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Therapist's Management of the Stiff Hand

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OUTLINE

CRITICAL POINTS

- Hand stiffness produces detrimental structural, neurologic, and emotional changes.
- Therapeutic interventions must be implemented expeditiously to minimize joint stiffness and preserve hand function.
- Three basic principles for postoperative rehabilitation are imperative:
  The effects of immobilization must be minimized.
  Healing tissue must not be overloaded.
  Stiff joints must not be forcefully and passively mobilized, which can exacerbate stiffness; on the contrary, in some cases, active motion alone can effectively increase the mobility of a stiff joint.
- Casting motion to mobilize stiffness (CMMS) is a technique that involves active motion redirection to reduce hand stiffness.
- Long-term use of a cast is sometimes needed to resolve joint stiffness and recover motion.
- Outcome measures reflect progress and can indicate the need to change the treatment plan.
- The patient's status will dictate the frequency, intensity, time, and type of exercise or treatment intervention.
- Concepts of neuroplasticity must be incorporated into the treatment program through the use of repetition and reinforcement of the correct movement patterns.
- External supports (orthotics, casting) are used to create a more efficient grasp pattern and are discontinued when the desired movement pattern can be replicated consistently.
- Practice compassionate listening to facilitate the individual's capacity to heal; encouragement, touch, optimism, and connection to the patient are vital.
- It is necessary to educate both surgeon and patient when promoting the use of the CMMS technique.

THE CHALLENGE OF THE STIFF HAND

One of the greatest rehabilitation challenges is the restoration of digital motion and functional use in a stiff hand. Many variables contribute to the development of digital stiffness, and an intensive approach is required to ensure a satisfactory functional outcome. At times and especially early on, typical treatments such as tendon gliding exercises, compression, and orthotic intervention can be very effective in resolving joint stiffness. However, at other times these same interventions can create a seemingly unremitting cycle of transient improvements, giving way to intermittent pain and swelling, causing frustration, prolonged therapy, and reduced patient compliance.

Hand therapists must continually evaluate the effectiveness of chosen interventions and modify treatment plans accordingly. Early identification and treatment of joint stiffness help to prevent debilitating functional loss. The line between an early and a chronically stiff hand is difficult to pinpoint. Complications such as infection may lead to adherence between multiple tissue planes and subsequently result in chronic stiffness. In cases with a high probability that the hand will become chronically stiff, the technique of Casting motion to mobilize stiffness (CMMS) should be considered as a treatment method when traditional methods fail to produce measurable improvements.

Time is of the essence because prolonged immobility is the greatest enemy of hand function. The challenge is to find the most effective way of addressing multiple problems simultaneously and as efficiently as possible to resolve digital stiffness.

As far back as the fifth century, Hippocrates, the father of modern medicine, stated that "one should bring the parts into their true natural position, both those that are twisted and those that are abnormally contracted, draw them into position by gentle means not violently....time is required for complete success, till the part has acquired growth in its proper position."[1]

UNDERSTANDING JOINT STIFFNESS

When injury occurs, the entire hand responds to the injury. Uninjured adjacent structures undergo fibroplasia, increased collagen turnover, and remodeling[2] and are at risk for loss of motion and function. Stiffness of the hand is not an increased rigidity of the tissues themselves [3] but a constraint created by crosslinking of the previously elastic configuration of the collagen fibers.[4]

Soft Tissue Biomechanics

The soft connective tissues of ligaments and tendons provide the stability necessary for proper joint function by enabling the transmission of forces across joints. The three-dimensional network of connective tissue fibers in the dermis provides the protective sheath around the body, which can withstand shear forces.

Soft connective tissues can be distinguished from hard mineralized tissues (e.g., bone) by their high flexibility and soft mechanical properties. Soft connective tissues are complex fiber-reinforced composite structures whose mechanical behavior is strongly influenced by the concentration and structural arrangement of collagen and elastin, the hydrated matrix of the proteoglycans, and the topographical site and respective function in the organism.[5]

Collagen and Elastin

Collagen is a fibrous protein that makes up one third of the protein in the human body and is found in the extracellular matrix (ECM). There are 16 different types of collagen, which all have different structures and functions, but only three of them (types I, II, and III) form regular fibers. In the dermis, collagen molecules form a fibrous network of cells called *fibroblasts*. Fibroblasts are the most common connective tissue cells found in animals and are the cells that synthesize the ECM and collagen. They play a critical role in wound healing. Injured dense connective tissue has a greater acceleration of collagen synthesis than skin wounds, which continues at a high level of activity for at least 1 month after injury, long after the skin wound has decreased its rate. As much as a 20-fold increase in collagen proliferation and deposition onto the areola surface of fascia occurs within 5 days of the injury. This surface activity forms a disorganized "coat" of collagen that may adhere to skin and restrict mobility of ligaments, tendons, or joint capsules.[6]

Elastin, like collagen, is a protein which is a major constituent of the ECM of connective tissue. It is present as long, flexible strands in the soft tissue. In contrast to collagen, elastin fibers do not exhibit a pronounced hierarchical organization. Elastin is a linearly elastic material that changes with deformation and has very small relaxation effects.

Collagen provides most of the tensile strength of the tissue in the hand. Collagen fibers themselves are inelastic, but movement between the collagen fibers imparts elasticity to the tissue. Normal hand motion occurs when these strong, dense connective tissue structures glide relative to one another.[7] Stiffness is caused by the fixation of the tissue layers so that the usual elastic relational motion is restricted by crosslinks binding the collagen fibers together.[4,8–12]

Viscoelastic Behavior of Connective Tissue

Soft tissues behave anisotropically, which is the property of being directionally dependent; it implies different properties in different directions. This is because of their fibers, which tend to have preferred directions. The tensile response of soft tissue in nonlinear stiffening and tensile strength depends on the strain rate. Soft tissue has the potential to undergo large deformations.

A viscous substance is one in which the stress (torque) is a function of the velocity. Plastic stiffness is a yielding stiffness. As force is applied, there is elastic behavior up to a certain point (the yield point), with further displacement, or with further application of (constant) force, there is progressive displacement (creep). Removal of the force is accompanied by partial return to the initial displacement (relaxation) or incomplete strain recovery.[13]

Connective tissue displays viscoelastic behavior (relaxation, creep, or both), which has been associated with the shear interaction of collagen with the matrix water-binding proteoglycans, which provides viscous lubrication between collagen fibrils. The viscoelastic response can be appreciated in the stiff hand's temporary response to passive stretch.

MOTOR CORTEX REPATTERNING

The primary motor cortex contains an organized map of movement representations, including the hand. Orthopedic disorders, which affect hand use, can cause reduced activation in the motor cortex, even in the absence of a neurological insult.[14] Human and animal studies have consistently demonstrated decreased cortical activity with lack of use.[15-18] These changes in brain patterning happen rapidly. For example, as little as 12 hours of arm immobilization begins to degrade one's motoric performance.[19] The stiff hand is essentially "immobilized" by edema and tissue adherence and is therefore used less and/or with a maladapted movement pattern for daily activities. While this is occurring, the motor and sensory cortical representation of normal, synergistic motion diminishes and is instead replaced by a maladaptive pattern. When abnormal patterns of movement are repeated over time, they give way to changes in the motor cortex.[16] As stiffness persists, these neural circuits are reinforced and are more challenging to alter.

Alternatively, neuroscience plasticity literature has highlighted the concepts of "use it or lose it" and "use it and improve it."[19a] It has been proven that repeated, challenging movements can enhance neural networks and expand the motor cortex representation.[20] In this way, heavily practiced behavior can increase the size of a specific muscle groups' representation in the brain.[19a] This has been demonstrated in Braille readers whose first dorsal interossei muscle of their dominant hand revealed a greater cortical representation than in the non-dominant hand or that of non-Braille readers.[19] Therapists treating the stiff hand cannot overlook the role they play in positively impacting the way the neural circuitry is adapted during the treatment process.

PREVENTION OF JOINT STIFFNESS

There is no pharmacologic agent or treatment modality that consistently prevents adhesions, increases wound strength, and minimizes stiffness.[2] The only practical method of modifying collagen response in traumatic conditions is through the application of prolonged stress that accommodates the physiological limits of the tissue and modifies crosslinking of the collagen fibers, enabling an elastic tissue response that facilitates joint motion.[21] Arem and Madden demonstrated that although stress will not modify collagen strength or deposition, it clearly modifies the shape and potential mobility when applied before crosslinking occurs.[22] It is imperative to recognize the risk to adjacent uninjured structures caused by generalized dense connective tissue inflammation throughout the hand after injury and to provide early motion to preserve these previously normal, uninjured structures. A poor functional outcome may occur because of the application of improper treatment methods. Although many treatment techniques have been developed to mobilize the stiff hand (Table 28.1), no basic research supports any particular exercise treatment regimen to regain

TABLE 28.1 Comparison of the Therapeutic Techniques Used to Treat the Stiff Hand

	DOSAGE OR STRESS LEVEL APPLIED					
Technique	**Intensity**	**Duration**	**Frequency**	**Benefit**	**Ease of Application**	**Evidence**
CMMS (dosage)	High	High	High	Viscoelastic response long term Induces neural plasticity via repetition Sensory feedback mechanism for motor control Uses active motion only Similar to rote exercises Reduces edema Extrinsic and intrinsic tendon gliding Inexpensive Strengthens the hand Reduces the number of therapy sessions required	Easy to moderate cast application 30-min to 1-hour cast application and instruction weekly to monthly Intensive for patient Education and support essential for cooperation	Level 5, expert opinion Two articles related to the hand[25,35]
Serial casting	High	High	High	Elongates tissues in chronic stage Inexpensive	Easy to moderate with single joint Challenging with multiple joint involvement	Retrospective study cites effectiveness in patients with arthritis[75]
Constraint-induced movement therapy	High 6 hr a day, 5 days per for 2 wk	High	High	Increases motivation and overcomes learned disuse through intense task-orientated approach Improves dexterity and motor function Induction of neural plasticity	Constant supervision required Intensive and requires high patient motivation Expensive Constraint of unaffected hand for 90% of waking hours Research on median and ulnar nerve injuries[63] but not the stiff hand	One study showed mCIMT improves hand function in patients with chronic median or ulnar nerve injuries[63] From CVA literature, may be more effective than traditional therapy[76] No studies on stiff hands
Mobilization splints or devices	High	Low (removable)	High	Viscoelastic response medium term to long term/stress relaxation Not proven to contribute growth of contracted connective tissue[9]	Please refer to the section on Mobilization Orthoses in this text	10 articles (2 level 2b, 8 level 4); 5 articles relating to elbow, 2 relating to wrist, and 3 relating to hand[50]
Task-specific therapy, OBI, or bilateral activities *Clients bring activities; must have therapeutic value, be meaningful, and be culturally relevant*	Medium	Medium 30 min twice a week for 6 wk plus 4 wk of HEP for 2 hr per week	Medium Occupation is used as a remediation to restore physical function in therapy and as a HEP	Induction of neural plasticity via repetition and specificity Motor–neural connectivity Practice and achieve performance competence Inexpensive	Can be incorporated into ADLs Caution against use in protective stages of healing	Level 1b evidence that incorporating OBI improves ROM, pain, and functional outcomes in hand injury rehabilitation[77] OBI leads to more satisfaction, motivation, better learning, and more generalization than rote exercise[63] Task-specific training enhances motor networks; ideal frequency and duration not determined in CVA rehabilitation[63] From CVA literature, conflicting evidence: insufficient evidence for effectiveness of bilateral[78]; strong training effect[79]

Sensorimotor input *Graded motor imagery* *Mirror therapy* *Laterality* *Attention* *Proprioceptive input (NB factor)* *Vibration* *Mental process of simulating an action without doing it physically* *Can be triggered implicitly by hand laterality task*				Induction of neural plasticity Pain relief, alleviate fear of moving, reduce central sensitization (motor imagery, laterality) Hemispheric preservation of sensorimotor networks after immobilization (proprioception and vibration) Motor cortex excitability Sensory feedback mechanism for motor control (proprioception)	Easy to moderate Refer to Chapter 100 on graded motor imagery	Level I evidence that mirror therapy as an adjunct treatment for orthopedic injuries improves AROM[63] Level II evidence that mirror therapy and guided plasticity training enhances recovery after nerve injuries[80] Low evidence for mirror therapy for treatment of CRPS, CVA, and phantom limb[31] Graded motor imagery and mirror therapy may be effective based on limited evidence in CRPS, PLP, and CVA[81] Two studies included laterality training but not in isolation; no known effect[81] One study supporting vibration and proprioceptive input in immobilized arms[82]
Active assisted therapeutic exercise	Low	Low	Low	Viscoelastic response ST	Easy	Single level 4 case series study (MCP and PIP joints)[50]
Passive ROM	Low	Low	Low	Not proven	Moderate to difficult	Three studies, all relating to shoulder (levels 2b, 3, and 4)[50]
Joint mobilization	Low	Low	Low	Pain relief	Therapist only	Six 6 (levels 2b–4): 3 studies relating to shoulder, 2 relating to wrist, and 1 relating to hand[50]

ADL, Activity of daily living; *AROM,* active range of motion; *CMMS,* casting motion to mobilize stiffness; *CRPS,* complex regional pain syndrome; *CVA,* cerebrovascular accident; *HEP,* home exercise program; *mCIMT,* modified constraint-induced movement therapy; *MCP,* metacarpophalangeal; *NB,* important; *OBI,* occupation-based intervention; *PIP,* proximal interphalangeal; *PLP,* phantom limb pain; *ROM,* range of motion; *ST,* short term.

mobility.[10,23] Three basic principles for postoperative rehabilitation are imperative:

1. The effects of immobilization must be minimized.
2. Healing tissue must not be overloaded.
3. Stiff joints must not be forcefully and passively mobilized which can increase pain and exacerbate stiffness; on the contrary, active motion alone can in some cases effectively increase the mobility of a stiff joint[24,25]

Although research support is limited, there is growing evidence to support the use of the CMMS technique as an effective method of addressing joint stiffness.[21,25,26,35,72] CMMS, which will be discussed at length later in this chapter, involves the use of a comfortable, nonremovable plaster of Paris cast that selectively immobilizes proximal joints in an ideal position while constraining distal joints to actively direct the desired motion in both directions over a long period of time.[21] The patient performs hourly exercises in the cast to regain both active and passive joint motion. The advantage of this technique is that the cast prevents the application of excessive mechanical force to the tissue and allows for an appropriate prolonged stress that accommodates the physiological limits of the tissue that is applied through active motion only. A reduction in collagen cross-linking is therefore facilitated, which enables an elastic tissue response.[21] Edema is reduced by the combination of tissue compression provided by the cast and skin motion that is created by digital flexion, which provides physical stimulation of superficial lymphatics. Prolonged low-load stress facilitates tissue elongation[27] and influences scar remodeling.[28] The unique properties of plaster of Paris enable the material to conform intimately to the tissue,[29] thereby reducing the possibility of pressure areas and reducing the sheer force of the cast on the skin.[29] Table 28.2 provides a comparison of plaster casts with thermoplastic orthoses.

The challenge for the hand therapist is to allow enough motion to nullify the negative effects of immobilization yet prevent excessive motion that will impede normal healing in the early postoperative period and simultaneously address the factors that perpetuate stiffness in the chronically stiff hand (Box 28.1).

FACTORS CONTRIBUTING TO JOINT STIFFNESS

Stages of Wound Healing

Tissue injury creates a relatively extended period of heightened collagen synthesis, degradation, and deposition within a wound compared with this same process within normal uninjured tissue.[30] Tissue progresses through three stages of healing: inflammatory, fibroplasia, and remodeling (or maturation).[31] Although these are chronological stages, they do not follow a precise timeline unless the wound has no complications. Every wound-healing response is individual and will determine the need for an individualized therapy program. Merritt[6] rightly said that therapists must believe what they see and not what they read and individualize their treatment accordingly. A profound example of individualized wound healing response can be seen in patients with complex regional pain syndrome (CRPS) in whom their disproportionate response to an injury will result in severe pain; inflammation; bone demineralization; and a dystrophic, functionless hand. Recent literature suggests that this adverse wound healing response could be mediated by the central nervous system because of individual intrinsic factors.[6]

During the inflammatory stage, the wound appears to heal; during the fibroblastic stage, the tissue structure is rebuilt; and during the remodeling stage, the final tissue configuration develops.[32] Wounds with massive tissue injury, infection, absence of wound closure, or delayed healing or wounds requiring repeated surgery have extended stages of healing far beyond the ideal time frame. Therapists treating complex injuries must be able to evaluate the characteristics of the wound and healing scar and determine the stage of wound healing to develop an appropriate treatment plan.

Inflammatory Stage

In an uncomplicated wound, the initial inflammatory phase of wound healing is completed within a few days. During this time, randomly oriented, matted collagen fibrils unite the injured structures. Because the intercellular forces are weak, wound healing may be easily disrupted, but involved structures are usually protected with immobilization in the days after the injury or repair.[7]

Fibroplasia Stage

The fibroplasia stage begins at the end of the first week of healing, when the fibroblast begins replacing the macrophage as the most common cell type; however, in complex wounds, this stage is greatly extended. Fibroblasts begin the process of collagen synthesis and outnumber the granulocytes and macrophages in the wound. They eventually evolve into myofibroblasts and are responsible for collagen fiber synthesis

TABLE 28.2 Comparison of the use of Orthoses versus Casting Motion to Mobilize Stiffness in Management of the Chronically Stiff Hand

	Orthotic Positioning	CMMS
Immobilizes stiff joints at end range	☑	
Increases total end range time	☑(Passively)	☑(Actively)
Active motion is possible in two opposite directions		☑
Uses principles of motor learning		☑
Provides circumferential pressure and pseudomassage to reduce edema		☑
Uses active lymphatic pumping to control edema		☑

CMMS, Casting motion to mobilize stiffness.

BOX 28.1 Stiff Hand Problems Simultaneously Addressed by Casting Motion to Mobilize Stiffness

Edema
- Light sustained pressure provided through circumferential nature of the cast
- Active motion facilitates lymphatic pumping

Intrinsic/Interossei Shortening
- With MCP joints in extension, the interossei muscles are lengthened during IP joint flexion–extension
- MCP joints can be extended serially as tissue elongation occurs

Motor Cortex Patterning
- Stabilizes wrist to direct force to DIP and PIP joints, allowing repetitive active motion, to cortically normalize grasp and tenodesis pattern

Mobilization
- The freedom of the IP joints in the cast allow for differential gliding of the FDP and FDS
- Active motion mobilizes joints at end range
- Active motion is possible in flexion and extension

DIP, Distal interphalangeal; *FDP*, flexor digitorum profundus; *FDS*, flexor digitorum superficialis; *IP*, interphalangeal; *MCP*, metacarpophalangeal; *PIP*, proximal interphalangeal.

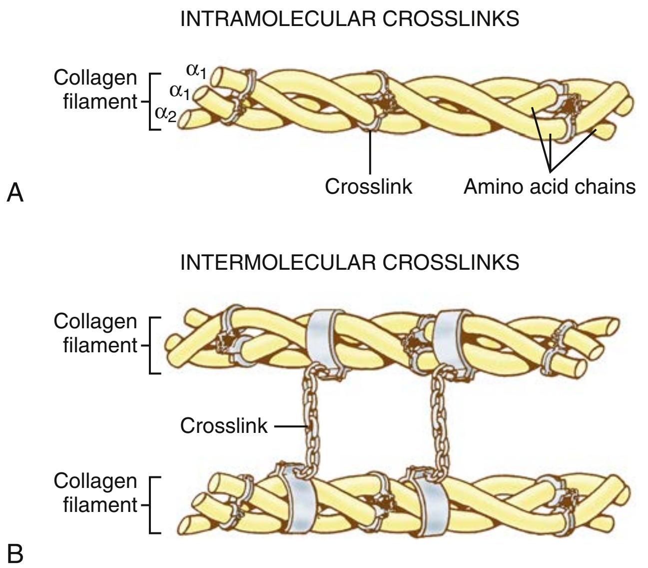

Fig. 28.1 Collagen crosslinking occurs between amino acid chains within one collagen filament (weak crosslinks; **A**) and between collagen filaments (**B**), locking them to one another (strong crosslinks). (From Hardy MA. The biology of scar formation. *Phys Ther.* 1989;69:1020.)

and concurrent contraction of the wound edges. Capillaries reestablish within the wound, forming a dense network. Collagen fibers are laid down between the capillaries, forming the scar needed to keep the wound closed. By the end of the second week, the wound is filled with newly synthesized but disorganized collagen fibers invading all areas of the wound.[7,33,34] Although the tensile strength of the wound remains diminished,[7] the random orientation of the collagen fibers limits their movement relative to one another. At this stage, the scar is not strong and cannot tolerate excessive stress.

Maturation Stage

In the uncomplicated wound, the maturation stage usually begins between 3 and 6 weeks after surgery or injury. This may be delayed by many months in the hand with infection, multiple tissue trauma, multiple surgeries, or delayed healing. As the cell population decreases, the number of scar collagen fibers increases.[7] The total collagen accumulation then stabilizes and remains constant. At this stage, collagen deposition is accompanied by collagen degradation, creating equilibrium. Alteration in the architecture of scar collagen fibers occurs as the scar matures. The tissue continues to respond to applied stress, but the response is greatly diminished compared with the earlier stages. Physical changes are caused by changes in the number of covalent bonds between collagen molecules (Fig. 28.1). Scars remain metabolically active for years, slowly changing in size, shape, color, texture, and strength,[7] and ultimately begin to resemble normal tissue.

The Influence of Wound Healing on Joint Stiffness

Joint stiffness and tissue adherence identified during the fibroplasia stage are characterized by a soft end-feel at the limitation of passive motion. This tissue is responsive because the crosslinking of the collagen fibers is weak, and stress causes the collagen fibers to align themselves with the direction of stress. The fluctuant end-feel of the passive joint limitation results primarily from edema filling the interstitial spaces and thus limits full passive mobility.

Because of the diminished strength of the healing tissue, excessive force can tear the fibrils, causing further injury and reinitiating the inflammatory process. Any movement must be applied slowly and gently and be sustained for a brief period of time. During fibroplasia, intermittent active motion is the ideal means of applying stress to the disorganized collagen to encourage realignment of the fibers. It is at this point that the therapist has the most influence on the ultimate outcome and prevention of persistent digital stiffness and the development of adherent scar tissue. Early response to resistive motions through the application of a gentle and sustained force applied using active redirection and low-load prolonged stress can prevent chronic joint stiffness[25,35,37] and morphologically alter young scars, which may be ineffective in older scars.[22]

Joint stiffness during the maturation phase of wound healing presents as a hard-end feel when passive motion is applied. The joint motion does not yield to gentle force and stops abruptly. This can be further compounded by the effects of prolonged immobilization that result in adaptive shortening of the periarticular tissues such as joint capsules, ligaments, fascia, and muscle–tendon units.[37] The application of stress to involved tissues can contribute to permanent changes in the periarticular structures and surrounding musculature, thereby improving joint stiffness and function.[37] It is up to the hand therapist to determine the duration and intensity of the stress applied. Optimal plastic deformation needed to effect change occurs with the application of low-load prolonged stress. Active stress across stiff joints can be applied through the use of the CMMS technique,[26] which is discussed in detail later in this chapter (see Treatment of the Stiff Hand).

TRAUMATIC AND FIBROPROLIFERATIVE CONDITIONS

In a severely traumatized hand, complications such as excessive inflammation, infection, pain, hematoma, delayed wound healing, complex regional pain syndrome, development of metacarpophalangeal (MCP) joint, proximal interphalangeal (PIP) joint, and distal interphalangeal (DIP) joint contractures and stiffness and scar contractures could occur and may all contribute to a poor functional outcome.[25]

The development of chronic joint stiffness is not isolated to traumatic hand injuries. Fibroproliferative conditions of the hand such as Dupuytren's disease can result in the development of joint contractures that may require surgical correction. To a certain degree, loss of extension after surgery is inevitable; however, loss of digital flexion is reported to occur in 40% of patients who have undergone Dupuytren's fasciectomy and can be more disabling than their original deformity.[38] Overly aggressive exercises and therapeutic regimens, and inappropriate orthotic, use can contribute to a flare response and poor results.[39]

EDEMA

Edema is the primary cause of immobility of the injured hand, and therefore reduction of edema to create motion is always a primary component of treating the stiff hand. The presence of mild postoperative edema actually facilitates wound healing by causing a moderate increase in the strength of the healing wound and an increase in macrophages and fibroblasts.[40,41] Greater amounts of edema destroy the continuity of the wound, breaking the fibrin seal and the integrity of the sutures.[40] After injury, edema develops when the lymphatic system becomes temporarily overloaded by the rate of capillary filtration, which results in a dynamic insufficiency that leads to the accumulation of excess fluid in the intercellular spaces.[42] This normal edema production in response to injury is to be clearly differentiated from lymphedema, which is a high plasma protein edema associated with a mechanical obstruction or insufficiency of the lymphatic system.

Movement of lymph fluid through the lymphatic system is aided greatly by external forces including adjacent muscle contraction, tissue compression (e.g., gentle massage, bandaging), and general stimulation (e.g., arterial pulsations, body movement). If wound healing progresses without complication, edema begins to subside, and motion is regained. However, injured hands that develop significant stiffness do not follow this path, and inflammation and edema persist. The persistent presence of edema plays a significant role in preventing full motion of the hand and must be controlled to prevent chronic joint stiffness.

Pitting versus Nonpitting Edema

Before pitting edema can become visible, the interstitial spaces must first become filled with fluid. This filling of the interstitium with lymphatic fluid increases the internal pressure, eliminating the ease of movement before edema is visible externally. Although this interstitial edema cannot be visually appreciated or measured as easily as pitting edema, it plays a significant role in preventing full motion of the hand.

Most of the interstitial fluid is trapped within the interstitial tissue gel. When edema exists in pockets of free fluid outside the interstitial spaces, it "pits" with pressure. These pockets of free fluid can hold more than half the volume of interstitial fluid.[42] One common location of pitting edema is the dorsum of the hand, where the loose dorsal skin pocket provides ample space for pockets of free fluid to accumulate. Manual pressure placed on the dorsal pocket causes the fluid to move, leaving the indentation (or pit), thus the term *pitting edema*.

EVALUATION OF THE STIFF HAND

Edema

A precise examination will assist in determining the level of interstitial edema. Fullness of the tissues is palpable, creating diminished tissue mobility in the injured hand. The hand must be examined for both pitting and non-pitting edema. External pitting edema can be measured accurately via water displacement.[43,44]

Loss or diminution of normal small skin wrinkles, tautness or obliteration of the dorsal finger joint creases, and obscurity of metacarpal head definition and of the dorsal finger extensor tendons can be observed and documented.

The use of digital photography is useful for monitoring progress and ensuring that observations are accurately recorded. Readers are referred to Chapter 57 for a thorough discussion of edema assessment and management.

Joint Tightness

Joint capsular tightness is identified by measuring the passive range of motion (PROM) of a joint to determine whether the PROM changes as proximal and distal joint positions are altered. If the joint range of motion (ROM) does not change regardless of the proximal or distal joint position, then isolated joint tightness is present. More often than not, a combination of joint tightness and other external constraints, such as muscle–tendon unit tightness or tendon adherence, are responsible for limited PROM. ROM measurements are taken on a regular basis to monitor changes in status in response to treatment.[45]

If there is a large discrepancy between active range of motion (AROM) and PROM, the emphasis should lie on active muscle–tendon unit pull-through. If AROM and PROM are equally limited orthotic intervention may be indicated to provide low-load, long-duration stress to gain further ROM. Active motion with blocking of the adjacent more flexible joints can help to increase joint mobility.

Interosseous Muscle Tightness

The interosseous muscles reside within a tight fascial compartment between the metacarpal bones. These small interosseous muscles have limited excursion, making them relatively intolerant of the adaptive shortening that occurs as a result of immobilization. Direct trauma to the metacarpal area may also create injury or ischemia to these muscles, causing potential for an even greater severity of interosseous muscle tightness. If the interosseous muscles are tight, full finger flexion will be limited by the tightness and elongation will be required. Because the line of pull of the interosseous muscles is volar to the MCP joint and dorsal to the interphalangeal (IP) joints, interosseous muscle tightness is determined by defining the amount of passive PIP joint flexion when the MCP joint is flexed and then determining if this amount of PIP joint flexion is less when the MCP joint is simultaneously brought into passive hyperextension. MCP hyperextension combined with PIP flexion is the position of maximum elongation of the interosseous muscles. If the range of passive PIP flexion is less when the MCP joint is held in full extension than it is when in MCP flexion, then the interosseous muscles are tight.

Muscle–Tendon Unit Tightness

Muscle–tendon unit tightness is a shortening of the muscle–tendon unit from origin to insertion, which limits full simultaneous motion of all joints crossed by the muscle–tendon unit. The muscle is the elastic part of this unit, which shortens with disuse. This tightness commonly occurs as a result of immobilization or restricted motion after injury or surgery. If a muscle–tendon unit is left in a shortened position in the presence of tissue inflammation, the tendon may also become adherent along its path even if there is no direct trauma to the tendon or tendon bed. Specific trauma to the tendon or tendon bed, however, creates distinct adherence at the site of injury. Tendon adherence may be isolated to the point of trauma or extend over a larger area of more extensive trauma or result from immobilization in the presence of inflammation. Tendon adherence affects movement only of the joint(s) distal to the point of adherence. Although both muscle–tendon unit tightness and tendon adherence may have similar clinical presentations, careful examination allows differentiation between the two.

Adherence

A tendon may be adherent anywhere along its path. Motion to decrease the adherence is accomplished by joint motion distal to the adherence. This can be *active* motion of the joints distal to the adherence that results in an active pull in a proximal direction on the adherent tendon, or it can be *passive* motion of the joints distal to the adherence, which are moved in the direction opposite to the active motion (i.e., passive extension if a flexor tendon is adherent). This insight allows correct positioning for exercise and determines the joint(s) to be included in any orthosis. Mobilization via orthoses to decrease tendon adherence is effective only in regaining distal glide of an adherent flexor or extensor tendon (e.g., using a composite wrist–digit extension orthosis to improve distal glide of flexor tendons). To gain proximal glide, the patient must isolate and strengthen the correct muscle to regain full excursion of the adherent muscle–tendon unit. Adherence after flexor tendon repair provides an example of the type of active motion necessary to gain proximal glide of an adherent tendon. Commonly, after flexor tendon repair, the patient may attempt flexion using the interosseous muscles, with minimal gliding of the repaired flexor tendon, especially if the injury has been within the flexor sheath (zone II). The MCP joints usually fully flex before the IP joints reach full flexion. When tendon healing permits, blocking the MCP joint in extension to demand flexor tendon excursion across the distal joints is required (Fig. 28.2).

Fig. 28.2 Blocking exercises with the metacarpophalangeal joints positioned in extension is used to promote excursion of an adherent flexor tendon. This is an active redirection orthosis.

Both tendon adherence and muscle–tendon unit tightness are demonstrated by a difference between the passive distal joint motion when the proximal joints are positioned in flexion. For example, with extrinsic extensor muscle tightness, the fingers will be unable to flex as far with the wrist in flexion as when the wrist is in extension. In the case of extrinsic flexor muscle tightness, with wrist extension, finger extension is limited, but when the wrist is flexed, the fingers can more fully extend.

Whereas in the case of interosseous muscle tightness, the primary joint which holds the key to evaluation is the position of the MCP joint, in the case of extrinsic muscle–tendon unit tightness or adherence, the primary key is the position of the wrist. Extrinsic extensor muscle tightness or adherence is present when the fingers can be flexed more with wrist extension and less with wrist in neutral or flexion. Extrinsic flexor muscles tightness or adherence is present when finger extension is limited when the wrist is extended but not limited when the wrist is in neutral or flexion.

Skin and Scar Tightness

All wounds heal with internal and external scar. Depending on the size, location, and extent of scar, external scars (especially linear scars) may limit joint motion. Even if the scar is not adherent to the underlying joint(s), the length of the scar may not allow multiple joints to move in the same direction simultaneously. For example, a split-thickness skin graft on the dorsum of the hand can tether the skin so that either IP joint flexion or MCP joint flexion is possible, but simultaneous MCP and IP joint flexion is not possible.[21]

To evaluate skin and scar tightness, position the joints so that the scar is at its maximum length. Blanching, palpable tightness, or immobility of the scar or skin displays the extent of tightness. If the skin is limiting motion, placing the skin in its shortest position allows increased joint motion proximally or distally. This motion is diminished as either joint is positioned to elongate the involved skin. This limitation may be difficult to determine in a severe injury that creates both skin and joint tightness.[21]

MUSCLE ISOLATION AND PATTERN OF MOTION

During periods of immobilization, the sensory-motor cortex is deprived of stimulation as functional use is not possible. Patients then become at risk of developing abnormal patterns of movement when they are permitted to resume motion. Initially, they can only move the most flexible joints. In an effort to regain functional hand use, patients often use excessive force to regain motion and overcome their hand stiffness. This strong muscle pull is counterproductive, as it recruits the strongest muscles and overpowers the weaker muscles. The result is co-contraction or loss of isolated muscle control. For example, the weakened wrist extensor muscles cannot adequately stabilize the wrist in extension to allow the finger flexor muscles to flex the digits. When the patient is asked to extend the wrist, the finger extensor muscles substitute for the wrist extensor muscles because they have been unrestrained in the cast and are stronger. If edema and finger stiffness are accompanying complications, little progress can be made with finger motion until the patient can stabilize the wrist with the wrist extensor muscles.[46]

WRIST TENODESIS PATTERN

The exquisite balance of muscle forces crossing the wrist and fingers creates a reciprocal motion called *tenodesis.* Finger extension occurs with wrist flexion as a result of the increased tension on the extrinsic extensor muscles when the wrist flexes. Conversely, when the wrist extends, tension is increased in the extrinsic flexor muscles that flex the fingers. This reciprocal action establishes the normal grasp and release pattern of the hand.

In the presence of joint stiffness, the tenodesis balance in the hand is frequently affected. In a minor injury, tenodesis is regained as motion at the injury site improves. In more severe injuries requiring long periods of immobilization, many joints may become stiff, and the muscles crossing them become weak, altering the reciprocal balanced motion.

The wrist is the key joint to reestablishing the tenodesis balance in the hand. Without the ability to stabilize the wrist in extension, the finger flexor muscles cannot transfer enough power to regain finger flexion. Usually, the primary goal is to regain digital flexion for grasp and manipulation of objects. However, when the fingers and the wrist all have limited motion, improvement in active finger flexion is compromised without first regaining some wrist extension

Pathological Patterns of Motion

Intrinsic-Plus Pattern or Dominant Interosseous Flexion Pattern

During normal finger flexion, the cascade of digital flexion is initiated at the DIP joints through the extrinsic flexor digitorum profundus (FDP) muscle followed by PIP joint flexion through the flexor digitorum superficialis (FDS) muscle and then by the MCP joint through the intrinsic interosseous muscles.[47,48]

If the hand is edematous and extrinsic flexor glide is limited (commonly seen after immobilization of wrist fractures or flexor tendon repair), the patient will initiate finger flexion with MCP joint flexion, and little IP joint flexion occurs.[46] In this pattern of motion, the interosseous and lumbrical muscles are never elongated to their maximum length, and they adaptively shorten, making the mobilization of the IP joints even more difficult.

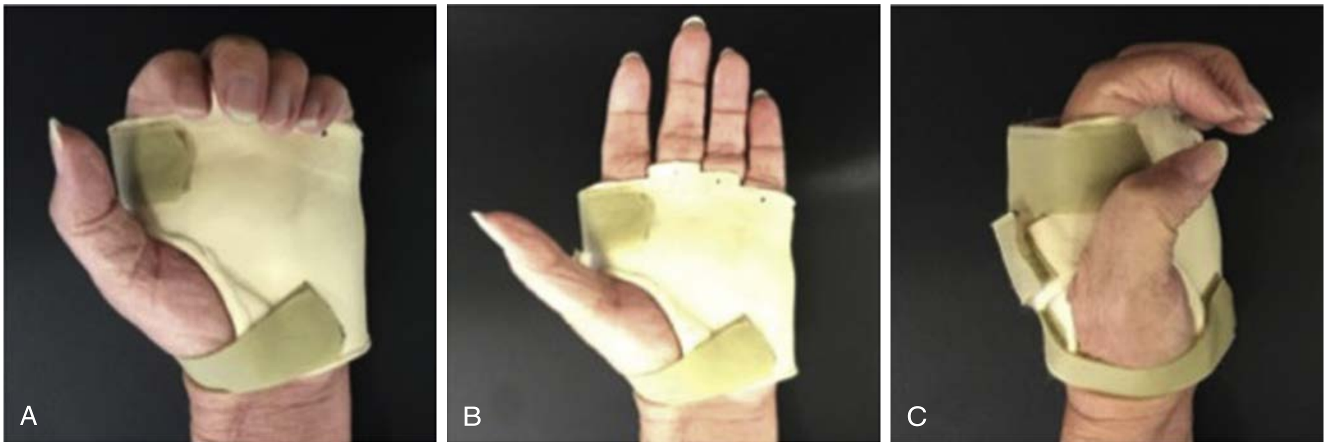

Fig. 28.3 Active redirection orthosis. **A,** Volar view of an active redirection orthosis that allows elongation of the interosseous and lumbrical muscles and encourage intrinsic minus pattern of motion. **B**, Volar view of orthosis shows intimate contour and well-distributed pressure over the metacarpophalangeal (MCP) Joints. **C**, Radial-lateral view shows the position of the MCP joints in maximum extension.

Early treatment—consisting of activities, exercises, and orthoses that block MCP joint flexion and require IP joint flexion (unless contraindicated by surgical repairs)—can convert global finger flexion into specific glide across the IP joints (Fig. 28.3). In a chronically stiff hand, longer periods of intervention may be necessary to change the pattern of motion. (See the Treatment of the Stiff Hand section.)

Intrinsic-Minus Pattern or Dominant Extrinsic Flexion Pattern

Although the dominant intrinsic flexion pattern is the most common pattern of stiffness seen in the hand, other patterns of stiffness present that require alternate interventions. When the intrinsic muscles are not actively participating in digital flexion, isolated MCP joint flexion is absent. Flexion occurs first at the IP joints, and only after full IP joint flexion do the extrinsic flexors pull the MCP joint into flexion. This may result from denervation of the intrinsic muscles, but in a stiff hand, it is more commonly a result of isolated capsular tightness of the MCP joints or the restraint created by adherence of the extensor tendons on the dorsum of the hand.

Blocking the wrist so that flexion forces from the extrinsic flexors can be directed to the MCP joints is required to actively mobilize the MCP joints. Without MCP joint flexion, the intrinsic hand muscles cannot participate in the digital flexion.

OUTCOME MEASURES

The objective, scientific assessment and meaningful analysis for each individual patient is crucial. The assessment results will encourage and reassure the patient to be optimistic to progress and will alert the therapist to changes that need to be made to the program.[6]

The use of outcome measures is essential and will enable the therapist to document case studies that can be used to analyze results, which can then contribute to the body of literature on the efficacy of our treatment interventions.

Objective means of quantifying the changes in pattern of movement or changes in soft tissue do not currently exist. Direct palpation is the only means of demonstrating the quality of soft tissue change.[21] Digital photography and video recording can be used to assure observations are accurately recorded. Table 28.3 provides an outline of outcome measures recommended for the evaluation of the stiff hand.

TREATMENT OF THE STIFF HAND

The management of joint stiffness over the past 4 decades has been based on the research and teachings of pioneering hand surgeons such as John Madden, MD; Erle Peacock, MD; and Paul Brand, MD, who described the principle of holding tissue in moderately lengthened positions for significant periods of time until it reaches a new length as the key to overcoming stiffness.[49] Therapists applied manual stretching techniques and mobilization orthoses in response to this theory. Subsequently, there is a high level of evidence for the use of mobilization orthoses in the management of joint contracture[50] versus the more recent CMMS technique, which only has level 5 evidence.[25,35,50a] Despite the lack of research for CMMS, the technique is theoretically sound according to the principles laid forth by Madden and colleagues because joints are placed in an ideal position for extended periods of time while being actively mobilized only.

In the early phases of hand stiffness, traditional treatments such as edema management, tendon gliding exercises, and facilitating functional hand use may be very effective in managing stiffness. However, because connective tissue has viscoelastic properties that result in tissue returning to its prestretch length,[49,51] stretching techniques and joint mobilization orthoses have a limited application when managing the stiff hand. Furthermore, we are now aware that immobilization must be minimized, healing tissue must not be overloaded, and an appropriate amount of stress that promotes favorable collagen orientation and increases tensile strength of healing tissue is required. Recent literature emphasizes that regaining motion is both a complex mechanical and cerebral challenge and demands that the problems of edema, fibrosis, extensive tissue adherence, and multiple joint stiffness are addressed simultaneously because these factors are interdependent. Changing one factor alone does not change the other factors. The benefits of CMMS when addressing these factors is that it facilitates motion, works the joints in both directions, applies principles of motor learning, uses active lymphatic pumping control of edema and provides circumferential pressure and pseudomassage simultaneously. Orthoses can increase total end range time and will immobilize stiff joints at end range but cannot treat multiple problems simultaneously (see Table 28.2).

If addressed early, chronic stiffness can be avoided. The authors highly recommend the application of the CMMS technique in an acutely stiff hand (from 2 weeks) to prevent chronic stiffness rather than apply it when the hand is already chronically stiff. The approach

TABLE 28.3 **Outcome Measures to Evaluate the Stiff Hand**

Category	Assessment	Description	Application	Test–Retest Reliability	Validity	References
ROM	Total active motion (TAM)	Measures TAM using a finger goniometer; the sum of all active flexion measurements of digit joints is calculated using the sum of all extension deficits of the digits and thumb subtracted from it to compute TAM	To assess: • Joint tightness • Tissue glide • Muscle–tendon unit tightness • Tendon adherence	High	High	83,84
	Interosseous muscle length testing	Quantifies the degree to which interosseous muscle tightness is present and may be contributing to hand stiffness; Compares calculated scores for the ROM measurements of involved digit(s) vs uninvolved digit(s)	To assess the length of the interosseous muscles	N/A	N/A	102,103,104
Strength	Grip strength	Measures grip strength using a dynamometer	To assess overall strength and function of the upper extremity	High	High	85–87
	Pinch strength	Measures pinch using a pinch gauge	To assess intrinsic hand function	High	High	84
Pain	Graphic numerical rating scale (GNRS)	Measures pain represented on a graphic scale from 0–10	To assess limiting factor to joint mobility or presence of anatomical deficit	N/A	N/A	84,88,89
Sensation	Semmes-Weinstein monofilament testing	Identifies deficits in threshold perception, protective sensation, and deep pressure	To assess sensory and motor nerve function	High	High	
Scar	Skin tightness	Measured by positioning joints so that the scar elongates to maximum length	Evaluation of multiple joint mobility, blanching, palpable tightness	Low	Low	21
Proprioception	Active wrist joint position sense testing	Patient is asked to reproduce predetermined wrist angle with eyes closed	Sensorimotor control	High (in distal radius fractures)	Low	90–92
Dexterity	Purdue peg board	Measures fine and gross dexterity function	Evaluate loss of dexterity in presence of joint stiffness	High	High	93,94
Function	Disability of the Arm Shoulder and Hand (DASH)	Measures symptoms and functional status after hand injury		High	High	95,96
	Upper Limb Functional Index-10 (ULFI)	Self-reported measure with 10 statements related to function		High	High	97–99
	Canadian Occupational Performance Model (COPM)	Used to determine the effect of the intervention on the participants self-determined occupational performance goals		High	High	100,101

N/A, Not applicable.

significantly reduces the duration of time needed in the cast. With a strategic approach to treatment of the stiff hand, therapists can be confident in their choice of treatment.

CLINICAL REASONING AND TREATMENT IMPLEMENTATION

Fig. 28.4 presents an algorithm to assist with the clinical reasoning process when planning treatment for the stiff hand and will be discussed in detail.

Identify the Problems

The location of joint tightness and abnormal pattern of motion must be identified to plan the treatment (see Evaluation of the Stiff hand).

It is useful to video the patient's movement pattern for evaluation purposes as well as for monitoring progress. Cues must be given to assist the patient to identify and contract the appropriate muscle to produce the desired motion, slowly, and with the minimal force needed to sustain the contraction. After the abnormal movement patterns have been identified, appropriate therapy interventions can be implemented and a home exercise program (HEP) devised.

Use of Traditional Principles

As research continues to evolve and guide practice, the lines between "biomechanical" and "motor learning" models begin to blur because concepts from each intermingle. This is true in the case of the stiff hand. A "therapeutic activity" may simultaneously encourage active

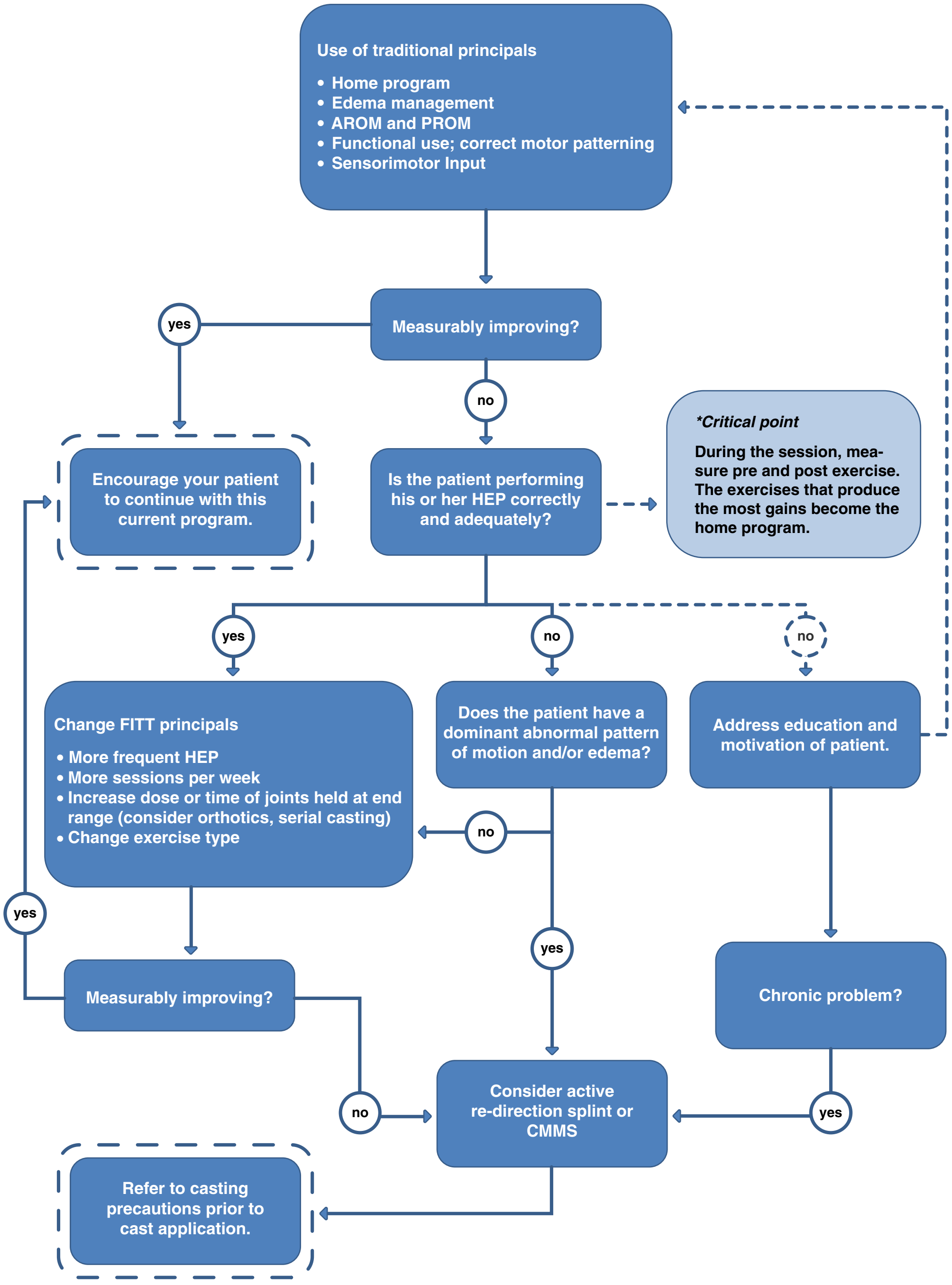

Fig. 28.4 Clinical reasoning flow chart for treatment planning for the stiff hand. *AROM,* Active range of motion; *CMMS,* casting motion to mobilize stiffness; *FITT,* frequency, intensity, time, type; HEP, home exercise program; *PROM,* passive range of motion.

tendon gliding, provide proprioceptive input, and engage the motor networks of bilateral cerebral hemispheres. The therapist will reinforce "normal" movement patterns and therefore "normal" cortical representation through available therapeutic techniques. At times, the most effective and efficient way to do this is simply by providing a verbal or visual cue. At other times, cues or traditional mobilization techniques may be frustratingly unsuccessful.

Although cortical representation changes rapidly based on use, for repatterning to become an ingrained, automatic, dominant motion, the motion must be repeated for long periods during the day and over many days and weeks.[52–57] Repatterning is enhanced by conscious, close attention to the desired motion,[58] but unattended repetitive motion and passive motion result in little or no significant plasticity changes in the cortex.[59,60] This helps to explain why, in practice, increased ROM is achieved with passive stretch but not carried over to the next session. A patient may be able to achieve wrist tenodesis with focused attention during rote exercise, but when his or her attention is divided in a relatively more complex task requiring grasp, the wrist falls back into flexion. Without external support of the wrist, this patient is constantly reinforcing a maladaptive grasp pattern during daily activities outside of the clinic. From a neurologic and therapeutic perspective, the patient requires more repetition to reinforce a normal grasp pattern.

The research that has been done in motor recovery after stroke has demonstrated that task-specific training is known to be a component of rehabilitation.[20,61] Additionally, activities that are occupation-based and functional have been shown to be more beneficial than standard or rote exercises.[62,63] Readers are encouraged to refer to the comparison of therapeutic techniques in Table 28.1 and select techniques that are of most benefit, provide high levels of dosage and stress, and have ease of application. The exact parameters of dosage in terms of intensity, duration, and frequency of stress are still unknown. We need to rely on our clinical observations of a positive influence of the dosage on healing tissue and the absence of a renewed inflammatory response. Skilled therapists blend programs to achieve favorable results. Time is of the essence when treating the stiff hand. Therapists must pay careful attention to early signs of an abnormal pattern of motion and the development of joint stiffness and apply the CMMS technique sooner rather than later. Once a normal pattern of motion is established and joint stiffness has resolved, functional and therapeutic home exercise programs can be prescribed.

HOME EXERCISE PROGRAMS

The importance of an individualized HEP cannot be overstated. Time should be spent in choosing the most effective and most efficient exercises. The patient must have an excellent understanding of both the technique and importance of adherence. Goniometric measurements of the involved joints must be performed before and after an exercise. Those exercises that demonstrate improvements in ROM can become the HEP. The exercises should be done frequently (four to six times per day) rather than forcefully. Each session should start with the therapist asking the patient to demonstrate the home program until independence is achieved because this is often where breakdown of communication between therapist and patient occurs.

After a treatment program has begun, it is crucial to monitor the patient's progress closely. Ideally, the individual with a stiff hand would be able to attend therapy on a very frequent basis initially, not necessarily to provide more hands-on treatments but rather to assess the effectiveness of the chosen interventions. Because the HEP is the cornerstone for a successful outcome, the therapist takes on the role of a "consultant," modifying the HEP each time no progress is made. Changes to a HEP can be made according to the FITT principle: frequency (i.e., times per day of performance of a HEP), intensity (i.e., how forcefully each exercise is performed), time (i.e., how long each exercise is held), and type (i.e., joint blocking vs active composite fist).

Repetition and review of proper technique will help ensure that the patient is carrying this over to the home program. A common therapeutic mistake is to move onto muscle strengthening before the patient relearns isolated muscle control, which can result in the strengthening of the "wrong" muscles. By emphasizing the above techniques, the patient learns to isolate the precise movement (rather than co-contracting), which will reestablish muscle balance in the hand.

Management of Edema

Minimizing the negative effects of immobilization caused by edema is the most useful initial treatment for an injured hand. Elevation, active muscle contraction, external pressure from various sources such as compressive wraps, and stimulation via gentle light massage can prevent the accumulation of excessive edema. Understanding which edema reduction technique to use when and the optimal type of force or pressure to use throughout the stages of healing is key to successful prevention of joint stiffness.

A key factor in managing edema is the use of intermittent active motion of the proximal joints, which ensures joint motion and recruitment of the large proximal muscle groups to assist with venous and lymphatic flow. Elevation of the extremity (i.e., hand above the elbow and elbow above the heart) to decrease the hydrostatic pressure in the vessels[64] and a gentle, even distribution of pressure to facilitate lymphatic flow while protecting the healing tissues will be effective. However, active motion across the site of injury immediately after surgery may disrupt healing and often cannot be immediately used. Because there are no muscles within the digits, skin movement and tissue compression from active flexion is the stimulus required for increased lymphatic flow in the digits. When digital motion must be limited to protect healing structures, gentle pressure to the digit, elevation, and active muscle contraction in adjacent uninjured areas must substitute for active motion of the digit itself. With many digital injuries, the MCP joint may safely be allowed full motion. This permits the patient to perform pumping exercises of digital adduction and abduction and MCP joint flexion and extension, which contracts the adjacent proximal intrinsic muscles. Conversely, blocking the MCP joints in extension assures active flexion forces are directed to the IP joints to effectively mobilize the digital edema.

Plaster of Paris can be used to either mold circumferentially to conform intimately to the hand or be applied as volar and dorsal slabs. This stabilizes the MCP joints in extension and provides the appropriate pressure to the dense lymphatic network in the palm, which assists with the reduction of digital edema. Applying gentle pressure to the volar–dorsal MCP joints while the plaster of Paris is drying provides a circumferential pressure that assists with reducing edema in the hand. The muscles proximal to the wrist are larger than the intrinsic muscles of the hand and thus are more effective stimulators of the lymphatic system. As long as the vascular status of the hand is stable, intermittent active motion of proximal muscles is begun as early as possible after surgery. Waste products that are evacuated from the injured hand are more effectively moved through the lymphatic system with this intermittent active motion. PROM does not stimulate muscle contraction and, for this reason, cannot be substituted for active motion to reduce edema.[65]

Active Motion: Pumping versus Gliding Motion

The muscles proximal to the wrist are larger than the intrinsic muscles of the hand and are thus more effective stimulators of the lymphatic system. As long as the vascular status of the hand is stable, intermittent active motion of the proximal muscles must begin as early as possible after surgery. Waste products that are evacuated from the injured hand are more effectively moved through the lymphatic system with intermittent active motion. PROM does not stimulate muscle contraction, so it cannot be substituted for active motion to reduce edema.

Because there are no muscles within the digits, skin movement and tissue compression from active flexion is the stimulus required for increased lymphatic flow of the digits. When digital motion must be limited to protect healing structures, gentle pressure to the digit, elevation, and active muscle contraction in adjacent uninjured areas must substitute for active motion of the digit itself. When active motion of the injured part is allowed, elevation and proximal pumping must be continued until enough motion is present at the injury site to allow local pumping that is sufficient for the tissues. During all stages of healing, compression dressings and gentle edema reduction techniques are needed to assist in reducing edema and inflammation.

Passive Range of Motion

Although joint motion can be maintained by either active or passive motion, passive motion provides limited glide of the tissue planes other than periarticular structures.[47] Increasing passive motion does not necessarily increase active motion. There are no clinical research data to dictate the ideal force, speed, and duration of passive motion applied to the stiff hand.[4] Although passive joint motion often is prescribed to overcome posttraumatic stiffness, no clinical research supports the efficacy of either intermittent passive motion or continuous passive motion to reduce joint stiffness in the hand.[10] Aggressive motion of the hand is detrimental and should be avoided.[9,65] Passive motion of the injured hand should be described as gentle encouragement of tissues to reach a maximum available length. The amount of force should respect the resistance of the tissues, and the position should be increased only when the tissues relax and decreased resistance is felt. When performing passive mobilization of the joint, one prolonged hold will allow the motion to be repeated actively more effectively than many repeated, quick, sudden passive stretches. Quick, forceful stretches may result in tissue damage and should be avoided at all times. In the hand with more mature stiffness caused by increased collagen crosslinking, the brief intermittent nature of passive motion alone is ineffective. This may seem contradictory because one may assume that the stiffer the joint, the more force required to mobilize it. On the contrary, it is the increased duration of a low-level force that best creates change. When patients with significantly stiff hand joints undergo PROM alone during a therapy session, there is an immediate response to tissue mobilization. However, when the patient returns, the progress gained in the previous session has not been retained.

Sensorimotor Input and Proprioceptive Feedback

Proprioceptive feedback is essential when working to regain finger flexion. Providing resistance to finger flexion to increase the patient's proprioceptive sense of digital motion is beneficial. This is particularly critical in the presence of diminished sensibility. This type of feedback can be accomplished by the patient holding an object slightly smaller than the available range of finger flexion. The size of the handle is decreased as the patient gains flexion range. The hood of the cast when using the CMMS technique also provides proprioceptive feedback.

Measured Improvements

If the patient is demonstrating measured improvements, then the prescribed program can continue. If not, then consider if the patient is performing the HEP adequately and attending sufficient therapy sessions. If the answer is yes, then consider changing the HEP, therapeutic techniques, intensity, and frequency of sessions or incorporate the use of orthoses or serial casting. If the patient is demonstrating measured improvements and has a normal pattern of motion then the prescribed program can continue. If not, and there is concern that chronic joint stiffness will develop, then apply the CMMS technique. An active redirection cast or orthosis can be applied if a single digit or joint is stiff. Active re-direction is described later on in the chapter. The CMMS technique will re-establish a normal pattern of motion and treat multiple problems simultaneously. If a normal pattern of motion is present, then consider changing the HEP, therapeutic techniques, intensity, and frequency of sessions.

If the patient is not performing the HEP, address education and motivation. If there is concern that chronic stiffness will develop, then consider applying the CMMS technique.

Balance of Exercise and Rest

The primary guideline for exercise progression should be the status of the hand after exercise. If edema, pain, and stiffness increase after exercise (or any treatment), the hand is not yet ready for that level of stress. Conversely, if the patient experiences sustained comfort and mobility, the amount of exercise is appropriate for the stage of recovery and may be slowly upgraded.

USE OF ORTHOTIC MOBILIZATION

Orthotic mobilization that repositions joints with serial application is the safest early means of mobilizing healing tissue and is optimally combined with an individualized exercise program. Conversely, even orthoses that apply passive mobilization impose immobilization and constriction; therefore, the benefits of the orthosis must outweigh the negative effects of restriction and immobilization.[9] Each orthosis applied to the injured hand must be designed based on the mobilization goals for that hand. Therapists must possess analytic skills, manual construction skills, and biomechanical knowledge to apply well-fitting and well-designed orthoses. Readers are referred to Chapters 108 and 109 for a detailed discussion of the principles and techniques of orthotic fabrication.

Human tissue responds to the application of low-load prolonged stress for defined periods of time that does not elicit an inflammatory response.[37,66–68]

The amount of temporary versus long-term change of tissues depends on the intensity and duration of the applied load.[37,49] If the stress is applied over a prolonged period, a plastic response occurs with increased length of the involved tissue.[66,69] One can understand this principle by thinking about how a rubber band, when quickly stretched, returns to its original length; a rubber band held stretched does not return to its original length as quickly or as completely. The patient's tissue response to the application of stress remains the primary guideline to its application. Because mobilization orthoses are applied intermittently, pressure exerted on the skin is less of a limiting factor.[9,70]

Periods of passive orthotic mobilization must be balanced with periods of active movement to ensure the maintenance of passive gains. The patient must understand that the goal is not to tolerate increasing amounts of tension but rather to tolerate low tension for longer periods. After an initial adjustment period, the tissues should comfortably tolerate the prolonged passive-mobilization force. The patient should be aware of the sensation of stretching while wearing the orthosis but should not experience pain. A motivated patient will eagerly wear an effective, well-fitting orthosis. However, chronic stiffness responds better to a nonremovable cast. Understanding the mechanical effect of each type of orthosis allows the therapist to choose the most effective means of regaining motion while facilitating healing.

TYPES OF MOBILIZATION ORTHOSES

There are three types of passive mobilization orthoses: serial static, dynamic, and static progressive. In addition, removable orthoses can

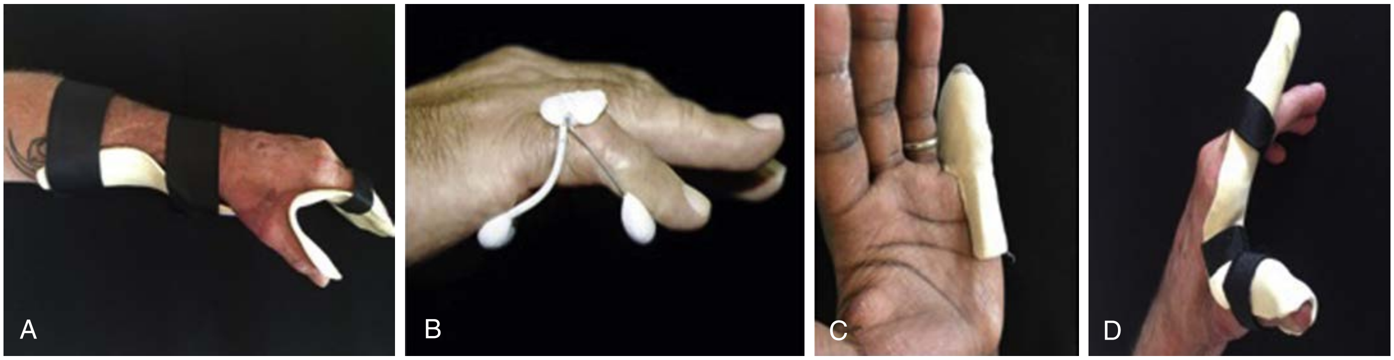

Fig. 28.5 Passive mobilization orthoses. **A,** Serial static hand and forearm orthosis to improve metacarpophalangeal joint extension. **B,** Serial static orthosis gains end-range proximal interphalangeal joint extension. **C,** Serial POP cast to improve a stiff proximal interphalangeal joint with flexion contracture. **D,** Serial static hand-based orthosis to increase the thumb webspace.

provide active redirection, which can be used in the early stiff hand or for single-joint stiffness. Understanding the mechanical effect of each type of orthosis allows the therapist to choose the most effective means of regaining motion while facilitating healing (see Tables 28.2 and Box 28.1).

Serial Static Mobilization Orthoses

A serial static orthosis immobilizes joints in a stationary position. The orthosis is applied with the tissue at its maximum length and is worn for long periods of time to allow the tissue to adapt. After a period of tissue accommodation, either a new orthosis is applied or the old orthosis is remolded to hold the tissue at a new maximum length. Although the orthosis is stationary, the repeated repositioning of the joint(s) increases the length of the tissues (Fig. 28.5).

Dynamic Mobilization Orthoses

A dynamic orthosis applies force to a specific joint or joints. A stretched rubber band, spring, or wire coil generates continuous force. As joint motion changes, the force of the orthosis continues. Although the force is constant while the orthosis is applied, the application of force is intermittent because the orthosis is periodically removed.[70a] In an early stiff hand in which collagen crosslinking is immature, intermittent dynamic force application may restore tissue mobility. If a dynamic orthosis is applied too early or with too much force, it can exacerbate the inflammatory response.

Static Progressive Mobilization Orthoses

Static progressive mobilization orthoses may appear identical to dynamic mobilization orthoses, but the applied force is not dynamic. Instead of the constant pull of a rubber band or spring, the tension on the joint is an adjustable static force. The force may be applied via hook-and-loop fastener or with commercially available components that adjust in small increments. When tension is applied, the joint is positioned at its maximum end range. The force is adjusted when the tissue response allows repositioning to a new length (Fig. 28.6).

Static progressive orthoses can be effective for joints with limited motion when there is significant resistance at the end of the available passive joint motion. Static progressive orthoses are especially recommended when positioning to regain end-range joint extension of the small joints of the hand. As with other passive mobilization orthoses, the patient removes the orthosis to work on active tendon gliding.

ACTIVE REDIRECTION ORTHOSIS

The term *active redirection* was coined by Judy Colditz and is used to describe the simple concept of blocking normal joints so that available muscle power is directed to the stiff joint(s) (see Fig. 28.3). The difference between blocking exercises and active redirection is duration. Blocking exercises are intermittent, whereas active redirection orthoses are worn for extended periods of time and facilitate repetitive, cyclical active motion (Fig. 28.7).

Rationale

PIP joint flexion contractures of the ring and little fingers that result from ulnar nerve palsy may regain full PIP joint extension when the MCP joint is blocked from full extension[72] (see Fig. 28.7). PROM is not always necessary to mobilize stiff joints if cyclical active motion is repeated frequently and the patient cannot revert to the previous imbalanced pattern of motion. Active redirection when MCP joint hyperextension is blocked and the stiff PIP joint moves actively into full extension throughout the day simultaneously accomplishes differential glide of tissue planes, reduction of digital edema ,and motor cortex remapping.

Design

Active redirection can be applied as a blocking orthosis worn during waking hours (see Fig. 28.3) or, if the stiffness is severe, as a nonremovable cast worn full time until rebalance of motion is achieved in the stiff joint(s) (Fig. 28.8). Active redirection is different than CMMS because only one of the components of CMMS is active redirection. CMMS also provides edema reduction via the cast with active movement as well as cortical repatterning. CMMS addresses multiple problems, whereas active redirection addresses joint stiffness in the absence of multiple problems. Either plaster of Paris or thermoplastic materials can be used to fabricate an active redirection orthosis. For digital stiffness caused by interosseous or lumbrical tightness, the wrist does not need to be included, provided the patient has wrist control. If the wrist needs to be included, then a circumferential plaster of Paris cast may be used.

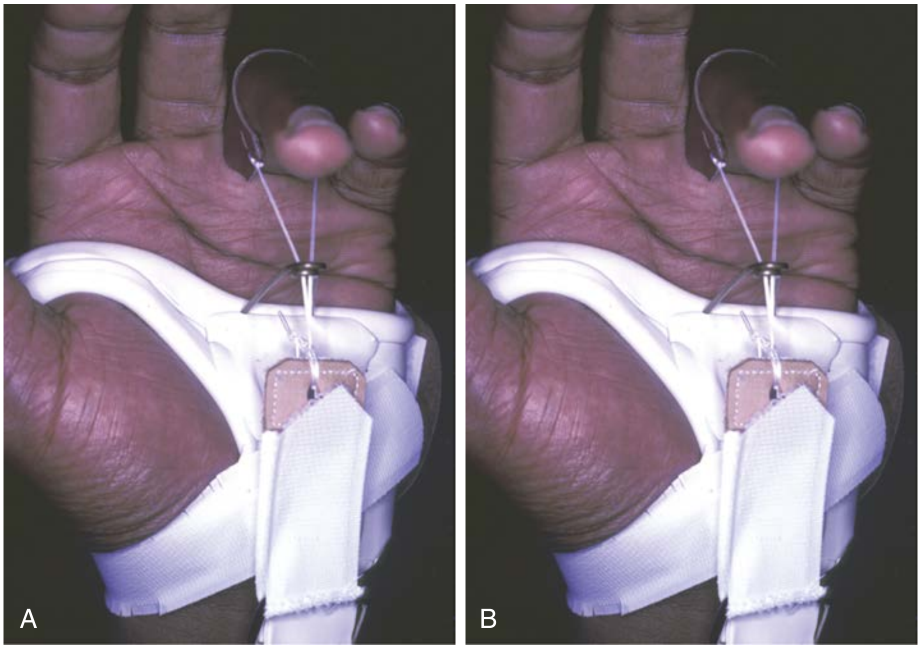

Fig. 28.6 **A** and **B,** This static progressive orthosis applies flexion force to gain metacarpophalangeal joint flexion. It is static progressive in that the force applied uses hook-and-loop fastener. If the force was applied with a rubber band, it would be considered a dynamic orthosis.

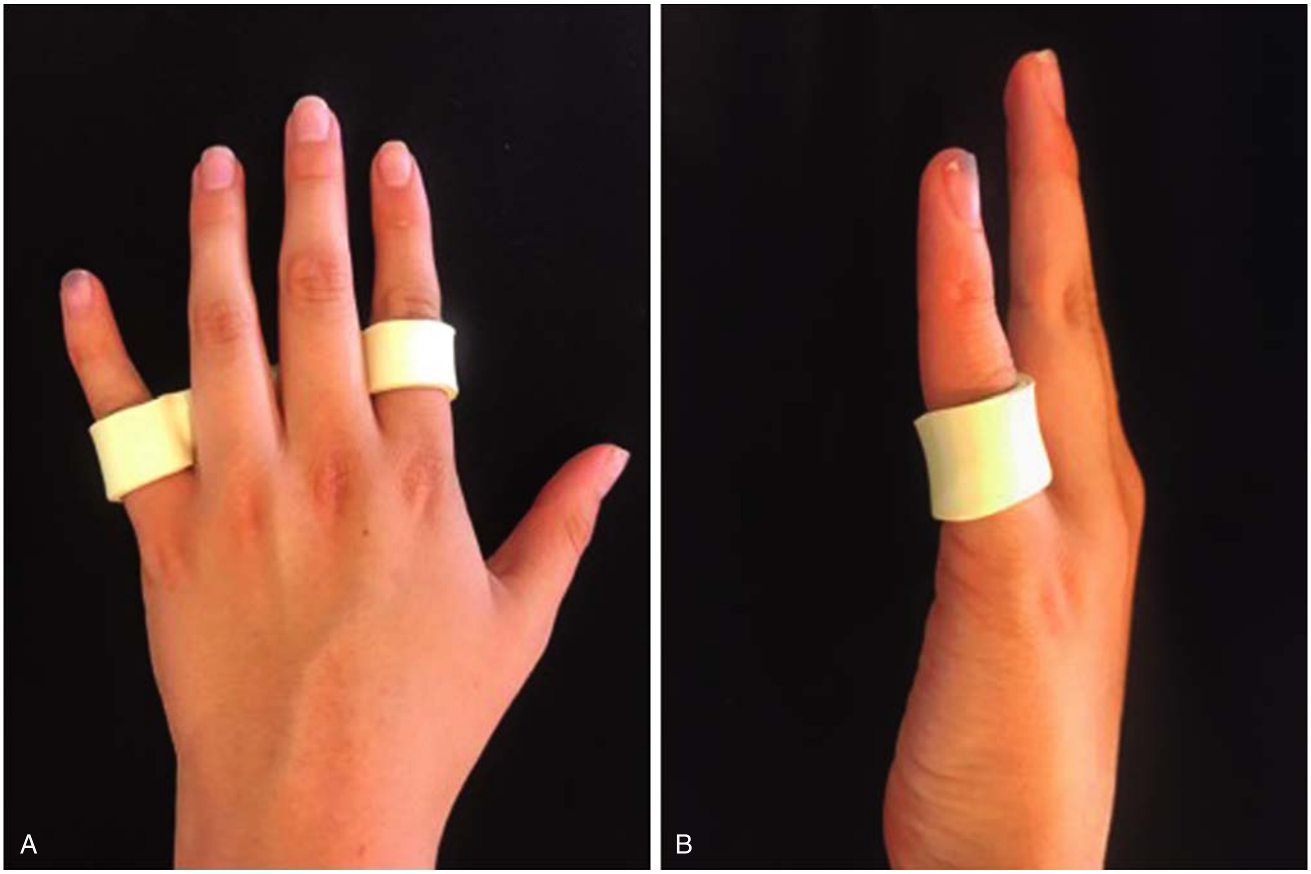

Fig. 28.7 Active redirection orthosis. **A,** Dorsal view of a small custom-molded orthosis that blocks metacarpophalangeal joint extension of the little finger to drive the extension force to the stiffer proximal interphalangeal joint. **B,** Lateral view of active redirection orthosis.

Fig. 28.8 Active redirection using nonremovable casts. **A**, Intrinsic minus cast with a dorsal hood that is positioned over the fingers so the distal interphalangeal (DIP) joints are in a starting position of relatively greater flexion than the proximal interphalangeal joints. This positioning facilitates initiation of finger flexion at the DIP joint(s). **B,** Intrinsic minus cast without a hood that provides optimal position to facilitate long-flexor glide and for active mobilization of the fingers into flexion. **C,** Intrinsic plus cast with a hood that provides optimal position to improve metacarpophalangeal joint flexion and composite flexion of the fingers.

CLINICAL APPLICATION OF CASTING MOTION TO MOBILIZE STIFFNESS

The CMMS technique is the use of plaster of Paris casting to selectively immobilize proximal joints in a desired position while constraining distal joints so that they move in a prescribed direction and range.[21] Unlike more traditional treatment methods, the CMMS technique aims to simultaneously mobilize stiff joints, reduce edema, and normalize the pattern of motion and its cortical representation. Traditional manual mobilization techniques and mobilization orthoses may be less effective in the chronically stiff hand than in the newly stiff hand because these techniques are intermittent and address only one problem at a time.

The CMMS technique challenges traditional treatment approaches in several ways. First and most dramatically, active motion can be effective in restoring joint mobility without the use of passive joint exercises. The CMMS technique utilizes no passive motion, modality, or manual treatment. Additionally, the cast immobilizes proximal joints, allowing only the stiff joints to move in the desired range and direction. CMMS focuses on gaining the motion that is needed most even though it may temporarily cause a loss of motion in the opposite direction or a loss of motion in the immobilized joints. This approach also challenges the common assumption that one should not allow gains in motion in one direction at the expense of the other direction. In the chronically stiff hand, the balance of motion is overwhelmingly in favor of the stiff pattern. The constrained motion within the cast allows the opposite pattern of motion to become dominant while simultaneously mobilizing adherent tissues and the stagnant edema. Reeducation of a more normal pattern of motion is facilitated by the restraints imposed by the cast and, when the patient is weaned from it, the motions temporarily lost while in the cast quickly return (unless there is some specific anatomic injury preventing such return). In cases of altered anatomy after injury, the therapist must understand that the reconstructed anatomy has the potential to return to the balanced motion before applying the CMMS technique. The same concern is not applicable to stiffness of uninjured joints resulting from immobilization.

The CMMS technique can successfully mobilize severe stiffness that is unresponsive to traditional treatment.[25] Because the patient is mobilizing only with active motion, treatment is not painful. Therapy sessions consist of reevaluation, cast changes, and home instructions, creating a cost-effective treatment approach that is overwhelming for neither the therapist nor the patient. As functional motion is regained, a very slow weaning from the cast is begun that allows for functional use of the hand to continue the progression of mobilization.

GENERAL PRINCIPLES

The wrist must always be positioned in slight extension and included in the cast so that the extrinsic muscle power is directed toward the digits. Because the extrinsic flexor and extensor muscles strongly influence digital motion, this position facilitates the most effective transmission of force to the joints of the hand to mobilize them into flexion. Even if the stiffness is limited to only one digit, the other digits should be included in the cast to allow the cortical representation of the uninjured digits to assist with accurate motion.[56] The only exception to this may be to allow slightly greater freedom of motion in the index finger if the stiffness is isolated to the ulnar digits.

It is important to use plaster of Paris for the CMMS technique because of its inherent intimate molding ability.[21] Other synthetic casting materials are more rigid and have sharp edges. Thermoplastic materials should not be substituted for the plaster of Paris because they are readily removed, and with prolonged wear, skin tolerance is poor. Only in cases in which the stiffness is not yet chronic and shorter periods of exercise are effective can the principles of the CMMS technique be applied with thermoplastic materials.

TREATMENT GUIDELINES

It is difficult to define detailed treatment protocols for the CMMS technique, but general guidelines are given in the following sections. Each treatment sequence is based on the patient's individual diagnosis, specific causes of tightness and/or lack of glide, pathologic pattern of motion, and response to the CMMS treatment. The therapist must be able to critically evaluate the stiff hand and determine the exact anatomic structures limiting motion. This knowledge determines the specific position needed to harness productive active motion within a cast. The algorithm flow chart (see Fig. 28.9 is an example of the thought process applicable to a stiff hand with limited finger flexion and interosseous muscles tightness.

Cast Design

The design of the CMMS cast is determined by the pattern of motion and location of tightness. The position for immobilizing proximal joints is not arbitrary. For example, if the PIP joint of the little finger is

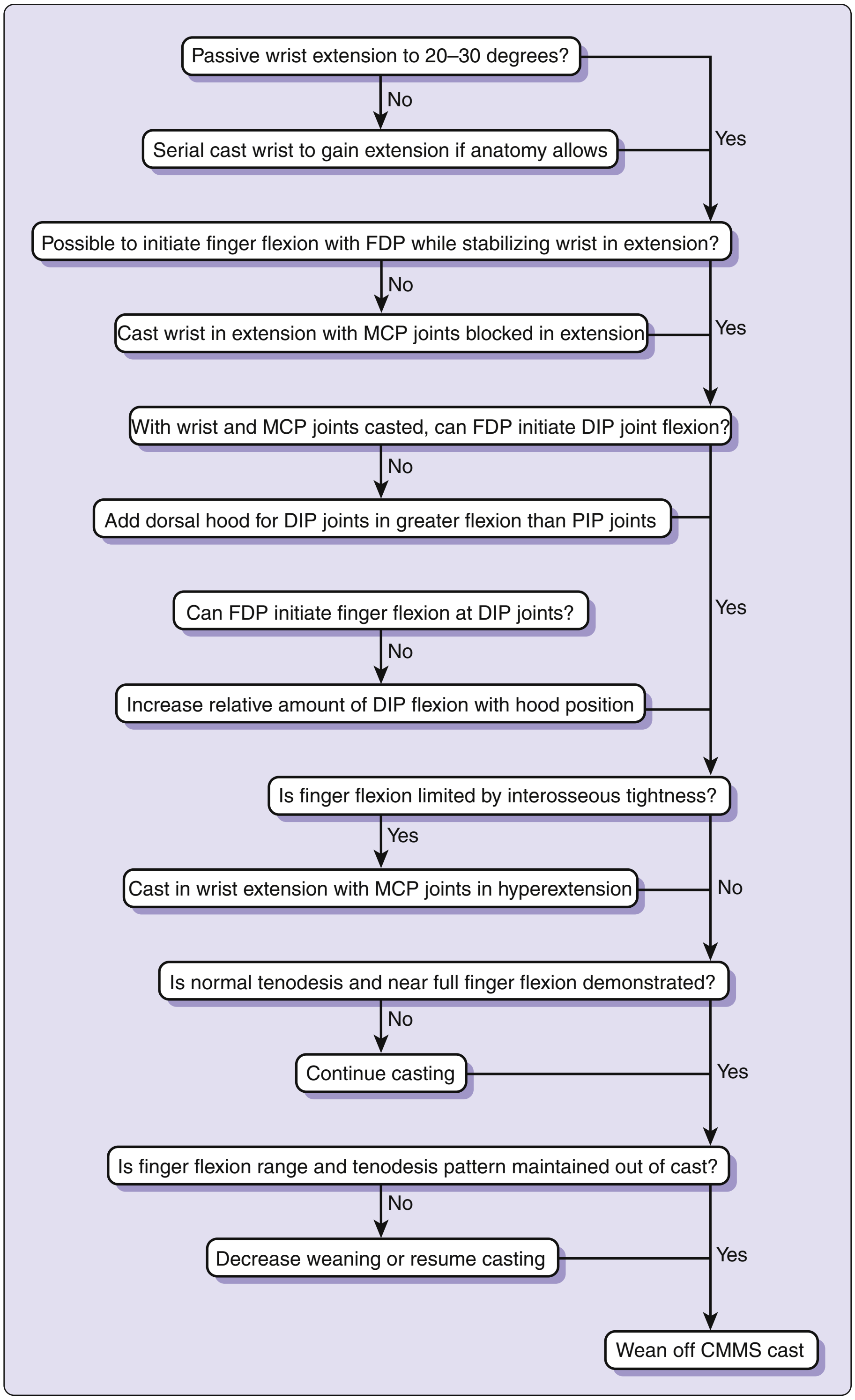

Fig. 28.9 Algorithm outlining the decision-making process for the application of the casting motion to mobilize stiffness technique to regain finger flexion in the chronically stiff hand. *CMMS,* Casting motion to mobilize stiffness; *DIP,* distal interphalangeal; *FDP,* flexor digitorum profundus; *MCP,* metacarpophalangeal. (Copyright Judy C. Colditz, 2008.)

primarily lacking extension, one might choose to immobilize the MCP joints in significant flexion to facilitate proximal excursion of the dorsal apparatus across the joint and invite greater participation of the extensor digitorum communis (see Fig. 28.8C). If the PIP joint of the little finger primarily lacks flexion, placing the MCP joints in full extension (or even hyperextension) drives more extrinsic flexor force across the joint toward flexion (see Fig. 28.8B). If both flexion and extension are equally limited in the little finger PIP joint, a position of about 45 degrees of MCP joint flexion would give the best mechanical advantage for mobility of the PIP joint in both directions.

An arbitrary time period for cast wear is initially chosen. The cast is then removed to reevaluate the active pattern of motion.

Observation of the new, altered, active pattern of motion determines the desired position of the proximal joints and the position of any dorsal blocks in the next cast. When the next cast is applied, usually only the position of the MCP and IP joints is changed. The exception to this would be if there is an element of extrinsic flexor or extensor muscle tightness that requires a change of wrist position.

Dominant Intrinsic-Plus/Dominant Interosseous Flexion Pattern

If someone has a dominant intrinsic-plus/dominant interosseous flexion pattern of motion, the initial cast will immobilize the MP joints in extension, with the IP joints free to capitalize on the extrinsic flexor tendon glide to restore a more normal grasp pattern. In the severely stiff hand with limited passive joint motion, diminished flexor tendon glide, and severe interosseous muscle tightness, it is difficult to mobilize all of these structures simultaneously. In such a circumstance, the initial cast should block the MCP joints in slight flexion. If initially the MCP joints are positioned in full hyperextension, it is more difficult for the patient to pull against the interosseous muscle and joint tightness, and initiate motion with the extrinsic flexor muscles.

Some patients cannot actively initiate IP flexion with the profundus muscles when the MCP joints are stabilized in extension. Although these patients are few, they have the most severe stiffness. To help the patient isolate and gain glide of the flexor tendon(s), a dorsal hood is placed over the IP joints (Fig. 67-28). This hood simply positions the DIP joints in relatively greater flexion than the PIP joints, so when the patient pulls away from the dorsal hood, the motion is initiated first with the FDP muscle(s). The patient is instructed to flex the IP joints by first moving the fingernails away from the hood.

The purpose of the dorsal hood is not to serially push the joints into more flexion but instead to capture the normal relative starting position of active IP joint motion. The hood positions the IP joints in the ideal relationship to one another to assure that finger flexion is instigated by the FDP muscles. Once IP joint motion is partially regained and excursion of the extrinsic flexors is reestablished within the finger, positioning the MCP joints in full extension (hyperextension) in the cast is necessary to elongate the interosseous and lumbrical muscles (Fig. 67-29). A stepwise treatment approach is the most effective; allow the FDP tendon to increase its glide and mobilize the stiff IP joints before also being required to pull against the interosseous muscle–tendon unit tightness.

When full IP flexion is possible while the MCP joints are held hyperextended, the MCP joint immobilization may be slowly discontinued (see later comments on weaning) and full composite finger flexion allowed. This should be considered only when the patient is able to spontaneously initiate flexion with the FDP muscles.

Some patients may however present initially with reasonable IP joint ROM and FDP tendon glide. These patients do not need to wear a cast with the MCP joints blocked in slight flexion but can immediately begin using the "intrinsic muscle stretch" cast in Fig. 67.29.

In the severely stiff hand the range of finger flexion may dramatically improve when the MCP joints are blocked in the initial cast. This dramatic mechanical mobilization may tempt the therapist to begin early weaning and to think that a cast with the MCP joints held in hyperextension is unnecessary. This treatment route will be a disservice to the patient, as it is impossible to have severe chronic limited finger flexion without developing secondary interosseous muscle tightness.

If the intrinsic finger muscles are extremely tight or the patient had great difficulty in regaining profundus glide, a dorsal hood may also be attached to this cast design (Fig. 67.30). The hood is infrequently needed at this stage. Although this cast position of MCP joint hyperextension does not support functional use of the hand, its use is mandatory to reach the ultimate goal of normal ROM and functional use of the hand.

Almost all surgical and therapy texts stress the importance of positioning the MCP joints in flexion to maintain the maximum length of the collateral ligaments. Inclusion of the extended MCP joints in the cast is contradictory to this traditional teaching. It is the increased IP active motion resulting from this position that reduces edema and demands glide of the tendons of the intrinsic muscles across the MCP joint, two factors that outweigh the temporary immobilization of these joints. Much of the cause of limited MP joint flexion is probably edema within the joint capsule, which is loose in extension but compressed in flexion. By blocking the MCP joint in full extension with the cast providing a contoured palmar pressure, edema within the MCP joints is reduced while the intrinsic muscle tendons are gliding past the MCP joint. Active IP joint flexion with the MCP joints blocked in extension demands elongation of the interosseous and lumbrical muscles. Active IP extension with the MCP joint in full extension also demands maximum muscle contraction of the interosseous muscles. Blocking the MP joints in full extension while allowing active IP joint flexion and extension thus elongates and tones the interosseous muscles. Since the interosseous muscles are the prime MCP joint flexor muscle(s), when casting is discontinued, MCP joint flexion can be regained without further specific intervention toward mobilizing the MCP joints into flexion. The only exception is if there is the presence of specific dorsal adherence resulting from dorsal trauma.

The inability to initiate finger flexion with the profundus muscles is commonly seen in the stiff hand following immobilization for a distal radius fracture or other trauma. In the initial CMMS cast, the desired relational position of DIP and PIP joint flexion may not be attainable because of joint stiffness, especially the DIP joint. After a few days in the cast the patient will gain digital flexion, but it is usually with the flexor digitorum superficialis (FDS) muscles rather than with the FDP muscles. If this occurs, the addition of a small piece of plaster of Paris or thermoplastic material over the distal edge of the dorsal hood (just over the distal phalanx) will aid in greater DIP joint flexion as the patient moves cyclically. It is important that each time the patient actively flexes, the fingernails move away from the dorsal hood before the PIP joint moves. The patient must look at and think about initiating this active motion to regain glide of the FDP tendon(s).

The complex decision-making process of applying the CMMS technique for the most common problem of regaining digital flexion in the chronically stiff hand is illustrated in Figure 67-31. This illustration only applies to stiff hands lacking full finger flexion.

Dominant Intrinsic-Minus/Dominant Extrinsic Flexion Pattern

Although the dominant intrinsic flexion pattern discussed above is the most common pattern of stiffness seen in the hand, other patterns of stiffness present that require other cast designs to effect change.

To regain MCP joint flexion, the wrist cast is applied in slight extension and a dorsal hood is placed only over the proximal phalanges, positioning them at their easy available maximum passive flexion range (Fig. 67.32). Care must be taken to assure the cast does not extend too far distally on the palmar surface, thus blocking MCP joint flexion. The patient works to actively pull the proximal phalanx away from the dorsal hood while flexing only the PIP joint with the superficialis muscle. The patient is instructed to place the fingertips on the cast and to "slide" the fingertips proximally. This exercise isolates the interosseous muscles, and cyclic loading increases the range of MCP joint flexion.

As MCP joint flexion increases, the cast may be changed to create a starting position of somewhat greater MCP joint flexion (Fig. 67-33). A small pad can also be inserted between the proximal phalanx and the dorsal block to position the MCP joints in slightly more flexion. The purpose is not to serially position the MCP joints in maximum flexion and hold them there, but instead to position the MCP joints in slightly greater flexion so active MCP joint flexion is within the end-range. The cast never holds the MCP joint immobile, because there is always room for the movement into and away from end-range flexion.

Cast Exercises

The importance of teaching the patient the correct cast exercises and how to balance exercise and rest cannot be overstated. It is key to regaining both active and passive motion and to reinforce the neural pathways responsible for efficient grasp. The patient will be instructed to perform exercises hourly, at least 15 to 20 repetitions, during all waking hours. Explicit instructions should be given to perform active exercises only, avoiding passive stretching, manual treatments, and any other modalities while in the cast, however tempting it may be. Regaining motion is both a complex cerebral and mechanical challenge. Therefore, patients must also be encouraged to consciously perform the exercises by looking at their fingers during active motion.

As with any cast wear, the patient should be instructed to move all uninvolved joints (i.e. shoulder, elbow) in all planes daily to prevent resultant joint stiffness.

If Casted in MCP Joint Extension

The patient will perform hook fists every hour actively. Cues are given to complete the entire movement, reaching end range at both flexion and extension, along with instructions to hold each motion for 2 to 3 seconds (vs. "wiggling" the fingers). A helpful technique can be to place dots using a marker on each of the patient's most distal finger pads and four other dots at the base of each finger and ask the patient to "connect the dots" during their exercises.

If Casted with a Dorsal Block Over the Metacarpophalangeal Joint

In this cast, the patient will be allowed MP flexion but not extension as the volar part of the cast will clear the distal palmar crease. (See Fig 28.8 C). The patient will perform two exercises at this point: hourly hook fists (as described earlier) and MCP joint flexion. This exercise should be performed with the PIP joints in slight, relaxed flexion. The emphasis is on moving the proximal phalanx away from the cast, and the cast provides visible feedback for the patient as to her or his progress.

Time in the Cast

The most challenging aspect of the CMMS treatment for therapists is the amount of time required in the cast to repattern the motor cortex and regain joint mobility in the chronically stiff hand. In Noyes' study of immobilization of monkey knees, it took 1 year to fully resolve the flexion contracture.[72a] Although initial mechanical gains will be rapid, the cortical change needed for the motion to be permanently retained requires a prolonged period of repeated constrained motion. Patients with chronic stiffness may wear the cast for many weeks or a few months with few or no cast changes. It is important to take into account the duration of time the hand has been stiff with an altered movement pattern. The longer this nonproductive pattern has been present, the longer the time required in the cast for the cortical change to be enduring (Fig. 28.10). Although this seems like a protracted period for the therapist, one must keep in mind that it is really a short period relative to the time the stiffness has been present. When the patient starts to regain motion in the hand, the therapist may be tempted to begin the weaning process. Experience has proven that this approach is fruitless because the patient immediately reverts to the old maladapted pattern of motion that has remained dominant in the motor cortex, and the stiffness returns. A weaning process is necessary.

Weaning Process

After a prolonged period of full-time cast wear, a period of slow weaning must occur to ensure that the patient can retain the active motion gained. The patient will quickly fatigue and revert to the previous maladaptive pattern of movement because of weakness resulting from the chronic stiffness as well as from partial immobilization in the cast. Initially, time spent out of the cast should be short. The frequency of the short periods of time spent out of the cast should be increased before each time period is increased.

When the patient is able to display the desired ROM out of the cast in addition to demonstrating a spontaneous tenodesis pattern, slow weaning can begin. The cast is opened on the radial and ulnar aspects, but the underneath padding and stockinette are cut only on the radial side. The edges of the opened cast are covered with adhesive tape to secure the padding and stockinette and to cover the raw edges of the plaster of Paris. Circumferential hook-and-loop straps are then applied. The cast can then be removed and reapplied to allow weaning to slowly begin (Fig. 28.11).

The weaning process starts with brief (~15-minute) periods out of the cast a few times a day. The patient works actively on nonresistive tasks that use the tenodesis pattern and concentrates on moving in the correct active pattern. After 1 or 2 weeks of slightly increasing the number of times out of the cast, functional activities are added that purposefully use the desired motion but do not provide excessive resistance. The patient learns to identify when the pattern of motion is disintegrating and returns to the cast. Awareness of how the hand should be used ensures that all motions out of the cast reinforce the gains made while in the cast.

Therapists are cautioned at this time to avoid focusing on regaining motion in the opposite direction. Only when the desired motion has been regained and the patient can maintain the motion out of the cast is emphasis placed on regaining motion in the opposite direction. In most cases, the motion will slowly return with normal hand use. The focus of therapy should remain on functional active motion.

Patients who are weaned too quickly and revert to an abnormal pattern of motion will require a period of repeated casting.

Requirements and Contraindications

The CMMS technique requires skill in the application and removal of a well-fitted and comfortable plaster of Paris cast. The cast must be applied with care to distribute pressure evenly and to block the joints needed to redirect motion to the target joints. Excessive exothermic reaction of the hardening plaster of Paris must be avoided.[73,74] Claustrophobic patients may not be able to tolerate the confines of the cast, and the technique should be applied judiciously with this population. The circumferential cast should never be applied to acute injuries, especially if vascular instability is present. In consultation with the referring surgeon, it may be used in select postsurgical cases such as flexor tenolysis to facilitate correct tendon glide but only after a few days in the postoperative compressive dressing.

This technique should not be indiscriminately applied to all patients with chronic stiffness. In the cases of severe trauma, the anatomic changes from an injury may eliminate the potential for regaining balanced motion, and the loss of motion created by the CMMS casting may not be regained.

Historically, a great deal of time, effort, and pain endurance has been required to restore motion to the chronically stiff hand.[9] In the

Fig. 28.10 Duration of casting required to resolve digital stiffness.

Fig. 28.11 Bivalve cast for the weaning phase. Hook-and-loop straps applied to secure it when reapplied.

era of increasing cost–benefit analysis, the amount of motion regained relative to the time and energy expended on treatment must be an efficient return. The CMMS treatment method simplifies the treatment approach, and even a severely stiff hand can be mobilized with only a few therapy visits and cast changes extended over a number of months. It is important to note that the CMMS treatment method was developed by Judy Colditz, OTR, CHT,[72] and although there is sound theoretical basis and the authors' clinical experience supporting this technique, prospective clinical research studies are needed to support its efficacy.

SUMMARY

Understanding the causes of stiffness in the hand as well as choosing the appropriate type and timing of intervention are fundamental to the successful mobilization of the stiff hand. A gentle approach aimed at reducing edema and avoiding stimulation of an inflammatory response is required. Gentle manual stretching, active motion, and use of the hand in conjunction with timely mobilizing orthoses can effectively transform a newly stiff hand into a mobile one. When stiffness and edema are prolonged and chronic joint tightness and tissue adherence limit motion and a nonfunctional pattern of motion results, the CMMS technique can be used to address these complex interrelated problems.

The delicate balance between tissue glide and freedom of motion can be restored, even after severe hand injuries, if treatment is provided based on a sound understanding of and respect for tissue response and the healing continuum.

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