduce edema has not specifically been investigated

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with burn survivors. Moreover, elevation alone has not been shown to be sufficient by itself to maintain and sustain the edema reduction benefits and therefore should be combined with other treatment approaches.[85,88]

Active exercises promote the gliding of associated musculoskeletal structures relative to their adjacent counterparts, with the muscle contractions acting as a pump to facilitate drainage from distal regions.[89,90] When active exercises cannot be performed or are inadequate to address the edema formation, electrical stimulation should be considered, which has been shown to have a positive impact both in burn survivors[86] and in the normal population.[90] Although the reduction of scar formation secondary to active exercises has not been investigated with burn survivors, there is evidence supporting that exercise has a beneficial effect on body composition, health-related quality of life, and a reduction in the need for surgical release of contractures,[91] and is therefore commonly incorporated as a component of burn survivor rehabilitation program.[92]

The use of retrograde massage or manual edema mobilization (MEM) has been recommended for acute hand injuries, particularly when the previously described treatment interventions have not been effective.[87] The use of any manual mobilization therapy for edema reduction should be complemented by conventional treatments such as compression, elevation, and exercise, as has been demonstrated when using these techniques following distal radius fractures.[93]

Although edema management has been described in the burn literature as a means to increase hand function (as reported by the disabilities of the arm, shoulder, and hand questionnaire (DASH)), ROM, strength, quality of life, and reduce volume and pain,[18] it is important to note that there is no conclusive evidence supporting that edema management directly mitigates the development of scar formation in the long-term.

Burn wound edema is the result of an imbalance of the filtration system between capillary and interstitial spaces,[94] exacerbated by prolonged inflammatory responses and persistent neutrophil aggregation, resulting in increased tissue pressure and vascular congestion.[95] Unbalanced inflammatory mechanisms will result in prolonged inflammation (greater than 2–3 days), which is a major contributing factor to HSc development.[96] Thus, prolonged inflammation is a common factor in the formation of edema and HSc. The lymphatic vasculature assumes a pivotal role in the modulation of the inflammatory response through its influence on the removal of extravasated fluid, inflammatory mediators, and leukocytes.[97] In theory, various edema management strategies have the potential to either mitigate protracted inflammation by enhancing lymphatic circulation or reduce the concentration of proinflammatory cytokines, thereby diminishing the development of scar formation.[80] Importantly, the increase in wound tension applied to the local fibroblasts while edema is present increases those fibroblasts profibrotic gene expression and contractile properties,[78] increasing the formation of HSc and propensity for contracture formation. Although direct translational investigations of conservative treatment interventions to reduce edema have generally not been designed to establish their long-term impact of HSc formation, the research described below does support that compression should be started early and results in reduced scar formation.

PRESSURE THERAPY

As mentioned earlier, pressure therapy is a conservative rehabilitation treatment that has been described in the literature for the management of HSc.[21,79,81,98–101] Pressure therapy can take a variety of different forms, such as custom-fabricated pressure garments,[99,102,103] compressive wraps,[79,81] face masks,[100,104] etc. However, the pathophysiological basis for their use remains the same: it reduces the number of fibroblasts and myofibroblasts, promotes fibroblast differentiation, increases apoptotic cell death of fibroblast/myofibroblasts and increases the production of matrix metalloproteinase-9 through hypoxia.[70,102,105–107] Pressure therapy has been shown to reduce scar thickness and erythema and increases pliability.[21] Pressure therapy also applies compression to the surface of a body part, which has been shown to promote the differentiation of keratinocytes by mechanical tension off-loading.[47,57,70]

Although pressure therapy has been shown to be an effective treatment modality for HSc, further research is required to determine the optimal therapeutic dosage and wearing schedule to achieve optimal outcomes. Despite this, most experts recommend achieving a consistent pressure ranging between 10 and 30 mm Hg[47,108] with 15–25 mm Hg being used in experimental investigations.[21] In order to achieve consistent pressure, a systematic review and meta-analysis by Ai et al. recommended to replace pressure garments every 2–3 months since they tend to lose their elasticity and thus become less effective with time.[21,24] Furthermore, Sharp et al.'s best evidence statement on the use of pressure therapy for HSc reported that custom-fabricated pressure garments should be initiated as soon as the healing skin can tolerate the pressure, which usually corresponds to when the wound is closed.[81] In animal studies, 16–24 hours of wear per day, 7 days a week, showed optimal outcomes,[98,109,110] but this is often difficult to achieve on a long-term basis unless therapists apply evidence-based knowledge and approaches to optimize patient treatment adherence.[47,111–113] The time at which pressure therapy should be discontinued is not clear; however, a study by DeBruler et al. on pressure garment therapy using the red Duroc pig model showed that early cessation (17 weeks postburn) of pressure therapy could potentially result in a significant loss of HSc improvements and that pressure should be initiated immediately postgrafting.[110,114]

Pressure inserts have been used to increase pressure in contoured regions under custom-fabricated pressure garments, where these garments were not providing adequate pressure. Many different insert materials, from elastomer to plastazote to neoprene or foam, have been discussed in the literature[115–121]; however, few studies provided data to support the benefits of these inserts.[47] Nonetheless, inserts are still widely used in the clinical setting since they provide increased pressure to contoured regions such as finger web spaces or the palm of the hand, where it is virtually impossible to get adequate pressure with custom garments alone.

As mentioned, there are several other ways to apply pressure, such as with the use of transparent face masks. Face masks have been shown to provide more pressure than pressure garments alone or pressure garments with inserts.[122–126] In more recent years, Wei et al. published 2 studies which demonstrated, using objective scar evaluation tools, that

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face masks reduced the thickness and increased the pliability of scars in adults and children postburn.[100,104] In addition, a recently published single-blinded RCT of face masks with embedded pressure sensors demonstrated improved scar thickness and hydration using objective instrumentation and significant improvements using subjective evaluations such as the Vancouver scar scale and the patient and observer assessment scale.[127] These benefits are likely generalizable to any orthoses that are form fitting and apply adequate pressure; however, currently, there is very little published evidence for the scar management benefits provided by orthoses other than face masks.[100,104,127]

GELS

Similar to pressure therapy, gels have been used as a conservative rehabilitation treatment for the management of HSc.[20,101,128–131] Gels are available in a variety of forms, ranging from gel sheets, liquid gels, or gel tubes.[19] Experts recommend the use of gels for burn scars that require more than 21 days to heal, which are at risk of HSc formation.[20] Studies have reported improvements in HSc erythema, pruritus, texture, and thickness with the use of nonsilicone or silicone-based gels, and a systematic review and expert consensus supported the use of gels for immature burn scars.[20]

The therapeutic benefit demonstrated with the application of gels was associated with their occlusive properties hydration (reduces trans-epidermal water loss (TEWL)) and a temperature increase that can decrease nociceptor activity, thus decreasing neurogenic inflammation.[19,20,70] Mustoe et al. also suggested that the occluded and hydrated environment produced by gels recreates a more "normal" skin barrier by imitating the normal stratum corneum. This occlusive barrier decreases TEWL and reduces keratinocyte stimulation by normalizing their hydration state, which ultimately reduces fibroblast activity.[19] Although Mustoe reports that this is a hypothesis, it does correlate with clinical findings that occlusion is essential to the benefits of gels. Furthermore, a study by Musgrave et al. demonstrated that HSc temperature under gel sheets increased by 1.7 °C,[132] which some studies have suggested increases the activation of collagenase with subsequent collagen breakdown.[132–134] Finally, although the mechanism of action is still being investigated, some studies attribute the primary effects of gel sheets to the relocation of tensile forces. Akaishi et al. reported that gel sheets reduced the mechanical tension on the scar by transferring it to the edge of the sheet, which is ideally located over normal skin.[128] Therefore, mechanotransduction could also be involved in the therapeutic mechanism of gel sheets.

Although the use of gel products has been shown to be beneficial in the prevention and treatment of HSc, some studies have reported issues with their use in the clinical setting. Firstly, some parts of the body make it more difficult to use gel sheets due to the complex contouring or motility of the skin (ex. face, near joints, etc.), which may result in suboptimal contact and conformity to the skin compared to other treatment interventions.[19,135,136] Furthermore, some individuals experienced skin maceration, pruritus, and irritation in hotter climates associated with the use of gels.[137,138] Therefore, some individuals may not be able to wear them for the recommended period of time, thereby reducing their efficacy.[138] Since gel sheets require consistent hygiene management to prevent skin reactions, and some individuals are reluctant to wear them on unclothed areas, patient adherence is also a concern.[19,139]

COMBINED PRESSURE THERAPY AND GELS

In recent years, two RCTs investigated the effect of combined pressure therapy and gel sheets on HSc with the adult burn survivor population[140,141] and one with pediatric burn survivors.[142] The first study was conducted with participants who presented posttraumatic HSc who were allocated to 1 of 4 groups: pressure garment, silicone gel, combined pressure garment, and silicone gel, and a no-treatment control group. They receive treatment for 6 months. The scar characteristics were evaluated using objective instrumentation. The results demonstrated that the greatest improvement in scar thickness was in the combined group.[140] The second study was conducted with 16 individuals who had HSc on their hands following burn injury. Each hand was separated into 3 treatment sites, which were randomized: pressure garment, liquid silicone gel with pressure garment, and silicone gel sheet with pressure garment. They evaluated the scars using 2 subjective scar assessments: the Vancouver scar scale and the patient and observer scar assessment scale (POSAS). The results showed that both combined groups had better POSAS scores, but no differences were reported between these two groups.[141] The scars in the second study were not evaluated using objective measures and were underpowered. The third was a parallel group, randomized trial where participants were randomized to silicone gel only, pressure garments only, or combined gel and pressure garments; however, there was no significant group difference.[142] Therefore, there is a lack of sufficient evidence to determine if combined pressure therapy and gel sheets are superior.

Interestingly, both studies reported earlier by Wei et al. investigating the efficacy of 3D printed facemasks reported that the combination of pressure produced by the mask and silicone lining contributed to reducing HSc thickness.[100,104] Although further studies are needed to compare regular facemasks to facemasks with silicone linings, these studies do suggest that a combination of pressure with gels could be beneficial on HSc thickness and pliability postburn; however, the precise contouring associated with a facemask may contribute to the reported outcomes.

SERIAL CASTING

In the clinical setting, serial casting is usually used as a last resort treatment when a patient does not respond to traditional therapy or is nonadherent to treatment recommendations for the management of joint contractures.[30,32,143] However, experts from the 2009 burn rehabilitation and research consensus summit[144] question this practice, as some studies have demonstrated that casting provides wound protection[33] and prevents scar contraction or reestablishes function joint ROM.[30,145–147]

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Interestingly, although serial casting is not used specifically for the management of HSc, some articles have described its beneficial effects on wound closure, an important factor in the formation of HSc. Ricks and Meagher[33] investigated the effects of casts following burn injury for the treatment of lower extremity burns after grafting in children and reported that this treatment resulted in more rapid wound closure and more complete graft take. Furthermore, Bennet et al.,[30] Johnson and Silverberg,[32] and Ridgeway et al.[34] also reported that open wounds present under the casts improved or stayed the same throughout serial casting.

Serial casting is typically used as a conservative treatment modality for scar contractures by applying a gentle passive force to the contracted tissue at end range. This allows the tissue to be maintained in gentle tension for an extended period of time, increasing tissue length and passive ROM (PROM).[148,149] Therefore, when considering the mechanotransduction theory, it is possible that when serial casting is used to increase joint ROM at a comfortable stretch (low-load long duration stretch), it could potentially be less harmful than cyclic stretching. Evidence supporting the value of a low load for a long duration has recently been reported in several retrospective chart reviews of pediatric burn survivors positioned at end range after axilla or palmar burns.[145,146]

SCAR MASSAGE

Scar massage to treat HSc has been advocated for various physical and psychological reasons.[26] However, the technique employed and the anatomical location where the massage is applied vary if the goal of therapy is relaxation or anxiety reduction,[150] as the therapist may choose to intentionally avoid the scar sites. Additionally, scar massage is typically combined with the application of moisturizing lotions, creams, or oils, which are common treatments recommended for scar. The application of these products alone relieves itch and induces the sensation that the skin becomes more supple.[151] Thus, the reported value of scar massage in reducing itch, pain, and anxiety[26] may be predominantly associated with the application of moisturizers or focusing massage therapy on normal skin locations. Moreover, reduction of itch, pain, or anxiety are not the focus of this review and will not be thoroughly addressed.

The value of scar massage for the prevention of HSc formation has not been investigated, which is an important clinical question that requires further investigation. However, the mechanotransduction literature described above provides evidence that an increase in mechanical tension, particularly cyclical tension, correlates with an increase in scar formation. Therefore, it has been suggested that massage therapy should be avoided in cases where there is a high risk of HSc formation.[152] More recent studies have suggested that there may be some short-term improvements immediately following the therapy session with scar massage but no substantial long-term benefits.[101,153,154]

Once HSc has formed, there is extensive clinical belief that massage can reduce the thickness and increase the pliability of the scar.[155,156] Nonetheless, the evidence examining massage therapy for established postburn scars does not strongly support this treatment intervention. In the 1990s, Silverberg et al. published a pilot study[157] and Patino et al. a RCT,[158] both of which found no significant between-group difference, but both studies used a subjective scar evaluation measure[159] that may have lacked adequate validity and reliability.[160] A later publication by Roh et al.[161] reported an improvement using the same subjective scar evaluation in a case-controlled study where the participants in the treatment and control groups were from different burn centers. However, the same researcher later reported a similarly designed prospective trial where participants were recruited from the same center and objective scar evaluation measures were employed, but there was no significant between-group difference.[162] However, participants were not randomized to the control or treatment group, and this study may have been underpowered. More recently, Cho et al. performed a RCT of burn survivors and reported a significant reduction in thickness, erythema, melanin, and TEWL and an increase in elasticity.[163] However, an expert consensus statement published by members of the Wound Healing Society[164] advocates for the use of intraindividual control scars when designing studies to determine the efficacy of treatment for scar. Most recently, an intraindividual RCT, using objective scar evaluation measures, reported an immediate difference in elasticity, erythema, and melanin. However, there was no long-term between-group difference.[154] These results may provide an explanation for why there is such a strong clinical belief that massage therapy is beneficial. The increase in erythema is likely associated with a temporary increase in blood flow, the decrease in pigmentation associated with an exfoliating effect, and the increase in pliability associated with the temporary mobilization of water away from the overabundant, hydrophilic proteoglycans, and glycosaminoglycans in HSc.[165] This transient expulsion of water from the scar is similar to the indentation that is seen in scars where a gel sheet is placed under the garment, but after the garment is removed for a substantial period of time, the indentation disappears as the water returns. Consequently, the immediate impact of the forces applied during massage therapy may lead patients and therapists to believe that there are long-term changes in elasticity, erythema, and pigmentation. However, once baseline measures, the control scar, and the spontaneous resolution with time are appropriately incorporated in the analysis, there is no evidence of long-term benefit.

Hence, considering the treatment time required (at least 30 minutes daily for 12 weeks), usually provided by a trained therapist or primary caregiver, there is a lack of robust evidence to support the resources required if the goal of massage therapy is to increase the elasticity or decrease the thickness of scar. In fact, when specific training of therapists and caregivers was compared to massage that lacked standardization, there was no difference found in scar height, vascularity, pliability, itch, or pain between groups, despite the fact that itch and pain did improve with time.[166] These results, once again, demonstrate the need for an intraindividual control group to appropriately account for the spontaneous improvement of HSc with time.[154] Furthermore, early in the wound healing process, or later in the recovery process if there is any skin breakdown, or if there is a sustained inflammatory response, the mechanotransduction literature provides evidence that massage therapy may increase scar formation. However, light effleurage to work in moisturizers or massage therapy in

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nonburned regions may reduce itch, facilitate relaxation, and reduce anxiety.[26]

PASSIVE STRETCHING

As described earlier, passive stretching is a conservative, widely used intervention to manage joint scar contractures after a burn injury.[167] However, whether it has a direct effect on scar formation has not been evaluated. Moreover, the conclusion of a 2017 Cochrane systematic review was that there was no clinically important effect of stretching in nonneurologic conditions.[168] However, research following burn injuries is extremely limited. Nonetheless, treatment of burn wounds over or near joints commonly includes passive stretching to prevent or reduce joint contractures. This increases mechanical forces and may increase the neurogenic inflammation, particularly if the force levels elicit a pain response associated with the release of neuropeptides and may facilitate the conversion of fibroblasts into myofibroblasts. This possibility has led many basic scientists to conclude that passive stretching may lead to further scarring.[169–171] Thus, when possible, active mobilization to prevent joint contractures and the application of a low load for a long duration to reduce joint contractures when present should be prioritized.[145,172]

The experimental data substantiating this conclusion are that cyclical stretching strongly activates fibrotic pathways and responses in in vitro and in vivo animal models. In Webb et al.'s in vitro evaluation of human fibroblast proliferation, type 1 collagen, and fibronectin production were increased in the cyclic strain group, combined with a significant increase in gene expression for type I collagen, transforming growth factor-β1, and connective tissue growth factor.[64] Interestingly, when Huang et al. subjected normal human dermal fibroblasts in vitro to cyclic stretching for 24 hours, fibroblast migration increased and apoptosis reduced, but

Table 1. Summary of the Evidence that Support of Refute the Use of Common Conservative Treatment Interventions for Scar Management Following Burn Injury

Treatment intervention		Proposed mechanism supporting (√) or refuting (*X*) this intervention	Clinical considerations
Edema management	**Compression**	(√) Mechanical off-loading[59-62] (√) Reduces inflammatory response, neurogenic inflammation[79–83]	• Compression for edema reduction should be initiated 48–72 hours postinjury.[77]
	Positioning/Elevation	(√) Gravity assists the drainage of edema, which could help reduce the inflammatory response, neurogenic inflammation[84,85]	• Elevation should be used in combination with other edema management techniques.[85,88]
	Pumping Exercises	(√) Muscle contractions assist the drainage of edema, which could help reduce the inflammatory response and neurogenic inflammation[85,89,90]	• Active pumping exercises or electrical stimulation should be used in combination with other edema management techniques.[89,90]
	Retrograde massage/ Manual edema mobilization (MEM)	(√) Manually assists the drainage of edema, which could help reduce the inflammatory response, neurogenic inflammation[87]	• Retrograde massage/MEM should be used in combination with other edema management techniques.[87]
Pressure therapy (Including: pressure garments, inserts, face masks, and low-load long duration orthoses)		(√) Mechanical off-loading[21,47,57–61,70,79,98–110,122–126] (√) Reduces neurogenic inflammation[10,68–74]	• Custom-fabricated garments should be applied once edema is stabilized and there is adequate healing. • Optimal pressure is >15 mm Hg.[21,47,108] • Use inserts when necessary.[47] • Wearing time should be >16 hours/day.[98,109,110]
Gels or Gel Sheets		(√) Mechanical off-loading[20,59–62,128–131] (√) Reduces Neurogenic inflammation[10,68–74] (√) Occlusion[19,20,72] (√) Hydration[19,20,72] (√) Temperature increase[19,20,72,128–131]	• Gels should extend beyond the scar edge.[128]
Serial casting		(√) Mechanotransduction[21,30,32,143,145,146,148–151]	• Serial casting should be applied when contractures interfere with function.[30,32,143]
Scar massage		(*X*) Mechanotransduction[46,51–57,152,154–158] (*X*) Neurogenic inflammation[10,70–74,152,154–158]	• Scar massage, other than light effleurage for moisturizer application, should be avoided early in the wound healing process or when scars are inflamed or break down.[26,152]
Passive stretching		(*X*) Mechanotransduction[10,46,51–57,63–74,168–171]	• Other treatment modalities such as AROM, strengthening exercises, or low-load long duration orthoses should be prioritized over passive stretching for scar management.[145,169–172]

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it did not alter their capacity to proliferate. They surmised that the increased migration of the fibroblasts left them with little spare energy for proliferation. Matrix metalloproteinase expression was increased, which is known to play a role in collagen degradation.[65] In Chin et al.'s studies, cyclical skin stretching in mice models resulted in a significant increase in the expression of neuropeptides and growth factors, whereas the corresponding peptide receptors were downregulated, suggesting a negative feedback loop.[63,67] However, as mentioned above, there are no studies examining passive stretching protocols in humans that have measured the effect of these interventions on scar characteristics; therefore, further research is required.

A summary of the evidence that supports or refutes the use of the common conservative treatment interventions for scar management following burn injury described above is provided in Table 1.

CONCLUSION

This review provides a summary and clinical application of the evidence that supports or refutes the use of common conservative treatment interventions for scar management following burn injury. There is increasing evidence that one of the main mechanisms, which is the foundation for the success of current conservative treatments for the reduction of HSc, is the reduction of mechanical forces and neurogenic inflammation. These concepts are especially important to consider in burn survivor rehabilitation, since some conservative treatment interventions may imply an increase of mechanical forces and/or stimulation of the skin's sensory properties, which can lead to further scarring.[169] This review encourages burn therapists to understand the basic science rationale underlying the various therapy modalities they commonly use in order to judiciously select the optimal treatment interventions and contribute to the advancement of knowledge to substantiate practice. In summary, this review supports the following statements:

- Compression for edema reduction should be initiated 48–72 hours postinjury and continued for wounds that require longer than 21 days to heal until scar maturation.
- Elevation, pumping exercises, and retrograde massage/MEM should be used in combination with other edema management techniques.
- Custom-fabricated pressure garments should be applied once the edema is stabilized and adequate healing has occurred. Garments should be monitored on a regular basis to ensure that optional pressure, >15 mm Hg, is maintained, adding inserts when necessary. The wearing time should be >16 hours/day.
- Gels for postburn scar management should extend beyond the scar.
- Serial casting should be applied when contractures interfere with function.
- Forceful scar massage should be avoided early in the wound healing process or when the scar is inflamed or breaks down.
- Other treatment modalities should be prioritized over passive stretching for scar management.

REFERENCES

1. Crowe CS, Massenburg BB, Morrison SD, Naghavi M, Pham TN, Gibran NS. Trends of Burn Injury in the United States: 1990 to 2016. *Ann Surg.* 2019;270:944–953.
2. WHO. *The Global Burden of Disease: 2004 Update*. 2008; Available from: www.who.int/healthinfo/global_burden_disease/GBD_report_2004update_full.pdf.
3. IHME. *The Global Burden of Disease: 2010 Update*. 2012; Available from: viz.healthmetricsandevaluation.org/gbd-compare/.
4. Deitch E et al. Hypertrophic burn scars: analysis of variables. *J Trauma.* 1983;23:895–898.
5. Gangemi EN, Gregori D, Berchialla P et al. Epidemiology and risk factors for pathologic scarring after burn wounds. *Arch Facial Plast Surg.* 2008;10:93–102.
6. Lawrence JW, Mason ST, Schomer K, Klein MB. Epidemiology and impact of scarring after burn injury: a systematic review of the literature. *J Burn Care Res.* 2012;33:136–146.
7. Wallace HJ, Fear MW, Crowe MM, Martin LJ, Wood FM. Identification of factors predicting scar outcome after burn injury in children: a prospective case-control study. *Burns Trauma.* 2017;5:19.
8. Wallace HJ, Fear MW, Crowe MM, Martin LJ, Wood FM. Identification of factors predicting scar outcome after burn in adults: a prospective case–control study. *Burns.* 2017;43:1271–1283.
9. Chiang RS, Borovikova AA, King K et al. Current concepts related to hypertrophic scarring in burn injuries. *Wound Repair Regen.* 2016;24:466–477.
10. Edger-Lacoursière Z, Nedelec B, Marois-Pagé E et al. Systematic quantification of hypertrophic scar in adult burn survivors. *Eur Burn J.* 2021;2:88–105.
11. Finnerty CC, Jeschke MG, Branski LK, Barret JP, Dziewulski P, Herndon DN. Hypertrophic scarring: the greatest unmet challenge after burn injury. *Lancet (London, England).* 2016;388:1427–1436.
12. Goel A, Shrivastava P. Post-burn scars and scar contractures. *Indian J Plast Surg.* 2010;43:S63–S71.
13. Limandjaja GC, Niessen FB, Scheper RJ, Gibbs S. Hypertrophic scars and keloids: overview of the evidence and practical guide for differentiating between these abnormal scars. *Exp Dermatol.* 2021;30:146–161.
14. Bock O, Schmid-Ott G, Malewski P, Mrowietz U. Quality of life of patients with keloid and hypertrophic scarring. *Arch Dermatol Res.* 2006;297:433–438.
15. Simons M, Price N, Kimble R, Tyack Z. Patient experiences of burn scars in adults and children and development of a health-related quality of life conceptual model: a qualitative study. *Burns.* 2016;42:620–632.
16. Oosterwijk AM, Mouton LJ, Schouten H, Disseldorp LM, van der Schans CP, Nieuwenhuis MK. Prevalence of scar contractures after burn: a systematic review. *Burns.* 2017;43:41–49.
17. Ghaed Chukamei Z, Mobayen M, Bagheri Toolaroud P, Ghalandari M, Delavari S. The length of stay and cost of burn patients and the affecting factors. *Int J Burns Trauma.* 2021;11:397–405.
18. Edger-Lacoursière Z, Deziel E, Nedelec B. Rehabilitation interventions after hand burn injury in adults: a systematic review. *Burns.* 2022;49:516–553.
19. Mustoe TA. Evolution of silicone therapy and mechanism of action in scar management. *Aesthetic Plast Surg.* 2008;32:82–92.
20. Nedelec B et al. Practice guidelines for the application of nonsilicone or silicone gels and gel sheets after burn injury. *J Burn Care Res.* 2015;36:345–374.
21. Ai J-W, Liu J-T, Pei S-D et al. The effectiveness of pressure therapy (15–25 mmHg) for hypertrophic burn scars: a systematic review and meta-analysis. *Sci Rep.* 2017;7:40185.
22. Atiyeh BS, El Khatib AM, Dibo SA. Pressure garment therapy (PGT) of burn scars: evidence-based efficacy. *Ann Burns Fire Disast.* 2013;26:205–212.
23. Johnson J, Greenspan B, Gorga D, Nagler W, Goodwin C. Compliance with pressure garment use in burn rehabilitation. *J Burn Care Rehabil.* 1994;15:180–188.
24. Nedelec B, Yick K-L, Kwan M-Y, et al. Are custom fabricated pressure garments applying adequate pressure? *J Burn Care Res.* 2019;40:S65–S65.
25. Ault P, Plaza A, Paratz J. Scar massage for hypertrophic burns scarring—a systematic review. *Burns.* 2018;44:24–38.
26. Lin TR, Chou F-H, Wang H-H, Wang R-H. Effects of scar massage on burn scars: a systematic review and meta-analysis. *J Clin Nurs.* 2023;32:3144–3154.
27. Parry IS, Schneider JC, Yelvington M, et al Systematic review and expert consensus on the use of orthoses (splints and casts) with adults and children after burn injury to determine practice guidelines. *J Burn Care Res.* 2020;41:503–534.
28. Jang KU, Choi JS, Mun JH, Jeon JH, Seo CH, Kim JH. Multi-axis shoulder abduction splint in acute burn rehabilitation: a randomized controlled pilot trial. *Clin Rehabil.* 2015;29:439–446.

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29. Kolmus AM, Holland AE, Byrne MJ, Cleland HJ. The effects of splinting on shoulder function in adult burns. *Burns.* 2012;38:638–644.
30. Bennett GB, Helm P, Purdue GF, Hunt JL. Serial casting: a method for treating burn contractures. *J Burn Care Rehabil.* 1989;10:543–545.
31. Choi YM, Nederveld C, Campbell K, Moulton S. A soft casting technique for managing pediatric hand and foot burns. *J Burn Care Res.* 2018;39:760–765.
32. Johnson J, Silverberg R. Serial casting of the lower extremity to correct contractures during the acute phase of burn care. *Phys Ther.* 1995;75:262–266.
33. Ricks NR, Meagher DPJr. The benefits of plaster casting for lower-extremity burns after grafting in children. *J Burn Care Rehabil.* 1992;13:465–468.
34. Ridgway CL, Daugherty MB, Warden GD. Serial casting as a technique to correct burn scar contractures. A case report. *J Burn Care Rehabil.* 1991;12:67–72.
35. Dewey WS, Richard RL, Parry IS. Positioning, splinting, and contracture management. *Phys Med Rehabil Clin N Am.* 2011;22:229–247, v.
36. Godleski M, Oeffling A, Bruflat AK, Craig E, Weitzenkamp D, Lindberg G. Treating burn-associated joint contracture: results of an inpatient rehabilitation stretching protocol. *J Burn Care Res.* 2013;34:420–426.
37. Huang TT, Blackwell SJ, Lewis SR. Ten years of experience in managing patients with burn contractures of axilla, elbow, wrist, and knee joints. *Plast Reconstr Surg.* 1978;61:70–76.
38. Richard R, Miller S, Staley M, Johnson RM. Multimodal versus progressive treatment techniques to correct burn scar contractures. *J Burn Care Rehabil.* 2000;21:506–512.
39. Mulder PPG, Vlig M, Boekema BKHL et al. Persistent systemic inflammation in patients with severe burn injury is accompanied by influx of immature neutrophils and shifts in T cell subsets and cytokine profiles. *Front Immunol.* 2020;11:621222.
40. Zhu M, Edger-Lacoursière Z, Marois-Pagé E, Nedelec B. Acute care strategies to reduce burn scarring. *Burns Open.* 2023;7:159–173.
41. Cubison TC, Pape SA, Parkhouse N. Evidence for the link between healing time and the development of hypertrophic scars (HTS) in paediatric burns due to scald injury. *Burns.* 2006;32:992–999.
42. Hassan S, Reynolds G, Clarkson J, Brooks P. Challenging the dogma: relationship between time to healing and formation of hypertrophic scars after burn injuries. *J Burn Care Res.* 2014;35:e118–e124.
43. Strassle PD, Williams FN, Weber DJ et al. Risk factors for healthcare-associated infections in adult burn patients. *Infect Control Hosp Epidemiol.* 2017;38:1441–1448.
44. Wong VW, Levi K, Akaishi S, Schultz G, Dauskardt RH. Scar zones: region-specific differences in skin tension may determine incisional scar formation. *Plast Reconstr Surg.* 2012;129:1272–1276.
45. Lee SY, Park J. Postoperative electron beam radiotherapy for keloids: treatment outcome and factors associated with occurrence and recurrence. *Ann Dermatol.* 2015;27:53–58.
46. Yin J, Zhang S, Yang C et al. Mechanotransduction in skin wound healing and scar formation: potential therapeutic targets for controlling hypertrophic scarring. *Front Immunol.* 2022;13:1028410.
47. Powell HM, Nedelec B. Mechanomodulation of burn scarring via pressure therapy. *Adv Wound Care.* 2021;11:179–191.
48. Varani J, Dame MK, Rittie L et al. Decreased collagen production in chronologically aged skin: roles of age-dependent alteration in fibroblast function and defective mechanical stimulation. *Am J Pathol.* 2006;168:1861–1868.
49. Wu M, Fannin J, Rice KM, Wang B, Blough ER. Effect of aging on cellular mechanotransduction. *Ageing Res Rev.* 2011;10:1–15.
50. Kant S, van den Kerckhove E, Colla C, van der Hulst R, Piatkowski de Grzymala A. Duration of scar maturation: retrospective analyses of 361 hypertrophic scars over 5 years. *Adv Skin Wound Care.* 2019;32:26–34.
51. Shutova MS, Boehncke W-H. Mechanotransduction in Skin Inflammation. *Cells.* 2022;11:2026.
52. Kuehlmann B, Bonham CA, Zucal I, Prantl L, Gurtner GC. Mechanotransduction in wound healing and fibrosis. *J Clin Med.* 2020;9:1423.
53. Tschumperlin DJ, Ligresti G, Hilscher MB, Shah VH. Mechanosensing and fibrosis. *J Clin Invest.* 2018;128:74–84.
54. Hosseini M, Brown J, Khosrotehrani K, Bayat A, Shafiee A. Skin biomechanics: a potential therapeutic intervention target to reduce scarring. *Burns Trauma.* 2022;10:tkac036.
55. Wong VW, Akaishi S, Longaker MT, Gurtner GC. Pushing back: wound mechanotransduction in repair and regeneration. *J Invest Dermatol.* 2011;131:2186–2196.
56. Wong VW, Beasley B, Zepeda J et al. A Mechanomodulatory device to minimize incisional scar formation. *Adv Wound Care (New Rochelle).* 2013;2:185–194.
57. Fernandes MG, da Silva LP, Cerqueira MT et al. Mechanomodulatory biomaterials prospects in scar prevention and treatment. *Acta Biomater.* 2022;150:22–33.
58. Aarabi S, Bhatt KA, Shi Y et al. Mechanical load initiates hypertrophic scar formation through decreased cellular apoptosis. *FASEB J.* 2007;21:3250–3261.
59. Gurtner GC, Dauskardt RH, Wong VW et al. Improving cutaneous scar formation by controlling the mechanical environment: large animal and phase I studies. *Ann Surg.* 2011;254:217–225.
60. Atkinson JA, McKenna KT, Barnett AG, McGrath DJ, Rudd M. A randomized, controlled trial to determine the efficacy of paper tape in preventing hypertrophic scar formation in surgical incisions that traverse Langer's skin tension lines. *Plast Reconstr Surg.* 2005;116:1648–56; discussion 1657.
61. Longaker MT, Rohrich RJ, Greenberg L et al. A randomized controlled trial of the embrace advanced scar therapy device to reduce incisional scar formation. *Plast Reconstr Surg.* 2014;134:536–546.
62. Lim AF, Weintraub J, Kaplan EN et al. The embrace device significantly decreases scarring following scar revision surgery in a randomized controlled trial. *Plast Reconstr Surg.* 2014;133:398–405.
63. Chin MS, Lancerotto L, Helm DL et al. Analysis of neuropeptides in stretched skin. *Plast Reconstr Surg.* 2009;124:102–113.
64. Webb K, Hitchcock RW, Smeal RM, Li W, Gray SD, Tresco PA. Cyclic strain increases fibroblast proliferation, matrix accumulation, and elastic modulus of fibroblast-seeded polyurethane constructs. *J Biomech.* 2006;39:1136–1144.
65. Huang C, Miyazaki K, Akaishi S, Watanabe A, Hyakusoku H, Ogawa R. Biological effects of cellular stretch on human dermal fibroblasts. *J Plast Reconstr Aesthet Surg.* 2013;66:e351–e361.
66. He J, Fang B, Shan S et al. Mechanical stretch promotes hypertrophic scar formation through mechanically activated cation channel Piezo1. *Cell Death Dis.* 2021;12:226.
67. Chin MS, Ogawa R, Lancerotto L et al. In vivo acceleration of skin growth using a servo-controlled stretching device. *Tissue Eng Part C Methods.* 2010;16:397–405.
68. Akaishi S, Ogawa R, Hyakusoku H. Keloid and hypertrophic scar: neurogenic inflammation hypotheses. *Med Hypotheses.* 2008;71:32–38.
69. Zegarska B, Lelińska A, Tyrakowski T. Clinical and experimental aspects of cutaneous neurogenic inflammation. *Pharmacol Rep.* 2006;58:13–21.
70. Yagmur C, Akaishi S, Ogawa R, Guneren E. Mechanical receptor-related mechanisms in scar management: a review and hypothesis. *Plast Reconstr Surg.* 2010;126:426–434.
71. Mauck MC, Shupp JW, Williams F et al. Hypertrophic scar severity at autograft sites is associated with increased pain and itch after major thermal burn injury. *J Burn Care Res.* 2018;39:536–544.
72. Scott JR, Muangman P, Gibran NS. Making sense of hypertrophic scar: a role for nerves. *Wound Repair Regen.* 2007;15:S27–S31.
73. Scott JR, Muangman PR, Tamura RN et al. Substance P levels and neutral endopeptidase activity in acute burn wounds and hypertrophic scar. *Plast Reconstr Surg.* 2005;115:1095–1102.
74. Ogawa R. Keloid and hypertrophic scarring may result from a mechanoreceptor or mechanosensitive nociceptor disorder. *Med Hypotheses.* 2008;71:493–500.
75. Cassuto J, Tarnow P, Yregård L, Lindblom L, Räntfors J. Adrenoceptor subtypes in the control of burn-induced plasma extravasation. *Burns.* 2005;31:123–129.
76. Lund T, Onarheim H, Reed RK. Pathogenesis of edema formation in burn injuries. *World J Surg.* 1992;16:2–9.
77. Edgar DW, Fish JS, Gomez M, Wood FM. Local and systemic treatments for acute edema after burn injury: a systematic review of the literature. *J Burn Care Res.* 2011;32:334–347.
78. Mascharak S, desJardins-Park HE, Davitt MF et al. Preventing engrailed-1 activation in fibroblasts yields wound regeneration without scarring. *Science.* 2021;372:eaba2374.
79. Edwick DO, Hince DA, Rawlins JM, Wood FM, Edgar DW. Randomized controlled trial of compression interventions for managing hand burn edema, as measured by bioimpedance spectroscopy. *J Burn Care Res.* 2020;41:992–999.
80. Park WY, Jung SJ, Joo SY, Jang KU, Seo CH, Jun AY. Effects of a modified hand compression bandage for treatment of post-burn hand edemas. *Ann Rehabil Med.* 2016;40:341–350.
81. Sharp PA, Pan B, Yakuboff KP, Rothchild D. Development of a best evidence statement for the use of pressure therapy for management of hypertrophic scarring. *J Burn Care Res.* 2016;37:255–264.
82. Lowell M, Pirc P, Ward RS et al. Effect of 3M Coban self-adherent wraps on edema and function of the burned hand: a case study. *J Burn Care Rehabil.* 2003;24:253–8; discussion 252.
83. Ward RS, Reddy R, Brockway C, Hayes-Lundy C, Mills P. Uses of Coban self-adherent wrap in management of postburn hand grafts: case reports. *J Burn Care Rehabil.* 1994;15:364–369.
84. Boland RA, Adams RD. The effects of arm elevation and overnight head-up tilt on forearm and hand volume. *J Hand Ther.* 1998;11:180–190.
85. Villeco JP. Edema: a silent but important factor. *J Hand Ther.* 2012;25:153–61; quiz 162.
86. Omar MT, El-Badawy AM, Borhan WH, Nossier AA. Improvement of edema and hand function in superficial second degree hand burn using electrical stimulation. *Egypt J Plast Reconstr Surg.* 2005;28:141–147.

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87. Miller LK, Jerosch-Herold C, Shepstone L. How should interventions to treat hand oedema be delivered? An online Delphi Consensus Method. *Hand Ther*. 2022;27:58–66.
88. Goddard AA, Pierce CS, McLeod KJ. Reversal of lower limb edema by calf muscle pump stimulation. *J Cardiopulm Rehabil Prev*. 2008;28:174–179.
89. Miller LK, Jerosch-Herold C, Shepstone L. Effectiveness of edema management techniques for subacute hand edema: a systematic review. *J Hand Ther*. 2017;30:432–446.
90. Vena D, Rubianto J, Popovic MR, Fernie GR, Yadollahi A. The effect of electrical stimulation of the calf muscle on leg fluid accumulation over a long period of sitting. *Sci Rep*. 2017;7:6055.
91. Flores O, Tyack Z, Stockton K, Ware R, Paratz JD. Exercise training for improving outcomes post-burns: a systematic review and meta-analysis. *Clin Rehabil*. 2018;32:734–746.
92. Flores O, Tyack Z, Stockton K, Paratz JD. The use of exercise in burns rehabilitation: a worldwide survey of practice. *Burns*. 2020;46:322–332.
93. Gutiérrez-Espinoza H, Araya-Quintanilla F, Olguín-Huerta C, Valenzuela-Fuenzalida J, Gutiérrez-Monclus R, Moncada-Ramírez V. Effectiveness of manual therapy in patients with distal radius fracture: a systematic review and meta-analysis. *J Man Manip Ther*. 2022;30:33–45.
94. Patel H, Skok C, DeMarco A. Peripheral edema: evaluation and management in primary care. *Am Fam Physician*. 2022;106:557–564.
95. Shupp JW, Nasabzadeh TJ, Rosenthal DS, Jordan MH, Fidler P, Jeng JC. A review of the local pathophysiologic bases of burn wound progression. *J Burn Care Res*. 2010;31:849–873.
96. El Ayadi A, Jay JW, Prasai A. Current approaches targeting the wound healing phases to attenuate fibrosis and scarring. *Int J Mol Sci*. 2020;21:1105.
97. Schwager S, Detmar M. Inflammation and lymphatic function. *Front Immunol*. 2019;10:308.
98. DeBruler DM, Baumann ME, Blackstone BN et al. Role of early application of pressure garments following burn injury and autografting. *Plast Reconstr Surg*. 2019;143:310e–321e.
99. Puzey G. The use, of pressure garments on hypertrophic scars. *J Tissue Viability*. 2002;12:11–15.
100. Wei Y, Wang Y, Zhang M et al. The application of 3D-printed transparent facemask for facial scar management and its biomechanical rationale. *Burns*. 2018;44:453–461.
101. Carney BC, Bailey JK, Powell HM, Supp DM, Travis TE. Scar management and dyschromia: a summary report from the 2021 American Burn Association State of the Science Meeting. *J Burn Care Res*. 2023;44:535–545.
102. Kim JY, Willard JJ, Supp DM et al. Burn scar biomechanics after pressure garment therapy. *Plast Reconstr Surg*. 2015;136:572–581.
103. Van den Kerckhove E, Stappaerts K, Fieuws S et al. The assessment of erythema and thickness on burn related scars during pressure garment therapy as a preventive measure for hypertrophic scarring. *Burns*. 2005;31:696–702.
104. Wei Y, Li-Tsang CWP, Liu J, Xie L, Yue S. 3D-printed transparent facemasks in the treatment of facial hypertrophic scars of young children with burns. *Burns*. 2017;43:e19–e26.
105. Li-Tsang CW, Feng B, Huang L et al. A histological study on the effect of pressure therapy on the activities of myofibroblasts and keratinocytes in hypertrophic scar tissues after burn. *Burns*. 2015;41:1008–1016.
106. Renò F, Grazianetti P, Stella M, Magliacani G, Pezzuto C, Cannas M. Release and activation of matrix metalloproteinase-9 during in vitro mechanical compression in hypertrophic scars. *Arch Dermatol*. 2002;138:475–478.
107. Renò F, Sabbatini M, Lombardi F et al. In vitro mechanical compression induces apoptosis and regulates cytokines release in hypertrophic scars. *Wound Repair Regen*. 2003;11:331–336.
108. Harris IM, Lee KC, Deeks JJ, Moore DJ, Moiemen NS, Dretzke J. Pressure-garment therapy for preventing hypertrophic scarring after burn injury. *Cochrane Database Syst Rev*. 2024;1:CD013530.
109. DeBruler DM, Baumann ME, Zbinden JC et al. Improved Scar Outcomes with Increased Daily Duration of Pressure Garment Therapy. *Adv Wound Care (New Rochelle)*. 2020;9:453–461.
110. DeBruler DM, Zbinden JC, Baumann ME et al. Early cessation of pressure garment therapy results in scar contraction and thickening. *PLoS One*. 2018;13:e0197558.
111. Crofton E, Meredith P, Gray P, O'Reilly S, Strong J. Non-adherence with compression garment wear in adult burns patients: a systematic review and meta-ethnography. *Burns*. 2020;46:472–482.
112. Coghlan N, Copley J, Aplin T, Strong J. Patient experience of wearing compression garments post burn injury: a review of the literature. *J Burn Care Res*. 2017;38:260–269.
113. Killey J, Simons M, Tyack Z. Effectiveness of interventions for optimising adherence to treatments for the prevention and management of scars: a systematic review. *Clin Rehabil*. 2021;35:656–668.
114. Alkhalil A, Tejiram S, Travis TE et al. A translational animal model for scar compression therapy using an automated pressure delivery system. *Eplasty*. 2015;15:e29.
115. Quan PE, Rau SB, Alston DW, Curreri PW. Control of scar tissue in the finger web spaces by use of graded pressure inserts. *J Burn Care Rehabil*. 1980;1:27–29.
116. Yelvington M, Brown S, Castro MM, Nick TG. The use of neoprene as a scar management modality. *Burns*. 2013;39:866–875.
117. Ward RS, Reddy R, Lundy CH, Brockway C, Saffle JR, Schnebly WA. A technique for control of hypertrophic scarring in the central region of the face. *J Burn Care Rehabil*. 1991;12:263–267.
118. Hwang YF, Chen CL, Chen-Sea MJ. Effectiveness of web space pressure inserts for postburn dorsal slant. *J Burn Care Res*. 2008;29:768–772.
119. Alston DW, Kozerefski P, Quan PE, Luterman A. Materials for pressure inserts in the control of hypertrophic scar tissue. *J Burn Care Rehabil*. 1981;2:40–43.
120. Malick MH, Carr JA. Flexible elastomer molds in burn scar control. *Am J Occup Ther*. 1980;34:603–608.
121. Tanaydin V, Beugels J, Piatkowski A et al. Efficacy of custom-made pressure clips for ear keloid treatment after surgical excision. *J Plast Reconstr Aesthet Surg*. 2016;69:115–121.
122. Rivers EA, Strate RG, Solem LD. The transparent face mask. *Am J Occup Ther*. 1979;33:108–113.
123. Fraulin FO, Illmayer SJ, Tredget EE. Assessment of cosmetic and functional results of conservative versus surgical management of facial burns. *J Burn Care Rehabil*. 1996;17:19–29.
124. Groce A, Meyers-Paal R, Herndon DN, McCauley RL. Are your thoughts of facial pressure transparent? *J Burn Care Rehabil*. 1999;20:478–481.
125. Parry I, Sen S, Palmieri T, Greenhalgh D. Nonsurgical scar management of the face: does early versus late intervention affect outcome? *J Burn Care Res*. 2013;34:569–575.
126. Kant SB, Colla C, Van den Kerckhove E, Van der Hulst RRWJ, Piatkowski de Grzymala A. Satisfaction with facial appearance and quality of life after treatment of face scars with a transparent facial pressure mask. *Facial Plast Surg*. 2018;34:394–399.
127. Hwang SJ, Seo J, Cha JY et al. Utility of customized 3D compression mask with pressure sensors on facial burn scars: a single-blinded, randomized controlled trial. *Burns*. 2024;50:1885–1897.
128. Akaishi S, Akimoto M, Hyakusoku H, Ogawa R. The tensile reduction effects of silicone gel sheeting. *Plast Reconstr Surg*. 2010;126:109e–111e.
129. Karagoz H, Yuksel F, Ulkur E, Evinc R. Comparison of efficacy of silicone gel, silicone gel sheeting, and topical onion extract including heparin and allantoin for the treatment of postburn hypertrophic scars. *Burns*. 2009;35:1097–1103.
130. Momeni M, Hafezi F, Rahbar H, Karimi H. Effects of silicone gel on burn scars. *Burns*. 2009;35:70–74.
131. van der Wal MBA, van Zuijlen PP, van de Ven P, Middelkoop E. Topical silicone gel versus placebo in promoting the maturation of burn scars: a randomized controlled trial. *Plast Reconstr Surg*. 2010;126:524–531.
132. Musgrave MA, Umraw N, Fish JS, Gomez M, Cartotto RC. The effect of silicone gel sheets on perfusion of hypertrophic burn scars. *J Burn Care Rehabil*. 2002;23:208–214.
133. Tredget EE, Nedelec B, Scott PG, Ghahary A. Hypertrophic scars, keloid, and contractures: the cellular and molecular basis for therapy. *Surg Clin North Am*. 1997;77:701–730.
134. Borgognoni L. Biological effects of silicone gel sheeting. *Wound Repair Regen*. 2002;10:118–121.
135. Lee SM, Ngim CK, Chan YY, Ho MJ. A comparison of Sil-K and Epiderm in scar management. *Burns*. 1996;22:483–487.
136. Bleasdale B, Finnegan S, Murray K, Kelly S, Percival SL. The use of silicone adhesives for scar reduction. *Adv Wound Care (New Rochelle)*. 2015;4:422–430.
137. Nikkonen MM, Pitkanen JM, Al-Qattan MM. Problems associated with the use of silicone gel sheeting for hypertrophic scars in the hot climate of Saudi Arabia. *Burns*. 2001;27:498–501.
138. De Decker I, Hoeksema H, Vanlerberghe E et al. Occlusion and hydration of scars: moisturizers versus silicone gels. *Burns*. 2023;49:365–379.
139. Carney SA, Cason CG, Gowar JP et al. Cica-Care gel sheeting in the management of hypertrophic scarring. *Burns*. 1994;20:163–167.
140. Li-Tsang CW, Zheng YP, Lau JC. A randomized clinical trial to study the effect of silicone gel dressing and pressure therapy on posttraumatic hypertrophic scars. *J Burn Care Res*. 2010;31:448–457.
141. Pruksapong C, Burusapat C, Hongkarnjanakul N. Efficacy of silicone gel versus silicone gel sheet in hypertrophic scar prevention of deep hand burn patients with skin graft: a prospective randomized controlled trial and systematic review. *Plast Reconstr Surg Glob Open*. 2020;8:e3190.
142. Wiseman J, Simons M, Kimble R, Ware RS, McPhail SM, Tyack Z. Effectiveness of topical silicone gel and pressure garment therapy for burn scar prevention and management in children 12-months postburn: a parallel group randomised controlled trial. *Clin Rehabil*. 2021;35:1126–1141.
143. Staley M, Serghiou M. Casting guidelines, tips, and techniques: proceedings from the 1997 American Burn Association PT/OT Casting Workshop. *J Burn Care Rehabil*. 1998;19:254–60; discussion 253.

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144. Richard R, Baryza MJ, Carr JA et al. Burn rehabilitation and research: proceedings of a consensus summit. *J Burn Care Res.* 2009;30:543–573.
145. Thomas R, Wicks S, Dale M, Pacey V. Outcomes of early and intensive use of a palm and digit extension orthosis in young children after burn injury. *J Burn Care Res.* 2021;42:245–257.
146. Thomas R, Wicks S, Toose C, Pacey V. Outcomes of early use of an end of range axilla orthotic in children following burn injury. *J Burn Care Res.* 2019;40:678–688.
147. Schetzsle S, Lin WWC, Purushothaman P, Ding J, Kwan P, Tredget EE. Serial casting as an effective method for burn scar contracture rehabilitation: a case series. *J Burn Care Res.* 2023;44:1062–1072.
148. Fess EE, McCollum M. The influence of splinting on healing tissues. *J Hand Ther.* 1998;11:157–161.
149. Bell-Krotoski JA, Figarola JH. Biomechanics of soft-tissue growth and remodeling with plaster casting. *J Hand Ther.* 1995;8:131–137.
150. Tappan FM, Benjamin PJ. *Tappan's Handbook of Healing Massage Techniques: Classic, Holistic, and Emerging Methods.* Upper Saddle River, NJ: Pearson/Prentice Hall; 2005.
151. Klotz T, Kurmis R, Munn Z, Heath K, Greenwood J. Moisturisers in scar management following burn: a survey report. *Burns.* 2017;43:965–972.
152. Ogawa R. The most current algorithms for the treatment and prevention of hypertrophic scars and keloids: a 2020 update of the algorithms published 10 years ago. *Plast Reconstr Surg.* 2022;149:79e–94e.
153. Nedelec B, Edger-Lacoursière Z, Gauthier N, Marois-Pagé E, Jean S. Randomized, controlled, within-patient, single-blinded pilot study to evaluate the efficacy of 12-weeks of endermotherapy with adult burn survivors. *Burns.* 7269;2024:107269.
154. Nedelec B, Couture M-A, Calva V et al. Randomized controlled trial of the immediate and long-term effect of massage on adult postburn scar. *Burns.* 2019;45:128–139.
155. Holavanahalli RK, Helm PA, Parry IS, Dolezal CA, Greenhalgh DG. Select practices in management and rehabilitation of burns: a survey report. *J Burn Care Res.* 2011;32:210–223.
156. Liuzzi F, Chadwick S, Shah M. Paediatric post-burn scar management in the UK: a national survey. *Burns.* 2015;41:252–256.
157. Silverberg R, Johnson J, Moffat M. The effects of soft tissue mobilization on the immature burn scar: results of a pilot study. *J Burn Care Rehabil.* 1996;17:252–259.
158. Patiño O, Novick C, Merlo A, Benaim F. Massage in hypertrophic scars. *J Burn Care Rehabil.* 1999;20:268–71; discussion 267.
159. Sullivan T, Smith J, Kermode J, McIver E, Courtemanche DJ. Rating the burn scar. *J Burn Care Rehabil.* 1990;11:256–260.
160. Lee KC, Dretzke J, Grover L, Logan A, Moiemen N. A systematic review of objective burn scar measurements. *Burns Trauma.* 2016;4:14.
161. Roh YS, Cho H, Oh JO, Yoon CJ. Effects of skin rehabilitation massage therapy on pruritus, skin status, and depression in burn survivors. *Taehan Kanho Hakhoe Chi.* 2007;37:221–226.
162. Roh YS, Seo CH, Jang KU. Effects of a skin rehabilitation nursing program on skin status, depression, and burn-specific health in burn survivors. *Rehabil Nurs.* 2010;35:65–69.
163. Cho YS, Jeon JH, Hong A et al. The effect of burn rehabilitation massage therapy on hypertrophic scar after burn: a randomized controlled trial. *Burns.* 2014;40:1513–1520.
164. Bush JA, McGrouther DA, Young VL et al. Recommendations on clinical proof of efficacy for potential scar prevention and reduction therapies. *Wound Repair Regen.* 2011;19:s32–s37.
165. Scott PG, Dodd CM, Tredget EE, Ghahary A, Rahemtulla F. Chemical characterization and quantification of proteoglycans in human post-burn hypertrophic and mature scars. *Clin Sci (Lond).* 1996;90:417–425.
166. Valladares-Poveda S, Avendaño-Leal O, Castillo-Hidalgo H, Murillo E, Palma C, Parry I. A comparison of two scar massage protocols in pediatric burn survivors. *Burns.* 2020;46:1867–1874.
167. Holavanahalli RK, Helm PA, Kowalske KJ, Hynan LS. Effectiveness of paraffin and sustained stretch in treatment of shoulder contractures following a burn injury. *Arch Phys Med Rehabil.* 2020;101:S42–S49.
168. Harvey LA, Katalinic OM, Herbert RD, Moseley AM, Lannin NA, Schurr K. Stretch for the treatment and prevention of contracture: an abridged republication of a Cochrane Systematic Review. *J Physiother.* 2017;63:67–75.
169. Singer AJ. Healing mechanisms in cutaneous wounds: tipping the balance. *Tissue Eng Part B Rev.* 2022;28:1151–1167.
170. Junker JP, Kratz C, Tollbäck A, Kratz G. Mechanical tension stimulates the transdifferentiation of fibroblasts into myofibroblasts in human burn scars. *Burns.* 2008;34:942–946.
171. Barnes LA, Marshall CD, Leavitt T et al. Mechanical forces in cutaneous wound healing: emerging therapies to minimize scar formation. *Adv Wound Care (New Rochelle).* 2018;7:47–56.
172. Parry IS, Schneider JC, Yelvington M et al. Systematic review and expert consensus on the use of orthoses (splints and casts) with adults and children after burn injury to determine practice guidelines. *J Burn Care Res.* 2020;41:503–534.

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