27

Comprehensive Rehabilitation of the Diseased and Injured Upper Extremity

Laurence A. Kirwan
and John A. Boswick, Jr.

It is well recognized that many minor injuries and disease processes of isolated joints and areas of the hand often result in severe impairment of function of the upper extremity. This chapter reviews the principles of rehabilitation of the upper extremity following disease and injury and presents techniques of management that will prevent or minimize loss of motion and decrease impairment of function.

Restoration of hand function and pain relief are the goals of rehabilitation of the diseased or injured upper extremity. The upper limb must be considered as a whole. Emotional instability and neuroticism are factors that predispose a patient to chronic pain. These must be evaluated and treated also.

Rehabilitation, as defined by the *Oxford English Dictionary,* is "the action of replacing a thing or restoring it to its previous condition or status." It may be argued that it is impossible to improve on a normal hand and that an injured hand is never normal again. If so, the term *rehabilitation* is incorrect. The aim of therapy is a level of recovery consistent with our previous experience of what is "possible." Often this will fall short of the preinjury or predisease level of function.

Two statements that encapsulate our philosophy and practice over the past decade are that "Early motion prevents stiffness," and that "The best form of occupational therapy is the person's own work."

"All uninjured parts should be kept unrestrained and free to move."[1] Patients are instructed on the operating table at the termination of surgery and again in the recovery room to exercise their shoulder and elbow as well as the unsplinted parts of their affected hand. The patient should actively abduct the shoulder to the side of the head and flex the elbow until the fingers touch the shoulder, in addition to exercising the wrist and fingers, 4 to 5 minutes every hour while awake. As postoperative pain and inflammation settle, exercises are performed for longer periods of time (25 to 30 minutes) two to three times daily.

The stiff hand is frequently stiff in several joints—the wrist, the metacarpophalangeal (MP) joints, the interphalangeal (IP) joints—rather than one or two individual ones. Stiffness may follow trauma or disease such as an infection or an arthropathy, for example, rheumatoid arthritis, dermatomyositis, or systemic lupus erythematosus.

The indications for rehabilitation are:

- a stiff hand with loss of motion in one or more joints in the hand, wrist, elbow, or shoulder
- loss of strength
- loss of dexterity
- sensory impairment—either diminished sensibility (protective or discriminative) or increased sensibility (hypersensitivity)
- swelling (edema)
- amputation, requiring reeducation, with or without a prosthesis
- pain
- emotional disturbance.

Acute Pain Versus Chronic Stiffness

Stiffness is sometimes described as acute or chronic. Acute stiffness is a result of the acute pain and the edema that follow injury or disease. It can usually be treated quickly with short-term measures, such as elevation and analgesics. In contrast, chronic stiffness is often painless unless attempts are made to move the tissues. Chronic stiffness does not result from pain or edema but is due to structural changes, for example, contractures of ligaments or adhesions of the joint capsule and tendons. Treatment is lengthy and the return of function is often incomplete. Peacock[2] described the fixation, which can occur in chronic stiffness, of joint ligaments to bone by new collagen synthesis in areas normally free of such attachments.

Preliminary measurements and tests are an integral part of therapy. They include grip and pinch strength, two-point discrimination, and active and passive range of motion. Serial measurements enable one to monitor progress and adjust therapy as indicated.

The causes of limitation of motion are tight skin, tight tendons or muscles, tight ligaments, tight joint capsules, and obstructions to motion such as adhesions (within a tendon sheath or a joint) or a bony block.

Tight Skin

Skin tightness can be distinguished by blanching of the overlying skin when the joint is stretched, for example, the dorsum of the MP joints following burn injury. Adherence of skin to underlying structures is diagnosed by observing skin puckering when the underlying tendons, ligaments, or muscles move.

Tight Tendons or Muscles

Tight extrinsic tendons and muscle-tendon units are diagnosed by examining for a tenodesis effect. For example, if the deep flexors are tight, as in Volkmann's ischemic contracture, wrist extension results in flexion of the MP and IP joints, which cannot be extended unless the wrist is flexed. Tightness of the intrinsic muscles is diagnosed by testing for laxity of flexion at the proximal interphalangeal (PIP) joint when the MP joint is extended. Differential tightness of the radial and ulnar intrinsics can be evaluated by deviation of the finger at the MP joint in an ulnar or radial direction, respectively. A tight oblique retinacular ligament (Landsmeer's ligament) is diagnosed by testing for laxity of the distal interphalangeal (DIP) joint when the PIP joint is extended.

Tight Ligaments

The collateral ligaments of the MP joints are tight in flexion and lax in extension. Contracture of these ligaments in extension prevents active or passive flexion of the joint. In contrast, the collateral ligaments of the IP joints are of the same length in all positions. Contractures of these ligaments may result in loss of flexion or extension dependent on a variety of factors (Table 27-1).

Tight Joint Capsules

Tight capsules, like tight ligaments, are detected when motion at the joint is unaltered by motion at adjacent joints. Adhesions between the volar plate and the overlying bone in the cul-de-sac proximal to the joint space may limit flexion or extension depending on the position in which the adhesions form.[3]

Obstructions to Motion

Adhesions within a joint or a tendon sheath, exostoses, or malunited fractures may be responsible for loss of joint motion. If the obstruction is springy, it is likely that movement can be improved, but if the block is abrupt, it is unlikely to improve with nonoperative therapy. Passive range that exceeds active range is due to tendon injury, tendon adhesions, or nerve injury.

A good range of motion is still possible in the IP joints, even with signs of significant radiological derangement. The reason for stiffness in such cases should be sought in tight collateral ligaments, tight intrinsics, or in other causes rather than bony incongruity (Table 27-1).

Specific Rehabilitation Techniques

A variety of techniques are used to restore function and rehabilitate the diseased or injured upper extremity. They include:

- active motion
- active assisted motion
- passive motion
- splinting (dynamic and static)
- sensory reeducation
- compression
- massage
- elevation
- biofeedback
- heat
- pain reduction
- drugs.

Active Motion

When the splint is removed at approximately 3 weeks after a tendon repair, active motion is allowed in the line of pull of the muscle tendon unit. Gentle

Table 27-1 Causes of PIP Joint Stiffness in Flexion and Extension

Flexion
Check-rein ligaments
PIP joint synovitis or effusion
Digital, wrist, or palmar flexor tenosynovitis (including trigger finger)
Flexor contracture or adhesions
Collateral ligament contracture or adhesions
Volar plate adhesions to bone
Capsular contracture (PIP joint)
Bony block
Joint fibrosis, ankylosis, or articular incongruity
Palmar skin contracture
Extension
Intrinsic muscle contracture or adhesions
Intrinsic tightness secondary to MP joint synovitis or contracture
Intrinsic muscle shortening secondary to mallet deformity (DIP joint)
PIP joint synovitis or effusion
Digital, wrist, or palmar flexor tenosynovitis
Distal profundus entrapment
Extensor contracture or adhesion
Collateral ligament contracture or adhesions
Volar plate adhesions to bone
Capsular contracture (PIP joint)
Retinacular ligament contracture or adhesions
Bony block
Joint fibrosis, ankylosis, or articular incongruity
Dorsal skin contracture

active motion is allowed in the opposite direction also. No further splintage is recommended unless the patient is untrustworthy because of infirmity or personality disorder or because his or her occupation may place unreasonable stress on the repair. Gentle resistive exercises are not permitted until approximately 6 weeks after repair.

Aggressive resistive exercises as well as passive stretching in a direction opposite to the pull of the repaired tendon are permitted 8 weeks after tendon surgery (repair or transfer). When restoring motion it is unnecessary to achieve 90° of flexion at the MP joints. Although normal MP joints will flex to 90°, the normal functional range is 75° at the index and middle fingers, 80° at the ring finger, and 80° to 85° at the little finger. Similarly, less flexion is required at the PIP joints of the index and long fingers and relatively more in the ring and little fingers. The index and long fingers are important for pinch, whereas the ring and little fingers are more important for power grip.

Improvement in active motion is the best sign of patient progress. Active motion is the safest and most effective exercise. If it achieves satisfactory results, there is no indication for other forms of treatment. However, in certain cases active motion may be harmful, for example, following tendon repair or transfer where active contraction may weaken or rupture the repair if applied too early. Active exercise such as making a fist has the added advantage of aiding venous return and decreasing edema.

Graded exercises against resistance are used to increase the strength and pull-through of tendons, provided they are well healed. The increased force will stretch adhesions more effectively than will unresisted active exercises alone. Resistive exercises are also of use in rehabilitating muscles after nerve injury.

Overly aggressive therapy by the patient or the therapist can cause an "overuse" syndrome resulting in an acute inflammatory reaction. Before initiating an exercise program, active or passive, any inflammatory response should have resolved, and edema in the limb must be reduced using one of the methods described later.

Active contraction may be hindered by co-contraction of antagonistic muscles. This may be a conscious or a subconscious attempt by the patient to prevent movement that he or she fears. Co-contraction can be avoided by showing the patient which muscles are contracting and by the use of biofeedback instruments. Electrodes that record muscle contractions are used to let patients know when they are contracting the muscle-tendon unit that has been injured or diseased (agonist) or its antagonist (co-contraction; see "Biofeedback" below).

Early motion prevents stiffness. It has been our policy to instruct the patient regarding exercises immediately after surgery, while still in the operating room, and again in the recovery room. The patient is instructed to move all the unsplinted parts of the hands, as well as the shoulder and the elbow. Initially, exercises should be performed for 4 to 5 minutes every hour while awake and later less frequently for 25 to 30 minutes two to three times daily. An early, aggressive exercise program will prevent most of the complications of immobi-

lization. Between active exercises, the parts may be splinted to prevent deformity. This is especially important in the burned upper extremity where adduction contractures of the shoulders, flexion contractures of the elbows and wrists, hyperextension deformities of the MP joints, flexion contractures of the IP joints, and adduction deformities of the thumb (Fig 27-1) may develop.

In the unburned patient, complications such as frozen shoulder may be prevented by early active motion. When active motion is impossible, passive exercises will help to retain a full range of motion. If the patient understands and is able to follow instructions, he or she need not be seen again until the dressing needs changing, which may be 2 to 3 weeks after surgery. If the patient fails to follow the initial instructions, he or she is seen as often as necessary until the exercises are being performed correctly.

Active Assisted Motion

When indicated, the proper use of active assisted motion plays a major role in the rehabilitation of the injured or diseased upper extremity. It is commonly used after tendon or joint surgery. It enables patients to increase the gains they might achieve with active motion alone. The best example of this is the so-called "blocking" of joints. If the available excursion of a tendon is used in flexing, or extending a joint proximal to the site of tendon injury, the excursion at the site of repair will be minimal. To increase distal tendon excursion, the proximal

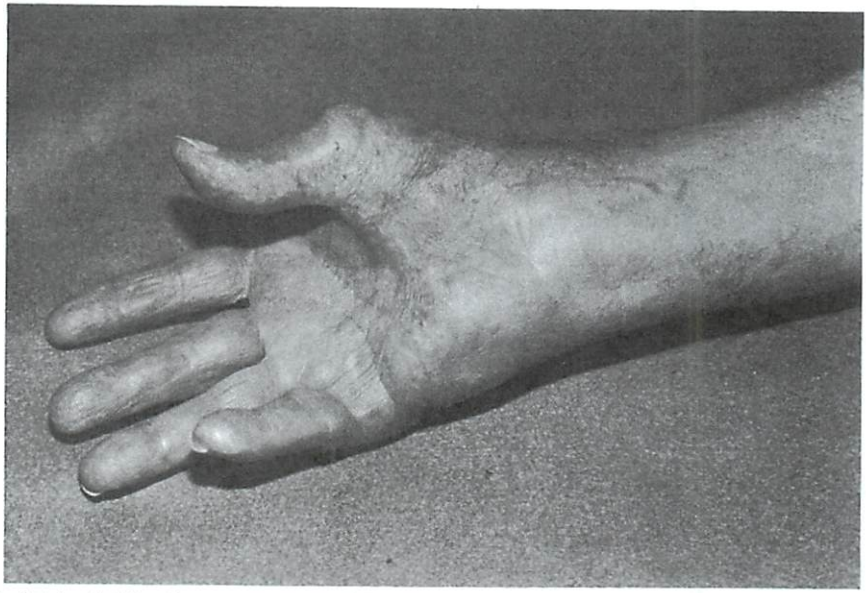

Fig 27-1. Adduction contracture of the thumb following partial-thickness burn injury to the first web.

joints can be blocked. Blocking is done with a finger or with a wood block. It should be started gently and is contraindicated in the early stages following tendon repair or transfer, lest it cause stretching or rupture of the tendon juncture. As time goes on and the repair becomes stronger, the proximal joints may be blocked in progressive degrees of extension or flexion for a flexor or extensor tendon repair, respectively.

Passive Motion

Passive motion is used to maintain or increase movement when active motion is insufficient or impossible, for example, as a result of tendon or nerve interruption. The patient must be able to distinguish between a sensation of pain and a sensation of stretch. The first must be avoided and the latter encouraged. Pain destroys the confidence of the patient and excites an inflammatory response which may cause further pain, stiffness, and scarring with subsequent loss of motion. Slow, gradual gains are safer than sudden, painful ones.

It should be noted that the tight conformity of the ligaments of the finger joints may impede gains in motion.[1] Distraction of the joint with a gentle axial pull, before therapy, stretches the joint capsule and collateral ligaments and may increase the range of motion subsequently obtained.

Passive motion can be increased with the aid of a splint, either static or dynamic (see "Dynamic and Static Splinting" below). A common static device for this purpose is the web strap. This is a simple strap which is tightened around the flexed finger, compressing the distal against the proximal phalanx. It increases motion at the extremes of the normal range.

Following a tendon repair in zone II, passive motion in the direction of the tendon pull is permitted in the immediate postoperative period. Passive motion in a direction opposite to the pull of the repaired tendon is the last activity to be allowed and is not permitted until the patient has been out of the splint for at least 4 weeks. If passive extension is indicated early, for example, because of incipient PIP joint contracture, it is applied to one joint at a time, keeping the adjacent joints flexed or extended so as to prevent tension on the repair. For example, the PIP joint can be extended after a flexor tendon repair, provided the MP joint and the wrist are fully flexed.

Dynamic and Static Splinting

The art of splint making has progressed from devices made of metal, cloth, or plaster, requiring great skill and time to fashion, to the present-day thermoplastics which can be molded accurately and adjusted quickly. In this way, hand splints have become readily available, disposable, and almost infinitely adjustable. Indeed, splint making is so deceptively easy that there is a danger of important mechanical principles being ignored[4] as well as simpler methods of treatment such as those already mentioned.

Static Splints

Static splinting may cause increased stiffness and should be interspersed with exercises and other measures to prevent stiffness and reduce swelling.

A common static splint is the wrist cock-up splint. It keeps the wrist in moderate dorsiflexion and by its tenodesis effect causes the MP joints and IP joints to flex, as well as the thumb to be abducted. Other factors that help maintain a functional posture in the wrist cock-up splint are activation of the intrinsic muscles and gravity.

Static splints may help to prevent deformity after burn injury. If indicated, the shoulder is splinted at 90° of abduction, the elbow in almost full extension, and the hand in the intrinsic plus position; the wrist is moderately dorsiflexed, the MP joints flexed 30° to 40°, the IP joints in almost full extension, and the thumb in palmar abduction parallel to the index metacarpal.

If the hand is not splinted, the wrist may fall into flexion and the hand may adopt a claw posture with hyperextension of the MP joints, flexion of the IP joints, loss of the transverse metacarpal arch, and contraction of the first web space (Fig 27-2).

All joints in the upper extremity may be deformed secondary to burn scar contracture, which usually occurs on the concave aspect of the joint. The exception is the MP joint, which contracts in a position of hyperextension. Timely static splinting interspersed with active and/or passive motion can prevent these deformities.

A splint designed to maintain elbow extension or shoulder abduction should not only immobilize the joint in this position (eg, an airplane splint for the

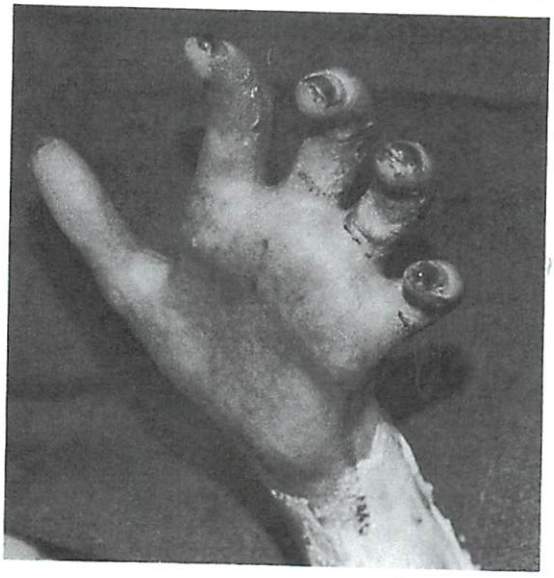

Fig 27-2. Claw hand posture following dorsal burns.

splint substitutes for intrinsic function by flexing the MP joints before the IP joints. Without the splint, digital balance is lost. The fingers will flex initially at the IP joints and then at the MP joints, without providing a wide enough grasp to enable the hand to pick up objects.

Following a radial nerve injury a static splint can be used to maintain the wrist in dorsiflexion with the thumb and fingers suspended in slings attached to rubber bands. This not only places the hand in a functional position for grasp but also permits resisted flexion exercises of the fingers and thumb against the dynamic traction and therefore helps to maintain power in the antagonistic muscles.

Dynamic Splints

Dynamic splints increase desirable movements, and static ones prevent undesirable movements (although static splints can also increase motion if used as serial splints, eg, finger casts for IP joint contractures).

Dynamic traction may be used early after tendon repair in zone II. Active extension is accompanied by synchronous relaxation of the finger flexors. Elastic bands flex the fingers passively, preventing active flexion. As a result of early motion, adhesions may be avoided or occur in such a manner as not to restrict tendon excursion.

In other conditions, dynamic traction may also be indicated, with or without static splints, if there is still significant stiffness after active and passive exercises.

Dynamic traction may be started when active motion plus a tolerance to mild stretch is present and as soon as the acute inflammatory response subsides (except following tendon repair, see above). The time to start therapy is assessed on an individual basis.

If therapy is begun too early or applied too vigorously, inflammation, new scarring, and increased stiffness will result. Other possible complications of dynamic traction are increased stiffness in other joints and injury due to failure to understand the mechanical forces involved.

Increased stiffness may be due to a bulky splint obstructing other joints as well as to lack of movement in the opposite direction from that in which traction is applied. Dynamic splinting may improve mobility in one plane at the expense of motion in the other planes. Hence, stiffness may occur in the other planes of motion. To prevent this, traction should be alternated with static or dynamic splinting in other planes or with active and passive exercises out of the splint.

Failure to appreciate the mechanical forces involved can cause skin ulceration or intra-articular damage. For example, when a dorsal splint is made to block MP extension and combined with dynamic traction of the IP joints, the fingers may pivot over the edge of the splint, causing necrosis of dorsal skin unless the splint applies pressure evenly and is designed so that the forces at the end of the finger are counteracted by opposing forces on the hand and dorsum of the proximal phalanx (Figs 27-4 and 27-5). If a joint is subluxed and the volar

shoulder) but should also conform to the contours of the skin so as to apply constant pressure to the underlying scar tissue. This encourages the healed burn to mature in the stretched position. If the splint is not conforming, bands of contracted scar cut corners, obliterating flexion creases and recreating the deformity when splinting is discontinued. In the hand, wedges of foam or plastic spacers can be inserted into the web spaces with compression, to prevent burn syndactyly.

Static splints can substitute for loss of muscle action after nerve injury or disease. Following median nerve injury, a thumb abduction splint prevents contracture of the first web space and overstretching of the thenar muscles, which would prevent recovery of full strength after nerve regeneration.

Following an ulnar nerve injury, early application of a static splint maintains the MP joints in flexion, preventing a hyperextension deformity which might lead to a collateral ligament contracture as well as overstretching of the volar plate. The splint allows active extension of the IP joints by preventing hyperextension at the MP joint[5] (Fig. 27-3). When the patient tries to grip an object, the

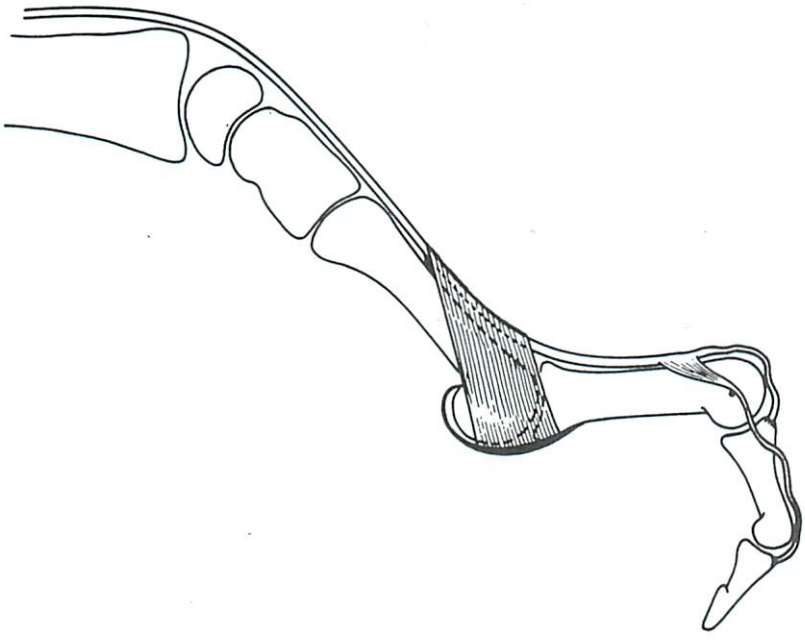

Fig 27-3. When the MP joint is hyperextended, the sagittal bands run progressively more transversely, finally blocking the extensor tendon. When thus blocked, the extensor tendon has less control over the IP joints, which then tend to fall into flexion. Source: Reprinted from "Intrinsic Muscles of the Fingers: Function, Dysfunction, and Surgical Reconstruction" by Smith RJ, *American Academy of Orthopaedic Surgeons Instructional Course Lectures* 24:203, with permission of CV Mosby Co, Copyright © 1975.

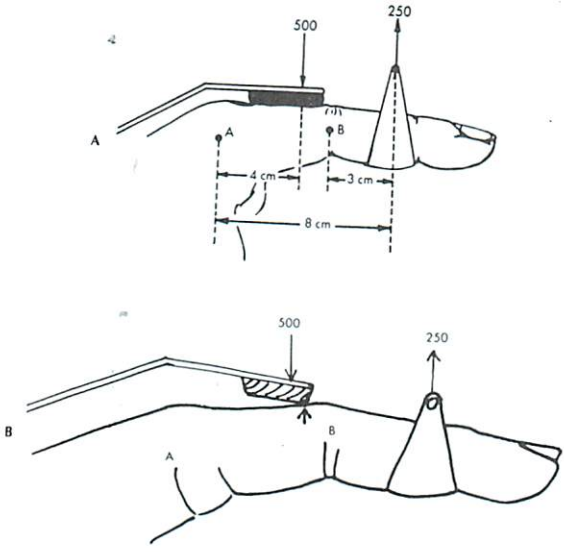

Fig 27-4. *a,* Diagram of finger subjected to force of 250 g through a sling 3 cm from the axis of the IP joint *(B).* The proximal segment of the finger is held in flexion by a felt pad, which is 4 cm from the axis of the MP joint *(A).* This pad receives a thrust of 500 g to balance the force on the sling (500 g × 4 cm = 250 g × 8 cm). *b,* Same finger as in *a,* but because the proximal part of the hand was not supported, the whole finger has tilted. Now only the edge of the felt pad presses on the finger, creating high pressure. Source: Reprinted from "The Forces of Dynamic Splinting: Ten Questions Before Applying a Dynamic Splint to the Hand" by Brand PW, in Hunter JM et al (eds): *Rehabilitation of the Hand,* ed 2, with permission of CV Mosby Co, Copyright © 1984.

plate and pericapsular structures are tight, as in the stiff type of rheumatoid arthritis, traction applied distally, instead of producing a gliding motion of the joint surfaces, may pivot the distal articular surface on the proximal. Pressure necrosis of the compressed articular surfaces may result. This is prevented by applying traction close to the joint so as to minimize the force applied and avoid angulation, thus encouraging a gliding motion of the joint surfaces.[4]

Dynamic splinting is monitored by measuring the volume of the hand and the skin temperature at affected joints. Frequent visual inspection for swelling, redness, and tenderness is essential. Increased volume of the whole or a part of the hand, as well as elevated temperature of a joint, are additional signs of inflammation. If any of these signs are present, traction should be discontinued until the process subsides.

A dynamic splint may constrict tissues, preventing venous return. Therefore splinting is used after the initial postoperative edema has settled but before intractable stiffness has occurred.

A hand that responds well to gentle passive stretch may receive no benefit from a dynamic splint. However, a joint that fails to respond to passive stretch

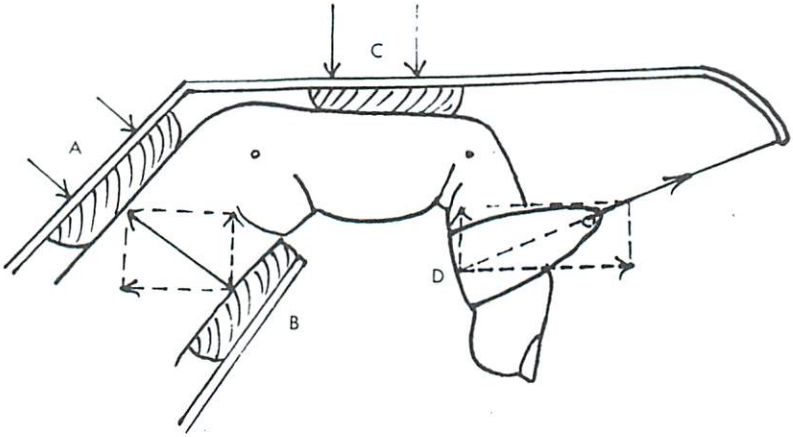

Fig 27-5. Vector diagram showing how both horizontal and vertical forces need to be balanced. Whereas in Fig 4*a* the sling was pulling dorsally, here it pulls obliquely because the IP joint is flexed. The horizontal vector at *D* is double the dorsal vector. Thus, the felt pad *(C),* which balances the dorsal force, will not take much force. However, the felt pad in the palm *(B)* must be capable of considerable horizontal restraint. It also has a dorsal vector *(C)* which keeps the hand snug against the pad and prevents titling as shown in Fig 4*b*. Pad *A* has no force to exert or absorb. It is there to stabilize against B and prevent unexpected shifts of position of the hand as a whole. Source: Reprinted from "The Forces of Dynamic Splinting: Ten Questions Before Applying a Dynamic Splint to the Hand" by Brand PW, in Hunter JM et al (eds): *Rehabilitation of the Hand,* ed 2, with permission of CV Mosby Co, Copyright © 1984.

or that has an almost full range of motion, but shows significant stiffness at the end points, is likely to benefit from dynamic traction.

Dynamic splinting should allow gains in range of motion while avoiding injury and stimulation of edema production through excessive force. *A dynamic splint will increase the passive range of motion but will neither improve power nor greatly affect tendon glide of the involved muscle-tendon units. Active and active resisted exercises must be performed for improvement to occur, particularly in power and also in tendon glide.* However, a dynamic splint can be used for performing active resisted exercises by muscle contraction in a direction opposite to the pull of the elastic bands.

Tight joints and tight muscle-tendon units may be found in the same hand. A dynamic splint is used to increase the passive range of motion of stiff joints, and later it may be used to stretch tight muscle-tendon units.

The aim of dynamic traction should be to maintain gains in movement as well as to increase passive motion between active exercises. Tension must be gentle enough not to excite an inflammatory response. The aim is not to increase the force but to increase the duration in which the force is applied. Ultimately the patient should feel comfortable wearing the splint throughout the day and/or the night.

Splinting throughout the night lessens morning stiffness and makes it easier for the patient to start active motion of the involved part earlier in the morning. Extension splinting is less constrictive and is tolerated better than flexion splinting at night. Flexion splinting is tolerated better in the day when there are distractions to divert the patient's mind from a less-than-comfortable splint.

Dynamic splints are poorly tolerated at night, a static splint may be used with continuation of the dynamic splint during the day.

It is vital to make patients aware that the gains they achieve with splinting can be lost within hours or days if they neglect to apply their splints. Even with full cooperation it may be several months before the gains made are retained without the constant wearing of splints. The lag represents the time it takes for the collagen fibers to realign themselves and for the scar to mature. Explaining this at the time therapy begins will give the patient realistic expectations and prevent dissatisfaction and noncompliance.

Sensory Reeducation

Hypersensitivity is defined as the condition of extreme discomfort or irritability at or near the injury site in response to normally nonnoxious tactile stimulation. More diffuse pain syndromes are discussed under "Pain Reduction" below.

The theory of desensitization of hypersensitive areas is based on the premise that if painful or potentially painful neuromas are repeatedly percussed, their nerve fibers will gradually degenerate and be replaced by fibrous tissue.[6]

A bulky bandage, a sock, or a mitten enclosing an injured part, "protecting" it from noxious stimuli, prevents sensory input and may lower the patient's sensory threshold, leading to hypersensitivity. Such practices should be discouraged. At best, such a limb is only of use as a paperweight to stabilize objects for the opposite limb, and at worst it amounts to a physiological amputation of the part.

The patient will usually avoid hypersensitive areas, and this leads to abnormal patterns of use. The patient must be informed that it is actually beneficial to use and stimulate the injured and hypersensitive areas.

Various methods of desensitization may be used. They include application of ultrasound and vibrators, rubbing and tapping with materials of different textures attached to dowels, and immersing the part in containers of particles of various consistency. Vibration and the application of textures or particles can be graded according to level of stimulation so that a program of therapy can be planned that starts with mild stimuli and progresses to more noxious ones as tolerance increases. Local infiltration or regional nerve blocks may be required to initiate therapy.

Dowel textures range from felt or moleskin to Velcro hooks. These may be tapped or rubbed adjacent to or directly on the sensitive site. Contact particles range from cotton to buckshot. Vibration varies from 83 cycles near the sensitive site but without actual contact to 100 cycles continuous contact over the site

itself. Exercises can be done at home. Rehabilitation should include simulated work activities as soon as the patient is able to cooperate. Using tools with which the patient is familiar may be more helpful than a formal desensitization program.

The end of treatment comes when the patient is able to return to work, not when he or she no longer has symptoms. With sufficient progress the patients cure themselves.

If these methods should fail, surgery may offer a solution, excising a neuroma in continuity and repairing the nerve or transposing an end-bulb neuroma into a well-padded location where it will be protected from noxious stimuli.

Compression

Intermittent and constant compression (including Ace bandage wrapping), elevation, string wrapping, retrograde massage, and active motion are all means of dispersing edema. Edema can be quantified by measuring the girth of a hand or a finger with a tape measure or by measuring its displacement of an equal volume of water according to Achimedes' law.

Constant compression (eg, Jobst compression garments) prevents edema and may prevent hypertrophic scarring. They are comfortable to wear and patients can become addicted to them. They should be examined often to be sure that they fit properly and should be replaced when loose. Because they take several weeks to be made, the patient should be measured before the wounds are healed. Small, unhealed wounds can be dressed and tolerated beneath a compression garment. Larger areas should be healed, or application and removal of the garments will traumatize the wound edges and retard epithelialization.

Retrograde Massage

Retrograde massage is a way of establishing the relationship between therapist and patient. It gives the patient positive feedback. As a therapeutic exercise, it helps to disperse edema and makes subsequent exercises easier to perform. Therefore, it is frequently used as a preliminary to an exercise session.

Elevation

The arm should be held in the "Statue of Liberty" position when the patient is standing upright and on two pillows when sitting and at night. A sling is not recommended. At best it keeps the hand over the heart. At worst, the hand is held immobile at waist level. A sling places the joints of the upper extremity in flexion, obstructing arterial inflow and venous return. Elevation should continue for as long as there is swelling or a tendency to swelling.

Biofeedback

The simplest form of biofeedback is the physician's or the therapist's tabulation of progress and advice about exercises as the patient recovers. Electrodes applied to the skin or placed within a muscle can record active contraction on a meter. This may be of use following tendon transfer when the patient is learning to use the transferred muscle-tendon unit. It is also indicated when co-contraction is a problem (see above). Other feedback aids are a grip dynamometer and a pinch meter with which the patient can record progress and attempt to exceed previous results.

Heat

Cold and inactive tissues have a low metabolic rate and are inelastic. Activity and heat increase the metabolic rate and increase tissue elasticity. Therefore, heat is a useful modality to increase tissue elasticity prior to an exercise program. Warm water soaks, whirlpool baths, and hot wax are all accepted methods of warming the limb. However, dependency in a whirlpool can aggravate edema, and we do not recommend this method.

Pain Reduction

Rehabilitation has two aims: to relieve pain and to improve function. The pain "threshold" is the least stimulus at which a patient perceives pain.[7] The pain "tolerance level" is the greatest stimulus intensity causing pain that a patient is prepared to tolerate.[7] A patient's readiness to communicate pain is a function of character and does not imply that the patient has a low pain threshold.

Personality traits can be measured by the Eysenck Personality Inventory (EPI) and the Minnesota Multiphasic Personality Inventory (MMPI). Patients with scores on the EPI, suggesting neuroticism, and with scores on the MMPI, suggesting depression, hypochondriasis, and hysteria, are those most likely to develop chronic pain syndromes.[8]

Passive exercises help to reduce pain and maintain mobility. However, active exercises build strength and dexterity, which are more important because they prepare patients for an independent existence and allow them to cure themselves.

Among the recognized causes of pain associated with upper extremity disease and injury are:

- peripheral painful stimulus
- reflex sympathetic dystrophy
- inflammatory pain of peripheral origin
- personality disorder
- cancer and other causes of intractable pain.

Peripheral Painful Stimulus

A peripheral painful stimulus is usually associated with a trigger point. A neuroma may be a terminal type or a neuroma in continuity. Terminal bulb neuroma is seen most commonly in amputation stumps.

Hypersensitivity or hyperpathia associated with a neuroma may be treated in several ways. Desensitization procedures are discussed above. Percutaneous injections of bupivacaine hydrochloride or triamcinolone acetonide combined with a short-acting local anesthetic may relieve symptoms.[9]

If these methods should fail, surgery may help, placing the neuroma in a new site where compression and traction are unlikely. A neuroma in continuity may be excised and the nerve repaired (end to end or with a graft).

This syndrome may be associated with tenderness of the respective nerve (median, ulnar, radial) proximally at the wrist, elbow, and in the supraclavicular fossa. If so, local treatment is less likely to help. A proximal 'neuritis' may respond to anti-convulsant medication such as Phenytoin (dilantin sodium) or Tegretol (carbamazepine). Consultation with a neurologist is recommended.

Reflex Sympathetic Dystrophy

"One must be alert to recognize early cases that will go on to edematous, immobile hands by recognizing signs of trophic disturbance and disposition on the part of the patient to hold his hand completely immobile."[1] Three conditions must be present for a dystrophy to develop. These are a persistent source of pain, a susceptibility on the patient's part, and an abnormal sympathetic reflex.

The cardinal signs are pain, swelling, stiffness, and vasomotor instability (temperature or color change). Pain is characteristically of a burning nature. The limb is usually warm. Hypersensitivity and cold intolerance are common. Technetium-99m-pertechnetate (^{99m}Tc04) and technetium-99m-distannous ethane-1-hydroxy-1-1-diphosphonate (^{99m}Tc-EHDP) scintigraphy may show localization of nuclide predominantly in the juxta-articular tissues. Radiographs may show osteoporosis in either a diffuse or a juxta-articular distribution, but this is not apparent for at least 6 weeks.[10]

Ultimately the extremity may become contracted, atrophic, stiff, pale, cool, sweaty, or dry. The syndrome is believed to be a prolongation of the normal sympathetic response to injury. The essential goal of treatment is interruption of the sympathetic reflex. The earlier this is done, the better the result.

Aggressive exercise programs at an early stage may prevent an incipient case from becoming established. The hand therapist should avoid any painful exercises as these may exacerbate the condition.

Median nerve compression at the wrist is associated with reflex sympathetic dystrophy. When present, decompression of the nerve is indicated. No subsequent surgery should be performed without cover of a sympathectomy or a stellate ganglion block.

Patients with Raynaud's phenomenon who have a positive cold stress test[11] and who do not show obstructive disease on arteriography may respond to medications that decrease peripheral sympathetic activity.

Drugs recommended for intra-arterial injection include α-receptor blockers such as tolazoline hydrochloride (Priscoline) and phenoxybenzamine hydrochloride as well as β-adrenergic blocking drugs such as propranolol hydrochloride and neuronal norepinephrine depletors such as reserpine, methyldopa, or guanethidine. Oral guanethidine, phenoxybenzamine, and propranolol have also been used.[11,12] Regional intravenous blocks with reserpine and guanethidine have been used effectively.[13-16]

A stellate ganglion block is a useful diagnostic test. A therapeutic response will usually confirm the value of a surgical sympathectomy, which may be indicated if a single or repeated blocks fail to relieve the pain for more than a brief duration. Surgery may relieve the burning pain of sympathetic overactivity. It will not affect pain secondary to a neuroma or arthritis.

If chemical or surgical sympathectomy fails, transcutaneous electrical nerve stimulation (TENS) may be considered. This is most effective when started within 3 months of the onset of pain.

The transcutaneous electrical nerve stimulator is an electric device that emits a pulsed current in the form of a biphasic, asymmetric wave form. The use of this device is based on the gate control theory proposed by Melzack and Wall[17] in 1965. Pain receptors (nocireceptors) transmit their impulses through A-delta and unmyelinated (C) fibers. When the impulses arrive in the posterior horn of the spinal cord, a gate mechanism controls the passage of signals to the spinothalamic tract and thence to the brain. Increased C fiber activity inhibits the gate, allowing transmission of more impulses. Increased large unmyelinated A fiber activity closes the gate, blocking pain. TENS is thought to stimulate the A fibers. The inhibitory agent released is an opioid peptide (endorphin).

"Conventional" TENS is usually administered at a frequency of 60 to 120 pulses per second (pps) with a low pulse width and a low amplitude, which is adjusted until the patient has a pleasant tingling sensation in the area that was previously painful. "Low-frequency" TENS has a frequency of 1 to 5 pps with a high pulse width and an amplitude adjusted until a visible muscle contraction occurs. Pain relief occurs 15 to 30 minutes after the start of conventional or low-frequency TENS and may last from 30 minutes to several hours after it is discontinued. "High-frequency" TENS has a frequency of 60 to 120 pps with a high pulse width and high amplitude adjusted until a strong muscle contraction occurs. This is in effect a form of counterstimulation. Pain relief is sudden and transient with variable carryover. This may be useful for brief procedures such as suture removal and wound débridement. Because an active muscle contraction occurs with low-frequency and high-frequency TENS, the patient must be cleared for active exercises. A muscle contraction does not occur with conventional TENS.

Therapy is usually initiated with conventional TENS, and converting to low frequency TENS if ineffective. For therapy to be evaluated properly, the

patient should refrain from analgesics for 6 to 8 hours prior to TENS and should be experiencing pain when treated. Too often, TENS becomes a 'beacon' of the patient's disability labelling them as an impaired member of the community. Although TENS may relieve pain, it has not, in our experience, speeded recovery. Frequently, the patient becomes addicted to this expensive and complex form of treatment making discontinuation impossible. It is often prescribed too readily by both physician and therapist.

Inflammatory Pain

See the section titled "Drugs" below, for a discussion of nonsteroidal anti-inflammatory agents (NSAIA). Steroids are of use in certain conditions such as rheumatoid arthritis. We have no experience with intravenous blocks using prednisone and lidocaine.[18] Intra-articular steroids are particularly helpful in degenerative joint disease involving a single joint (eg, thumb basal joint arthritis). Flexor tenosynovitis may also respond to local injections of steroids. Half to one cc of Triamcinolone acetonide (Kenalog-40) combined with 0.5–1.0 cc of 1% lidocaine is the usual amount injected.

As pain subsides, massage and heat are combined with a program of exercises and NSAIA.

Personality Dysfunction

Medications for emotional instability and depression may be required. Phenytoin (Dilantin) and amitriptyline hydrochloride (Elavil) are commonly used drugs. These may be combined. Hypnosis may be helpful.

Cancer and Other Causes of Intractable Pain

When pain is intractable, invasive procedures such as spinal dorsal sensory root rhizotomy[19] may be of use in cases of unilateral pain involving the brachial plexus when the involved extremity is useless. Cordotomy is indicated for diffuse pain involving areas innervated by many nerve roots. Destructive lesions at higher levels (eg, cingulotomy) are of temporary benefit only.

Drugs

Infiltration with bupivacaine (Marcaine) at the end of an operation (with or without indwelling catheters) to provide continuous pain relief following tenolysis or capsulotomy, will enable the patient to begin early exercises.

Following trauma or disease, NSAIAs are the first-line medications after the acute stage, when narcotic analgesics can be used. Of the NSAIAs, phenylbutazone and indomethacin are not recommended for long-term therapy because of their gastric irritation. NSAIAs that we use regularly are ibuprofen (Motrin), naproxen (Naprosyn), diflunisal (Dolobid), piroxicam (Feldene), and meclofenamate sodium (Meclomen). In long-standing, active arthropathies, remitting agents such as penicillamine (Cuprimine) and aurothioglucose (Solganal) are of use in controlling the frequency of attacks.

Conclusion

The indications for rehabilitation are loss of function (ie, movement, strength, dexterity, and sensibility) as well as loss of parts. Pain and emotional disturbance may cause impairment of function. The best treatment, as always, is prevention; early motion prevents stiffness. The best therapist is the patient himself or herself. The best therapy is the patient's own work. A motivated patient will do well with a minimum of therapy.

Postoperatively, patients are instructed on the operating table at the termination of surgery and in the recovery room to exercise the shoulder and elbow as well as the unsplinted parts of the injured hand. Exercises are done every hour for 4 to 5 minutes and, after the acute stage, two to three times daily for 25 to 30 minutes.

In all cases, whether after injury or disease, patients are taught to distinguish between the sensation of stretch and the sensation of pain. The first is encouraged and the latter is not. The axiom, "It may hurt but it won't harm," is wrong. If it hurts it may harm, and it may excite an inflammatory reaction that leads to further cicatricization and stiffness. "The inevitability of gradualness" is to be stressed. Gentle exercises done with increasing frequency or duration will achieve more than sudden, impatient jerks performed on the unsuspecting patient.

Active motion is the safest and most effective exercise. It improves tendon glide and strength as well as range of motion. Active assistive and active resisted exercises are complementary. Active exercises are not possible after a recent tendon repair, however. Dynamic traction is used early after a flexor tendon repair in zone II.

Static splinting is used most frequently in the acute stage to prevent undesirable positions of rest. The wrist cock-up splint as well as the intrinsic plus splint are those in common use. The former places the hand in a "position of function." The wrist is in 20° to 30° of dorsiflexion, the MP joints in 20° to 30° of flexion, and the IP joints in 10° to 20° of flexion. The latter places the hand in the intrinsic plus position. The wrist is in the same position, but the MP joints are now flexed to 50° to 60° and the IP joints are in neutral. The thumb is held in 45° of palmar abduction and is opposite the index metacarpal.

Dynamic splinting may be started when active motion plus a tolerance to mild stretch is present. Dynamic splinting encourages motion in desirable planes.

Although splints are much easier to construct with thermoplastic materials, the mechanical principles involved in their construction are frequently misunderstood, and it behooves the physician to be cognizant of these principles and to be sure that splints are made and applied correctly. The price of misunderstanding is skin and joint injury. Splints should be reviewed by the physician at each office visit and adjusted as often as necessary. Frequently, one splint will not achieve all that is necessary, and then two or more splints should be used during the day or night to achieve the desired goals.

Other methods such as compression, massage, elevation, biofeedback, heat, and anti-inflammatory drugs may be used singly or in combination to decrease swelling, stiffness, and inflammation.

In the acute stage, elevation and static splinting are all that is indicated. As the inflammatory reaction subsides, gentle active and passive range-of-motion exercises are suitable. Gentle traction may also be of use at this time. As time progresses, active assistive and resistive exercises as well as more aggressive passive stretching, particularly in planes opposite to the plane of motion activated by the repaired tendon, are indicated. This sequence may be altered depending on the individual patient or the structure injured; for example, dynamic traction is used early following a tendon repair in zone II.

Pain may be localized to the site of a neuroma or may be diffuse as in reflex sympathetic dystrophy. A desensitization program is useful for a local cause of hypersensitivity. If the patient is encouraged to use the part normally from the beginning, the incidence of hypersensitivity is lessened considerably.[20]

Reflex sympathetic dystrophy can be prevented by "being alert to recognize early cases that will go on to edematous hands by recognizing signs of trophic disturbance and disposition on the part of the patient to hold his hand completely immobile."[1] An early, aggressive program of therapy may help. Other modalities of treatment have been discussed. Despite the best treatment, there will always remain a small percentage of patients who will do badly and develop chronic pain syndromes that are refractory to treatment. Personality dysfunction is frequent in this minority and may respond to treatment.

Inflammatory pain may respond to splinting and drugs, intractable pain such as that due to cancer may require surgery.

Early motion as well as functional activities prevent stiffness, swelling, and pain. An understanding of the purpose of each method of treatment and the correct timing for application of each will prevent injury and encourage rapid recovery of the hand to a level of maximum function consistent with the initial injury or the disease.

References

1. Bunnell S: *Surgery of the Hand,* ed 3. Philadelphia, JB Lippincott Co, 1948.
2. Peacock EE: Some biochemical and biophysical aspects of joint stiffness: Role of collagen synthesis as opposed to ultra molecular binding. *Ann Surg* 1966; 163:1-6.
3. Scott FA, Boswick JA: Palmar arthroplasty for the treatment of the stiff swan-neck deformity. *J Hand Surg* 1983; 8:267-272.
4. Brand PW: The forces of dynamic splinting: Ten questions before applying a dynamic splint to the hand, in Hunter JM, Schneider LH, Mackin EJ (eds): *Rehabilitation of the Hand.* St Louis, CV Mosby Co, 1984, pp 847-852.
5. Smith RJ: Intrinsic muscles of the fingers: Function, dysfunction and surgical reconstruction. *American Academy of Orthopaedic Surgeons Instructional Course Lectures* 1975; 24:200-220.

6. Russell WR, Spalding JMK: Treatment of painful amputation stumps. *Br Med J* 1950; 2:68–73.
7. Mersky H: Pain terms: A list with definitions and notes on usage (International Association for the Study of Pain [IASP] Subcommittee on Taxonomy). *Pain* 1979; 6:249–252.
8. Bond MR: *Pain: Its Nature, Analysis and Treatment.* Edinburgh, Churchill Livingstone, 1979.
9. Smith JR, Gomez NH: Local injection therapy of neuromata of the hand with triamcinolone acetonide: A preliminary study of twenty-two patients. *J Bone Joint Surg Am* 1970; 52A:71–83.
10. Kozin F, Genant HK, Bekerman C, et al.: The reflex sympathetic dystrophy syndrome II. Roentgenographic and scintigraphic evidence of bilaterality and of periarticular accentuation. *Am J Med* 1976; 60:332–338.
11. Porter JM, Snider RL, Bardana EJ, et al.: The diagnosis and treatment of Raynaud's phenomenon. *Surgery* 1975; 77:11–23.
12. Simson G: Propranolol for causalgia and Sudek atrophy. *JAMA* 1974; 227:327.
13. Chuinard RG, Dabezies JE, Gould JS, et al.: Intravenous reserpine for treatment of reflex sympathetic dystrophy. *J Hand Surg* 1980; 5:289.
14. Hannington-Kiff JG: Intravenous regional sympathetic block with guanethidine. *Lancet* 1974; 1:1019–1020.
15. Hannington-Kiff JG: Relief of Sudek's atrophy by regional intravenous guanethidine. *Lancet* 1977; 1:1132–1133.
16. Wynn Parry CB: Brachial plexus lesions, causalgia and Sudek's atrophy. *Am Soc Surg Hand Newsletter* 1981; 2.
17. Melzack R, Wall PD: Pain mechanisms: A new theory. *Science* 1965; 150:971–979.
18. Wiley AM, Poplawski ZB, Murray J: Post-traumatic dystrophy of the hand. *Orthop Rev* 1977; 6:59–61.
19. Leavens ME: Neurosurgical relief of pain in cancer patients. *Cancer Bull* 1981; 33:98–100.
20. Fisher GT, Boswick JA: Neuroma formation following digital amputations. *J Trauma* 1983; 23:136–142.
21. Omer GE: Management of pain syndromes in the upper extremity, in Hunter JM, Schneider LH, Mackin EJ (eds): *Rehabilitation of the Hand.* St Louis, CV Mosby Co, 1984, pp 503–508.