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Introduction

When Dr. Ketch, Editor of Operative Techniques in Plastic and Reconstructive Surgery, asked me to be the Guest Editor for this issue of the journal, my initial response was ‘let me think about it.’ He wanted the topic of peripheral nerve injuries reviewed using a clinically relevant format. Contacting individuals to help write appropriate material is always challenging. Imagine calling Drs. MacKinnon and Humphreys and asking them to contribute yet one more article. Once the individuals I wanted to contribute agreed to help with this edition, I was able to give an affirmative answer to Dr. Ketch. It is impressive and a tribute to the contributing authors that they are willing to share their experience and knowledge with readers. Their time and schedules are beyond congested; they have other professional commitments, families, avocations, and still they agreed to contribute. A sincere ‘thank you’ to Paul Lim, Douglas B. Humphreys, Susan E. Mackinnon, Linda L. Zeineh, Bradon J. Wilhelmi, and Elvin G. Zook for their contributions.

This issue focuses on nerve injuries. The degeneration and regeneration processes of the peripheral nerve are understood, but the ability to impact this biological process has not been significantly enhanced. However, the surgical management of nerve injuries has changed, and this issue illustrates those changes. The acute management of peripheral nerve injuries using microsurgical techniques has greatly decreased the need for nerve grafts. The injury that produces a nerve gap has alternatives for repair not previously available. Nerve injuries proximally with consequence distally might need a nerve transfer rather than a tendon transfer. The common nerve compression syndromes are well-known to most readers, but the less common compressions and two-level compressions of the same nerve make the clinical assessments and treatment more challenging. The ultimate reason these authors have contributed is to better serve those patients that entrust us with their care, indeed their lives.

Emphasis on technique will again be the dominant theme in this edition, and the numerous illustrations provide the reader with applicable information. The adage ‘a picture is worth a thousand words’ has always made sense to me!

Allen L. Van Beek, MD
Guest Editor
Nerve injuries can result in severe functional deficits. There have been many different methods proposed for reconstruction of the deficits incurred as a result of these injuries. Direct repair of nerve injuries is the preferred method for reconstruction whenever possible. When this is not possible, nerve grafts have typically been used to attempt to restore function to the affected extremity. For long gaps, nerve allografts have been used with some success, and for short gaps, less than 3 cm, nerve conduits have found clinical use. While sensory recovery is relatively time independent, motor recovery is directly influenced by the duration of muscle denervation. Results from long nerve grafts often result in disappointing functional recoveries. Nerve transfers deliver nerve fibers closer to the sensory or motor target to enhance ultimate recovery, thus obviating the time dependence of nerve regeneration.

The idea of nerve transfers is not a new one; Lurje, in 1948, published his article on nerve transfers to restore function after brachial plexus injuries. He proposed that normal functioning nerves be transferred directly to adjacent injured nerves to restore arm function. His original article recommended the use of the long thoracic nerve, the thoracodorsal nerve, and the triceps branch of the radial nerve for reconstruction of the suprascapular, the musculocutaneous, and the axillary nerves, respectively. Similarly, intercostal nerve transfers are an accepted method of nerve reconstruction after brachial plexus avulsion injuries. However, the excellent results of the nerve grafting techniques of Millesi and Narakas, introduced in the 1960s and 1970s, relatively obscured these nerve transfer techniques and long nerve grafts became the standard for reconstructing nerve lesions. Nerve transfers were considered a last option reserved for avulsion injuries of the brachial plexus.

Proximal nerve injuries, including brachial plexus injuries, have poor outcomes because of the finite length of time available to innervate motor endplates. Typically, the motor endplates become refractory to reinnervation after approximately 12 to 18 months in adults. Nerves regenerate at a rate of 1 to 1.5 mm/day, thus there is less chance of meaningful recovery for nerve regeneration over distances greater than 18 inches. The transfer of expendable functioning nerves closer to the motor endplates of the affected muscles would theoretically expedite reinnervation and recovery. While tendon transfers are very useful reconstructive options, they require significantly more dissection and postoperative immobilization which, in some patients, may be problematic. Most importantly, Guelinckx has shown the detrimental effect of tenotomy on muscle function. He has shown that there is a decrease in the specific force developed by a muscle after tenotomy. By contrast, a nerve transfer maintains the exquisite tension/insertion balance of the muscle/tendon unit.

There are six basic principles of motor nerve transfers. (1) The donor nerve should be repaired near the motor endplates of the target muscles to ensure the shortest amount of time for reinnervation in an attempt to minimize motor endplate loss. (2) The donor nerve should be from a muscle whose function is expendable or has redundant innervation. (3) The use of a donor nerve with pure motor fibers will maximize muscle fiber reinnervation. (4) The donor nerve should have a large number of motor axons and be a good size match to the recipient nerve. (5) To facilitate motor re-education, a donor nerve which has a function synergistic to the motor function of the muscle to be reconstructed should be used. (6) Motor re-education will improve functional recovery postoperatively.

The principles for a sensory transfer are similar. (1) The donor nerve should be repaired as close to the sensory receptors as possible. (2) The donor nerve should be relatively expendable. (3) A pure sensory nerve with a good size match should be used. (4) If possible, the distal denervated end of the donor nerve can be repaired end-to-side to an adjacent normal sensory nerve. (5) Sensory re-education will improve functional recovery.

Musculocutaneous Nerve and Elbow Flexion

Denervation of the biceps and brachialis muscles can be due to damage of the upper brachial plexus, as well as direct damage to the musculocutaneous nerve. Reconstructive priorities after brachial plexus injury include elbow flexion followed by shoulder external rotation and abduction. Thus, the best donor nerves should be reserved to restore these critical functions.
Fig 1. Medial Pectoral to Musculocutaneous Nerve Transfer: lateral antebrachial cutaneous nerve turned back into the biceps muscle to redirect motor axons into the muscle.

Multiple muscle transfers have been used to restore elbow flexion including the pectoralis major, latissimus dorsi, triceps, and the Steindler flexorplasty. Similarly, the nerves that innervate these muscles can be utilized to reconstruct elbow flexion.

Surgical Technique

The musculocutaneous nerve is dissected with exploration of the brachial plexus, exposing the branches to the coracobrachialis, biceps and brachialis muscles, along with the lateral antebrachial cutaneous nerve (LABC). When choosing where to connect the donor nerve into the recipient, it is best performed as close as possible to the motor endplates to decrease the time to reinnervation and attempt to increase functional recovery. If possible, the donor nerve should be transferred to the musculocutaneous nerve distal to the branches to the coracobrachialis, as the latter does not contribute to elbow flexion. Nerve regeneration should be directed toward the branches that innervate the brachialis muscle, as well as the biceps muscle, as these are both important in restoring elbow flexion, with the biceps providing elbow flexion and supination and the brachialis being the stronger elbow flexor. The lateral antebrachial cutaneous nerve is identified and excluded from the repairs to prevent critical donor motor axons being directed to this sensory nerve. Alternatively, the lateral antebrachial cutaneous nerve can be turned back and inserted into the biceps to redirect motor axons into the muscle to augment elbow flexion (Fig 1) or used to carry motor axons into the forearm for future free muscle transfer to restore hand function.

The use of medial pectoral nerves as donors for reconstruction of the musculocutaneous nerve is well documented (Fig 2A). Exploration of the brachial plexus requires detachment and reflection of the insertion of the pectoralis major and minor muscles. The medial pectoral nerves can be identified and isolated on the undersurface of the pectoralis minor muscle. Identification and conformation of the medial pectoral nerves is obtained intraoperatively with a hand held nerve stimulator (Medtronic VARI-STIM® III). Usually between 2 to 4 branches can be found and these are traced distally into the muscle as far as possible. It is important to obtain as much length as possible as this will often allow direct coaptation of the medial pectoral to the musculocutaneous nerve.

Oberlin described using redundant flexor carpi ulnaris (FCU) fascicles of the ulnar nerve to transfer to the biceps branch of the musculocutaneous nerve to restore elbow flexion (Fig 2B). A careful neurolysis of the ulnar nerve along a distance of about 2 cm is performed in the midarm to identify the FCU fascicles. These nerve fibers are usually found along the lateral side of the ulnar nerve and their function is confirmed with the use of a hand held nerve stimulator (Medtronic VARI-STIM® III). It is important to ensure that stimulation of the segment to be transferred results in contraction of the FCU. Equally, or more important, is to confirm that stimulation of
the remaining ulnar nerve results in intact motor function to the hand intrinsics, as well as the flexor digitorum profundus of the 4th and 5th digits. Approximately 20% of the ulnar nerve is used in the transfer. Direct nerve repair to the biceps branch of the musculocutaneous nerve close to the motor endplates is always possible with this nerve transfer as the location of the neurolysis of the ulnar nerve is located so as to allow a direct transfer. This transfer has proven effective to reconstruct elbow flexion with no significant deficit in hand function noted.20

Similarly, we have used the median nerve to provide expendable donor motor fascicles from the flexor carpi radialis (FCR) or flexor digitorum superficialis (FDS) muscles (Fig 2C).23 A similar neurolysis technique of the median nerve with direct nerve stimulation is performed. As the median nerve is located closer to the biceps branch than is the ulnar nerve, it may be a more satisfactory transfer in some patients. The technique for identifying the redundant donor fascicles is the same. A neurolysis technique over a distance of approximately 2 cm will separate the median nerve along the fascicular planes. Intraoperative direct nerve stimulation will identify the potential donors (FCR, FDS). Stimulation of the remainder of the median nerve will confirm that the function remaining in the nerve includes all the anterior interosseous nerve function, pronation, and thenar muscle functions.

The thoracodorsal nerve is an excellent donor for elbow flexion (Fig 2D).20,24 We reserve this transfer for when other suitable donors are not available (i.e., the medial pectoral, median or ulnar nerves). As the latissimus dorsi muscle is an excellent fall back muscle transfer for shoulder reconstruction. The thoracodorsal nerve can be isolated directly at the level of the infracoacicular plexus as it separates off the posterior cord and through a secondary incision along the anterior border of the latissimus dorsi. The thoracodorsal nerve should be dissected distally as far as possible to allow direct transfer to the musculocutaneous nerve without a nerve graft. The entire thoracodorsal nerve is used in the transfer. Even if there are associated severe traumatic injuries involving the axilla making exploration difficult (e.g., gunshot wound), the thoracodorsal nerve can be easily identified on the chest wall for transfer. Outcomes with this transfer have been very good and it is very useful if other options are not available. This transfer is also valuable if the latissimus dorsi muscle has been used for local coverage and the thoracodorsal nerve remains intact.

Other nerve transfers available to restore elbow flexion include the distal spinal accessory nerve.10-13 This transfer has been well described but requires a long nerve graft to reach the upper arm for repair to the musculocutaneous nerve. The use of the intercostal motor nerves has been well described but the dissection can be tedious, the nerves are small with a limited number of motor axons, and a nerve graft may be required.9,13 We have used the triceps branch of the radial nerve to restore elbow flexion. This transfer will successfully result in reinnervation of the biceps but because of its antagonistic function is sometimes difficult to re-train. Another option is to use the distal portion of the long thoracic nerve.5 Careful dissection of the long thoracic nerve is undertaken to maintain innervation to the proximal part of the serratus anterior muscle to prevent scapular winging. Motor re-education with this transfer is difficult because of the difficulty in generating voluntary contraction of the serratus anterior muscle. Other motor donors include the phrenic nerve and the contralateral C7 nerve root.25,26

The deficit that results from using the contralateral C7 is minimal but its use requires a long vascularized nerve graft and cross cortical re-education is difficult. Use of the phrenic nerve is controversial and rarely indicated as the long-term outcome from loss of diaphragmatic function may be significant if not recovered.

Authors Preferred Technique To Maximize Elbow Flexion

If both the biceps and brachialis muscles are not functioning and there is no elbow flexion from brachioradialis, we prefer either the partial median or ulnar nerve transfer to the biceps and the medial pectoral nerve transfer, with a medial antebrachial cutaneous nerve graft, to the brachialis (Fig 2E). If brachioradialis is already providing elbow flexion, then we transfer either the ulnar or median nerve transfer to the biceps and do not feel it is necessary to also innervate the brachialis. By not having to also innervate the brachialis we avoid having to take down the pectoralis muscle to identify the medial pectoral nerves. The musculocutaneous nerve can be identified by digital palpation within the biceps muscle. Thus, a shorter incision in the midarm will allow transfer of the median or ulnar nerve to the biceps.

Suprascapular Nerve For Shoulder Abduction and External Rotation

Reconstruction of the suprascapular nerve is critical not only for shoulder function but also because external rotation of the shoulder allows elbow flexion through a full functional range. The suprascapular nerve originates from the upper trunk of the brachial plexus and passes across the posterior triangle of the neck and through the scapular notch. This nerve functions to initiate abduction and externally rotate the arm, as well as stabilize the humeral head in the glenoid fossa. Suprascapular reconstruction is the second priority after elbow flexion in the completely paralytic upper extremity after brachial plexus injury. We have used the medial pectoral nerve as a transfer with good results, but our preferred transfer is the distal accessory nerve.5,20 The distal accessory nerve transfer is a good option for reinnervation of the suprascapular nerve, as it is synergistic and a direct repair is almost always possible.27,28

Surgical Technique

The suprascapular nerve can be exposed through a supraclavicular approach by following the C5 root and upper trunk distally. The nerve is also palpable with the surgeon's finger behind and below the clavicle from the infracoacicular approach and can be manipulated superiorly toward the neck exposure. The suprascapular nerve is divided as proximally as possible to allow for repair to the accessory nerve.

The distal accessory nerve is exposed on the anterior border of the trapezius and dissection is carried deep to the muscle. Reflection of the clavicular attachment of the trapezius muscle will allow easy identification of the accessory nerve, which is intimate with the trapezius muscle. The nerve is identified along the deep surface of the trapezius and verified by nerve stimulation (Medtronic VARI-STIM® III). The dissection is then carried distally to expose as many of the muscular branches to the trapezius as possible. The nerve is divided distally to leave some of these branches to the trapezius intact yet still allow the nerve transfer. The nerve is then repaired in the standard fashion without tension to the suprascapular
erve (Fig 3A). If it is impossible to repair the nerve without enison an interpositional nerve graft is used.

Another option to consider is to repair the suprascapular ervice to the accessory nerve in an end-to-side fashion (Fig 3B). The nerve transfer is augmented by performing a proximal rush on the donor accessory nerve or a partial neurectomy. This has been shown experimentally to improve the motor xonal sprouting and subsequent nerve regeneration into the recipient nerve. The added injury to the donor accessory nerve is critical to encourage donor motor sprouting. Without this donor nerve injury, spontaneous motor sprouting from the donor accessory nerve to the suprascapular nerve will not occur. The benefit of this, as opposed to the direct transfer of the entire distal accessory nerve to the suprascapular nerve, is that it allows the accessory nerve to remain in continuity so as not to significantly downgrade trapezius function. The trapezius plays critical role in scapular stabilization and when the spinal accessory nerve is sacrificed, even though it is distal to many of the branches of the trapezius, it has been noted that there can be some associated scapular winging. Similarly, if good shoulder function is not forthcoming, a shoulder fusion (dependent on some trapezius function) will still be a useful fallback option. The end-to-side technique with injury to the accessory nerve is similar to the concept used for a partial hypoglossal to facial nerve transfer.39

**Axillary Nerve and Shoulder Abduction**

The axillary nerve originates from the posterior cord of the brachial plexus and passes posteriorly through the quadrangular space. Accompanying it through the quadrangular space are the posterior circumflex humeral vessels. Once the axillary nerve emerges from the quadrangular space it travels around the surgical neck of the humerus and supplies motor function to the teres minor and deltoide muscles. The axillary nerve primarily functions to abduct the arm. The axillary nerve terminates as the upper lateral brachial cutaneous nerve and supplies sensation to the skin over the inferior half of the deltoid and adjacent arm. Occasionally the injury to the axillary nerve is localized to the quadrangular space area, and a direct nerve graft can be done between the healthy proximal and distal nerve stumps. More frequently, there is an associated proximal root or trunk injury to the nerve fibers that supply the axillary nerve, and a nerve transfer is a more appropriate technique. Nerve transfers have been described using the medial pectoral nerves, intercostal nerves, accessory nerve,
found coming off the posterior cord of the plexus just proximal to the posterior circumflex humeral artery. The posterior circumflex humeral artery is found by following the brachial artery proximally. The first branch found (the last branch off the axillary artery in Zone 3) will be the posterior circumflex humeral artery, and the axillary nerve is found adjacent and just superior to this. The

Surgical Technique

The brachial plexus is exposed in the usual fashion with reflection of the pectoralis major and minor muscles. The axillary nerve is

redundant branches of the triceps, and the thoracodorsal nerve to reconstruct the axillary nerve.37,38
axillary nerve is divided as proximal as possible to its takeoff from the posterior trunk of the brachial plexus. The appropriate motor donor is then transferred to the axillary nerve. The repair is done to the superior/lateral part of the axillary nerve, as this is where the motor component to the deltoid is located. It is important to direct motor axons to the deltoid muscle to avoid loss of motor axons into the lateral shoulder skin.

Leechavengyong describes using a posterior approach for transfer of the triceps branch to the axillary nerve. The axillary nerve is located posteriorly as it comes through the quadrangular space. The triceps branches of the radial nerve are found posteriorly between the lateral and long head of the triceps muscles. The triceps branches are then isolated and can be transferred to the axillary nerve. This posterior approach may be useful in the rare cases where the trauma to the axillary extends very distally and/or severe scarring anteriorly makes the standard approach difficult.

**Authors Preferred Technique To Restore Axillary Nerve Function**

Our transfer of choice is the medial pectoral to axillary nerve transfer which if necessary can be supplemented with a branch from the triceps.

**Ulnar Nerve and Intrinsic Muscle Function and Sensation**

High ulnar nerve injuries result in a significant functional deficit to the hand due to the loss of intrinsic muscle function. Even with sharp transection of the ulnar nerve and early repair, recovery of intrinsic function has been poor. There have been multiple tendon transfers devised in an attempt to correct established ulnar nerve deficits but none are entirely satisfactory.

We have been happy with the restoration of motor function to the hand intrinsics using the distal portion of the anterior interosseous nerve (AIN) as a motor donor (Fig 4A). If median nerve function remains intact, then the distal portion of the AIN innervating the pronator quadratus can be transferred to the deep motor branch of the ulnar nerve. Despite the fact that the AIN is not synergistic with the intrinsic musculature of the hand and there is a size mismatch, it does provide a nearby, expendable, and relatively pure motor nerve that does not require a nerve graft. Although, there is a discrepancy in the number of myelinated nerve fibers in the anterior interosseous nerve innervating the pronator quadratus (1165 ± 351) compared with the number of fibers in the motor branch of the ulnar nerve (4767 ± 810), the results from this transfer provide
Fig 4. Nerve transfers to restore ulnar nerve function. (A) Transfer of the terminal motor branch of the anterior interosseous nerve to the deep motor branch of the ulnar nerve. (B) Transfer of the dorsal and superficial sensory branches of the ulnar nerve to restore sensation in the ulnar nerve distribution.

Surgical Technique

An incision is made ulnar to the thenar crease to expose Guyon's canal and then extended into the forearm to identify the pronator quadratus muscle. The deep motor branch of the ulnar nerve is identified and released in the hand well passed the hook of the hamate. The ulnar artery and vena comitantes are retracted ulnarily, along with the sensory branches of the ulnar nerve. The fibrous, proximal leading edge of the hypothenar muscles is identified and divided to expose the deep motor branch of the ulnar nerve. The motor branch is then neurolysed proximally from the ulnar nerve proper under magnification. There is no plexus formation within the ulnar nerve this distal n in the forearm which allows the motor branch to be separated from the sensory component. This neurolysis does not have to be "physically" performed but can be done "visually" by following the distinct motor fascicles proximally to the level of the pronator quadratus. The neurolysis of the ulnar nerve is performed proximally enough in the forearm to allow a direct repair to the distal anterior interosseous nerve (AIN) without ension. Proximally, the pronator quadratus muscle is identified and the AIN is followed into the muscle. The AIN will rerorize at approximately the mid portion of the pronator quadratus and is sharply divided just proximal to that. Direct repair of the AIN to the deep motor branch of the ulnar nerve is always possible.

Restoration of sensory function to the ulnar aspect of the hand is achieved by transferring the superficial sensory and dorsal sensory branches of the ulnar nerve to the median nerve in an end-to-side fashion (Fig 4B). It has been well documented that the sensory donor nerves will reliably, spontaneously prout in an end-to-side repair. The distal ulnar nerve is exposed from the mid forearm into Guyon's canal. The sensory branch is located and "visually" neurolysed under loop magnification from the motor branch, back to a point were it can easily reach the median nerve. Proximally the dorsal sensory branch of the ulnar nerve is located in a similar fashion and is neurolysed under magnification from the ulnar nerve. The median nerve is then identified and the previously isolated sensory branches of the ulnar nerve repaired in an end-to-side fashion to the median nerve after removing the epineurium from the median nerve. The wrist is splinted in a neutral position for approximately 7 days and then free wrist and finger motion is begun to minimize adhesions and prevent stiffness.

Radial Nerve, Finger, and Wrist Extension

Typically, radial nerve palsies are reconstructed by direct nerve repair, nerve grafts, or tendon transfers. These reconstructive techniques provide reliable recovery of wrist and finger extension. However, there will be some reconstructive situations where these standard techniques are not possible or ideal. In these situations, nerve transfers using expendable median nerve motor donors to the radial nerve can provide another option for reconstruction after radial nerve injury (Fig 5).

Surgical Technique

The median and radial nerves are both identified through a longitudinal volar forearm incision. The radial nerve is found deep to the brachioradialis muscle before its division into the superficial sensory and posterior interosseous nerves (PIN). The superficial sensory branch should be neurolysed from the motor fibers of the radial nerve and excluded from the transfer. The posterior interosseous nerve is dissected proximally to a point above the branch which innervates the extensor carpi radialis brevis (ECRB). Any intraoperative stimulation that is necessary is done at this point to confirm lack of radial nerve
function. The radial nerve is then divided and transferred into the anterior forearm for repair to the median nerve.

The median nerve is then isolated in the proximal forearm. The tendon of the superficial head of the pronator teres muscle is released and the deep head excised to allow visualization of the median nerve. The branches of the median nerve are exposed and, using a hand held nerve stimulator (Medtronic VARI-STIM® III), the branches to the pronator teres, flexor digitorum superficialis (FDS), the anterior interosseous nerve (AIN), the flexor carpi radialis (FCR), and palmaris longus (PL) are identified. The large sensory component of the median nerve does not respond to this direct stimulation. A formal internal neurolysis of the median nerve is not required because at this level of the extremity the branches of the median nerve are distinct and not positioned “within” the median nerve proper. The anatomy in this area has been well studied and is consistent.23 There are consistently two or more branches to the pronator teres and FDS, 73% and 94% of the time, respectively. The AIN usually originates proximal to the distal FDS branch and comes off the median nerve either radially or posteriorly. Due to the limited anatomic variation in this area, the median nerve provides several redundant branches for potential nerve transfer. The expendable branches of the median nerve are transferred to the branch innervating the ECRB and to the PIN.

Authors Preferred Technique To Restore Radial Nerve Function

Our best results for radial nerve reconstruction were with FCR nerve fascicles transferred to the ECRB and FDS fascicles trans-ferred to the PIN. The sensory branch of the radial nerve can be transferred in an end-to-side fashion to the median nerve in an attempt to regain some radial nerve sensation. Postoperatively, patients are placed in a dressing which maintains the elbow in flexion and shoulder in abduction. This is worn for seven days, after which gentle range of motion is begun to maintain joint mobility until radial nerve recovery.

Median Nerve, Forearm Pronation, Thumb Opposition, and Sensation

Forearm Pronation

Partial loss of median nerve function is uncommon but can be managed with nerve transfers either from FCU branches of the ulnar nerve or more simply using expendable motor branches from the median nerve itself (Fig 6).23 If the loss of pronation is an isolated finding and the median nerve function to the FDS remains intact, this innervation to the FDS provides a good motor donor to restore pronator function. Another option is to transfer redundant fascicles of the ulnar nerve innervating the FCU muscle.

Surgical Technique

The median nerve in the proximal forearm is exposed through a standard approach. The two branches of the median nerve to the pronator teres, which will not respond to electrical stimulation, are identified, as are the AIN, FCR, PL, and FDS.
branches. Intraoperative nerve stimulation allows confirmation that there are at least two functioning branches to the FDS. One or both of these branches to the FDS can be transferred to the two nonfunctioning branches of the pronator teres and a direct repair is performed. The palmaris longus or flexor carpi radialis branches can also be used but our preference is to use the branches to the FDS.

If the median nerve is not functioning at all, then a branch of the ulnar nerve that innervates the flexor carpi ulnaris can be used as a motor donor to recover pronation and standard tendon transfers used to restore finger and wrist flexion. This transfer is performed by exposing both the median and ulnar nerves. The nerve transfer can be facilitated if the ulnar nerve is transposed anteriorly, as this brings the ulnar and median nerves closer together. Redundant fascicles to the FCU are isolated and neurolysed from the ulnar nerve proper. Intraoperative nerve stimulation (Medtronic VARI-STIM® III) is used to identify the fascicles to the FCU, as well as to ensure that adequate innervation remains to the ulnar innervated muscles. The branches to the pronator teres are isolated and, using standard microsurgical technique, the FCU branch is transferred to the pronator teres branches.

**Thumb Opposition**

Tendon transfers are a reliable reconstructive option for thumb opposition. However, transfer of the distal AIN that innervates the pronator quadratus to the recurrent branch of the median nerve can be considered as a reconstructive option. With median nerve injuries, direct repair may not always result in intrinsic median motor recovery, as the vast majority of the nerve is composed of sensory fibers and the motor topography of the proximal nerve stump is difficult to delineate. In such cases, the AIN innervating the pronator quadratus can provide an expendable motor donor to reinnervate the median innervated intrinsic muscles. The procedure is performed through an extended carpal tunnel incision that allows identification of the recurrent motor branch of the median nerve as well as the distal AIN. The recurrent motor branch of the median nerve is isolated. The AIN is then found in the distal forearm as it enters the pronator quadratus muscle. The AIN will arborize within the pronator quadratus muscle, and the nerve is divided just proximal to this. The AIN is then transferred into the recurrent motor branch. This transfer will usually require a nerve graft, depending on the location of the median nerve injury in the forearm.

**Median Nerve Sensation**

Loss of median nerve sensation in the hand is a tremendous functional deficit, as it provides important proprioceptive information for pinch and fine motor movements. As with motor nerve deficits, reconstruction should be attempted with direct nerve repair or nerve grafts when possible. When reconstructing median nerve sensation, it is especially important to focus on restoring sensation to the radial side of the index finger and the ulnar side of the thumb. Multiple nerve transfers have been devised to reconstruct median nerve sensation using the radial and ulnar nerves as donors (Fig 7). With upper trunk brachial plexus injuries, sensation in the third web space (ulnar side of long finger and radial side of ring finger) will be intact and can be transferred to the first web space (ulnar side of thumb and radial side of index). With high median nerve injuries, the ulnar nerve is most commonly used, as it is readily accessible and has branches which can be sacrificed without significant impairment to the fourth web space. The ulnar nerve branch that innervates the fourth web space can be transferred across the palm and used to preferentially innervate the digital nerves to the radial side of the index finger and the ulnar side of the thumb. Direct repair is possible and no nerve grafts are required (Fig 7C). The digital nerves that innervate the second and third web spaces can then be transferred to the sensory component of the ulnar nerve in an end-to-side fashion.

Other less favorable options include the dorsal cutaneous branch of the ulnar nerve, which can be transferred volarly for repair to the sensory component of the median nerve (Fig 7B). As well, the sensory branch of the radial nerve can be used as a donor to regain median nerve sensation in the hand (Fig 7A). In all cases, the distal part of the donor sensory nerve is repaired directly to the radial side of the median nerve to direct sensation.
Fig 7. Nerve transfers to restore median nerve sensation in the hand. (A) Transfer of radial sensory nerve to the median nerve. Common digital nerve to the 3rd web space transferred to ulnar in end-to-side fashion. (B) Transfer of the dorsal sensory branch to the median nerve. Common digital nerve to 3rd web space transferred to ulnar nerve in end-to-side fashion. (C) Transfer of the sensory branch of the ulnar nerve to the 4th web space to restore sensation to the ulnar side of the thumb and radial side of the index finger. The common digital nerves to the 2nd and 3rd web spaces transferred to the ulnar nerve in an end-to-side fashion.

toward the thumb and index finger. The ulnar part of the median nerve can then be neurolysed from the median nerve proper and transferred in an end-to-side fashion to the ulnar nerve. In all of these cases, if possible, the distal part of the donor nerve is connected to a normal adjacent sensory nerve in an end-to-side fashion to attempt to provide some sensation back to the donor site.

Conclusion

Nerve transfers are a viable option for reconstructing significant nerve deficits after devastating nerve injuries. These transfers provide new options to obtain significant functional recovery in reconstructing what was once thought impossible. Nerve transfers which were once reserved for only the most severe of nerve injuries are now finding increased use in all types of nerve injuries with very promising success.

References

NERVE TRANSFERS


Surgical interventions for nerve compression neuropathy are some of the most common operative procedures performed on the upper extremity. While neuropathy can be objectively evaluated using various parameters, controversy persists regarding etiology, incidence, operative indications and the types of procedures recommended. Despite the controversies, surgical intervention for well documented cases of compressive neuropathy results in a very high percentage of successful outcomes when compared with other procedures to give relief of pain.\(^1\)\(^2\)\(^3\) This continuing education review will focus on operative indications, surgical technique and postoperative care for carpal tunnel syndrome, ulnar tunnel syndrome, cubital tunnel syndrome, pronator syndrome, and posterior interosseous nerve syndrome. These maladies are common enough that plastic surgeons should be able to diagnose the problem and provide surgical intervention for relief from these maladies.

Carpal Tunnel Syndrome

The symptoms of carpal tunnel syndrome (CTS) are: hystesthesia in the distribution of the median nerve, nocturnal hystesthesia, positional hystesthesia, proximally radiating pain, thermal regulatory changes, weakness of grip, object dropping, muscle spasms, and muscle atrophy.

These symptoms are produced by changes occurring within the median nerve as it courses through the carpal tunnel's confining structures (Fig 1). The neuropathy has been associated with anatomic anomalies, synovial hypertrophy, lumbrical hypertrophy, tendon volume change, strain demyelination, nerve vascular changes, endocrine abnormalities, inherited lysosomal storage disorders, skeletal alterations, acute and chronic infections and even emotional predispositions.\(^4\)\(^5\) Simply put, anyone can develop CTS, and all etiologies need to be considered and evaluated.

To confirm the clinical impression of median nerve neuropathy in the forearm, both motor and sensory testing should be documented preoperatively as a baseline study utilizing both clinical and electrophysiologic testing. While the majority of patients do well following decompression, for those who have persistent or recurrent symptoms, the preoperative documentation will be invaluable in determining potential reasons for their problems.\(^6\)

Surgical intervention is indicated in individuals with persistent symptoms after conservative measures such as splinting, decreased activity, anti-inflammatory medications, and other supportive measures have failed.\(^7\) Carpal tunnel decompression is often thought of as an “easy” operation; however, nerve injuries associated with carpal tunnel decompression have been reported in multiple series.\(^8\) The goal of surgery is to decrease irritation of the nerve as it crosses the wrist. This involves release of the transverse carpal ligament (TCL), volar carpal ligament (VCL) and the distal forearm fascia as it transitions into the VCL above the wrist (Fig 2 and 3). Important anatomic landmarks are shown in illustration 4.

**Open Carpal Tunnel Release**

The operative details depend to some degree on the surgeon’s preference, but the following steps help establish a process of planning, performing surgery, and rehabilitating patients after surgery that is thorough and effective.

**Anesthesia.** Anesthesia for carpal tunnel release is most commonly done with local or regional anesthesia. The type of anesthesia should be designed to match the patient’s health status, the patient’s anxiety level, and the surgeon’s operative plan. Tourniquet control is essential to provide clear visibility of vital structures. Bier blocks provide both anesthesia and tourniquet control and is the most common anesthetic technique used by the author. Local anesthesia with tourniquet control is another common technique. The tourniquet discomfort is often difficult for patients to tolerate with local anesthesia.

**Operative Technique.** The determining factors for incision location are dominated by concern for the cutaneous sensory branches to the palm from both the median and ulnar nerves (Fig 4). Severance of these nerves or their main branches can produce painful dysesthesias in patients and lead to complaints of painful incision. The preferred incision by the author is shown in Fig 5. These two incisions are preferred to one long incision because of the propensity for a continuous incision across the wrist to create a prominent, unsightly scar in some patients. The single palm incision is possible in some patients but makes assuring proximal release more difficult and injury to the median nerve more likely by blind “snipping or slicing” of the proximal elements of the VCL. The palm incision is extended to the level of the palmar fascia (PF) through the septated fat of the palm. The PF is opened in a linear direction on the presumed location of the ulnar border of the carpal tunnel. If too ulnar, the ulnar artery and vein are cut and the patient will be exposed to inadvertent injury (Fig 6). Located immediately under the PF are the arborizations of the ulnar artery into the superficial vascular arch. The ulnar and distal border of the TCL can be identified by locating the flexor mechanism or by palpating the hook of the hamate. The ligament is divided adjacent to the hook of the hamate (Fig 7). Failure to divide the ligament on the ulnar border will expose the recurrent motor branch of the median nerve to injury as it can be found traversing the ligament rather than being found distal to the ligament. Once identified and visualized the ligament is divided. In the
Fig 1. The confines of the carpal and ulnar tunnels are osseous or rigid soft tissues maintained across a major fulcrum.

After releasing the structures, the surgeon's fifth finger will readily pass under the wrist through the tunnel. The contents of the carpal tunnel are elevated out of the tunnel and the tunnel examined for ganglions or other abnormal structures that could be contributing to the compression of the nerve. It is important to note the always-present fat pad located in the distal aspect of the tunnel, which should remain; it should not be mistaken for a lipoma to be removed. Steroidal anti-inflammatory pharmaceuticals are placed within the tunnel and dispersed around the tendons.

Elements of the PF are approximated and bupivacaine-containing epinephrine injected around the incision margins. The tourniquet is deflated and hemostasis completed. After skin closure is completed, additional bupivacaine is instilled into the surgical site by pushing it through gaps in the sutures. The local anesthesia greatly decreases complaints about postoperative pain.

In the immediate postoperative period, movement of the digits and the wrist is encouraged. Providing a removable, custom splint (Fig 8) facilitates this. It is worn for 2 to 3 weeks to protect the incision and restrict utilization but is removed at least 6 times each day to exercise the wrist. Return to work at a light duty status occurs 7 to 10 days after surgery and return to unrestricted work requires 4 to 10 weeks healing time depending on occupation.

Fig 2. The contents of the tunnels are variable in volume, function and adaptability

Endoscopic Carpal Tunnel Release

Endoscopic carpal tunnel release (ECTR) was popularized by Chow* and subsequently heralded and criticized. It has become an established and effective method of decompression of the median nerve as it traverses the wrist. Its advantages are: avoiding injury to the palmar cutaneous branches, causing less postoperative pain, and having quicker rehabilitation. Its disadvantages are: lack of visibility to search for abnormalities in the tunnel, steeper learning curve, and requirement of special equipment. When planning for an ECTR, the patient must be counseled regarding the potential for conversion to the open technique if visibility is unusually obscured. When it occurs, there will be less motivation, having counseled the patient properly, to continue despite compromised visibility: the likely predecessor of nerve injury.

The topographic landmarks for planning ECTR are shown in Fig 9. The two portal techniques are utilized because visibility is improved distally and direct inspection for common digital nerve displacement is feasible. Tourniquet control is essential to visualization. Bier block and local anesthesia with tourniquet control are the two common methods of providing anesthesia.

Operative Technique. The first incision is placed 1 to 2 cm proximal to the wrist crease. The incision must be located ulnar

Fig 3. The forearm's investing fascia transitions into the volar carpal ligament and its components join the transverse carpal ligament. The authors perform a subcutaneous distal forearm fascia for a distance of 6 to 8 cm above the wrist crease when the fascia seems tight or constrictive proximally.

Fig 4. Palmar cutaneous branches of both the median and ulnar nerve are illustrated and if injured during surgery may result in dysesthesia in the palm.

Fig 5. TRAP-CARPAL ULNAR TUNNEL
to the palmaris longus or ulnar to the 3rd metacarpal to avoid the course of the palmar cutaneous branch of the median nerve. The incision is 1.5 cm in length. The transition between the forearm fascia and the VCL is identified and carefully opened in a transverse direction because the median nerve is located just under the fascia. Using a blunt dissector (Instratec), the ulnar border of the tunnel can be identified by the washboard-type feeling created by the ligament. The slotted cannula is introduced with the slot at the twelve o'clock position. The tip of the cannula's obturator is palpated in the palm distal to the TCL but under the PF. This tip will usually be palpated 3.5 to 4.0 distal to the wrist crease. A second 1.5-cm incision is made over the obturator tip and extended down to the PF. The probe is pushed through the fascia, the fascia is opened, and the probe checked for the potential of having a common digital nerve trapped on top of the cannula. The obturator is removed. The wrist is then placed in 45 to 60 degree extension (Fig 10) and a 4-mm 30 degree angle scope is positioned in the cannula using the distal port. The scope is used to visualize the tunnel and assure that nothing lies between the cannula and the overlying tunnel. If the distinct transverse bands of the tunnel are visualized clearly (Fig 11), the technique can be completed by dividing the ligaments on the ulnar border of the tunnel adjacent to the hook of the hamate. The author prefers to divide all structures completely, the adequacy of which is confirmed by visualizing subcutaneous fat extruding into the tunnel. This may require two passes of the knife in some areas. Once division of the TCL is completed, attention is directed proximally. The cannula is removed and redirected proximally under the VCL and/or forearm fascia. Utilizing a meniscotome blade directed along the slot of the cannula, the VCL and distal forearm fascia are released for a distance of 6 to 8 cm. A long acting steroid and anti-inflammatory and bupivacaine with epinephrine are irrigated into the surgical site for relief of pain. A similar technique of rehabilitation is utilized as with the open technique, but the patient’s ability to return to work usually occurs earlier depending on the occupation.11

Ulnar Tunnel Syndrome

Ulnar tunnel syndrome refers to compression of the ulnar nerve as it courses through Guyon’s Canal (Fig 12). This syndrome is rare but because of the canal’s location adjacent to the median nerve and the carpal tunnel, it is often released because of its “close proximity.”

Ulnar tunnel syndrome can be confused with its more proximal malady, cubital tunnel syndrome. When the nerve is compressed at the wrist, the symptoms and findings can be insidious and overlooked by both patient and physician. Ulnar nerve compression in the Canal of Guyon can manifest with numbness in ring and small fingers, numbness combined with motor...
weakness in the hand, or motor weakness alone. The onset of motor weakness as an isolated finding is insidious and only when substantial atrophy of intrinsic muscles has occurred is the diagnosis investigated. In contrast, the physician typically investigates numbness, either by itself or in conjunction with muscle involvement, sooner because it is a symptom troubling to the patient and often thought to be cubital tunnel.

Differentiating ulnar tunnel syndrome from cubital tunnel syndrome is important. This can usually be done clinically by eliciting a thoughtful history and performing a careful examination of the hand. With cubital tunnel syndrome there is usually tenderness and a Tinel's sign at the elbow. Numbness extends proximal and is specifically found on the dorsum of the hand. Weakness and atrophy involves all of the hypothenar muscles. Ulnar tunnel syndrome does not produce numbness on the dorsal hand because the dorsal sensory branch of the ulnar nerve takes origin central to Guyon's Canal. Tinel's sign or tenderness over the nerve is usually present at the wrist. Atrophy of the hypothenar muscles is often spared because their motor branches are proximal to the site of most compres-

Fig 11. Unless clear visualization of the ligament is present, the endoscopic technique should be converted to an open technique.

sion in Guyon's Canal. The most common site of compression in ulnar tunnel syndrome is where the most dominant distal motor branch leaves the ulnar nerve proper and courses under the pisohamate (arcuate) ligament (Fig 13). The branches continuing distal are the sensory branches and are often spared from compression. The confluence of structures creates a foramen like structure that tethers the nerve's position and makes it prone to compression. Synovial swelling, ganglions, tumors, aneurysms and edema impact the nerve most commonly at this site. Compression at this site specifically causes atrophy of the volar intrinsics, dorsal intrinsic, first dorsal interosseous and adductor pollicis brevis muscles. Most noticeable to patients is the appearance of a concavity between the metacarpals and first web space. Since sensory loss does not occur, detection may be delayed. Nerve conduction studies utilizing sensory tests and even motor tests with electrodes over the thenar muscles are often normal. To avoid confusion with disuse atrophy as can be

Fig 12. The motor branch of the ulnar nerve passes under the ligament extending between the pisiform and the hamate. This structure will not be visualized unless the ulnar nerve and the ulnar nerve's sensory branches are mobilized and elevated.
seen in the elderly, electromyography is used to conclusively demonstrate denervation of the muscle as indicated by a fibrillation potential or positive sharp waves.

When ulnar tunnel syndrome is associated with constriction associated with the volar carpal ligaments constriction, releasing it will solve the problem. In the author’s experience, the more common etiology is compression of the nerve where it divides into motor and sensory branches. Simply releasing the ligament will not alleviate this compression. The etiology of compression is often obscured by the overlying ulnar artery and venae comitantes. Unless they are lifted and the motor branch visualized the operation may not alleviated the problem.

Operative Technique

Operative technique to explore the ulnar tunnel can be completed through an incision that is just ulnar to the third metacarpal similar to CTR. This incision site is preferred because it avoids injury to the palmar cutaneous branch of the ulnar nerve (PCUN). PCUN takes origin approximately 5 to 8 cm above the wrist crease and is located ulnar to the 4th metacarpal. The volar carpal ligament courses from the hook of the hamate to the pisiform and forms the roof of the canal. It is 1 to 2 cm wide, located proximal to and separate from the palmaris brevis muscle. To see the most likely site of compression the palmaris brevis muscle has to be divided. The muscle should be divided on its radial side to prevent denervation and reflected lateral. The ulnar artery and veins will cover the nerve and have to be retracted. Small branches are cauterized with the bipolar cautery. The ulnar nerve is identified and lifted from bed. The motor branch is immediately visible and can be seen to be coursing under the tethering edge of the arcuate fibrous arch partly formed by the flexor digiti minimi and opponens digiti minimi aponeuroses Fig 13. It is this fibrous edge that must be removed. It is also this edge that the nerve is impacted against by expanding masses such as tumors or ganglions Fig 14. Postoperative protective splinting for 2 to 3 weeks combined with early mobilization and strengthening activity usually results in substantial improvement even when advanced atrophy is present. In some circumstances, cubital tunnel and ulnar tunnel syndrome can coexist or, more problematically, the site of injury may not be definitively ascertainable. When either circumstance presents, both sites should be decompressed simultaneously.

Cubital Tunnel Syndrome

Irritation of the ulnar nerve as it traverses the cubital tunnel is the second most common form of nerve compression involving the upper extremity. It produces pain originating in the area of the medial epicondyle and radiating both proximally and distally from the elbow. A common complaint is numbness along the ulnar border of the hand usually isolated to the small finger and one half of the ring finger, though involvement of the
long finger can occur. The numbness will specifically include the dorsal aspect of the hands. This differentiates neuropathy at the level of the elbow from neuropathy located at the wrist. Neuropathy at the wrist does not involve the dorsal sensory branch of the nerve. If hypesthesia occurs, it involves the palm skin of the ulnar border digits but not the dorsal hand. Patients in advanced cases will notice the interdigital and web space areas as sunken, and may also note the accompanying weakness from intrinsic atrophy. Examination of the flexor profundus of the fifth finger and the flexor carpi ulnaris may also demonstrate weakness associated with ulnar nerve compression through the cubital tunnel.

The anatomy of the cubital tunnel is shown in Fig 15.

The surgical indications for decompression of the cubital tunnel are clinical signs and persistent symptoms that support the diagnosis in the face of electrophysiologic studies that demonstrate a significant conduction velocity decrease across the tunnel, a fibrillation potential, or positive sharp waves in muscles supplied by the nerve.13

The surgical procedures most commonly advocated for cubital tunnel release are: decompression, decompression with subcutaneous transfer, decompression with submuscular transfer, medial epicondylectomy, and endoscopic release.2 All of the procedures report success, but the procedure that seems most applied and most successful in review of the literature is decompression combined with submuscular transfer.2 14

Anesthesia

Because an axillary block may confuse or complicate the setting of a more proximal ulnar neuropathy, the operation is best performed using general anesthesia. The upper extremity is washed from axilla distally and then draped. A sterile pneumatic tourniquet should then be applied. This permits accurate identification of small sensory nerves during the procedure by keeping a bloodless field. Bupivacaine with epinephrine injected in the skin incision edges and flushed into the surgical site after closure helps with pain management.

Operative Technique

The incision is placed as shown in Fig 16. It is located midway between the medial epicondyle and the tip of the olecranon. The incision extends 6 to 8 cm distal to the epicondyle and 8 cm proximal to the epicondyle. This will allow excellent visibility and provide for a smooth transition for the nerve after it is transferred anteriorly. After incising the skin, loupe magnification is used to identify branches of the medial antebrachial

Fig 17. The nerve is mobilized out of the tunnel leaving branches of the inferior ulnar collateral vessels with the nerve.

Fig 19. The medial intermuscular septum must be removed if the nerve is transposed anterior and sub-muscular in location. If it remains it will compress the nerve.

Fig 18. A sub-muscular plane is created underneath the muscle but superficial to the ulnar collateral ligament.

Fig 20. Distal release of the FCU aponeurosis and muscle origins off the proximal ulna permit smooth anterior transposition. The motor branch to the FCU will be seen leaving the ulnar nerve.
cutaneous (MABC) nerve. Also in the superior aspect of the incision the entire MABC nerve will often be visualized (Fig 15). These nerves should be protected or painful neuromas and distal hypesthesia can result. Both are usually interpreted as persistent problems by the patient.

Once the tunnel is identified, it is opened and the nerve mobilized out of the tunnel (Fig 17). The distal branch of the ulnar collateral artery is preserved and transferred with the nerve to decrease any chance of an ischemic neuropathy as an outcome of the transfer. In most instances a small articular sensory branch of the ulnar nerve takes origin in the tunnel (Fig 18). This branch should be protected during the transposition of the nerve but may require dissection distally to allow adequate length for the transfer. Again, it is the potential of neuroma formation by the sensory branch that is of concern and mandates preservation of the nerve if possible. Once the nerve is lifted out of the tunnel, the medial intermuscular septum is identified and removed (Fig 19). After the nerve is transferred, this structure would be a sharp band compressing the nerve unless it is removed. Removal will result in the medial epicondyle looking more prominent on a permanent basis, and it is important to inform the patient preoperatively of that outcome.

The common origin of the flexor carpi radialis, flexor carpi ulnaris, palmaris longus, pronator teres, and flexor sublimis is released from the medial epicondyle to create a submuscular location for the transposed nerve (Fig 20). The flexor profundus muscle is divided off the ulnar proximal shaft. The oblique incision should leave a 1 to 2 cm cuff of the fibrous common origin to permit reattachment of the common origin after transposition (Fig 21). When elevating the common origin it is not possible to create a completely “submuscular” plane. Attempts to do so will result in division of the ulnar collateral ligament (Fig 18).

In fact, the nerve will be covered with a composite musculotendinous cover. It is important to release the specific aponeurosis of the flexor carpi ulnaris during transposition to allow a smooth angle distal to the epicondyle. Following transposition of the nerve, the muscles are reattached to the medial epicondyle. The oblique residual cuff on the epicondyle allows for easy approximation in a slightly elongated position. Patients are returned to light work restricted mostly to using their other hand at 5 to 10 days following the surgery. Most are able to return to unrestricted work by 6 to 8 weeks postoperatively.

A removable, long arm splint, set at 100 to 120° extension, is supplied to the patient. The splint is removed at least 6 times a day and the elbow flexed and extended to the degree tolerated.
Fig 25. Struther's ligament, a rare abnormality, can also compress the nerve. It can be detected through the incision shown using lighted retractors.

by the patient. The splint is used to protect the incision and surgical site for 2 to 3 weeks. Because of the detached muscles, resistive activity is avoided for one month and maximum resistance for six weeks.

Pronator Syndrome

Compression of the median nerve and its anterior interosseous branch (AIN) at the level of the elbow is a more common etiology of median nerve compression than previously realized. Failure to respond and recover from CTS may occur because of a second, more proximal compression. It is essential to exclude this diagnosis before CTS to eliminate the possibility of nerve decompression in the wrong area and to eliminate the possibility of dual level irritation of the median nerve. Components of this more proximal neuropathy include hypesthesia in the distribution of the median nerve and weakness in the thenar muscle similar to CTS. Radiating pain into the hand and forearm as well as generalized aching and cramping in the forearm are common complaints. Differentiation from CTS is possible because in advanced cases, weakness of the profundus tendon of the index finger or weakness of the flexor pollicis longus are features. In less advanced cases, provocative compression testing over the median nerve, Tinel's signs at the elbow, tenderness over the nerve, and hypesthesia in the distribution of the palmar cutaneous branch help differentiate between the two sites.

Pronator syndrome and anterior interosseous nerve syndrome (AINS) have been distinguished by motor and/or sensory losses: AINS was defined as involving solely motor loss while pronator syndrome involved both motor and sensory losses. However, the most common finding during surgery in the author's practice has been the deep head of the pronator crossing both of the nerves to join the superficial head but having a greater impact on one or the other nerve (Fig 19). The deep head varies from being almost entirely a tendinous structure to, more commonly, a small muscle with a tendon within its substance. The origin of the deep head can span a distance of 1 to 3 cm along the ulna's lateral edge. Where the deep head and superficial head of the pronator join each other, a distinct acutely angled arch is formed. This arch encompasses both the anterior interosseous and the median nerves and will often result in narrow 1 to 3 mm indentations of the nerves. Hyperesthesia so notable in broader areas of compression in CTS is seldomly noted. The arch always has a significant tendon com-
component and the arch must be divided with, preferably, the deep component excised (Fig 22). This arch can be located 6 to 8 cm below the cubital crease. It is said that sublimus tendon bands can compress the nerve. This has not been observed in the author’s experience, and it is possible that a distally located union of the two pronator heads could be mistaken as a sublimus band. The important point is to realize that the compression can be located either distal to the take off of the AIN or across both the median nerve and the AIN. The author has never observed the AIN being entrapped under this arch alone without the median nerve. It is possible that the proximal versus distal location of this arch is why, in some circumstances, this syndrome may present with only sensory findings or a combination of sensory and motor findings. The most prevalent electrophysiologic assessment can also support the diagnosis by detecting fibrillation potentials in the pronator quadratus or flexor pollicis longus muscle and by detecting a decreased conduction velocity across the elbow area, which has been noted in some patients. Unless the clinical findings are very conclusive, surgeons should be reluctant to operate on patients who do not have electrophysiologic abnormalities.

Operative Technique

A sterile tourniquet to maintain a bloodless field combined with a general anesthetic is essential to this operation. The structures within the surgical field are of critical importance and must be accurately identified and protected during exploration and decompression.

The incision, 6 to 8 cm in length, is made entirely below the cubital crease. Incisions across the crease lead to untoward scars and are, in the author’s experience, unnecessary. The incision extends down to the lacertus fibrosis; avoiding injury to the anterior branch of the medial antebrachial cutaneous nerve is essential. The brachialis and biceps tendon insertion comprise the lateral border of dissection and the flexor mass of the forearm, the lateral border. Following complete excision of the lacertus fibrosis, the brachial artery and veins are readily displaced laterally to reveal the median nerve located medial to them and adjacent to the flexor wad. The anterior interosseous nerve has different anatomical characteristics but usually is about 1 to 2 cm in length as a discreet branch and then begins toborize. The nerve can be better visualized after removing the compressive deep head of the Pronator (Fig 23 and 24). While removing the entire length of the deep pronator component, the trifurcation of the brachial artery must be protected. Proximal dissection by elevating the skin with a retractor off of the vessels and nerve permit examination by palpation and direct inspection to assure that the extremely rare Struthers ligament type entrapment is not present (Fig 25). The tourniquet is deflated and hemostasis assured. Steroidal antiinflammatory and bupivacaine are irrigated into the incision. Postoperatively, a removable, long arm splint is used. The splint is removed at least six times a day to allow mobilization of the elbow and wrist. Light duty is resumed within 5 to 7 days and regular work activity 4 to 8 weeks after surgery. The incision scar may become reactive in this location, and topical self-adherent silicone sheeting helps prevent hypertrophy.

Posterior Interosseous Nerve Syndrome

Posterior Interosseous Nerve Syndrome (PINS) is a compression of the posterior interosseous nerve (PIN) as it traverses under the proximal edge of the supinator muscle Fig 26. Some authors include this syndrome in the broader term, radial tunnel syndrome. In this discussion, PINS is distinguished as a separate entity because of its specific etiology and clinical features. Patients with this diagnosis will have symptoms of pain radiating along the dorsal aspect of the forearm and lateral aspect of the upper arm. PINS and lateral epicondylitis are usually considered separate entities; however, in some patients the diseases seem to coexist. PINS is usually most painful approximately 5 cm distal to the lateral epicondyle. A trigger point of pain is detected at this distance when one palpates between the mobile and immobile portions of the extensor muscles (Fig 27). Reproduction of pain symptoms is usually possible by putting strain across the nerve by exerting static supination force across the nerve and also by compression over the nerve trigger point for 30 seconds. Usually, lateral epicondylitis pain is over the epicondyle and accentuated by extensor mechanism stress to the area. MRI of the lateral epicondyle is helpful in establishing the diagnosis of lateral epicondylitis if the diagnosis is not clinically obvious. If clinical testing demonstrates sensory deficits and motor deficits, one must be suspicious of compression at the humerus groove or radial head. If the deficit is only sensory compression of the sensory branch as it exits from under the brachioradialis, Wartenberg’s Syndrome may be responsible. Electrophysiologic testing is seldom helpful unless the degree of neuropathy is advanced. The extensor digitorum communis, extensor indicis proprius, abductor pollicis longus, extensor pollicis brevis, and extensor pollicis longus are innervated by the PIN after it courses under the Arcade of Froshe.

Operative Technique

Similar to pronator syndrome, the release of the PIN is performed with tourniquet control and general anesthesia. We prefer not to perform axillary or stellate ganglion blocks in patients that already have evidence of neuropathy.

The incision is centered over the point 5 cm distal to the lateral epicondyle landmark. If a lateral epicondylectomy is required, the incision is extended proximally to the lateral epicondyle. Cutaneous branches of the lateral antebrachial cutaneous nerve are located in this area and must be avoided. An intermuscular plane is developed between the brachioradialis
and the extensor carpi radialis longus muscles (Fig 26). This plane is not well defined. The muscle fibers are spread in a linear direction. On going through the depth of the plane, the perineural fat of the PIN and radial nerve will be noted (Fig 28). The PIN courses under the proximal head of the supinator muscle. Supinator fibers are oriented obliquely across the PIN while the superficial component of the nerve follows the linear fibers of the brachioradialis. The vascular leash of Henry is usually present but has never seemed to be the reason for compression. The proximal edge of the supinator, however, does compress the nerve producing a narrow segment indentation on the nerve; a pseudoneuroma may be present (Fig 29). Be particularly alert to this diagnosis in individuals with a history of fracture, blunt trauma, or hemorrhage in this area. Once the edge of the supinator is identified, the entire proximal fibrous edge is removed until only muscle fibers are present. There are reports that the nerve can be compressed at the distal margin of the supinator; the author has never observed this variation. Postoperative management is similar to that for pronator syndrome and the result predictable if careful clinical screening is observed.

Summary

At least three nerve compression syndromes affecting the forearm are associated with the three major nerves as they cross the elbow; two more syndromes are associated with them as they cross the wrist. Thorough establishment of the diagnosis combined with meticulous surgical technique are rewarded with successful outcomes for patients suffering from compressive neuropathies of the upper extremity.

References

Peripheral nerve injury has been the focus of surgical research for centuries. Initial advancements included not only identifying nerves in vivo and differentiating them from surrounding structures such as tendons, but also understanding their function and unique anatomy. The understanding of the physiology of neurotrophic factors and the relationship to axonal regeneration has contributed to developing new methods of nerve repair. Operative exploration of an injured nerve involves assessment of many factors that will decide if a primary or secondary repair will be performed. The vascularity of surrounding tissue, the amount and quality of soft tissue coverage, the type of nerve injury, the age and general medical condition of the patient, and associated injuries are some of the factors that are assessed. Types of primary repair include epineurial and perineurial. Different methods of facilitating differentiation of motor and sensory fascicles include electrostimulation and histochemical staining techniques. Once the nerve is identified and trimmed back to viable tissue, any type of nerve gap must be assessed. If there is tension at the reapproximation site or significant gap, a nerve graft can be done at a secondary surgery. Other options include using polyglycolic acid or vein graft conduits. New contributions continue to be made as our understanding of peripheral nerves advance on the biochemical, physiologic, and anatomic levels.

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"A nerve is a soft and pervious tube from brain or spinal marrow to bring sensation or motion." The words of Daniel Turner in 1736 echo the fundamental appreciation of the structure and function of peripheral nerves. For centuries, surgeons have been striving for excellence in the field of acute nerve injuries. From the concepts of primary and secondary nerve repair to nerve grafts, the contributions continue to flourish amidst the current technological and biochemical advancements.

History

Acknowledgement of the existence of nerves in peripheral anatomy is found in the works of Hippocrates. The studies of Galen revealed his disbelief in nerve regeneration after nerve transection. In 1795, Cruikshank and Haighton published their research on nerve regeneration in bilateral vagotomies in dogs. Muller and Schwann then studied transactions of rabbit sciatic nerves and confirmed distal stump axons. In 1847, Paget documented complete recovery of median nerve function follow-

Anatomy

The peripheral nerve is composed of three fundamental connective tissue layers: the epineurium, perineurium, and endoneurium (Figs 1 and 2). The epineurium is subdivided into outer and inner components. The outer epineurium surrounds the fascicles and is associated with nutrient vessels during entry into the nerve. The inner epineurium absorbs external pressure and stress while permitting motion. A larger proportion of epineurium is found in high pressure and stress locations of the body, such as the joints. The fibroblasts of the epineurium aggressively proliferate after nerve injury.

The perineurium forms the sheath of individual fascicles. It contains the elastic properties thereby resisting longitudinal traction. The protective characteristic of the perineurium is similar to the blood-brain barrier. Maintaining the ionic balance and pressure ensure proper function of the nerve. Outer and inner layers also form this sheath. Tight junctions connect the multiple layers of lamellae interspaced between collagen fibers. Localized conduction blocks result from damage to the perineurium.

The endoneurium is composed of structures such as collagen, the Schwann cell basement membrane, and ground substance. This layer surrounds the axon, provides additional protection, and also resists longitudinal traction.

The blood supply to the peripheral nerve is provided by the longitudinal vessels within the epineurium. These vessels then penetrate into the perineurium. This connects into the endoneurium via a longitudinal vascular network.

Fascicles are composed of groups of axons. Current thought reveals that fascicles are proximally differentiated based on function.

Knowledge of the topography of peripheral nerves is impor-
through the antebrachial fascia approximately 0.8 cm proximal to the wrist crease and then separates into ulnar and radial branches.

The radial nerve divides into the posterior interosseous (motor) and superficial radial nerves (sensory) in proximity of the lateral elbow. Approximately 7 cm proximal to the wrist, the superficial branch emerges through the forearm fascia between the brachioradialis and the extensor carpi radialis longus tendons. After traveling through both superficial and deep portions of the supinator, the posterior interosseous nerve bifurcates into the superficial and deep divisions. Muscles that are innervated by the deep branch of the posterior interosseous nerve include the extensor pollicis longus, abductor pollicis longus, extensor pollicis brevis, and extensor indicis proprius. The extensor digiti minimi, extensor digitorum communis, and extensor carpi ulnaris are innervated by the superficial branch of the posterior interosseous nerve.

**Management**

Assessment of an acute peripheral nerve injury is multifaceted. A complete history and physical examination, if possible, is necessary preoperatively. Assessment of prior injuries or abnormalities, a detailed account of the injury noting the time of occurrence, deficits noted on physical examination, and identification of additional surrounding injuries are critical in deciding an appropriate treatment plan. Occupation and hand dominance are also noted.

The level of injury is considered one of the most important prognostic factors for regaining function. For several reasons, distal injuries portend better return of function when compared with more proximal injuries. Because of the greater distance from the target organ, more time is required for axonal regeneration. Also, proximal injuries involve a greater proportion of cell mass. There is less fascicular homogeneity in the more proximal nerve.

Mechanism of injury is an additional factor in the clinical assessment of nerve injury. Sharp transections in the horizontal plane are less likely to damage surrounding tissue and the nerve proximally and distally. More significant injuries involving devitalization of tissue, or significant torque or force with dis-

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**Fig 1. Diagram of the peripheral nerve anatomy. Reprinted with permission from "Repair and Grafting of the peripheral Nerve," by Terzis JK and Smith KL, McCarthy J (ed), Plastic Surgery, Volume 1, pg 643.**

**Fig 2. Photograph demonstrating cross-sectional view of median nerve.**
sipation of energy result in a markedly different nerve recovery when compared with sharp transections. Adequate visualization of the involved nerve proximally and distally is imperative given the likely extension of destructive forces longitudinally.\textsuperscript{2} The status of surrounding soft tissue must also be assessed. Good vascular supply, stable bone, adequate range of motion of joints, and appropriate skin cover are required criteria to perform a primary nerve repair.\textsuperscript{2} Treatment should be directed toward obtaining this stable environment to allow repair of the nerve as soon as possible.\textsuperscript{2}

Younger patients appear to obtain better functional results.\textsuperscript{6} There is less axonal degeneration and a higher rate of regeneration for younger patients.\textsuperscript{2} The concept of cortical plasticity also favors the young. A greater capacity for the cerebral cortex to process the rearranged information from the disorganized axons of the injured nerve results in improved return of function for young patients.\textsuperscript{2} In the peripheral nerve of a child, the axonal pressure is higher, fascicular tissue occupies more cross-sectional area (80% in a child vs. 60% in an adult), and the shorter extremities equate to less distance for regeneration.\textsuperscript{7} These factors contribute to improved results of nerve repair in children.\textsuperscript{7} Hunt and Bora discuss that the percentage of patients that obtain functional sensory and motor recovery after a sharp nerve transection with early end-to-end coaptation progressively decreases as age increases from childhood, adolescents, and adults, approximately 95%, 75%, and 30 to 70% respectively.\textsuperscript{7}

Adequate anesthesia is imperative for optimal nerve repair.\textsuperscript{1} The injured nerve must be dissected in a meticulous manner. The extremity is exsanguinated and the tourniquet is then inflated to 250 mm Hg. Adequate exposure is obtained by dissecting the surrounding tissue from the nerve under magnification, maintaining the integrity of the mesoneurium and the nerve.\textsuperscript{2} Exploration initially begins outside of the zone of injury and is extended into the injured tissue, first the proximal and then the distal nerve stump.\textsuperscript{2} It is advantageous to remove interfascicular blood clot because it can obstruct alignment.\textsuperscript{7} To avoid rotational deformities, the orientation of the nerve can be marked with 10-0 nylon sutures placed in the 6 and 12 o'clock positions.\textsuperscript{2} 8-0 nylon sutures can be used in larger nerves for orientation.\textsuperscript{3} These orientation sutures are left long to allow rotation of the nerve during repair.\textsuperscript{3} Uninjured epineurium is opened longitudinally, dissected toward the injured tissue, and excised.\textsuperscript{2} The damaged portion of the nerve should be carefully examined for any intact fascicles. Continuity should be maintained. Furthermore, an additional technique for determining orientation of the nerve is its longitudinal blood vessel, which can be used to align the nerve ends.\textsuperscript{2}

The decision to perform primary versus secondary repair is based on several factors. Primary repairs are performed within 8 hours of the initial injury, early secondary repairs within 3 to 6 weeks, and late secondary repairs after 3 months.\textsuperscript{3} Significant wound contamination, devascularized surrounding tissue bed, inadequate soft tissue coverage, significant crush injury to the nerve, and skeletal instability are all factors that favor secondary repair.\textsuperscript{3} Waiting approximately 8 to 16 weeks may allow for better delineation of viable and nonviable nerve tissue.\textsuperscript{2} The advantages of early repair include shorter time interval for reinnervation of muscle and sensory receptor end-organs, subjects the patient to one operation, eliminates the need for nerve grafting and subsequent donor site morbidity, and results in better sensory and motor recovery.\textsuperscript{7} The optimal scenario for early nerve repair is a sharp transection of a nerve with minimal contamination and a stable, well-vascularized milieu.\textsuperscript{7} Secondary nerve repair offers the opportunity to adequately evaluate the nerve stumps and also the thick epineurium for easier suture placement.\textsuperscript{7}

Intraoperative distinction of individual fascicles of the proximal and distal nerve stumps in an acute nerve transection can allow appropriate realignment in the repair.\textsuperscript{2} Differentiation between motor and sensory fascicles in a mixed nerve is essential in achieving maximal return of function. This technique is facilitated by knowledge of nerve topography at the various levels of injury. In stimulating the distal stump, motor contraction will usually be apparent. Stimulation of motor axons in the distal stump will be successful within the first 3 to 4 days after injury.\textsuperscript{4} Therefore, early repair may be better because of this ability to differentiate at least distal motor fibers through electrostimulation within 3 to 4 days. For proximal mixed nerves, which are difficult to reorient, muscle twitching with stimulation indicates a motor fascicle, while the absence of twitching indicates a sensory fascicle.\textsuperscript{4} A level of anesthesia can be achieved such that the patient is alert enough to communicate feeling or pain in the corresponding body part.\textsuperscript{2} Identification of the sensory portion of the nerve can be achieved by asking the patient to respond "yes or no" to direct questioning of sensation in the involved portion of the extremity. Both proximal and distal stumps are stimulated within a range of 0.5 to 2 mA with a nerve stimulator.\textsuperscript{2} Stimulation is begun at the lower setting and progressively increased.\textsuperscript{4} As results are obtained, the information is mapped out. Disposable nerve stimulators facilitate intraoperative electrical stimulation.\textsuperscript{2}

Another method for fascicle identification involves histochemical staining. A transverse section of nerve is stained to identify motor and sensory components.\textsuperscript{4} Since motor axons have a cholinergic origin, acetylcholinesterase activity can identify motor axons.\textsuperscript{2} However, a prolonged period of incubation is required for analysis, thereby making this technique not applicable for one stage nerve reconstruction.\textsuperscript{2} Carbonic anhydrase has been found in sensory fascicles, but difficulty in identification of the stain and inconsistency in staining the distal stump also categorize this technique as still experimental.\textsuperscript{2} Assays of choline acetylase activity have been used in identification of motor axons.\textsuperscript{2} These assays are only effective if used within 3 days of the injury.\textsuperscript{2} Histochemical identification is undergoing continual evolution to improve clinical applicability.\textsuperscript{2}

Two types of primary nerve repair are generally used, epineurial and perineurial (Fig 3). The external epineurial repair is the more common of the two (Fig 4). In a completely transected nerve, dissection and mobilization are performed as previously described. The ends of the nerves usually retract, thereby creating a gap of approximately 10 to 15 mm.\textsuperscript{2} To achieve a tension free repair, the mesoneurium can be divided.\textsuperscript{2} Flexion of the joint to decrease the tension is not advised because of the additional stress applied to the repair with joint movement. The ulnar nerve can be transposed anteriorly at the elbow to allow less tension.\textsuperscript{2} 2 to 3 cm of additional length can be achieved with this transposition in the region of the cubital tunnel.\textsuperscript{3} The nerve stumps are then cut in a single clean stroke with a No. 11 scalpel blade against a firm background, i.e., a sterile moistened tongue blade.\textsuperscript{7} Scissors may cause further damage to the nerve endings.\textsuperscript{4} Once the nerve stumps are trimmed, the fascicles protrude or "mushroom" outwards.\textsuperscript{7} De-
vitalized tissue is removed. A minimal number of interrupted 9-0 nylon sutures are then used to reapproximate the external epineurium. 8-0 nylon sutures may be used for larger nerves. The sutures are not secured too tightly, because they act more for guidance of axonal regeneration rather than creating a watertight seal. A conscious effort must be made to realign the fascicles, using surface landmarks such as the mesoneurium and vessels to assist in reorientation. Tension must be avoided in the repair. Tension leads to gap formation and results in failure of the nerve repair. 4 cm is the maximal amount of gap tolerated for primary end-to-end repair depending on the location. Overlapping of fascicles is also an undesirable result in the repair. Poor axonal alignment and undue tension are evidenced by mushrooming of the fascicles at the repair site.

The proximal and distal stumps of fascicles are reapproximated in the perineural repair, most commonly indicated in

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Fig 3. Diagram demonstrating 3 types of nerve repair. (A) External epineurial repair. (B) Interfascicular perineurial repair. (C) Group fascicular perineurial repair. Reprinted with permission from Jabaley ME.

Fig 4. Epineurial repair of median nerve transection.
The repair of partially transected nerves is dissected off the nerve, leaving the inner epineurium which is adherent to the perineurium. The length of external epineurium that is excised is approximately double the diameter of the corresponding nerve. The spiral bands of Fontana hat are in the perineurium help identify individual fascicles. These bands are not visible when longitudinal traction is applied to the fascicle. Under the microscope, proximal and distal stumps of fascicles are matched using the previously described nerve stimulation and histochemical techniques. 0.0 nylon sutures incorporate the inner epineurium (Fig 5). The goal should be adequate tension-free reapproximation with the least amount of sutures. Reapproximation of groups of fascicles rather than each individual fascicle is more time efficient and decreases the amount of foreign material in the nerve. Group fascicular repair is considered the optimal repair of distal nerve trunks such as the median and ulnar nerves at the elbow because the sensory and motor components are separated. Because of the presence of the anterior interosseous fibers with the main trunk of the median nerve, perineurial repair is an option. The bifurcation of the common digital nerve into the proper digital nerves at the base of the finger is also an area for perineurial repair.

Another consideration for nerve injuries at the level of the wrist is decompression. Release of guyon’s canal for ulnar nerve injuries and the carpal tunnel for median nerve injuries allows regeneration without compression. Proper axonal alignment is also facilitated with decompression.

If the severed nerve endings cannot be reapproximated because of significant gap, options include nerve graft or nerve conduits. A polyglycolic acid or vein graft conduit can be used for severed digital nerves to coapt the nerve gap and neurotrophic factors allow alignment of the axons. Results have shown better 2-point discrimination when compared with nerve grafts for short defects. Polyglycolic acid tubes resorb within 90 to 120 days. The conduit is used for nerve gaps of 3 cm or less and in sensory nerves that were completely divided distal to the wrist crease. Autogenous vein grafts are preferred over conduits for large/major nerves. As previously described, nerve
ends are debrided to normal appearing tissue. The inner diameter of the conduit is approximately 0.5 to 1.0 mm larger than the involved nerve. The bioabsorbable polyglycolic acid tube is then sutured initially to the proximal and then the distal nerve stumps. 8-0 or 10-0 nylon is used as a U-stitch, suturing the conduit to the most distal end of the epineurium of the nerve stump, and then the conduit again, such that approximately 5 mm of the nerve lay within the conduit. The suture is tied with a knot lying on the outer portion of the tube. Before the distal nerve stump is secured to the tube, a solution of 1000 units of heparin per 100 mL of normal saline is infused into the lumen of the tube to prevent clot formation. Even for less than 4 mm digital nerve gaps, in which primary end-to-end repair is acceptable, polyglycolic acid conduit repair has been shown to result in better 2-point discrimination in one study. However, primary nerve repair is recommended if it can be performed tension free.

If nerve grafting is necessary at the secondary repair, the proximal and distal nerve stumps are marked with 8-0 nylon sutures with long tails, 6-0 prolene sutures for larger nerves, and secured in position to prevent further retraction. For nerve grafts to be successful, adequate vascularity and soft tissue coverage are necessary. In the setting of an acute nerve injury, these two criteria usually are not present and therefore nerve grafting is not done acutely. Vascularized nerve grafts can be performed secondarily in areas that are poorly vascularized. Hemostasis is achieved in all wounds after the tourniquet is let down and before skin closure. The extremity is splinted in a position placing the least amount of tension on the nerve repair. Immobilization for 3 to 4 weeks is followed by functional splitting and active range of motion. Passive range of motion is started 6 weeks postoperatively. Tinel's sign indicates the level of axonal regeneration. Progression of Tinel's sign and return of strength and muscle bulk are measured along with nerve conduction and electromyographic studies. One expects the distal advancement of Tinel's sign to be 1 mm per day. If there is no evidence of return of nerve function within 4 to 6 months after surgery, reexploration is warranted and internal neurolysis will be required because of the likely encasement of regenerating fascicles in scar. The risk of reoperation and formation of new scar tissue must be weighed against the risk of inadequate nerve coaptation and causing further nerve injury.

Possible complications associated with nerve repair include hypersensitivity, pain, joint contracture, scar formation at the reapproximation site interfering with axonal regeneration, neurovascular injury, and infection. An awareness of these complications can prevent their occurrence.

Conclusion

Clinical decision making skills combined with new technological advancements continue to advance the field of nerve injuries. As researchers continue to strive for achieving complete neurologic return of function, new findings of nerve anatomy and the interrelation of physiologic hormones to axonal regeneration continue to be discovered.

References