Splinting the hand with a peripheral-nerve injury is both easy and difficult. The ease of splinting results from the readily recognizable and often identical deformities. Therefore, unlike many other hand injuries, the deformities resulting from isolated peripheral nerve paralysis are usually effectively splinted using standard splinting designs. The difficulty, however, in splinting peripheral nerve paralysis arises from the impossibility of building a static external device that substitutes for the intricately balanced muscles the splint attempts to replace.

**PRINCIPLES OF SPLINTS**

The purposes common to all splints used for peripheral nerve injuries are as follows:

1. To keep denervated muscles from remaining in an overstretched position
2. To prevent joint contractures
3. To prevent the development of strong substitution patterns
4. To maximize functional use of the hand

**Overstretching of muscles**

In all cases of isolated upper extremity peripheral nerve injury, the denervated muscle group will have normal unopposed antagonist muscles still present. These normal muscles will continuously overpower the denervated muscles and maintain them on constant stretch. A muscle undergoing reinnervation must first overcome this stretched position and achieve a normal resting length before it is able to contract enough to bring about joint motion. Short periods during which the unopposed muscles overpower the denervated muscles will not cause harm, but a denervated muscle constantly held in a stretched position decreases the potential for early return of normal muscle activity. A denervated muscle protected by appropriate splinting allows the earliest perception of returning motion. Properly executed splints will enhance returning muscle function instead of allowing substitution patterns.

**Joint contractures**

Joints held continually in one position cannot experience the habitual movement of the capsular structures. Even without accompanying trauma, prolonged immobility will result in restriction of joint motion. To maintain motion, the joint must be frequently taken through its full range. It is not adequate to immobilize joints at one extreme at night and the other extreme during the day. Although positional splinting at night can be useful, it is the full active motion of joints during the day that maintains joint lubrication and accompanying soft tissue glide.

**Substitution patterns**

In an isolated peripheral nerve injury, there is no opposing force to the intact active muscle group and muscle imbalance is created. Without an external splinting constraint, the patient constantly reinforces the strength of the normal muscles and overpowers the denervated muscles. An excellent example is low median palsy, in which the intact flexor pollicis longus (FPL) and extensor pollicis longus (EPL) carry the thumb back and forth across the palm in an abducted position (Fig. 34-1). With the opponens pollicis (OP) and abductor pollicis brevis (APB) absent, the extrinsic muscle pattern becomes dominant in the motor cortex. As the OP and APB are reinnervated, they have difficulty participating in thumb motion because the stronger extrinsic muscles con-
Chapter 34  Splinting the hand with a peripheral-nerve injury

623

Fig. 34-1. In low median palsy, the thumb is carried back and forth across the palm in an adducted position by extrinsic muscles because of the absence of the opponens pollicis and the abductor pollicis brevis.

Fig. 34-2. In the nerve-injured hand, a neoprene mitten aids in cold tolerance.

Functional use of the hand

A peripheral-nerve lesion with significant sensory loss prevents functional use of the hand even when a splint substitutes for absent muscles. The combination of sensory loss and motor imbalance in median and ulnar nerve lesions makes it impossible to use the hand normally. A splint can assist in maximizing function only when the sensibility returns to a functional level.

Cold intolerance is common with peripheral-nerve injuries, particularly during reinnervation. Protective neoprene mittens, gloves, or finger sleeves are helpful for patients in colder climates or for those who work in cold environments (Fig. 34-2).

SPLINT REQUIREMENTS

Design

Splints should be kept as simple as possible. Bulky splints, if worn by the patient, will impede function of the hand rather than reinforce it. Early after the nerve injury, it is acceptable to cover a denervated sensory area with the splint. In cases of partial nerve laceration or returning sensibility, the tactile surfaces should be left free if possible.

Splints for peripheral-nerve injuries should not totally immobilize any joint. For example, splinting of the wrist in radial palsy with a static wrist immobilization splint prevents the returning wrist extensor muscles from gaining strength or excursion while in the splint.

Splint timing

Many peripheral-nerve injuries are associated with accompanying trauma to skin, tendon, bone, or vascular structures. Tension on the healing tissues must be avoided immediately after injury to allow adequate healing. Thereafter, glide of tendons and joint movement take precedence over splinting for the peripheral-nerve deformity. The peripheral-nerve splint will often restrict full tendon excursion. For example, in a laceration at the wrist involving the median and ulnar nerves and wrist and finger flexor tendons, the wrist must be held in some flexion immediately postoperatively to prevent tension at the repair sites. Only as wrist motion is gained and excursion of the flexor tendons is achieved will the claw deformity of the fingers be evident. The initial adherence of the flexor tendons at the wrist will create tenodesis of the finger flexors, providing a built-in splint. Only when the deformity becomes clinically evident should one splint for it. In longstanding denervation in which joint contractures have already developed, one must first splint to regain joint motion before splinting the dynamic deformity.
SPECIFIC NERVE LESIONS

It is easiest to understand the deformity of a pure lesion of the three primary upper extremity peripheral nerves: ulnar, median, and radial. Combined nerve injuries, especially when associated with other injuries, require more skill to determine treatment priorities, although the splinting principles are the same.

Ulnar nerve

Low lesion. Laceration of the ulnar nerve at the wrist (low ulnar palsy) results in denervation of the majority of the intrinsic muscles of the hand (Fig. 34-3). The ulnar nerve innervates all of the hypothenar muscles: abductor digiti minimi, flexor digiti minimi, and the opponens digiti minimi. The ulnar border of the transverse metacarpal arch is lost. Absence of the dorsal and volar interossei muscles creates the inability to abduct and adduct the fingers, causing loss of the fine manipulative power of the hand. In addition to the loss of the interossei, the ring and little fingers also lose function of the lumbricales, removing any intrinsic muscle balancing force in these two digits. The extrinsic muscles dominate.

Fig. 34-4. In ulnar palsy, the loss of intrinsic muscle control in the ring and little fingers allows the metacarpophalangeal joints to hyperextend. The denervated muscles are stretched in this claw position. ED, Extensor digitorum communis; FDP, flexor digitorum profundii; FDS, flexor digitorum superficialis.

Fig. 34-5. A blocking force over the dorsum of the proximal phalanx prevents metacarpophalangeal hyperextension and allows the extrinsic extensor to transmit power to the interphalangeal joints when the intrinsic muscles are absent. ED, Extensor digitorum communis.
The resulting deformity is clawing of the ring and little fingers. The intrinsic muscles normally flex the metacarpophalangeal (MP) joint and extend the interphalangeal (IP) joints. When they are absent, there are no prime flexors of the MP joint. Because there is no intrinsic muscle control in these digits, the tension of the long flexors is opposed only by the extrinsic extensors, which primarily extend the MP joints (Fig. 34-4). In this claw position, both the lumbricales and the interossei are held in a stretched position. The patient can flex the MP joints actively, but only after the IP joints are fully flexed. The greatest functional loss is the inability to open the hand in a large span to grasp objects and the inability to handle small objects with precision.

The goal in splinting ulnar palsy is to prevent overstrecthing of the denervated intrinsic muscles of the ring and little fingers. The ring and little finger MP joints must be prevented from fully extending (Fig. 34-5). Any splint that blocks the MP joints in slight flexion prevents the claw deformity by forcing the extrinsic extensors to transmit force into the dorsal hood mechanism of the finger. This extends the IP joints in the absence of active intrinsic muscle pull (Fig. 34-6). The dorsal block should be molded carefully to distribute pressure over the dorsum of each proximal phalanx and should end exactly at the axis of the proximal interphalangeal (PIP) joint.

A bulky splint that blocks the MP joints will impede function of the hand. In isolated low ulnar palsy, two thirds of the palmar surface of the hand has intact median nerve sensibility. Ulnar palsy splints should cover a minimal surface of the palm. Total finger flexion must also remain unimpeded. Early after injury, the MP joint block can be incorporated into the immobilization splint that prevents tension on the nerve and tendon repairs at the wrist. When wrist movement is allowed, a small molded thermoplas-
PART SEVEN ACUTE NERVE INJURIES

Fig. 34-7. An ulnar palsy splint may be made of leather wrist and finger cuffs with a static line to block metacarpophalangeal hyperextension.

Flexor carpi ulnaris
Flexor digitorum profundus to IV & V
Flexor pollicis brevis
Adductor pollicis
Palmaris brevis
Dorsal & volar interossei
Flexor pollicis brevis
Opponens pollicis
Abductor pollicis brevis
Lumbricals to II & III

Median nerve

Fig. 34-8. The path of the ulnar nerve and the muscles involved in high ulnar palsy.

Ulnar palsy splints will not create flexion contractures of the MP joint. Every time the splint is removed for skin care, the extrinsic extensor will still hyperextend the MP joint.

High lesion. High ulnar palsy lesions are commonly a result of trauma at or above the elbow. In addition to the muscles previously mentioned in low ulnar palsy, the flexor digitorum profundus (FDP) of the ring and little fingers and the flexor carpi ulnaris are absent in a high lesion (Fig. 34-8). With absence of the FDP and all intrinsic muscles to the ring and little fingers, clawing in the high ulnar nerve lesion is rarely present. As reinnervation of the FDP occurs, clawing becomes evident and splinting becomes mandatory rather than optional. The same MP hyperextension restriction splint is used for both the high and low ulnar palsy lesions (see Fig 34-5). In a high lesion, the patient must maintain full passive IP flexion of the ring and little fingers when the FDP is absent.

Low lesion. A laceration of the median nerve at the wrist produces low median palsy, a devastating injury. The median nerve innervates the majority of the thenar muscles and construction is challenging because the tension of the spring must be exact. It must be strong enough to block MP extension when the patient attempts finger extension while also following the arc of the proximal phalanx when the MP joint flexes. Therapists who apply a dynamic force for MP flexion (e.g., the Bunnell knuckle-bender splint) are missing the concept of blocking MP hyperextension. In such a splint, the patient hyperextends actively against the force of the rubber bands, which gives resistance and proprioceptive input to the extrinsic extensors, strengthening them. Unless one is able to construct a coil splint with precise tension, it is recommended that static splinting that prevents the MP joint from hyperextending be applied.

Ulnar palsy splints will not create flexion contractures of the MP joint. Every time the splint is removed for skin care, the extrinsic extensor will still hyperextend the MP joint.

High lesion. High ulnar palsy lesions are commonly a result of trauma at or above the elbow. In addition to the muscles previously mentioned in low ulnar palsy, the flexor digitorum profundus (FDP) of the ring and little fingers and the flexor carpi ulnaris are absent in a high lesion (Fig. 34-8). With absence of the FDP and all intrinsic muscles to the ring and little fingers, clawing in the high ulnar nerve lesion is rarely present. As reinnervation of the FDP occurs, clawing becomes evident and splinting becomes mandatory rather than optional. The same MP hyperextension restriction splint is used for both the high and low ulnar palsy lesions (see Fig 34-5). In a high lesion, the patient must maintain full passive IP flexion of the ring and little fingers when the FDP is absent.

Low lesion. A laceration of the median nerve at the wrist produces low median palsy, a devastating injury. The median nerve innervates the majority of the thenar muscles and...
Adduction contracture of the thumb in median palsy is caused by an unopposed adductor pollicis muscle.

Motor loss is present in only the radial portion of the hand (Fig. 34-9). Loss of the OP and APB renders it impossible to pull the thumb away from the palm. The thumb is carried across the palm in an adducted position (see Fig. 34-1). If the adduction deformity is not obvious, the nerve injury may be partial or one may be observing the not uncommon cross-innervation from the ulnar nerve. In low median palsy, the lumbrical muscles of the index and long fingers are also absent. Because the ulnarly innervated palmar and dorsal interossei are still present, clawing is nearly always absent in these two digits; only in combined median and ulnar palsy is clawing of the index and long fingers present (along with the ring and little fingers).

Adduction contractures of the thumb are the most common deformity following a low lesion of the median nerve. To provide a balance to the unopposed adductor pollicis, some means of holding the first metacarpal abducted from the second metacarpal is necessary (Fig. 34-10). Thumb abduction splinting prevents the OP and APB from resting in a stretched position and reinforces early return of their action. Abduction splinting also maintains the soft tissue length of the first web space. Much of the functional abduction of the thumb requires the elongation of the soft tissue of the entire first web.

It is difficult to maintain the full length of the first web space while still allowing MP flexion of the index finger. A night splint that holds the index finger extended and the thumb abducted is helpful in maintaining the motion necessary for functional abduction of the thumb (Fig. 34-11).

A small daytime splint made of neoprene, leather, or thermoplastic material is used during the day to hold the thumb in a stable opposed, but less than fully abducted, position (Fig. 34-12). If a daytime splint prevents full flexion of the MP joint of the index finger, it can lead to an unnecessary extension contracture of this joint.
Fig. 34-12. Neoprene (A) or leather (B) splint for maintaining functional abduction of the thumb while awaiting median nerve return.

Fig. 34-13. High median palsy robs the radial aspect of the hand of the majority of flexor muscle function.
Chapter 34  Splinting the hand with a peripheral-nerve injury  629

Fig. 34-14. In high median palsy, the long finger is carried into flexion because of the common flexor profundus muscle belly.

The thumb, index, and long fingers are the prime digits for manipulating objects. In median palsy, loss of sensibility in these digits makes it extremely difficult for the patient to use the hand for normal activity. Because reinnervation proceeds proximally to distally, it is common that the motor return precedes full sensory return. At times the splint may be discontinued before sensory return reaches maximum. For this reason, early after a median nerve repair at the wrist, one need not hesitate to cover the palm to position the thumb. As the nerve regenerates, a small leather or neoprene splint (see Fig. 34-12) positions the thumb in slight abduction and opposition so that the strong extrinsic flexor and extensor does not overpower the returning thenar intrinsic muscles.

In many adults, especially those with a high nerve lesion, OP function often does not return even though sensibility may recover to a protective level. In these patients a tendon transfer to restore thumb opposition increases functional thumb use.

High lesion at the elbow. High median nerve injuries result from injury to the nerve at or near the elbow. In these lesions, loss of the FDP of the index and long fingers and the flexor digitorum superficialis to all fingers robs the hand of all except minimal gross grasp function (Fig. 34-13). It is often difficult to maintain passive flexion of the index finger. Buddy taping it to the long finger does not provide adequate passive motion. Even though the long finger appears to flex actively, it is actually being carried along into flexion as part of the common muscle belly of the profund of the long, ring, and little fingers (the ring and little FDP are innervated by the ulnar nerve) (Fig. 34-14).

Active pronation is also lost with denervation of the pronator teres and pronator quadratus, but slight abduction of the arm allows gravity to assist with pronation.

In the adult patient with a high median or ulnar nerve lesion, normal motor and sensory return is rare. Splinting high-level deformities to maintain passive range of motion is appropriate in preparation for tendon transfers. Although no splint need be cumbersome if correctly designed, some authors suggest very early tendon transfers as an internal splint to prevent the need for cumbersome external devices.

High lesion: anterior interosseous nerve. The median nerve bifurcates distal to the elbow, just after it passes between the two heads of the pronator (see Fig. 34-13). One branch continues with sensory and motor fibers to the hand. The other purely motor branch, the anterior interosseous nerve, terminates in the forearm as it innervates the FDP of the index and long fingers, the FPL, and the pronator quadratus.

Absence of anterior interosseous nerve function prevents the patient from successfully transmitting force to the tips of the index and long fingers, as well as the thumb. This can

Fig. 34-15. A, Fingertip pinch is impossible in anterior nerve palsy. B, Small splints that prevent joint extension re-create fingertip pinch.
most easily be demonstrated by asking the patient to pinch the thumb tip against the index tip. The index distal interphalangeal (DIP) joint and the thumb IP joint fall into extension or hyperextension (Fig. 34-15, A). In isolated anterior interosseous nerve palsy, all intrinsic muscles are normal and sensibility throughout the hand is intact. Thus the primary functional loss is the inability of the thumb tip to transmit force because the remainder of the thumb is functioning normally.

While awaiting return of anterior interosseous nerve function, splinting the thumb IP joint (and perhaps also the index DIP joint) allows the patient to continue writing, dressing, and performing other pinching activities. The splint blocks joint extension but can allow further flexion. Either a temporary custom molded thermoplastic splint (Fig. 34-15, B) or a more streamlined and durable sterling silver Siris splint (Silver Ring Splint Company, P.O. Box 2856, Charlottesville, Virginia) may be used as a functional splint until definitive nerve return occurs or tendon transfer is completed.

Radial nerve

High lesion. Unlike the median and ulnar nerve, the radial nerve is more commonly injured at the higher level where it spirals around the humerus (Fig. 34-16). Injury at the spiral groove of the humerus is most commonly associated with humeral fractures and direct compression. Sensory loss with radial nerve palsy is of little functional concern because it lies over the dorsoradial aspect of the hand. In isolated radial palsy, the entire palmar surface of the hand retains normal sensibility. Injury at or below the spiral groove spares the innervation of the triceps, leaving elbow function intact. There is absence of all wrist and finger extensors as well as the supinator, but all flexor and intrinsic muscles in the hand retain full function.

Unlike patients with median or ulnar nerve palsy, in whom sensory and intrinsic muscle loss impedes hand function, the patient with radial palsy has the potential for relatively normal use of the hand. For this to be possible, a splint must be appropriately designed to harnesses wrist motion while allowing finger flexion. Although some authors advocate early tendon transfers to eliminate the need for external splinting, this is not the standard management of radial palsy. Radial nerve palsy often recovers spontaneously; therefore effective splinting may be needed for months during regeneration.

The primary functional loss in radial palsy is inability to stabilize the wrist in extension so that the finger flexors can be used normally. The loss of the wrist and finger extensors destroys the essential reciprocal tenodesis action vital to the normal grasp-and-release pattern of the hand (Fig. 34-17). The ideal splint re-creates this natural harmony of tenodesis action: finger extension with wrist flexion and wrist extension with finger flexion. Many authors suggest the use of a static wrist support splint. Wrist immobilization splints do not address the problem of absent finger and thumb extension. Any grasp or release activity must be assisted by the uninvolved hand. Wrist immobilization splints also commonly cover the valuable palmar sensibility.
A splint first described by Crochtiere et al.\textsuperscript{10} and a modified design described by Hollis\textsuperscript{17} and Colditz\textsuperscript{9} provide a static nylon cord rather than dynamic rubber bands to suspend the proximal phalangeal area (Fig. 34-18). This splint allows full finger flexion. Because the wrist never drops below neutral, the powerful flexors have the ability to bring the wrist into slight extension. During relaxation, gravity drops the wrist and the blocking force of the loops under the proximal phalanges achieves extension of the MP joints. Full finger extension is accomplished by the intrinsic muscles acting in concert with the blocking action of the splint. Because the splint receives a facsimile of the normal tenodesis motion, training is rarely required for the patient to adapt to the splint.

The EPL, extensor pollicis brevis, and abductor pollicis longus are extrinsic muscles that lie on the dorsoradial surface of the forearm. They are taken off maximum stretch when the splint harnesses the wrist. In this splint the thumb is not included in the outrigger system because of the awkwardness of an outrigger projecting radially. Occasionally, the absence of the extrinsic extensor and abductor muscles of the thumb allows it to rest under the index finger, impeding finger flexion. In such circumstances, a small splint is used to immobilize the thumb MP joint in extension but allow unrestricted thumb carpometacarpal (CMC) and IP joint motion. The intrinsic muscles of the thumb can extend the IP joint in the absence of the extrinsic extensors.
The advantages of this splint design are numerous. The suspension design allows partial wrist motion and full finger motion (Fig. 34-19). It maintains the normal hand arches as the thumb and ring and little CMC joints are unimpeded. This free motion is impossible in static splints or when rigid bars hold the metacarpal or proximal phalangeal area. Most strikingly, this splint has an absence of splinting material on the palmar surface, allowing normal grasp. The greatest advantage of this splint is its comparatively low profile, which allows it to be used effectively by the patient in the daily routine.

As function begins to return, the splint facilitates strengthening of the wrist extensors instead of impeding them. One should be cautioned against designs for dynamic wrist and finger extension because the powerful unopposed flexors often overcome the force of the dynamic splint during finger flexion.

Low lesion: posterior interosseous nerve. After the radial nerve crosses the elbow and plunges below the supinator, it divides, forming the posterior interosseous branch (deep motor) and the superficial sensory branch (see Fig. 34-16). Posterior interosseous palsy is the isolated involvement of the deep motor branch of the radial nerve. In posterior interosseous palsy, radial wrist extension is nearly always spared and brachioradialis function will always be present. Clinical presentation will be strong radial deviation.
Chapter 34  Splinting the hand with a peripheral-nerve injury  633

Fig. 34-21. A median/ulnar palsy splint must have a firm palmar bar to provide a point of counterforce to effectively prevent metacarpophalangeal joint hyperextension and position the thumb in abduction.

of the wrist during attempted wrist extension (Fig. 34-20). Attempted finger extension will demonstrate a pattern of MP flexion and IP extension in view of the absent extensor digitorum communis, extensor digiti minimi, and extensor indicis proprius muscles and the intact interosseous and lumbrical muscles.

Although the clinical picture differs from that of the high radial nerve lesion, the same mechanics for splinting apply to the low lesion. The splint design described earlier allows the radial wrist extensors to function normally to stabilize the wrist during finger flexion. Finger extension is achieved by the patient dropping the wrist into flexion, as the statically suspended proximal phalanges area supports the MP joints in extension. No joint has been unnecessarily immobilized, and the normal tenodesis pattern of the hand is maintained.

In the unusual circumstance of a partial lesion of the posterior interosseous nerve, normal finger extension can be harnessed to lift a weak finger. A custom thermoplastic splint molded under the weak finger or fingers and over the adjacent normal fingers at the proximal phalanx may be of functional value to the patient and hasten the return of the isolated weak muscle.

MIXED NERVE LESIONS

Mixed nerve lesions provide the ultimate splinting challenge. Because multiple nerve involvement nearly always accompanies trauma to many other structures, a balance between increasing glide of soft tissues and constraint splinting for the denervated muscles is required. Monitoring the kinesiologic balance of motion is the guidepost for splinting.

The most common mixed lesion is concurrent injury to the median and ulnar nerves, because they both lie on the palmar surface of the wrist. Division of these nerves robs the hand of all intrinsic muscles, and clawing of all four digits is present. A splint must block MP hyperextension. Likewise, there are no intrinsic muscles stabilizing the thumb, and the thumb should be held in an abducted position (Fig. 34-21).

When only the radial nerve is functioning, one may harness wrist extension power into finger flexion by fitting the Rehabilitation Institute of Chicago tenodesis splint. This splint can occasionally be applicable to the mixed nerve palsy patient to provide both a minimal grasp function and to maintain some range of finger flexion (Fig. 34-22).

In combined lesions in which muscle loss requires periodic static splinting, it is essential that the patient frequently remove the splint and carry out passive ranging of the joints. In isolated nerve injuries, one muscle group is denervated with the antagonist still working. In combined lesions, the imbalance may not be so simple. As the nerve return changes the muscle balance, splinting needs change.

The possible combinations of mixed nerve lesions are endless. A complete manual muscle test and a clear clinical picture of the balance of motion are the only guides to determining the splinting regimen.
CONCLUSION

Splinting deformities that result from isolated nerve injuries in the upper extremity requires knowledge of normal and pathologic kinesiology. One must understand the correct design and mechanics of splints. A thorough knowledge of the anatomy and the expected course of reinnervation, as well as manual muscle testing skill, are necessary to evaluate the nerve-injured hand. The external constraints of splinting should maximize the balance of motion while awaiting return. Changes in splinting must occur in response to muscle and sensory return to assist in maximizing function.

REFERENCES


Fig. 34-22. The Rehabilitation Institute of Chicago tenodesis splint harnesses the power of wrist extension into functional pinch.