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Mobilizing the Stiff Hand: Combining Theory and Evidence to Improve Clinical Outcomes

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ABSTRACT: The purpose of this narrative review is to provide a clinically reasonable guide to intervention choices, by combining a sound understanding of theory with available research evidence. The pathology of contracture formation is presented within the context of tissue repair. The soft tissue response to stress is explained and the optimal "dose" of treatment is discussed. The evidence behind the use of exercise, joint mobilization, continuous passive motion, casting motion to mobilize stiffness, and mobilizing splinting is examined. Recommendations regarding treatment implementation and future research needs are highlighted. The importance of mobilizing splinting and exercise as treatment modalities in the management of joint contracture is demonstrated.

Level of Evidence: 5.

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"The joints of the hand are the means by which the power of the muscles moving the bones of the hand bring about useful function. When these joints are stiffened by fibrosis, destroyed by disease, or deformed by dislocation or fracture, the function of the hand is impaired or even destroyed. This alteration in function may occur despite all our efforts; however, it more frequently occurs because of improper methods of treatment" (p. 1129).[1]

According to Innis et al.[1] poor clinical management after hand injury contributes to further hand dysfunction, joint stiffness, and joint contracture. As these authors suggest, the implementation of inappropriate treatment techniques in a particular situation can result in suboptimal effects on patient outcomes. The purpose of this narrative review is to facilitate effective clinical reasoning in the management of joint contracture in the hand, as the result of the integration of an understanding of biological theory with an evaluation of available research evidence. The pathology of contracture formation is described first in conjunction with the process of tissue repair.

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PATHOLOGY OF CONTRACTURE FORMATION

Joint contracture is a common sequelae of traumatic hand injury.[2–5] Immobilization in the context of tissue repair after trauma predisposes the hand to contracture formation. After injury, tissue repair follows three distinct phases: the acute inflammatory response, fibroplasia (collagen deposition and scar formation), and maturation or remodeling.[6–8] A period of enforced immobilization in a splint or cast may be required immediately after injury to rest and protect involved structures. Once gentle motion may be commenced, pain and edema experienced by the patient as part of the inflammatory response may prohibit the involved joint from being moved through full normal range of motion (ROM). The patient will be reluctant to move a joint that is painful, in the manner in which that joint is accustomed. Likewise, even if a joint is not painful but remains slightly edematous, it will be unable to move throughout its normal full ROM. Brand et al.[9] use the analogy of a ship anchored at sea to explain the effect of edema on a joint. When the tide is low, the ship is able to float across a much greater area on the surface of the ocean. When the tide is high, a much smaller ROM is possible.

At around four or five days after injury, fibroplasia commences and is characterized by a period of collagen formation that continues for two to four weeks.[7] Collagen is the major component of all soft tissues comprising approximately 77% of the

fat-free dry weight.[2] During fibroplasia, collagen fibers are deposited randomly into the wound matrix to form a rudimentary bridge of scar tissue across the wound gap. If continued immobilization is required during fibroplasia, detrimental changes within the joint commence. These include disorganization of cellular and fibrillar components of ligaments and the joint capsule, the development of adhesions between the folds of synovial lining, formation of fibrofatty connective tissue within the joint space, atrophy of cartilage and osteoporosis.[10–12] The rate of collagen synthesis and degradation is accelerated with the overall collagen mass reducing, resulting in weakening of soft tissues in and around the joint.[10]

Normal joint motion promotes collagen formation that is compatible with the functional requirements of that joint. Specifically, the forces that act across the joint during motion determine the quantity, alignment, length, and structural organization of collagen fibers. In the joint before injury, crosslinks are developed to help hold collagen fibers in place while allowing for maximum elongation (required to achieve full joint ROM). After injury, the combination of pain and edema prohibit normal joint motion, and new collagen deposited during fibroplasia is allowed to form in a shortened disorganized fashion with a greater number of interfibrillar crosslinks.[10] The meshwork of the extracellular matrix becomes progressively more disorganized, allowing key structures, such as the collateral ligaments, joint capsule, and volar plate, to shorten and tighten forming contracture.[12–14] Once this joint contracture is formed, unless adequate stimulus is provided to promote tissue lengthening, restriction in passive ROM (PROM) will continue into the final maturation stage of tissue healing. This stage commences at three to six weeks postinjury and involves remodeling of scar tissue into a more organized and stronger structure.[7,8,15] During this phase, collagen fibers deposited during fibroplasia are replaced and reorganized according to the tensile loads placed across them.[6] Once again, if the joint is not able to be moved through its normal ROM, new collagen will be laid down in a shortened position, and restrictions in ROM become more fixed as remodeling continues. This process of remodeling may continue for several years postinjury.[15–17] In summary, immobilization within the context of tissue repair after injury produces a number of pathological processes in the surrounding soft connective tissue, which result in joint contracture.

MOBILIZING THE HEALING HAND-TREATMENT DOSE AND THE SOFT TISSUE RESPONSE TO STRESS

Hand therapy has been described as ''behavioural modification of the fibroblast.''[18] Successful remodeling of healing tissue to form a functional scar occurs as a result of the application of an appropriate level of stress at the right time in the healing process.[18] An understanding of the soft tissue response to stress is required to allow the clinician to choose the most suitable ''dose'' of treatment required for a given situation.

The application of stress to soft tissues causes both viscous and elastic changes in the extracellular matrix that are responsible for increased ROM. Initially, the application of low levels of stress causes fluid in the extracellular matrix to begin to move away from the area under tension, allowing collagen fibers room to unfold. This uncoiling of collagen fibers is known as ''creep,'' and this initial phase of the soft tissue response to stress has been called ''adaption'' or ''unfolding.''[9,19,20]

As stress continues and more fluid is moved away from the joint, collagen fibers have further room within the extracellular matrix to shuffle and slide in relation to each other. This allows for greater overall elongation of the soft tissue and fiber realignment. Hence, phase 2 of the soft tissue response to stress is known as ''alignment.''[9,19,20] Once further elongation and sliding are no longer possible, collagen fibers will stiffen (known as phase three ''stiffening'') and, if stress continues, will eventually fail (phase 4, ''failure'').[9,20] Figure 1[21] provides a graphical representation of the four stages of the soft tissue response to stress.

To promote collagen growth and reorganization, stress needs to be applied ideally in phases 2 and 3 of the graph shown in Figure 1.[9] This results in the ''living response'' to stress with subsequent tissue lengthening as the joint adapts to its new functional demands.[9,22] The longer the joint is held at end range under adequate tension, the greater will be the gains in ROM.[23] Applying stress to the level of stage 4 will result in tissue failure. This restimulates the inflammatory response and subsequently increases the risk of further scar tissue production, increased stiffness, and increased joint contracture.[24]

The dose of treatment provided with any given therapy technique is the product of the applied level

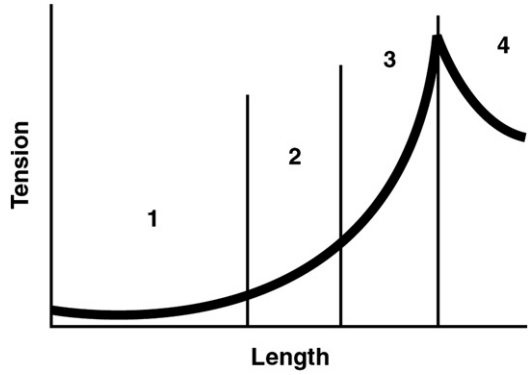

FIGURE 1. *The four phases of the soft tissue response to stress. 1 = unfolding, 2 = alignment, 3 = stiffening, 4 = failure. Adapted from Wilton.*[21]

of stress and the duration over which this is applied.[25] Treatments that apply high loads over short periods of time include techniques, such as joint manipulation and passive mobilization. They are known as "high load brief stress" (HLBS) techniques.[25,26] Conversely, techniques that apply a low force to mobilize a stiff joint over long periods of time are called "low load prolonged stress" (LLPS) techniques.[25,26] LLPS techniques include splinting and casting. The term "stress" is used rather than "stretch" to indicate that tissues are not being lengthened beyond their elastic limit.

Both techniques of long and short duration can provide a large "dose" of treatment to healing tissues. For example, HLBS techniques (e.g., joint mobilization, passive exercise) may provide a large dose of treatment, even though they are applied over very short periods of time (generally several seconds at a time). The higher force involved with the application of HLBS techniques increases the treatment dose. LLPS techniques, such as splinting and casting, in contrast, use a lower level of force, yet, apply this over longer periods of time (e.g., over many hours). The dose of treatment is increased by the longer duration of application.

COMBINING THEORY AND RESEARCH EVIDENCE

Effective clinical reasoning in the management of joint contracture requires the integration of an understanding of theoretical concepts with research evidence. Treatment options to be examined include exercise, joint mobilization, continuous passive motion (CPM), casting motion to mobilize stiffness (CMMS), and mobilizing splinting. A summary of the evidence for the various treatments is provided in Table 1, along with recommendations for clinical implementation and future research.

Exercise—Active, Assisted, and Passive Range of Motion

Active ROM (AROM) has been defined as the arc of motion through which a joint can be moved as a result of the patient using his or her own muscle power.[27] In assisted ROM, the patient moves his or her joint through a part of the available ROM using an isotonic muscle contraction, and the movement is then gently completed with the assistance of the therapist or by the patient using his or her unaffected hand.[28] Active and assisted motion generally provide a lower dose of treatment than PROM and, as such, may often be commenced in the inflammatory and early fibroblastic stages of tissue repair, after a period of rest and provided that injured structures are stable postsurgery.[25,29]

TABLE 1. **Combining Evidence and Theory—Recommendations for Clinical Implementation and Further Research**

Technique	*Stage of Tissue Healing*	*Treatment Dose*	*Evidence*	*Conclusions*
Active/assisted Exercise	Inflammatory, fibroplastic, and remodeling	Low dose	Single level 4 case series study (MCP and PIP joints)	Active exercise has a positive effect in the hand, but further higher level evidence is needed
Passive exercise	Fibroblastic and remodeling	High dose	Three studies, all relating to shoulder (level 2b, 3, and 4)	Passive exercise has a positive in the shoulder. Hand-specific research needed
Joint mobilization	Remodeling	High dose	Six studies (level 2b–4). Three studies relating to shoulder, 2 relating to wrist, and 1 relating to hand	Clinical merit of technique in the hand unclear. Further hand-specific research needed
CPM	Inflammatory	Moderate dose depending on length of time in situ	Six studies (2b–4). One study relating to shoulder, 1 study relating to elbow, and 4 relating to hand	Lack of evidence to support use of CPM in the management of established joint contracture in the hand
Casting motion to mobilize stiffness	Remodeling/chronic joint stiffness	Low to moderate dose according to frequency of motion repetition	Level 5 expert opinion. No research articles to date	Research needed to evaluate effectiveness
Mobilizing splinting	Fibroplastic and remodeling	Varying dose according to length of time splint used	Ten articles (2 level 2b, 8 level 4). Five articles relating to elbow, 2 relating to wrist, and 3 relating to hand	Highest level of evidence of all techniques

CPM = continuous passive motion.

Passive range of motion has been defined as the arc of motion through which a joint can be moved using external forces. This occurs when the therapist or the patient manually moves the joint of the affected hand to the end of pain-free ROM.[27] As PROM may provide a higher dose of treatment than AROM and assisted ROM, it is usually avoided in the inflammatory phase of tissue healing.[25,29]

Michlovitz et al.[30] published a systematic review examining research relating to therapeutic techniques for improving ROM in the upper limb. Articles published before June 2003 were included in the review. Each study was ranked using Sackett's[31] levels of evidence. These levels range from level 1a (high-quality systematic review of randomized controlled trials) to level 5 (expert opinion), with level 1a being the highest level of evidence and level 5 the lowest level of evidence.

Michlovitz et al.[30] identified four articles that focused specifically on the effectiveness of exercise in managing joint contracture. Three of these studies examined the effect of passive exercise to improve ROM in the shoulder, whereas the fourth study evaluated the effect of both AROM and PROM in improving ROM in the metacarpophalangeal (MCP) and proximal interphalangeal (PIP) joints of the hand.[30] Two of the articles reviewed by Michlovitz et al.[30] were level 4 studies (one case report and one case series).[32,33] One article was a level 3 case series study,[34] and one article was a level 2b study (prospective cohort study).[35] Findings from each of the four studies demonstrated a positive effect with the use of exercise. Hence, Michlovitz et al.[30] concluded that, overall, there was moderate evidence to support the use of exercise in the management of joint contracture in the upper limb.

Joint Mobilization Techniques

Joint mobilization techniques combine the use of an HLBS (<30 sec) with manually applied traction and gliding of one joint surface in relation to another.[30] Joint mobilization techniques have a dual purpose to relieve pain and to improve motion in a stiff joint.[36–38] In the case of joint contracture, mobilization techniques are based on the hypothesis that faulty motion of one joint surface in relation to another will result in restricted ROM.[36] Hence, restoration of the normal joint kinematics is believed to improve joint contracture. Additionally, it has been suggested that, if the mobilization technique is performed in the end ranges of available motion, connective tissue will be stretched beyond its elastic limit allowing for plastic deformation. This plastic deformation occurs as the result of microfailure of collagen fibers. According to Threlkeld,[38] this low level of tissue damage is essential to restimulate the cycle of inflammation, tissue repair, and remodeling, required to achieve permanent tissue lengthening and subsequent increased ROM. The use of modalities, compression, and elevation is recommended to control the inflammatory response stimulated by the use of joint mobilization techniques.[38] This rationale for the use of joint mobilization is level 5 evidence, that is, "expert opinion."

Michlovitz et al.[30] have reviewed research relating to the use of joint mobilization techniques in the upper limb. Of the six articles examined, four were level 2b (prospective cohort studies), one was level 3b (individual case–control study), and another was level 4 (case series). Two of the level 2b articles supported the use of joint mobilization in the shoulder and the hand,[39,40] whereas the remaining two level 2b articles did not support the use of joint mobilization in the shoulder or wrist.[41,42] The two case series articles (levels 3b and 4)[43,44] were found to support the use of joint mobilization techniques in the wrist and shoulder.

The only available article relating directly to joint mobilization in the hand examines MCP joint mobilization post-metacarpophalangeal fracture.[39] This level 2b study by Randall et al.[39] was included in the Michlovitz et al.[30] systematic review. Participants in this study were immobilized postfracture in a cast for an average of four weeks in the "safe" position, with the MCP joints in 70–90 degrees of flexion. After removal of the cast, participants were randomly allocated to treatment or control group and attended three therapy sessions over a one-week period. The treatment group received a set program of MCP joint mobilization in addition to the standard treatment program. Progress over the week was evaluated in addition to progress within each of the sessions. Findings indicated that the improvements in joint excursion within session were greater in the treatment versus the control group. However, both groups were found to improve significantly across sessions.[39] This appears to indicate that the permanent gains made in ROM in both groups may have resulted from the overall treatment program rather than simply joint mobilization alone. It is possible that the cumulative stress delivered by the home exercise program, in particular, produced the permanent change in ROM observed across sessions. It is the amount of time spent under tension at the end of available ROM that will have the greatest impact on contracture resolution.[23]

The largest amount of improvement in joint excursion in the study by Randall et al.[39] was found with MCP extension because of the initial cast position having been in MCP flexion (70–90 degrees). However, MCP joints posttrauma tend to produce extension rather than flexion contractures.[45] This is the result of the involvement of the collateral ligaments and the tendency for MCP joints to rest in extension in the presence of edema because of pooling of fluid

over the extensor tendons on the dorsum of the hand.[9] Hence, although a moderate level of evidence may exist to justify the use of joint mobilization techniques in the upper limb generally,[30] it does not appear that the clinical merit of using joint mobilization techniques to improve motion specifically in joint contractures of the hand has been clearly demonstrated. Additionally, empirical evidence is needed to demonstrate that plastic deformation of connective tissue, resulting from microfailure of collagen fibers, increases PROM without causing further scar tissue production and subsequent increased joint contracture.

Continuous Passive Motion

The concept of CPM was developed by Salter et al.[46] in the 1970s in response to findings from earlier research that highlighted the detrimental effects of immobilization on synovial joints.[46,47] One of the most notable detrimental effects associated with immobilization was found to be cartilage degeneration. Cartilage is essential for healthy joint function and is known to have limited regenerative capacity.[46] Salter et al.[46] hypothesized that if intermittent mobilization is better for healthy and injured joints than immobilization, continuous motion may be still better to prevent cartilage degeneration and promote healthy joint function.[48] Because a joint's capacity for active motion is limited by skeletal muscle fatigue, continuous motion is passively applied by means of a machine.[46] Salter et al.,[46] throughout the 1970s and 1980s, demonstrated the positive effect of CPM in the prevention of cartilage degeneration as well as in facilitating cartilage regeneration through a sequence of case series in both animal and human subjects (level 4 evidence).[46] Additionally, CPM was found to promote faster healing of fractures and tendons.[46]

Continuous passive motion was designed for use in the early phase of tissue repair (inflammation) to prevent formation of joint contracture.[48] It is hypothesized that CPM prevents bleeding into the joint and pumps excess fluid away, reducing edema and subsequent fibrosis.[48] Research has indicated that after the first week of treatment, CPM is less effective.[46,48,49] The dose of treatment provided by a CPM machine is influenced by the duration of use, force applied by means of the machine, velocity, and the extent of ROM.[50] Hence, CPM may potentially provide a high dose of treatment, and it should be used with care when implemented in the inflammatory phase of tissue repair.[25]

Michlovitz et al.[30] examined recent evidence relating to the use of CPM in the upper limb and found two articles that met their criteria. One shoulder article was a level 2b study (prospective cohort), examining the use of CPM for four weeks post-rotator cuff repair.[30,51] No significant difference was found between the CPM versus the non-CPM group. A second level 3 article (case−control) assessed the use of CPM post-anterior release for elbow flexion contracture.[30,52] CPM was found to improve ROM into flexion but not extension.[30,52]

On further examination of the literature, several additional articles were identified relating to the use of CPM with the hand. These articles were not included in the Michlovitz et al.[30] review. Covey et al.[53] prospectively studied the use of CPM for 10 patients with bilateral second- and third-degree burns of the hand (level 2b prospective cohort). For each patient, one hand received the conventional hand therapy program, including active and passive exercise. The second hand received CPM as the primary treatment. No difference was identified between the control and CPM groups in ROM gained before discharge. Both groups took an average of nine days to regain full AROM in eight out of the 10 cases.[53] Data were analyzed descriptively because of low patient numbers in each group.

Ring et al.[54] examined the use of CPM after MCP joint arthroplasty and were unable to demonstrate a significant difference in digit ROM in the CPM versus the non-CPM group (level 2b study).[31] Likewise, Sampson et al.[55] found no difference between the CPM and the non-CPM group in postoperative rehabilitation for 25 patients with Dupuytrens disease (level 2b study).[31] Schwartz and Chafetz,[56] in a retrospective chart review post-digital tenolysis and capsulectomy (level 3b case−control), found no significant difference between CPM and non-CPM groups in change in total active motion or duration of rehabilitation.

Salter et al.[46] reported positive effects with the use of CPM in their case series in the inflammatory phase of tissue repair. Yet, there appears to be limited higher-level evidence to support the use of CPM in the management of established joint contracture in the hand. According to Michlovitz et al.,[30] ''there is a dearth of studies in the literature on this technique, which is time and cost expensive'' (p. 129).

Casting Motion to Mobilize Stiffness

Casting motion to mobilize stiffness is a technique developed and promoted by Colditz.[57] Originally designed for use in the remodeling phase of tissue repair with the chronically stiff hand, this technique advocates that persistent joint contracture may be reinforced by the presence of a dysfunctional active movement pattern and subsequent dysfunctional cortical programming.[57] Colditz[57] advocates that if a joint contracture has been present for a long period of time, the patient will no longer be able to flex and extend his or her digits in the usual way. As such, they will be forced to recruit muscles in a different

motor pattern to perform functional tasks. Colditz[57] states that, because of substitution with alternative muscles, cortical representation and cortical programming is altered. Colditz[57] advocates that CMMS may correct this problem.

Colditz's[29,57,58] CMMS technique involves identifying the predominant dysfunctional movement pattern and then casting the wrist and proximal joints of the hand in the position most likely to restore the desired motion. Once in the cast, patients are requested to perform flexion and extension exercises using their stiff joints frequently. They are also requested to use their hand as much as possible in activities of daily living with the cast in situ.[47] Colditz[29,57] bases her technique on the notion that digit flexion is always initiated by the flexor digitorum profundus (FDP) tendon at the interphalangeal (IP) joints and that movement at the MCP joints occurs later in the flexion cascade, once the IP joints are already flexed. Colditz[57] quotes the work of Long and Brown[59] and Arbunckle and McGrouther[60] in support of her theory of normal digit flexion. Evidence to support the use of CMMS in clinical practice is currently low at level 5 (expert opinion).[31] Research is needed to examine the effectiveness of this technique compared with other treatment modalities aimed at restoring ROM in the stiff hand (e.g., splinting).

Mobilizing Splinting

Mobilizing splinting has been described as the ''modality of choice'' for facilitating contracture resolution in the hand.[61,62] Mobilizing splints are used to apply LLPS to a contracted joint with the goal of improving PROM. A small force applied through the splint traction system is used to hold the joint at the end of available ROM, for long periods of time.[21,29] Mobilizing splinting is a suitable technique for use in the fibroplastic and maturation phases of tissue healing, once scar tissue formation has commenced and injured structures are considered stable (Table 1).[48] A useful guide to clinical reasoning in splint prescription and application has been provided by McClure et al.[63] These authors outline an algorithm that considers parameters of splint intensity, total end range time (TERT), pain, and progress with ROM.

Unlike joint mobilization, the focus of splinting is to apply stress below stage 4 of the soft tissue response to stress (i.e., before tissue failure). Splinting is based on the concept that an adequate level of stress, applied over long periods of time, will stimulate the connective tissue growth and reorganization needed to achieve permanent lengthening.[22,48] Advocates of splinting believe that plastic deformation of connective tissue achieved through microfailure of collagen fibers will restimulate the inflammatory response and ultimately produce more scar tissue and promote increased joint contracture.

There is considerable biological evidence based on animal models to support the use of splinting in restoring PROM to a contracted joint. Kottke et al.[14] conducted an early study of soft tissue response to stress and hypothesized that a greater number of intermolecular crosslinks exist between collagen fibers of contracted or immobile tissues, and that these crosslinks are a short distance apart. Alternatively, in mobile tissues placed under tension for adequate periods of time, loose areolar tissue is formed with fewer crosslinks and greater distances between them.

Other studies have provided further support for this theory. Akeson et al.[12] found a significant increase in the number of intermolecular crosslinks after nine weeks' immobilization in New Zealand rabbits. Arem and Madden[13] studied the effect of stress on healing wounds in rats. These authors used magnets and found that wounds that healed under tension produced an elongated scar with collagen fibers orientated in the direction of tension. Wounds that did not heal under tension produced a much shorter, thicker scar, with considerable disorganization of collagen fibers.

Michlovitz et al.[30] examined research evidence in human subjects and identified a substantial number of articles, all of which reported positive effects with splinting in the management of joint contracture. Eight of the nine articles identified were level 4 (prospective and retrospective case series or case reports) and one article was level 2b (randomized controlled trial). Five articles related to the elbow, two involved the wrist, and two involved the PIP joint. One PIP joint article was level 4,[64] whereas the other PIP article by Flowers and LaStayo[23] provided the highest level of evidence at level 2b.

A subsequent level 2b article was identified on our review of the literature. This article by Glasgow et al.[45] was not included in the Michlovitz et al.[30] review, as it was published after the review period (1966 to June 2003). Glasgow et al.[45] explored the effects of splinting in the MCP and PIP joints of the hand and found splinting to positively influence joint contracture.[45] The addition of this article by Glasgow et al.[45] brings the total of articles examining the effects of splinting on humans up to 10, all of which support its use in the management of joint contracture. Hence, on combining research findings from both animal and human studies, there is a strong body of evidence to support the use of splinting to mobilize the contracted joint in clinical practice.

DISCUSSION

The best method of managing joint contracture is to prevent it. Poor clinical management after hand

injury can contribute to further dysfunction and the formation of joint contracture. Hence, effective clinical reasoning is crucial to promote optimal functional outcomes in our patients. According to Brand and Hollister,[65] "Restoring the balance, beauty, and power to a damaged hand is an adventure. The stakes are high. The rewards are exciting. The penalties of failure are grievous … Somewhere out there are patients who do not use their hands and who hide them from view because of what we did or failed to do. We should not forget such patients. They should stand beside us while we plan treatment for others. They should look over our shoulders at surgery and be with us at therapy sessions, whispering reminders to be gentle and warnings to stop and think" (p. 247). Ultimately, the responsibility for improving clinical practice in the management of joint contracture is shared among hand therapists. We all have a role to play, in evaluating the effectiveness of our own intervention and in improving the current body of research evidence.

Our review of theory and research has highlighted a lack of evidence to support several of the techniques currently in use. For example, examining the biological evidence behind CPM indicates that it is most appropriately used in the first week after injury or surgery as a preventative measure, rather than as a treatment for established joint contracture. Research evidence relating to CPM use in the hand shows no advantage to the use of this technique as an addition to traditional therapy.

Likewise, the theory behind the use of joint mobilization to improve PROM in the stiff hand is controversial. Advocates of this technique state that the inflammatory process that is stimulated through microfailure of collagen fibers can be managed adequately through the use of modalities, compression, and elevation. This is to ensure that further scar tissue formation and increased joint contracture do not result. Research is needed to substantiate this theory. Advocates of splinting would suggest that applying stress to induce plastic deformation and microfailure will exacerbate the inflammatory response and result in fibrosis and increased joint contracture. The only clinical article dealing with joint mobilization to improve motion in the stiff hand focuses on regaining MCP joint extension after fracture. This article found a positive effect with the use of joint mobilization; yet, the practical application of the findings is limited. Clinically, MCP joints after injury are usually stiff into flexion and not extension.

No research data were identified on our examination of the literature that evaluated the use of CMMS in managing joint contracture. Research is needed to examine the effectiveness of this technique compared with more traditional interventions. The theory of cortical involvement in sustaining established joint contracture seems logical; however, it is yet to be validated empirically.

Compared with these other techniques, active or passive exercise demonstrated a moderately high level of evidence to justify their use in clinical practice. Despite this, three of the four studies identified examined the effectiveness of exercise in the shoulder rather than the hand. Further hand-specific studies focusing on the effectiveness of exercise in the management of joint contracture are warranted.

The greatest body of evidence relates to the use of mobilizing splinting in the treatment of joint contracture. We identified 10 research articles, all reporting positive effects of splinting. Additionally, findings from animal studies provide a theoretical basis for the mechanism by which splinting acts on contracted tissue to produce increased PROM. Thus, combining theory and research evidence from human and animal studies provides a strong case for the use of mobilizing splinting in the management of joint contracture.

SUMMARY AND CONCLUSIONS

Immobilization and inflammation combine to predispose the hand to contracture formation after trauma. Effective clinical reasoning in the management of joint contracture relies on careful consideration of both theory and research before treatment implementation. This article has reviewed research relating to techniques commonly used by therapists in the management of joint contracture, in conjunction with the theory of the pathology of contracture formation and the soft tissue response to stress. Treatment techniques examined include, active or passive exercise, CPM, joint mobilization, CMMS, and mobilizing splinting.

Combining theory with research findings indicates a high level of evidence for the use of mobilizing splinting in the management of joint contracture. A moderate level of evidence exists to support the use of active or passive exercise; however, further hand-specific research is needed. There is a low level of evidence to support CPM, joint mobilization, and CMMS in the treatment of joint contracture, and further research is needed to justify regular use in everyday clinical practice.

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#1. The thrust of this study is to
   a. present new data
   b. review evidence
   c. present the traditional wisdom
   d. highlight controversy

#2. The total stress dosage of treatment incorporates
   a. direction and duration
   b. amplitude and frequency
   c. intensity and frequency
   d. intensity and duration

#3. The key to managing stiffness is to
   a. alter the viscosity of the synovial fluid
   b. stretch shortened connective tissue
   c. induce growth of shortened connective tissue
   d. break adhesions

#4. There is evidence for the use of CPM in which phase of healing
   a. fibroplastic
   b. inflammatory
   c. early maturation
   d. late maturation

#5. The article suggests that the most effective intervention for joint stiffness is
   a. mobilizing splinting
   b. casting motion (CMMS)
   c. passive exercise
   d. joint mobilization

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