Orthopaedic Surgery Board Review Manual

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Compression Neuropathies of the Upper Extremity

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COMPRESSION NEUROPATHIES OF THE UPPER EXTREMITY

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INTRODUCTION

Orthopaedic surgeons frequently treat patients with compression neuropathies of the upper extremity. Anything that compromises the space available for a specific nerve at a specific location can result in a local compression neuropathy. This includes masses, trauma, medical conditions associated with edema, vasculopathies, and inflammation of surrounding tissues and structures. Additionally, compression neuropathies often have no identifiable cause, in which case they are termed idiopathic. These neuropathies may be associated with many medical conditions, including diabetes, thyroid disease, rheumatoid arthritis, pregnancy, and diseases requiring hemodialysis.1 Presenting symptoms commonly include pain, paresthesias, numbness, and weakness of the arm and hand.1

The role of the orthopaedic surgeon is to elucidate the site of compression, determine the clinical severity, relieve the clinical symptoms, and prevent progression of nerve injury. History, physical examination, and electrophysiologic testing are all essential to determine the site of compression and define the severity of nerve involvement. Nerve compression can occur at any level along the course of the nerve, from the nerve roots (radiculopathy), to the brachial plexus, to the main nerve branches themselves, which is the main topic of this review. In terms of clinical severity, occasional numbness and tingling in the hand can signal the early stages of nerve compression, whereas constant numbness with weakness and muscle atrophy represents more severe nerve involvement. Once the site and severity of compression are determined, a comprehensive treatment plan can be implemented. The goals of treatment are to minimize symptoms using different nonoperative modalities while the compression is mild and potentially reversible. Surgical decompression is performed when the affected nerves have severe or progressive compression.

This manual presents the orthopaedic surgeon’s approach to compression neuropathies of the upper extremity, beginning with an overview of pathophysiology and electrophysiologic testing. The primary focus of the manual is on the specific neuropathies, which are reviewed in terms of their anatomy, diagnosis, and treatment.
Discussion of the associated disease states and an in-depth presentation of the etiologic mechanisms and their relation to occupational risk factors are beyond the scope of this review.

PATHOPHYSIOLOGY

Nerve compression can be caused by various mechanisms. Regardless of the cause—for example, idiopathic or by mass effect from a tumor, kinking around an enlarged muscle (triceps), or compression from inflamed tenosynovium—the effects on the nerve are similar.

Most accepted theories of nerve pathophysiology from compression involve ischemia and altered axonal transport. In animal (rabbit) models, venular flow in the epineurium is slowed with 20 to 30 mm Hg of external compression and is blocked at a pressure of 80 mm Hg. Slow axonal transport becomes altered after 8 hours of applying a pressure of 30 mm Hg and shows accumulated proteins. Fast axonal transport is altered after applying a pressure of 20 mm Hg for 8 hours. In humans, experimental paresthesias have been created in volunteers with 30 mm Hg of compression on the median nerve. Complete motor and sensory nerve conduction blocks have been induced with compression of 60 mm Hg.

The histopathologic changes of chronic compression that occur within the nerve start with damage to the blood–nerve barrier, followed by endoneurial edema and perineural thickening, which alter the microcirculation. With continued compression, demyelination occurs and axons begin to degenerate, as shown in a rat model. Additionally, longitudinal nerve tension can play a role in this process: in a rabbit model, stretching of 8% of the nerve length led to a block in venous outflow, whereas stretching of 15% of the nerve length led to neural ischemia. This mechanism of ischemia is especially important in the human ulnar nerve at the elbow, where the nerve runs posterior to the axis of the joint rotation and undergoes stretching on elbow flexion.

ELECTROPHYSIOLOGIC TESTING

Often used to complement the patient history and physical examination, electrophysiologic testing evaluates the function of skeletal muscle and motor and sensory nerves. To identify the muscles involved and to determine the level and degree of dysfunction, this testing typically involves 2 types of studies: nerve conduction studies (NCS) and electromyography (EMG). For NCS, electrical activity is recorded extracellularly from muscle or nerves with the use of surface electrodes; for EMG, a needle is inserted into the studied muscle. The key elements of these studies are briefly described below.

NERVE CONDUCTION STUDIES

Motor Nerve Conduction

In a motor NCS, motor nerves are supramaximally stimulated at a superficial location with distal response from a muscle.

Latency. Latency denotes the time from the application of the stimulus to the initial deflection from muscular contraction.

Compound muscle action potentials (CMAPs). CMAPs are recorded and represent the muscle response after stimulation of the motor nerve. CMAPs display the evoked motor response as a sum of the action potentials of the individual muscle fibers. CMAP amplitude is directly proportional to the number of muscle fibers depolarized, which provides an estimate of the number of functioning axons and muscle fibers.

Conduction velocity (CV). CV for motor nerves is influenced by temperature, patient age, myelin sheath thickness, and internode distance.
**F waves.** F waves represent the delayed muscle stimulation that occurs after the initial motor nerve stimulation. In addition to the anterograde (orthodromic) transmission of the nerve impulse that leads to muscle contraction, some retrograde (antidromic) transmission of the electrical impulse also occurs. This antidromic impulse conduction can stimulate the neuron in the spinal cord, sending a recurrent orthodromic nerve impulse that results in a second, delayed muscle contraction. F-wave measurements reflect the orthodromic and antidromic conduction along the entire nerve and are useful in the study of general polyneuropathy.

**Sensory Nerve Conduction**

Sensory action potentials are unaffected by lesions proximal to the dorsal root ganglia and are useful to detect pathology proximal (cervical root) or distal (brachial plexus or peripheral nerve) to the dorsal root ganglia. The amplitudes recorded for sensory NCS are much smaller than those in motor NCS because the study is conducted on the nerve itself. The stimulation recording and CV calculation can be performed in both the orthodromic and antidromic directions. In sensory NCS: (1) a surface electrode stimulates the sensory nerve under study, which causes production of a measurable sensory nerve action potential; (2) latency denotes the time from the stimulus application to the initial deflection from muscular contraction; (3) CV is calculated by dividing the length of the nerve segment from the stimulus point to the recording point by the latency; and (4) the amplitude is proportional to the total number of nerve fibers activated.

**Electromyography**

EMG is used to study the electrical activity of individual muscle fibers and motor units, which can be helpful to differentiate primary nerve and muscle dysfunction. It may also differentiate between partial and complete nerve dysfunction. Key findings in EMG are:

- **Insertional activity.** Insertional activity is a burst of muscle activity that occurs because of the mechanical stimulation when the needle is inserted into muscle. Insertional activity is increased in nerve compression syndromes and in nerve injury and is decreased in prolonged denervation caused by loss of muscle fibers.

- **Rest activity.** Although muscle is normally silent during rest, denervated muscle fibers can become spontaneously active and produce fibrillations (action potentials that arise from single muscle fibers) and positive sharp waves. Spontaneous activity during rest may not be seen until 3 to 5 weeks after nerve injury.

**Clinical utility and limitations of electrophysiologic testing**

The NCS latency and CV findings should be reviewed in the context of the clinical picture and EMG results, because the recorded speed of conduction (CV) relates only to the fastest and healthiest myelinated fibers. The results of both NCS and EMG are operator dependent and are not comparable among physicians or machines. Additionally, these tests cannot detect some cases of early compression because the first changes to the nerve affect the unmyelinated axons; large, healthy myelinated axons are still working and transmitting potentials, and therefore the initial changes may not be detected on NCS/EMG. Finally, electrophysiologic testing may miss dynamic issues, such as a dynamic compression caused by muscular contraction or changes in blood flow. In fact, results of NCS in median nerve compression have been shown to be normal in 20% of patients.
with clinically or surgically proven nerve compressions.7

Compression of the Median Nerve

MEDIAN NEUROPATHY AT THE WRIST (CARPAL TUNNEL SYNDROME)

Compression of the median nerve at the wrist is the most common compression neuropathy of the upper extremity.

Anatomy

The median nerve enters the wrist and hand through the carpal tunnel (Figure 1). The flexor retinaculum serves as the roof of the carpal tunnel and consists of 3 parts. Proximally, the flexor retinaculum runs deep to the flexor carpi radialis (FCR) and flexor carpi ulnaris (FCU). In the midportion, the transverse carpal ligament runs from the hamate and pisiform bones to the scaphoid and trapezium bones. Distally, an aponeurosis is found between the thenar and hypothenar muscles.

Proximal to the wrist, the palmar cutaneous branch of the median nerve arises from the radial aspect of the nerve and runs radial to the median nerve proper and ulnar to the FCR. The nerve then pierces the volar carpal ligament, the transverse carpal ligament, or the antebrachial fascia to become superficial and supply sensation to the palmar skin. The recurrent motor branch arises distal to the transverse carpal ligament to innervate the thenar muscles; the nerve then divides into the digital nerves for the thumb, index, long, and radial ring finger.

For both the palmar cutaneous branch and recurrent motor branch, many anatomic variations have been reported of which the surgeon must be aware to avoid iatrogenic injury. For example, the palmar cutaneous branch has been found passing through the palmaris tendon. Lanz8 classified the variations of the motor branch into subgroups; the most common variation is the branch arising extraligamentous, recurrent, and distal to the retinaculum (Figure 2). Incising the transverse carpal ligament from the ulnar side is recommended to avoid motor branch injury.9

Diagnosis

History. Patients typically complain of pain and paresthesias in the hand along with the thumb, index, long, and radial side of the ring finger. The pain often worsens at night, may be exacerbated by certain wrist positions (flexion/extension), and may be elicited after repetitive activities. Often, patients describe the need to “shake” the hand to relieve the symptoms. Dropping objects and an inability to perform fine manipulation are common because of altered sensation. Finally, thenar muscle weakness may signify more advanced disease.1,10

Figure 1. Schematic cross-section of the wrist, illustrating nerve entrapment syndromes and showing the relationship of the carpal tunnel and the ulnar tunnel. A = ulnar artery; C = capitate; H = hamate; M = median nerve; P = pisiform; PCL = palmar carpal ligament; S = scaphoid; t = flexor tendon; T = triquetrum; TCL = transverse carpal ligament; U = ulnar nerve. (Reprinted with permission of Elsevier from Szabo RM, Steinberg DR. Nerve entrapment syndromes in the wrist. J Am Acad Orthop Surg 1994;2:116.)
Physical examination. Physical examination is useful to aid in the diagnosis of carpal tunnel syndrome and to differentiate it from other proximal compression neuropathies. The Phalen’s test, reverse Phalen’s test, carpal compression test, and percussion test (Tinel’s sign) are most commonly used to determine if the compression occurs at the carpal tunnel. Threshold tests (vibrometry and Semmes-Weinstein monofilaments) are useful in the detection of early disease. Innervation density tests (static and moving 2-point discrimination) are only abnormal when the disease process is more severe. Weakness can be tested clinically by asking the patient to pull the thumb away from the palm in an abducted position and assessing thumb strength compared with that of the opposite hand; however, if the patient has bilateral disease, interpretation of strength testing may be difficult. These tests may also be repeated during the examination after a repetitive activity (eg, typing, playing an instrument) to determine if a dynamic condition is present.1

Testing. Although the diagnosis of carpal tunnel syndrome is clinical, several tests assist in diagnosis and quantification of the severity. Electrophysiologic testing has become the test of choice for most physicians and is the gold standard for evaluation of compression to confirm the diagnosis and assist in quantifying the severity. NCS evidence of

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Figure 2. Variations in median nerve anatomy in the carpal tunnel. (A) The most common pattern of the motor branch is extraligamentous and recurrent. (B) Subligamentous branching of a recurrent median nerve. (C) Transligamentous course of the recurrent branch of the median nerve. (D) The motor branch can uncommonly originate from the ulnar border of the median nerve. (E) The motor branch can lie on top of the transverse carpal ligament. (Reprinted with permission of Elsevier from Lanz U. Anatomical variations of the median nerve in the carpal tunnel. J Hand Surg [Am] 1977;2:44–53.)
carpal tunnel syndrome consists of a distal motor latency greater than 4.5 msec or an asymmetry of 1.0 msec between hands, a distal sensory latency greater than 3.5 msec or an asymmetry of 0.5 msec between hands, and a CV less than 50 m/sec. EMG assessment of the abductor pollicis brevis muscle may show fibrillations, sharp waves, or increased insertional activity in carpal tunnel syndrome. Other tests less commonly performed include the hand-volume stress test and direct carpal tunnel pressure measurement.11,12

Treatment

Nonoperative management. Conservative treatment consists of splinting the wrist in the neutral position to limit the flexion and extension compression in the carpal tunnel. Splinting can provide relief for approximately 54% of patients after 3 months of treatment.13 Oral anti-inflammatory medication and corticosteroid injections can help reduce symptoms, and diuretics can be useful for patients with fluid imbalances. A recent Cochrane systematic review found that there was no benefit from more than 1 injection.14 In 1 study, corticosteroid injections provided transient relief in 80% of patients, but only 22% were symptom-free after 12 months.15 Patients who benefit most from corticosteroid injections combined with splinting are those with mild, intermittent symptoms for less than 1 year’s duration.14 The ideal candidate has no weakness or atrophy and has neurophysiologic tests showing no denervation potentials and slightly prolonged distal motor and sensory latencies. Of these patients, 40% will be symptom-free after 1 year.15 Kaplan et al16 reported that 5 factors (patient age > 50 years, symptom duration > 10 months, constant paresthesias, stenosing flexor tenosynovitis, and a positive Phalen’s test result in < 30 sec) all predicted a worse outcome with nonoperative management. All patients should be instructed on wrist positioning, ergonomic work evaluations, and limiting repetitive activities.

Surgery. Surgical release of the compression is indicated after failure of conservative treatment or for patients who present in the more severe stages of disease. The surgical approach can be either an open technique (mini or small open) or an endoscopic technique, using 1 or 2 portals.

The standard open approach provides a wide exposure, which lessens the possibility of an incomplete release, allows identification of anatomic variations, and minimizes the risk of injury to the median nerve and its branches, the superficial arch, and the flexor tendons. With this approach, however, the incision site can be painful postoperatively and can limit activities and a return to work.17 These considerations led to the introduction of the small open incision and endoscopic carpal tunnel release. The endoscopic techniques have been associated with a slightly more rapid return to work (2 weeks) and have shown equivalent results to open release at 1 year.17 Nevertheless, the reported complication rate for the endoscopic approach is higher.17 A meta-analysis by Thoma et al18 found that the incidence of transient nerve injury was 3 times higher with the endoscopic method. Additionally, many prospective randomized studies show no difference in short- and long-term outcomes for endoscopic release compared with an open approach.17,19,20 One study found higher rates of reoperation and patient dissatisfaction with results of endoscopic release.19 Concannon et al21 found a much higher recurrence rate with the endoscopic method.

There are obvious patient expectations associated with each of these procedures, which must be considered when deciding on the surgical approach. The incision site and length, scar formation, pilar pain, risk of complications, and risk of
persistent symptoms must be explained to each patient as these will vary depending on the severity of the disease and the choice of the surgical approach. What is most important is accurately diagnosing the condition and performing a release before the compression progresses to permanent changes in the nerve, thereby minimizing the possibility of residual symptoms.

**PROXIMAL COMPRESSION OF THE MEDIAN NERVE**

**Anatomy**

The median nerve forms from branches of the medial and lateral cords of the brachial plexus. The nerve crosses over the brachial artery and lies on the medial side of the arm near the brachialis muscle, between the brachialis and the medial intermuscular septum. The nerve then descends into the forearm after passing under the bicipital aponeurosis (lacertus fibrosis) (**Figure 3**). It runs between the deep and superficial heads of the pronator teres muscle; proximal to this muscle, the median nerve branches to the palmaris longus, FCR, and flexor digitorum superficialis (FDS) muscles. Distal to the pronator teres, the nerve passes deep to the fibrous arch of the FDS.22,23

Proximal sites of compression include the following (from proximal to distal): (1) Struthers' ligament, which arises from a supracondylar process from the medial humerus, proximal to the medial epicondyle; (2) a snapping brachialis tendon, which may lead to sensory neuropathy; (3) lacertus fibrosis; (4) the interval between the deep and superficial heads of the pronator teres muscle (compression here is termed pronator syndrome); (5) the FDS arch; and (6) anomalous muscles, including the accessory head of the flexor pollicis longus (also termed Gantzer's muscle), palmaris profundus, and FCR brevis.22,23 Pronator syndrome is the most common type of proximal compression of the median nerve.

The following diagnosis and treatment discussion focuses on this syndrome.

**Pronator Syndrome**

**Diagnosis.** Pronator syndrome is more common in weightlifters and in persons whose occupations involve activities that require excessive pronation of the forearm with the elbow in extension. Although Tinel's sign is not present at the wrist, this syndrome can be confused with carpal tunnel syndrome because of the similar clinical presentation. With the forearm supinated and the wrist in a neutral position, pressure over the leading edge of the pronator teres can
reproduce the symptoms. Dysesthesias should be present in the thenar eminence as the palmar cutaneous branch arises proximal to the carpal tunnel.\textsuperscript{23} Additionally, the frequency of nighttime symptoms in pronator syndrome is reduced compared with those in carpal tunnel syndrome.\textsuperscript{24} These 2 nerve compressions can coexist, and thus a careful examination and workup should be performed. Electrophysiologic testing is usually negative. Radiography of the distal humerus may show the supracondylar process, which would indicate the presence of a Struthers’ ligament.\textsuperscript{22,23}

**Treatment.** Surgical treatment is usually not required for pronator syndrome. Activity modification that limits the offending motion or stress is the mainstay of treatment.\textsuperscript{23} Surgical release may be necessary for refractory cases or cases in which a space-occupying lesion is identified. Preoperative radiographs should be obtained to assess for the presence of a supracondylar process. The area requiring decompression extends from 5 cm proximal to the elbow to assess for a Struthers’ ligament, passes distal through the lacertus fibrosis and pronator teres, and ends with the FDS. Outcomes of surgical release are favorable, with 1 study reporting a good or excellent result from decompression in 28 of 36 patients.\textsuperscript{24}

**ANTEROIOR INTEROSSEOUS NERVE SYNDROME**

**Anatomy**

Anterior interosseous nerve (AIN) syndrome is compression of the motor branch of the median nerve in the forearm leading to weakness of the flexor pollicis longus, pronator quadratus, and index/long flexor digitorum profundis.

**Diagnosis**

Pain may be elicited by resisted flexion of the long-finger FDS. No sensory changes are present because the AIN is primarily a motor nerve. Testing of the pronator quadratus should be performed with the elbow flexed to minimize the humeral origin of the pronator teres. Electrophysiologic testing is useful in diagnosing AIN syndrome.\textsuperscript{23}

**Treatment**

Treatment is largely nonoperative. Anti-inflammatory medications, stretching, and strengthening the surrounding musculature can all be useful. In 1 study, all patients were successfully treated without surgery.\textsuperscript{25} Observation should be continue for 3 to 6 months before considering surgical release.\textsuperscript{23} Surgical treatment is similar to the decompression for pronator syndrome as previously described. Tendon transfers may be performed if improvement does not occur after surgical release. One study reported on the outcomes of exploration and decompression for 15 of 20 patients with AIN syndrome.\textsuperscript{26} Of the 15 patients who underwent surgery, 11 had satisfactory function and 3 required tendon transfers; of the 5 cases that were nonoperative, 2 patients recovered and 3 continued to have paralysis.\textsuperscript{26}

**COMPRESSION OF THE ULNAR NERVE**

**ULNAR TUNNEL SYNDROME**

**Anatomy**

The ulnar nerve passes through the ulnar tunnel (Guyon’s canal) at the wrist (Figure 1). This triangular area is bordered posteriorly by the transverse carpal ligament, anteriorly by the volar carpal ligament, and medially by the pisiform. The anatomy of the ulnar tunnel can be subdivided into 3 zones (Figure 4). The nerve enters in zone 1 on the lateral side of the pisiform, proximal to its bifurcation into motor and sensory branches. The nerve begins at the proximal part of the volar carpal ligament and...
runs approximately 3 cm. Zone 2 includes the deep motor branch and is in close proximity to the hook of the hamate. Zone 3 is most distal, involving the superficial branch of the ulnar nerve in close proximity to the ulnar artery. The superficial branch supplies sensation to the small finger and medial ring finger and innervates the palmaris brevis muscle. The deep motor branch innervates the hypothenar muscles, the medial 2 lumbricals, the interossei muscles, and the adductor pollicis.\textsuperscript{1,27–29}

**Diagnosis**

Symptoms vary based on the anatomic site (zone) of compression of the ulnar nerve. Ulnar tunnel syndrome does not involve numbness over the dorsal ulnar hand and wrist because the dorsal ulnar sensory nerve, which branches proximal to the wrist, supplies this area. Zone 1 compression causes combined motor and sensory symptoms because the nerve has not yet split into motor and sensory branches. This compression presents as weakness of the hypothenar and intrinsic muscles as well as sensory deficit of the palmar ulnar hand, small finger, and ulnar ring finger. Often ganglia (most commonly), anomalous muscles, or fractures of the hook of the hamate cause compression in this region. Zone 2 involves the deep motor branch and causes strictly motor deficits of the intrinsic muscles, sparing the hypothenar muscles. Common causes are ganglia (most common), anomalous muscles, and fractures of and fracture nonunion of the hook of the hamate.\textsuperscript{1,27} Zone 3 compression causes sensory abnormalities without any motor deficits affecting the palmar ulnar hand, small finger, and ulnar ring finger. Causes include synovial inflammation, thrombosis of the ulnar artery, and aneurysm of the ulnar artery.\textsuperscript{27–29} Often imaging (MRI for a suspected mass, radiography or CT for suspected hamate fracture) should be obtained prior to surgical decompression because of the high incidence of compression caused by a mass or fractured hamate.\textsuperscript{28,30}

A careful history should be taken concerning recreational and occupational activities that may apply excessive pressure to the ulnar hand and wrist. Smoking and using pneumatic drills can predispose to vascular thrombosis of the ulnar artery. Any prior history of trauma should be carefully evaluated. Evidence of proximal nerve compression should be investigated on history and physical examination. Any clawing of the fingers should be noted. Strength testing should be performed on the intrinsic muscles and hypothenar muscles. Electrophysiologic testing can aid in the diagnosis.\textsuperscript{28} Distal sensory latency measured from the wrist to the small finger is delayed in compression of zones 1 and 3, whereas distal motor latency is abnormal.
in the first dorsal interosseous and abductor digiti quinti muscles in compression of zones 1 and 2. Nerve CV should be greater than 50 m/sec.

**Treatment**

Conservative treatment is indicated for cases in which no extrinsic cause for compression is obvious. A careful physical examination must be performed to identify potential masses, fractures, or vascular malformations; if present or suspected, imaging should be obtained (MRI for a mass, CT for a fracture, vascular study for a malformation/aneurysm). If any of these findings is present, nonoperative management is likely to fail. Conservative options include limiting repetitive activities, splinting in neutral position, and anti-inflammatory medications.31

Surgical decompression is indicated in patients with an identifiable lesion or in cases refractory to nonoperative management. Also, if a mass, hook of the hamate fracture, anomalous muscle, or vascular malformation is the cause of compression, surgical removal is indicated. For patients who do not have an identifiable cause of compression, the entire length of the ulnar tunnel should be released with a zigzag incision and visualized because fascial or tendinous bands may be present at the level of the forearm or distally at the hamate.1

**CUBITAL TUNNEL SYNDROME**

**Anatomy**

The ulnar nerve is the terminal branch of the medial cord (cervical nerve 8 and thoracic nerve 1) of the brachial plexus. The nerve travels posteromedial to the brachial artery, posterior to the medial intermuscular septum, and anterior to the medial head of the triceps muscle. Approximately 8 cm proximal to the medial epicondyle, a deep fascial band arises from the triceps muscle (termed the *arcade of Struthers*), which may cause compression (Figure 5). The nerve runs posterior to the medial epicondyle and medial to the olecranon under the

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**Figure 5.** The 5 sites for potential ulnar nerve compression and the causes of compression at each site. (Adapted with permission of Springer Science+Business Media from Amadio PC. Anatomical basis for a technique for ulnar nerve transposition. Surg Radiol Anat 1986;8:155–61.)
cover of Osborne’s ligament. This pathway defines the cubital tunnel. The nerve then passes between the 2 heads of the FCU into the forearm.

The ulnar nerve is superficial and easily compressed against hard surfaces. Apfelberg and Larson found that the volume of the cubital tunnel can decrease up to 55% with elbow flexion. Gelberman et al. found increased intraneural pressure and decreased tunnel volume with elbow flexion. Additionally, the nerve runs posterior to the axis of rotation of the elbow and thus undergoes a stretch or traction phenomenon during routine elbow flexion. Finally, subluxation of the nerve out of the tunnel can also contribute to compression symptoms.

**Diagnosis**

Symptoms usually consist of numbness and paresthesias in the small finger and ulnar half of the ring finger. Associated pain may be present in the medial elbow or forearm. Weakness may be present initially, and atrophy of the interosseous muscles may be evident on physical examination. Weakness will be present in the flexor digitorum profund of the small and ring fingers and the FCU; however, in some cases the FCU receives its innervation proximal to the level of compression. This differs from a more distal compression, where these muscles are never affected.

Cubital tunnel syndrome is a clinical diagnosis that can be supplemented with electrophysiologic tests, although test results can be negative. NCS should be performed with the elbow flexed, as this may slow the CV, and the study should include the dorsal ulnar sensory branch. A CV less than 50 m/sec is abnormal, and the difference between the CV observed above and below the elbow should be less than 10 m/sec. EMG of the first dorsal interosseous muscles and the FCU can assist in localizing the level and determining the severity of the compression.

The finding of a positive Tinel’s sign at the cubital tunnel or just proximal to the tunnel can aid in diagnosis; however, this sign may be positive in up to 25% of normal elbows. The elbow flexion compression test may also provide useful clinical information, but a positive result may be noted in up to 13% of normal elbows.

When severe neuropathy is present, clawing may occur in addition to a positive Froment’s paper sign and Wartenberg’s sign. Alteration in threshold nerve testing may also be present with mild compression. Careful sensory examination should show decreased sensation over the dorsal ulnar aspect of the hand. This finding helps distinguish cubital tunnel syndrome from ulnar tunnel syndrome at the wrist.

**Treatment**

The main objectives for conservative management include altering activities to limit the causative positions and pressures. Positions that place the elbow in flexion (eg, driving, telephone use, sleeping) or cause compression by leaning onto a hard, flat surface are to be avoided. An elbow pad placed medially may be helpful to cushion the area and remind the patient not to cause pressure over the nerve. The pad can be placed anteriorly at night to prevent elbow flexion and to minimize nighttime symptoms. Rigid splints holding the elbow in extension can also be useful but are cumbersome and less likely to be tolerated by the patient.

Operative treatment consists of nerve decompression along the entire course of the ulnar nerve from the mid-arm to the proximal forearm. Many operative approaches are described; however, all must include care to protect the medial brachial...
Compression Neuropathies of the Upper Extremity

and antebrachial cutaneous nerve branches as well as the first motor branch to the FCU. Options include in-situ decompression, anterior subcutaneous transposition, anterior intramuscular transposition, anterior submuscular transposition, and medial epicondylectomy. A review of 50 reports found that 90% of patients with mild symptoms had relief with any surgical procedure; however, for patients with moderate or severe disease, in situ decompression was rarely successful.39

In-situ decompression. In-situ decompression can be used for cases of mild neuropathy in which alteration of the traction phenomenon may not be necessary to achieve symptom relief. During this procedure, the entire length of the nerve is decompressed but left in its original tissue bed without disturbing the extrinsic or intrinsic circulation. A recent prospective, randomized study found that both in situ decompression and submuscular transposition are effective treatment options; however, in-situ decompression had fewer complications.40 Careful examination must show no nerve subluxation after in-situ decompression. It is important to understand that postoperative residual symptoms necessitate repeat surgery for transposition through a scarred bed, which technically complicates reoperation.36

Subcutaneous transposition. Subcutaneous transposition decompresses the nerve and relieves the tension by placing the nerve anterior to the axis of the elbow joint rotation. Care must be taken not to create any kinking of the nerve; thus, adequate exposure must be achieved. The arcade of Struthers must be released, the intermuscular septum excised, the cubital tunnel released, and the FCU released distal enough to allow anterior placement of the nerve without any new compression points.32,38 Either the subcutaneous tissue can be sewn to the flexor pronator fascia to create a boundary and prevent subluxation, or a more formal fascial flap can be raised and used as a sling to prevent subluxation.41 Good results for subcutaneous transposition were noted in 90% of cases of mild disease, 70% of moderate disease, and 50% of severe disease.39

Intramuscular transposition. Intramuscular transposition is performed less frequently because many surgeons believe it increases scarring around the nerve. This approach allows more protection from compression than subcutaneous transposition, especially in thin patients. Despite the concern about increased scarring, a primate model showed that, compared with submuscular placement, intramuscular placement did not increase scar formation at the muscle–nerve interface.42

Submuscular transposition. Submuscular transposition places the nerve deep to the flexor pronator mass, which protects it from outside compression and relieves traction forces. This approach is commonly performed for moderate to severe disease and as revision surgery for failed subcutaneous transposition. One study showed that, for moderate disease, submuscular transposition has the best results with the fewest complications.39 Disadvantages include postoperative immobilization necessary to allow healing of the flexor pronator mass and potentially increased scarring.38

Medial epicondylectomy. Medial epicondylectomy allows removal of the prominence against which the ulnar nerve may be compressed. The nerve is allowed to follow its own path of least resistance.38 Care must be taken to avoid injury to the medial collateral ligament of the elbow joint. O’Driscoll et al.43 found that only 20% of the epicondyle may be removed before the medial collateral ligament is affected and recommended avoiding the anteroinferior region. Disadvantages are that the most distal compression site is not addressed,
the arcade of Struthers is not decompressed, and the traction phenomenon is not altered. Also a high recurrence rate has been noted.39

**COMPRESSION OF THE RADIAL NERVE**

**RADIAL TUNNEL AND POSTERIOR INTEROSSEOUS NERVE SYNDROMES**

**Anatomy**

The radial nerve arises from the posterior cord of the brachial plexus. It then travels anterior to the subscapularis, teres major, and latissimus dorsi muscles; continues laterally; and exits the axilla through the triangular space and lateral triceps. The nerve pierces the lateral intermuscular septum and enters the anterior compartment of the arm 10 cm proximal to the lateral epicondyle (Figure 6). It then enters the forearm anterior to the lateral epicondyle and splits into the superficial sensory branch and the posterior interosseous nerve (PIN). The PIN passes below the arcade of Frohse (the proximal edge of the supinator muscle) before traveling between the two heads of the supinator muscle. The nerve exits the supinator muscle to innervate the dorsal forearm muscles. The radial tunnel runs approximately 5 cm and is located anterior to the radiocapitellar joint. Its lateral border is the extensor carpi radialis longus and brevis and brachioradialis. The medial border is the biceps tendon and brachialis. The brachioradialis also forms the roof as it runs from lateral to anterior to the nerve. The tunnel extends distally to the arcade of Frohse, but tendinous bands may exit distally through the supinator muscle.23,44

**Radial Tunnel Syndrome**

**Diagnosis.** Radial tunnel syndrome is a poorly described clinical entity, primarily because the objective methods of diagnosis are few. In addition, the clinical picture can often be confounded with lateral epicondylitis and radiocapitellar pathology. The principal complaint in radial tunnel syndrome is aching pain. A point of maximal tenderness is usually noted at the nerve compression site. This point occurs approximately 5 cm distal to the lateral epicondyle and is 2 to 3 cm distal to the radial head. Pain can be reproduced by compression of the mobile wad or by resisted active extension of the long finger and may have associated paresthesias in the distribution of the superficial
radial nerve. Compression is maximal with the elbow extended, the forearm pronated, and the wrist flexed. Another provocative maneuver is elbow extension with forearm supination and the long finger extended, thereby compressing each potential site.

Electrophysiologic testing is routinely negative for this compression; however, in 1 study, 17% of patients had compression of other nerves. Additionally, 60% of patients who had a poor or fair result after radial tunnel release had evidence of a cervical nerve 7 radiculopathy on EMG, whereas only 16% of patients who had a good or excellent result had evidence of a radiculopathy. An injection of local anesthetic causing transient wrist drop and temporary improvement of pain can be used as a diagnostic tool.

Treatment. Initial treatment of radial tunnel syndrome is nonoperative and includes nerve gliding, activity limitations to avoid the causative arm positions, and splinting, which may entail limiting elbow extension and forearm pronation. An ergonomic work evaluation may be beneficial. Nonsteroidal anti-inflammatory drugs or even corticosteroid injection into the radial tunnel but outside of the nerve may be useful in difficult cases. Lateral epicondylitis may also be present in these patients and requires separate evaluation and treatment. Surgical release is indicated after 6 to 12 months of failed conservative treatment. Potential sites of compression that must be released include the fibrous edge of the extensor carpi radialis brevis, fibrous bands at the radiocapitellar joint, the radial recurrent artery, the arcade of Frohse, and fibrous bands in the supinator. Lister et al advocated decompression through a transverse incision over the supinator if the supinator was confirmed as the cause of compression (Figure 7).

PIN Syndrome

Diagnosis. PIN syndrome consists of compression of the branch of the radial nerve with resultant loss of motor function. Clinically, patients present with weakness and radial deviation in wrist extension in addition to loss of finger and thumb extension. Typically, no sensory loss occurs because the PIN carries only few sensory fibers. A major difference between PIN syndrome and radial tunnel is that results of electrophysiologic tests are abnormal in PIN syndrome. Common causes are elbow synovitis (rheumatoid arthritis) and benign tumors (e.g., ganglion cyst, lipoma); PIN can also occur idiopathically. PIN occurs in men twice as often as in women and is twice as common in the dominant arm. It is important to remember that the extensor carpi radialis longus is preserved; therefore, wrist extension remains intact, despite

Figure 7. Anatomy of the posterior interosseous nerve in the forearm as it is crossed by the supinator muscle. The approach shown is through the interval between the brachioradialis and the extensor carpi radialis longus. ECRL = extensor carpi radialis brevis; ECRL = extensor carpi radialis longus. (Reprinted with permission of Elsevier from Mackinnon SE, Novack CB. Compression neuropathies. In: Green DP, Hotchkiss RN, Pederson WC, Wolfe SW, editors. Green's operative hand surgery. 5th ed. New York: Churchill Livingstone; 2005:1039.)
increased radial deviation caused by malfunction of the extensor carpi ulnaris and extensor carpi radialis brevis.\textsuperscript{23,44}

**Treatment.** PIN syndrome can be treated with rest, activity modifications, a cock-up wrist splint, and a course of physical therapy to maintain joint mobility. Gentle stretching of the wrist extensors with the elbow extended is begun after a spontaneous recovery is documented. Electrophysiologic studies can be obtained after 1 month (as a baseline study) and at 4 or 5 months if no clinical evidence of recovery is noted. Observation should last 3 to 6 months before considering surgical release. Other surgical indications include the presence of a mass, chronic radial head dislocation, and rheumatoid arthritis because these findings are unlikely to resolve with conservative management.\textsuperscript{44} Chronic cases (duration > 12 months) tend to be irreversible because of muscle fibrosis and the lack of motor end-plate function and may require tendon transfers. If a mass is suspected, MRI should be performed and the mass removed. Surgical decompression is aimed at a release of the supinator and arcade of Frohse.\textsuperscript{44,49}

**SUPERFICIAL RADIAL NERVE COMPRESSION**

**Anatomy**

The superficial radial nerve branches from the main radial nerve near the lateral humeral epicondyle, travels deep to the brachioradialis, and enters the superficial distal forearm between the brachioradialis and the extensor carpi radialis longus (Figure 8). In some patients the nerve actually pierces the tendon of the brachioradialis. The nerve branches approximately 5 cm proximal to the radial styloid; the palmar branch continues to innervate the skin on the dorsal aspect of the radial thumb, while the dorsal branch continues to innervate the dorsal aspect of the ulnar thumb and radial index finger. A third branch may be present and innervate the dorsal aspect of the ulnar index finger and radial long finger.\textsuperscript{44}

**Diagnosis**

Compression of the superficial radial nerve at the wrist, termed cheiralgia paresthetica or Wartenberg's syndrome, is typically caused by tight wristwatches, handcuffs, and repetitive forearm pronation/supination. Patients typically report par-
esthiasias, pain, or numbness in the distal dorsal radial forearm, wrist, and hand.

Pronation with ulnar wrist flexion can reproduce the symptoms on physical examination, and Tinel’s sign may be present. Concomitant de Quervain’s tenosynovitis must be ruled out. Patients with compression of the lateral antebrachial cutaneous nerve may present with similar symptoms.\(^5\)

Electrophysiologic testing is not typically useful in diagnosing superficial radial nerve compression.\(^4\) However, diagnostic injection of the lateral antebrachial cutaneous nerve more proximally in the forearm followed by injection of the superficial radial nerve can aid in diagnosis. A thorough history and physical examination with appropriate imaging of the cervical spine may be necessary to rule out any cervical disk disease.

Treatment

Conservative treatment includes modifying activities to limit pronation/supination motions, splinting, anti-inflammatory medications, physical therapy, and avoiding ulnar wrist flexion to limit stretching the nerve. One study reported a success rate of 37% with nonoperative treatment.\(^5\) Corticosteroid injection may also be beneficial for symptom relief. Surgical release should be reserved for recalcitrant cases.\(^4\) Surgical release resulted in excellent to good subjective improvement in 86% of patients, fair improvement in 6%, and no improvement in 8%; however, only 43% of patients returned to their previous (preoperative) occupation.\(^5\)

**SUMMARY**

Compression neuropathies are some of the most common clinical problems treated by orthopaedic surgeons. The keys to successful treatment include taking a detailed patient history, performing a meticulous physical examination, and ordering imaging or electrophysiologic tests when indicated to support or rule out a diagnosis. Conservative management is usually the initial treatment implemented, but in certain cases of severe or progressive nerve injury surgical treatment may be the initial recommended treatment. It is of the utmost importance to discuss the diagnosis and the severity of the disease with the patient so that their expectations for success mirror those of the surgeon.

**REFERENCES**