Entrapment and Compressive Neuropathies

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Entrapment and compressive neuropathies of the upper and lower extremities are frequently encountered disorders in the office.1–3 Early in their course, they may be mistaken for more proximal lesions of the plexus or nerve roots, orthopedic disorders, or occasionally central nervous system disorders. Certain clinical clues in the history and examination, however, often will suggest the correct diagnosis, aided by appropriate electrodiagnostic (EDX) and imaging studies when indicated. Some of the more common neuropathies are discussed, along with suggestions regarding testing and treatment.

UPPER EXTREMITY
Carpal Tunnel Syndrome

Case vignette
A 34-year-old right-handed woman described intermittent pain of the right hand and wrist with tingling of the index and middle fingers. Symptoms had been present for 6 months, often awakening her from sleep at night. She also noted aching in her arm extending into the shoulder. Symptoms worsened while holding a telephone or book, or driving. Shaking out her hand or placing it under warm running water offered minimal relief of symptoms. She had difficulty buttoning her shirt and opening jars, and reported dropping things from her hands. Neurologic examination showed decreased light touch sensation over the finger pads of the right index and middle fingers, with slight weakness of thumb abduction. There was a Tinel’s sign at the wrist, and Phalen’s maneuver produced paresthesias in the right middle finger after 30 seconds of wrist flexion.

What is carpal tunnel syndrome, and what are its clinical manifestations?
Carpal tunnel syndrome (CTS), or median neuropathy across the wrist, is the most common entrapment neuropathy in the upper extremity. It is more prevalent in women

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than men.\textsuperscript{4} Although symptoms are usually bilateral, they are almost without exception more prominent in the dominant hand, as in this patient. Paresthesias are frequently present in the distribution of the median nerve, including the medial thumb, index, middle and lateral fourth fingers. Sensory loss may be noted in the same distribution, notably sparing the thenar eminence, which is supplied by the palmar cutaneous sensory branch of the median nerve that comes off proximal to the carpal tunnel (Fig. 1). Although sensory symptoms predominate, in more advanced cases, thenar wasting with weakness of thumb abduction and opposition may be present, both mediated by the distal median nerve. Tinel’s sign (paresthesias radiating into the fingers with tapping over the median nerve at the wrist) is present in over half the cases, although this is a nonspecific sign. In contrast, paresthesias produced with the Phalen’s maneuver (holding the wrist in a flexed position) are more sensitive than the Tinel’s sign, and more specific to CTS, commonly producing paresthesias in the middle or index fingers after 1 or 2 minutes of wrist flexion.\textsuperscript{5}

Neurologic examination may show decreased light touch sensation over the finger pads of the index and middle fingers, with slight weakness of thumb abduction. There may be a Tinel’s sign at the wrist, and Phalen’s maneuver may produce paresthesias in the median innervated fingers after several seconds of wrist flexion.

Symptoms often are provoked during sleep, when persistent wrist flexion or extension leads to increased pressure in the carpal tunnel, resulting in nerve ischemia and paresthesias. Patients often have difficulty buttoning their shirt or opening jars, or describe suddenly dropping things from their hand. Intermittent wrist pain and paresthesias affecting the index and middle fingers, worsened by movements that involve wrist flexion or extension, such as holding a book or telephone, or driving a car, are suggestive of CTS.\textsuperscript{3} Arm discomfort extending into the shoulder is not uncommon, although neck pain should not be seen unless there is a superimposed problem, such as cervical radiculopathy.

\textbf{Fig. 1.} Typical median nerve territory sensory loss pattern in carpal tunnel syndrome. Note that the thenar eminence is spared, supplied by the palmar cutaneous sensory branch of the median nerve. (\textit{From} Stopford JSB. The variation in distribution of the cutaneous nerves of the hand and digits. J Anat 1918;53:14–25.)
What are the causes and pathophysiology of carpal tunnel syndrome?

Most cases of CTS are idiopathic, a result of repeated stress to connective tissue. Patients often report activities that involve repetitive hand use, such as typing on a keyboard. Individuals with a narrow carpal canal are more prone to entrapment. Other medical conditions that predispose to CTS include hypothyroidism, rheumatoid arthritis, diabetes, and various inflammatory, infectious, and systemic disorders such as Lyme disease, sarcoid, and amyloidosis. Routine blood screening for these disorders in patients who have CTS is generally unrevealing, however, unless other symptoms suggestive of systemic disease are present. Pregnancy and hemodialysis also are associated with an increased risk for CTS. Rarely, mass or infiltrating lesions of the carpal tunnel can be seen, such as ganglion cysts, neurofibromas, schwannomas, or arteriovenous malformations.

What is the differential diagnosis of carpal tunnel syndrome?

The differential diagnosis of CTS includes C6-C7 radiculopathy, brachial plexopathy, and proximal median neuropathy. Cervical radiculopathy generally is accompanied by neck pain, and in C6 or C7 radiculopathy, biceps or triceps reflexes are hypoactive or absent. Brachial plexopathy may be accompanied by weakness outside of the median nerve distribution, and reflex changes also may be present. Proximal median neuropathy is rare, accompanied by sensory loss over the thenar eminence, which is not seen in CTS, and weakness of proximal median innervated muscles, such as the long flexors of the thumb, index and middle fingers, and in some cases, arm pronation.

How is Carpal Tunnel Syndrome Diagnosed?

If CTS is suspected clinically, EDX studies, including nerve conduction studies (NCSs) and electromyography (EMG), are helpful in confirming the diagnosis and evaluating for other disorders such as cervical radiculopathy, brachial plexopathy, or proximal median neuropathy that can mimic CTS. In most cases of CTS, EDX studies easily demonstrate demyelination of the median nerve in the form of conduction velocity slowing across the wrist, resulting in prolonged distal median motor and sensory latencies. If there is secondary axonal loss, median sensory and motor amplitudes are reduced or absent. In mild cases of CTS, however, routine median motor and sensory NCSs may be normal, and the diagnosis may be missed in up to 25% of patients. Referral to an EDX laboratory where sensitive internal comparison studies can be done substantially increases the yield in making the diagnosis of CTS. In these studies, the distal latency of the median nerve is compared with that of an adjacent nerve of similar length and caliber, stimulating and recording across the carpal tunnel. Any significant difference in latency implies a demyelinating lesion along the median nerve. Common internal comparison studies include median versus ulnar palmar mixed nerve studies; median versus ulnar distal motor latencies recording the second lumbrical and interossei respectively; median versus ulnar digit four sensory latencies; and median versus radial digit one sensory latencies.

Imaging studies, especially high resolution sonography, have been proposed as sensitive diagnostic tools in clinically suspected CTS, although EDX studies remain the gold standard.

Mass lesions, however, especially should be evaluated in the rare patient who presents with prominent symptoms in the nondominant hand, with a palpable mass in the wrist or palm, or with a slowly progressive syndrome that does not wax and wane. In such cases, imaging with MRI or CT scan of the wrist is indicated to look for a structural lesion.
How is carpal tunnel syndrome treated?
Mild CTS often responds to conservative therapy, including removing causative factors, such as repetitive hand use, and applying a neutral wrist splint to the symptomatic hand, especially during sleep. A 1- to 2-week course of nonsteroidal anti-inflammatory medications may help reduce symptoms if there is no medical contraindication. If these measures do not reduce symptoms, a local corticosteroid injection adjacent to the carpal tunnel may be helpful. Repeated injections beyond two to three injections may damage the flexor tendons, and are not recommended. Steroid injections are most useful only in mild cases, or as a temporizing measure, such as during pregnancy, or while treating a reversible medical condition, such as hypothyroidism. Surgical decompression is indicated in those patients who have severe wasting and weakness of the thumb abductors, persistent numbness or paresthesias, or in patients who have failed conservative measures or have ongoing denervation in the abductor pollicis brevis on EMG studies. Those patients who have more functional upper extremity limitation preoperatively may not do as well postoperatively. Of course surgery also is indicated if a mass lesion is present, or in cases of acute CTS following local trauma. Surgery entails sectioning of the transverse carpal ligament to decompress the median nerve, either through a longitudinal incision from the wrist to the palm, or through endoscopic release. Endoscopic release affords a shorter recovery time, yet the transverse carpal ligament is not visualized directly using this technique, which might result in an incomplete section or damage to the median nerve or other nearby structures.

Ulnar Neuropathy at the Elbow

Case vignette
A 65-year-old man was referred for slowly progressive weakness of hand grip, accompanied by numbness and tingling of the right fourth and fifth fingers. He described pain in his arm and elbow. There was no neck or shoulder pain. Examination showed decreased light touch and pinprick sensation in the right fifth finger and medial hand, with moderate weakness of the intrinsic hand muscles. Deep tendon reflexes were normal. There was tenderness of the right ulnar nerve in the groove.

What is ulnar neuropathy at the elbow and what are its clinical manifestations?
Ulnar neuropathy at the elbow is the second most common entrapment of the upper extremity. The most common cause of ulnar neuropathy at the elbow is chronic compression or stretch of the ulnar nerve in the elbow, either at the ulnar groove or more distally in the cubital tunnel. Both present in a similar manner, with the slow progression of intrinsic hand muscle weakness, loss of grip and pinch strength, and wasting of the thenar and hypothenar eminences. In contrast to CTS, in which sensory symptoms predominate, patients may experience no sensory symptoms, despite sensory loss on examination. Elbow pain is common, in some patients radiating down the medial forearm and wrist. The ulnar nerve may be palpably enlarged and tender in the groove. Paresthesias may be provoked by flexing the elbow or applying pressure to the ulnar groove. Weakness of hand grip is common, because of weakness of the ulnar innervated intrinsic hand muscles. Thumb abduction, innervated by the median and radial nerves, is spared. Asking the patient to make a fist may reveal his or her inability to bury digits four and five in the palm, because of weakness of the ulnar innervated flexor digitorum profundus to digits four and five (Fig. 2). Sensory disturbance, when present, involves the dorsal and volar fifth finger, medial fourth finger, and dorsal and volar aspects of the medial hand. Sensory loss may extend just beyond the wrist crease. If there is altered sensation beyond the wrist crease into
the medial forearm, however, a more proximal lesion such as in the brachial plexus or C8-T1 nerve roots should be investigated.

Several classic hand postures may occur with ulnar neuropathy at the elbow. The most common, the Benediction posture, results in clawing of the fourth and fifth fingers, (hyperextension of the metacarpophalangeal joints and flexion of the interphalangeal joints of the second and third fingers (Fig. 3, top). The fingers and thumb are held slightly abducted because of weakness of the interossei and adductor pollicis. The Wartenberg’s sign results in abduction of the little finger with the hand at rest (Fig. 3, middle), making it difficult for patients to put their hand in their pocket without the little finger getting caught outside. The Froment’s sign occurs when the patient is asked to use the thumb and index finger to pinch an object (Fig. 3, bottom). Patients flex the thumb and index fingers (median innervated), akin to an “OK” sign, because of weakness of the ulnar intrinsic hand muscles that does not allow them to extend the proximal and distal interphalangeal joints of the thumb and index fingers.

What are the causes and pathophysiology of ulnar neuropathy at the elbow?

Ulnar neuropathy at the elbow usually occurs as a result of chronic mechanical compression or stretch, either at the groove or at the cubital tunnel. The underlying pathophysiology may be demyelination or axonal loss. At the groove, most cases are caused by external compression and repeated trauma, although rare cases of ulnar neuropathy at the groove are caused by ganglia, tumors, fibrous bands, or accessory muscles. So-called tardy ulnar palsy may be seen as a result of elbow fracture, often years before, with subsequent arthritic changes of the elbow joint. In addition, chronic minor trauma and compression, such as chronic leaning on the elbow, can exacerbate or cause ulnar neuropathy at the groove. Ulnar neuropathy at
Fig. 3. The Benediction posture results in clawing of the fourth and fifth fingers (A), while the fingers and thumb are held slightly abducted because of weakness of the interossei and adductor pollicis. The Wartenberg'sign (B) results in abduction of the little finger with the hand at rest, because of preferential weakness of the third palmar interosseous muscle, making it difficult to adduct the little finger. The Froment's sign (C) occurs when the patient is asked to use the thumb and index finger to pinch an object. Because of weakness of the ulnar intrinsic hand muscles, patients cannot extend the proximal and distal interphalangeal joints of the thumb and index fingers, and flex the thumb and index fingers (median innervated) to compensate, akin to an *OK* sign. (From Preston DC, Shapiro BE. Electromyography and neuromuscular disorders. 2nd edition. Philadelphia: Elsevier Butterworth Heinemann; 2005; with permission.)
the groove also can be seen in patients who have been immobilized because of surgery or who sustain compression during anesthesia or coma. Some contend that repeated subluxation of the ulnar nerve out of the groove, such as during elbow flexion, also leads to ulnar neuropathy.

The cubital tunnel, which is just distal to the groove, is the other major site of compression of the ulnar nerve in the region of the elbow. Although some use the term cubital tunnel syndrome to refer to all lesions of the ulnar nerve around the elbow, it more properly denotes compression of the ulnar nerve under the humeral–ulnar aponeurosis. Some individuals have congenitally tight cubital tunnels that predispose them to compression. Repeated and persistent flexion stretches the ulnar nerve and increases the pressure in the cubital tunnel, leading to subsequent ulnar neuropathy.

**What is the differential diagnosis of ulnar neuropathy at the elbow?**

The differential diagnosis of ulnar neuropathy at the elbow includes C8-T1 radiculopathy, lower trunk or medial cord brachial plexopathy, or rare cases of ulnar nerve entrapment in the forearm or wrist. Patients who have cervical radiculopathy generally report neck pain radiating into the arm, with sensory disturbance. In both cervical radiculopathy and lower trunk/medial cord brachial plexopathies, which are far less common, weakness is seen in ulnar and nonulnar innervated muscles, such as the thumb abductors (median innervated) and finger extensors (radial innervated), with sensory changes that extend into the medial forearm.

**How is ulnar neuropathy at the elbow diagnosed?**

In suspected ulnar neuropathy at the elbow, EDX studies can help confirm the diagnosis, and exclude other diagnoses such as cervical radiculopathy or brachial plexopathy. Motor NCSs may demonstrate focal demyelination in the form of conduction block or conduction velocity slowing across the elbow, yielding a definitive diagnosis of ulnar neuropathy at the elbow. If these are not seen, short segmental stimulation studies on the ulnar nerve across the elbow may help localize the lesion either at the groove or the cubital tunnel, which may have implications for the best surgical approach. Sensory and mixed ulnar NCSs across the elbow also may increase the yield of identifying focal slowing in some patients. In many patients who have ulnar neuropathy at the elbow, focal slowing or conduction block cannot be demonstrated, because the underlying pathophysiology is axonal loss. In such cases, findings on needle EMG can localize only the lesion to at or above the take off to the most proximal muscle affected on EMG, although in practical terms the lesion is nearly always at the elbow, either in the groove or the cubital tunnel. EDX studies also are used to look for evidence of a C8-T1 radiculopathy or lower trunk/medial cord brachial plexopathy.

**How is ulnar neuropathy at the elbow treated?**

Conservative measures are tried first. Activities that require repetitive or sustained elbow flexion should be discontinued, and the patient should be advised to avoid leaning on the elbow. A simple elbow pad may be helpful, or an elbow splint can be applied to prevent sustained elbow flexion. If there is no improvement with conservative measures, especially in a patient who has progressive weakness and wasting of the hand, surgery is indicated. Surgical options include decompression of the cubital tunnel, submuscular transposition of the ulnar nerve, and medial epicondylectomy. Although submuscular transposition has the best success rate, it also has the greatest morbidity, including the risk of nerve devascularization. If the lesion is localized to the ulnar groove, transposition generally is indicated. If the lesion can be localized precisely to the cubital tunnel, however, some believe that simple decompression may be optimal. Some studies have shown more favorable outcomes in patients...
who have demyelination, either conduction block or conduction velocity slowing across the elbow. Prognosis is poorest in older individuals, those who have exacerbating medical conditions such as diabetes, or in those in whom symptoms have been present for over a year.

### Ulnar Neuropathy at the Wrist

#### Case vignette
A 24-year-old right-handed man complained of progressive wasting and loss of grip strength of the right hand. He was an avid bicyclist, often bicycling over 20 miles a day. Examination revealed atrophy of the intrinsic hand muscles, most marked in the first dorsal interosseous, with weakness of the abductor digiti minimi and interossei muscles on the right. The thumb abductors and long flexors of digits four and five were strong. There was no tenderness of the ulnar nerve in the groove. Deep tendon reflexes and sensation in the upper extremities were normal.

**What is ulnar neuropathy at the wrist and what are its clinical manifestations?**

Four clinical presentations of ulnar neuropathy at the wrist are described, depending on the location of the lesion. The first two subtypes are purely motor. In the first subtype, muscles innervated by the deep palmar motor branch are involved, including the interossei, adductor pollicis, and third and fourth lumbricals. In the second subtype, the lesion involves the deep palmar motor branch and the hypothenar muscles. These purely motor subtypes are the most common, accounting for over 75% of all cases of ulnar neuropathy at the wrist. Because of the lack of sensory findings, these patients often are confused with early amyotrophic lateral sclerosis. In the third subtype, there is weakness of hypothenar and deep palmar motor innervated muscles and sensory loss in the medial fourth and volar fifth digits. The fourth subtype presents with isolated sensory loss over the medial fourth and volar fifth digits. Thus, if sensory loss is present, it spares the dorsal medial hand, innervated by the dorsal ulnar cutaneous sensory branch, which arises several centimeters proximal to the wrist.

**What are the causes and pathophysiology of ulnar neuropathy at the wrist?**

Activities or occupations that involve repetitive movement or force against the ulnar aspect of the wrist predispose to lesions here. This is especially true for bikers, or manual laborers who use tools that cause repetitive pressure against the hypothenar eminence. In these patients, the hypothenar area may become calloused at the compression site. Other risk factors include wrist fracture, trauma, distal musculotendinous fibrous arch, thrombosed ulnar artery, and mass lesions, often a ganglion cyst, within Guyon’s canal.

**What is the differential diagnosis of ulnar neuropathy at the wrist?**

Progressive weakness and wasting of the ulnar intrinsic hand muscles, sparing the median and proximal ulnar innervated muscles, are consistent with ulnar neuropathy at the wrist.

This is a rare condition, yet important to recognize, because it often is confused with early amyotrophic lateral sclerosis, especially in an older patient. In most cases, there are no sensory findings, although there may be sensory loss in digits four and five. Other disorders to consider in the differential diagnosis of ulnar neuropathy at the wrist include C8-T1 radiculopathy, lower trunk or medial cord brachial plexopathy, or rare cases of ulnar nerve entrapment in the forearm. Patients who have cervical radiculopathy generally report neck pain radiating into the arm, with sensory disturbance. In both cervical radiculopathy and lower trunk/medial cord brachial plexopathies, which are far less common, weakness is seen in ulnar and nonulnar innervated muscles,
such as the thumb abductors (median innervated) and finger extensors (radial innervated), with sensory changes that extend into the medial forearm.

**How is ulnar neuropathy at the wrist diagnosed?**

If there is a strong suspicion of ulnar neuropathy at the wrist, MRI or CT scan of the wrist and hand are done to look for a structural lesion such as ganglion cyst or tumor. Referral for EDX testing is often instrumental in clarifying the diagnosis, while excluding other disorders. Testing should include ulnar motor NCSs recording both the abductor digiti minimi and the first dorsal interosseous, because the pattern of abnormalities that emerges can help localize the lesion and determine which branches of the ulnar nerve are affected. The other helpful NCS to perform is the lumbrical–interossei distal latency comparison. Because the ulnar interossei are innervated by the deep palmar motor branch, this test may identify differential ulnar slowing at the wrist in lesions that involve the deep palmar motor branch. Likewise, short segmental inching studies of the ulnar nerve across the wrist, recording the first dorsal interosseous, can be extremely helpful in detecting abrupt changes in latency and amplitude, which help localize the lesion at the wrist. Routine ulnar sensory NCSs recording the fifth digit and the dorsal ulnar cutaneous sensory NCS are done to help determine the level of the lesion. The needle EMG examination of suspected ulnar neuropathy at the wrist entails sampling deep palmar motor and hypothenar innervated muscles, proximal ulnar innervated muscles, and median and radial C8-T1 innervated muscles to confirm that the abnormalities are limited to ulnar muscles distal to the wrist and to exclude a cervical root or motor neuron lesion. In pure ulnar motor lesions, however, it remains difficult to exclude early amyotrophic lateral sclerosis. Clinical correlation, questioning for possible risk factors for ulnar neuropathy at the wrist, and serial follow-up remain important.

**How is ulnar neuropathy at the wrist treated?**

Conservative treatment is usually successful in those patients who have an obvious risk factor, such as bicyclists and those who have work-related risk factors such as manual laborers. In mild cases, symptoms often improve simply by stopping the offending activity. Surgical exploration is indicated for any mass lesion in the canal, and in cases where symptoms are progressive or severe, or do not resolve when the offending activity is discontinued.

**Radial Neuropathy at the Spiral Groove**

**Case vignette**

A 56-year-old man was referred for a right wrist drop. He awoke from a heavy sleep 4 weeks ago with complete inability to extend his right wrist or fingers, and numbness over the back of his hand between the thumb and index finger. There was no arm, neck, or shoulder pain. Neurologic examination showed complete paralysis of the right wrist and finger extensors. Finger abduction was normal strength when tested with the hand passively extended to the neutral position. Wrist and finger flexion and elbow flexion and extension were normal strength. Light touch sensation was reduced over the first dorsal web space between the thumb and index fingers, and extending into the proximal phalanges of the index, middle, and ring fingers. The right brachioradialis reflex was absent.

**What is radial neuropathy at the spiral groove, and what are its clinical manifestations?**

The presentation of a complete wrist and finger drop, with numbness in the lateral dorsal aspect of the hand, is most consistent with proximal radial neuropathy. The most
common site of compression is at the spiral groove. Patients who have radial neuropathy at the spiral groove, also known as Saturday night palsy, present with the acute onset of a marked wrist and finger drop, accompanied by mild weakness of supination and elbow flexion from involvement of the supinator and brachioradialis muscles, respectively. As in this case, the weakness is accompanied by numbness in the lateral dorsal aspect of the hand. Elbow extension remains strong, because the branch to the triceps muscle comes off proximal to the spiral groove. Median and ulnar innervated muscles are also normal. Finger abduction should be tested with the hand held passively extended, by placing the hand on a flat surface, or it may appear to be weak. There is altered sensation over the lateral dorsal hand and dorsal aspects of digits one through four, in the distribution of the superficial radial sensory nerve (Fig. 4). The triceps reflex remains intact, and the brachioradialis reflex is depressed or absent.

**What are the causes and pathophysiology of radial neuropathy at the spiral groove?**

At the spiral groove, the radial nerve lies juxtaposed to the humerus, making it prone to compression, especially following prolonged immobilization. This usually occurs when a person falls into a deep sleep with their arm draped over a chair or bench, such as while intoxicated. The prolonged immobilization leads to compression and demyelination of the radial nerve at the spiral groove. Less commonly, radial neuropathy at the

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**Fig. 4.** Typical superficial radial nerve territory sensory loss pattern in radial neuropathy at the spiral groove. The superficial radial sensory nerve supplies sensation over the lateral dorsum of the hand and part of the thumb and dorsal proximal phalanges of the index, middle, and ring fingers. *(From Preston DC, Shapiro BE. Electromyography and neuromuscular disorders. 2nd edition. Philadelphia: Elsevier Butterworth Heinemann; 2005; with permission.)*
spiral groove occurs secondary to a humeral fracture, nerve infarction such as from vasculitis, or strenuous muscular effort.

**What is the differential diagnosis of radial neuropathy at the spiral groove?**
Radial neuropathies with resultant wrist drop can be caused by compression at other sites, including the axilla, or by entrapment of the posterior interosseous nerve branch of the radial nerve. Radial neuropathy from prolonged compression may occur in the axilla, for example, in patients who use crutches incorrectly, applying prolonged pressure to the axilla. In this case, there is weakness of arm extension from involvement of the triceps; the triceps reflex is depressed or absent, and sensory disturbance extends from the lateral dorsal hand up into the posterior forearm and arm. None of these are seen in radial neuropathy at the spiral groove.

Other diagnoses that should be considered in a patient who has wrist and finger drop include posterior interosseous neuropathy (PIN), a lesion of the posterior cord of the brachial plexus, an unusual C7 radiculopathy, or a central lesion. These, however, usually can be excluded based on key clinical clues. For example, patients with PIN have a characteristic response to attempted wrist extension. The wrist deviates radially in extension because of the relative preservation of the extensor carpi radialis, which comes off proximal to the PIN, compared with the weak extensor carpi ulnaris, which comes off distal to the lesion. Furthermore, there is no reflex loss or cutaneous sensory loss in PIN, although there may be pain in the forearm from dysfunction of the deep sensory fibers supplying the interosseous membrane and joint capsules. A lesion in the posterior cord of the brachial plexus results in weakness of the deltoid and latissimus dorsi, in addition to radial innervated muscles. In C7 radiculopathy, radial and nonradial innervated C7 muscles are weak, including arm pronation and wrist flexion. Although a central lesion such as stroke can cause arm weakness with wrist and finger drop, there should be accompanying upper motor neuron signs such as spasticity and brisk reflexes.

**How is radial neuropathy at the spiral groove diagnosed?**
NCSs and EMG are useful for evaluating a patient who has a wrist and finger drop to identify a radial neuropathy, localize the lesion, assess its severity, and establish a prognosis by defining the underlying pathophysiology. EDX evaluation includes a radial motor study looking for evidence of a focal conduction block at the spiral groove or axonal loss. The superficial radial sensory nerve also is examined to look for evidence of axonal loss. If the clinical examination suggests weakness beyond the radial distribution, investigation for a more widespread neuropathy or brachial plexopathy is indicated. If a focal conduction block at the spiral groove cannot be demonstrated, the needle EMG examination may be helpful in distinguishing between PIN, radial neuropathy at the spiral groove, radial neuropathy in the axilla, a lesion of the posterior cord of the brachial plexus, or a C7 radiculopathy.

**How is radial neuropathy at the spiral groove treated?**
Radial neuropathies from external compression can be managed conservatively in nearly all cases. Most cases at the spiral groove occur from prolonged compression from a one-time episode. In patients who have a significant finger or wrist drop, a cock-up wrist splint is used to keep the wrist and fingers extended. Any offending factors are eliminated, such as improper use of crutches. Length of recovery depends on whether the compression results in demyelination or axonal loss. Patients who have demyelinative lesions usually recover well after several weeks. If the lesion involves axonal loss, recovery may take several months to over a year. Decisions regarding surgical treatment are more controversial. In patients who have progressive
symptoms, or those who have not responded to conservative treatment such as splinting and anti-inflammatory agents, surgical exploration may be advisable to exclude a structural lesion (eg, nerve sheath tumor, lipoma) or release a fibrous band.42,43

Suprascapular Neuropathy

Case vignette
A 32-year-old woman noted progressive atrophy of the left posterior shoulder. She described deep pain in her left posterior shoulder with progressive wasting over the scapula for the past year, giving the appearance of a hole in the scapular area. She frequently lifted weights at the gym, and was aware that lifting and external rotation of her left shoulder had become more difficult. There was no numbness, previous episodes of pain or weakness, or family history of similar problems. Examination showed prominent atrophy of the left posterior inferior scapular area, scapular winging, mild weakness of shoulder abduction, and moderate weakness of external rotation of the shoulder. Deep tendon reflexes and sensation were intact.

What is suprascapular neuropathy, and what are its clinical manifestations?
The classic presentation of suprascapular neuropathy is that of insidious onset of muscle wasting over the scapula with reduced ability to externally rotate or abduct the shoulder, and pain in the posterior shoulder. There often appears to be an indentation or hole in the suprascapular area (Fig. 5). Weight lifters are particularly prone to this entrapment, because of the repetitive movements about the shoulder. The most common site of suprascapular entrapment is at the suprascapular notch, beneath the transverse scapular ligament (Fig. 6). Because the suprascapular nerve is relatively immobile at the suprascapular notch and at its origin at the upper trunk, while the shoulder and scapula are quite mobile, repetitive movements about the shoulder

![Fig. 5. Suprascapular neuropathy. Note the prominent atrophy of the inferior scapular area on the right. Suprascapular neuropathy results in weakness of shoulder abduction and external rotation, without any cutaneous sensory loss. (From Preston DC, Shapiro BE. Electromyography and neuromuscular disorders. 2nd edition. Philadelphia: Elsevier Butterworth Heinemann; 2005; with permission.)](image-url)
result in stretch and nerve injury. Less commonly, the nerve becomes entrapped more distally at the spinoglenoid notch (see Fig. 6).

Symptoms and signs depend on the site of nerve entrapment. Entrapment at the suprascapular notch results in prominent shoulder pain because of involvement of the deep sensory fibers to the glenoacromial and acromioclavicular joints. Patients describe deep, boring pain along the superior scapula radiating to the shoulder, exacerbated by shoulder movements, especially adduction of the extended arm. There may be tenderness at the suprascapular notch. Atrophy is recognized most easily over the infraspinatus, which is not covered by the trapezius. There is weakness of shoulder abduction and external rotation. Because other muscles also can perform abduction and external rotation of the shoulder, however, the patient may not notice these limitations. If the entrapment occurs more distally at the spinoglenoid notch, the syndrome is isolated to atrophy and weakness of the infraspinatus muscle. Unlike the more proximal lesions, pain is absent, because the deep sensory fibers to the shoulder joint exit proximal to the motor branch to the infraspinatus.

**What are the causes and pathophysiology of suprascapular neuropathy?**

People working in certain professions, including professional volleyball players, baseball pitchers, and dancers, and activities that require repetitive movements about the shoulder, such as weight lifting, are associated with a high incidence of suprascapular nerve entrapment, likely as a consequence of repetitive movement of the scapular...
that involves shoulder abduction and protraction. In these professions, the clinical and EDX findings most often suggest a distal lesion at the spinoglenoid notch. Occasionally, the suprascapular nerve becomes entrapped because of mass lesions, including ganglion cysts (especially at the spinoglenoid notch), sarcomas, and metastatic carcinomas. Certain positions also are associated with suprascapular entrapment, such as positioning during surgical procedures, whereby patients are placed in a knee chest position with the scapula protracted. Other etiologic causes include direct trauma to the shoulder. Brachial neuritis may result in suprascapular neuropathy, although in this case weakness usually is not isolated to the suprascapular nerve, and the reflexes may be depressed.

**What is the differential diagnosis of suprascapular neuropathy?**

Shoulder weakness also can be caused by C5-C6 radiculopathy, brachial plexopathy, or a lesion of one of the nerves that comes off the upper trunk, or lateral or posterior cord of the brachial plexus. Cervical radiculopathy, however, usually is accompanied by neck pain, and in C5-C6 radiculopathy, one should see a depressed biceps and brachioradialis reflex, along with weakness in other C5-C6 innervated muscles, and sensory complaints. An upper brachial plexopathy, or neuropathy involving one of the nerves that comes off the upper trunk, or lateral or posterior cord, likewise should result in weakness beyond the distribution of the suprascapular nerve, and in some cases reflex and sensory changes. Other conditions that should be considered include rotator cuff injuries and other orthopedic conditions. Local orthopedic conditions may be difficult to differentiate clinically from suprascapular neuropathy. Although weakness should not be present, pain often prevents full muscle activation. Exacerbation of pain by palpation (other than at the suprascapular notch) or by passive shoulder movement (other than abduction and protraction of the shoulder) is unusual for suprascapular entrapment, and suggests an orthopedic problem.

**How is suprascapular neuropathy diagnosed?**

EDX testing is extremely useful to determine if abnormalities are restricted to the suprascapular innervated muscles, and evaluate for C5-C6 radiculopathy and brachial plexopathy. Motor NCSs of the suprascapular nerve may show reduced amplitudes, as these lesions typically involve axonal loss rather than demyelination. The needle EMG, however, easily demonstrates axonal loss. Both the supraspinatus and infraspinatus muscles should be sampled. In lesions at the suprascapular notch, both are abnormal. With spinoglenoid lesions, however, only the infraspinatus is involved. Other C5-C6 innervated muscles and the cervical paraspinal muscles are also sampled, to exclude a cervical radiculopathy or more widespread brachial plexus lesion. Because the suprascapular nerve has no cutaneous distribution, there is no corresponding sensory nerve to be recorded. Studies of the sensory nerves that pass through the upper trunk of the brachial plexus are performed to help exclude a more widespread plexus lesion. Imaging studies including CT or MRI scan of the shoulder should be done, especially in those patients who have no risk factors for developing a suprascapular neuropathy, to evaluate for a mass lesion.

**How is suprascapular neuropathy treated?**

Treatment often begins with a trial of conservative therapy, such as rest, physical therapy, and eliminating offending activities such as weightlifting. In one prospective series, lesions caused by traction or repetitive overuse responded equally well to nonoperative and surgical management. Those who do not respond to conservative measures likely will require surgical exploration, especially those who have a ganglion cyst or other known compressive lesion. Surgery entails exploration of the
suprascapular notch or spinoglenoid notch, depending on results obtained with EDX and imaging studies,\textsuperscript{51} with the goal being to decompress and release the nerve from the notch. One series found equally good results with open and arthroscopic treatment of ganglion cysts at the spinoglenoid notch.\textsuperscript{51} Surgery is not indicated in those patients with suprascapular neuropathy secondary to brachial neuritis, who improve without surgery.

**LOWER EXTREMITY**

**Peroneal Neuropathy at the Fibular Neck**

**Case vignette**

A 20-year-old man developed a right foot drop after leaving a movie theater. He was unable to dorsiflex the right foot and toes, and described tingling over the top of the foot. He tended to slap the right foot when walking. Examination showed a thin young man who had severe weakness of the right ankle and toe dorsiflexors and ankle evertors. Ankle inversion appeared slightly weak. The remainder of his strength was normal, including ankle and toe plantar flexors, knee flexors and extensors, and proximal muscles around the hip. Deep tendon reflexes were intact throughout, including the ankle reflexes. There was decreased pinprick and cold over the dorsum of the right foot extending up to the lateral calf to just below the knee. There was no pain or Tinel’s sign palpatung the peroneal nerve across the fibular neck.

**What is peroneal neuropathy at the fibular neck, and what are its clinical manifestations?**

The classic presentation of a peroneal neuropathy at the fibular neck usually involves both the deep and superficial peroneal nerves (common peroneal neuropathy). This combination of deep and superficial peroneal neuropathy results in weakness of toe and ankle dorsiflexion, ankle eversion, and sensory disturbance over the dorsum of the foot and the lateral calf below the knee.\textsuperscript{52–55} There may be pain and a Tinel’s sign over the lateral fibular neck. Ankle inversion is spared, which is innervated by the tibial nerve. Ankle inversion, however, must be tested with the ankle in a dorsiflexed position, to avoid the mistaken impression that the tibialis posterior is weak.

**What are the causes and pathophysiology of peroneal neuropathy at the fibular neck?**

Peroneal neuropathy has several etiologies.\textsuperscript{4} Habitual leg crossing may cause repetitive injury to the peroneal nerve because of its superficial location at the fibular neck.\textsuperscript{56} Similarly, repetitive stretch from squatting, such as in gardeners, has been associated with peroneal neuropathy. Patients who are thin or have recently lost a great deal of weight are prone to peroneal palsy, probably from the lack of protective supporting adipose tissue at the fibular neck.\textsuperscript{57–59} Slowly progressive lesions often suggest a mass lesion, such as a ganglion cyst or nerve sheath tumor.\textsuperscript{60} Entrapment of the peroneal nerve at the fibular tunnel, although uncommon, also may present in a progressive manner. Acute peroneal neuropathy may follow trauma, stretch injury such as when the ankle is forcibly inverted, or compression from prolonged immobilization. In the hospital, this occurs most often postoperatively in patients who have received anesthesia or heavy sedation.

**What is the differential diagnosis of peroneal neuropathy at the fibular neck?**

Other conditions that should be considered in a patient who presents with a foot drop and numbness over the dorsum of the foot include high sciatic neuropathy,\textsuperscript{61} lower lumbosacral plexopathy, and L5 radiculopathy. These conditions, however, result in weakness of ankle inversion and sensory loss that extends to the lateral knee, sole
of the foot, or lateral foot, none of which is seen in peroneal neuropathy at the fibular neck. Other signs of a more proximal lesion include a depressed or absent ankle reflex or weakness of hip abduction, extension, or internal rotation.

**How is peroneal neuropathy at the fibular neck diagnosed?**

EDX studies are especially helpful for evaluating peroneal neuropathy at the fibular neck \(^{52,53,62}\) and differentiating this from a more proximal lesion. In demyelinating lesions, the peroneal motor NCS can be used to localize the lesion if focal slowing or conduction block is demonstrated across the fibular neck. The yield of finding a demyelinating lesion is increased when the peroneal motor NCS is performed recording the extensor digitorum brevis and the tibialis anterior. \(^{63}\) In purely demyelinating lesions at the fibular neck, the distal superficial peroneal sensory response remains normal. The presence of a predominantly demyelinating lesion has important prognostic implications. As the underlying axons remain intact, the prognosis for full recovery over a relatively short period is excellent, provided that the cause of the entrapment is no longer present.

If the lesion is primarily axonal loss, peroneal motor and superficial peroneal sensory amplitudes are reduced or absent in common peroneal neuropathy, and one cannot localize the lesion. The degree of axonal loss can be estimated by comparing the distal motor amplitude on the involved side with that on the asymptomatic side, to determine prognosis. Note that in an isolated deep peroneal neuropathy, the superficial peroneal sensory potential remains normal, making it difficult to differentiate from sciatic neuropathy, lumbosacral plexopathy, and L5 radiculopathy. Tibial motor and sural sensory NCSs should be performed to evaluate lesions of the sciatic nerve, lumbosacral plexus, and lumbosacral nerve roots.

The needle EMG is used to confirm the localization, assess the severity of the lesion, and exclude a more proximal lesion. In addition to peroneal innervated muscles, nonperoneal innervated L5 muscles are sampled to exclude a sciatic neuropathy, lumbosacral plexopathy, or radiculopathy. Even if the lesion is localized to the peroneal nerve at the fibular neck by the NCSs, nonperoneal innervated L5 muscles should be sampled to exclude a superimposed lesion. In slowly progressive lesions, MRI scan of the leg and thigh should be done to evaluate for a mass lesion.

**How is peroneal neuropathy at the fibular neck treated?**

EDX studies are useful in determining the prognosis and treatment options. Those who have predominantly demyelinating lesions at the fibular neck generally have a good prognosis, with good recovery in the first 3 months, once offending factors are eliminated. These patients usually can be managed conservatively, with an ankle foot orthosis to prevent falls and ankle sprains if the foot drop is severe enough to interfere with the patient’s gait. Physical therapy exercises are used to prevent contractures. Exacerbating factors, such as habitual leg crossing, should be eliminated. Patients who have lost a great deal of weight recently may benefit from protective padding over the fibular neck. Patients who have axonal loss lesions based on EDX studies (either primary or secondary) have a longer time to recovery and a poorer prognosis. Rarely, surgical exploration is warranted, usually in cases of severe trauma or stretch where the nerve has been so severely damaged that there is no evidence of continuity or reinnervation based on EDX studies after 2 to 6 months, or if there is little to no recovery based on the clinical examination after this time period. \(^{64-68}\) Some patients may obtain benefit from surgical decompression as far as a year out after their original nerve injury. \(^{67}\) Surgical exploration also is indicated in slowly progressive lesions when a mass lesion is suspected or seen on the MRI scan.
**Meralgia Paresthetica**

**Case vignette**
A 53-year-old woman was referred for burning, pain, and numbness over the right anterolateral thigh, of 3 months’ duration. She had a 3-year history of diabetes mellitus, and had recently gained 20 pounds. Strength testing and deep tendon reflexes were normal in the upper and lower extremities. There was an oval-shaped patch of decreased light touch and pinprick sensation over the right anterolateral thigh, and a Tinel’s sign at the inguinal ligament on the right side.

**What is meralgia paresthetica, and what are its clinical manifestations?**
The classic clinical presentation of lateral femoral cutaneous neuropathy, also known as meralgia paresthetica, is burning pain and numbness over the anterolateral thigh, without weakness of reflex changes.\(^6^9\) The lateral femoral cutaneous nerve of the thigh is a pure sensory nerve that runs directly off the L2-L3 roots around the pelvic brim, and it passes under the inguinal ligament to supply an oval-shaped area of skin over the anterolateral thigh. Entrapment of the nerve may occur as it passes under the inguinal ligament, resulting in a painful, burning, numb patch of skin that can be delineated over the anterolateral thigh (Fig. 7). Many patients report hypersensitivity of the numb area. Symptoms are worsened with standing and walking, and relieved by flexing the hip. Because there is no muscular innervation from this nerve, there is no associated muscle atrophy, weakness, or loss of reflexes.

**What are the causes and pathophysiology of meralgia paresthetica?**
Most cases of meralgia paresthetica are idiopathic, occurring most commonly in patients who are obese; wear tight underwear, pantyhose, or pants; or have diabetes mellitus. Certain occupations also can predispose to compression of the lateral femoral cutaneous nerve, such as workers who wear heavy tool belts. It also can be precipitated by pregnancy. Although most cases are caused by entrapment at the inguinal ligament, some cases have resulted from tumors and other mass lesions such as abdominal aortic aneurysms compressing the upper lumbar plexus more proximally. Compression also may occur perioperatively or postoperatively following hernia repair, renal transplant, hip replacement surgery, iliac crest bone graft harvesting, gastric bypass surgery, and after aortic valve and coronary artery bypass surgery.\(^7^0–^7^2\) Rare lesions are caused by direct trauma.

**What is the differential diagnosis of meralgia paresthetica?**
The differential diagnosis includes femoral neuropathy, lumbar plexopathy, and high lumbar radiculopathy. Lumbar radiculopathy usually is accompanied by back pain, and the sensory loss generally is ill defined, in contrast to the fairly well demarcated sensory loss in meralgia paresthetica. In femoral neuropathy and lumbar plexopathy, the sensory loss usually involves the entire anterior thigh, and extends into the medial thigh and leg. Finally, lumbar radiculopathy, plexopathy, and femoral neuropathy may be accompanied by weakness and a depressed knee jerk, which are not seen in meralgia paresthetica.

**How is meralgia paresthetica diagnosed?**
The diagnosis often is recognized based on the patient’s classic clinical presentation of burning pain and numbness restricted to the anterolateral thigh, unaccompanied by back pain, weakness, or reflex changes. Unfortunately, the lateral femoral cutaneous sensory potential is not recorded easily in the EMG laboratory, even in healthy asymptomatic individuals, and especially in obese or older individuals. If the sensory potential is recordable on the asymptomatic side, however, then a low or absent
potential on the symptomatic side is certainly consistent with a diagnosis of lateral femoral cutaneous neuropathy. EDX testing is probably most useful in excluding a femoral neuropathy, lumbosacral plexopathy, or lumbar radiculopathy, if the diagnosis is in question. In this case, femoral and peroneal motor NCSs, and superficial peroneal and saphenous sensory NCSs can be performed, in addition to lateral femoral cutaneous sensory NCS. Note, however, that the saphenous sensory potential may be absent in healthy, asymptomatic individuals, especially in the older population, and both the symptomatic and asymptomatic side must be tested. Needle EMG examination of L3-L4 innervated muscles should be entirely normal in lateral femoral cutaneous neuropathy, being a purely sensory nerve, while denervating changes might be found in lumbar radiculopathy, plexopathy, and in femoral innervated

Fig. 7. Anatomy of the lateral femoral cutaneous nerve. This pure sensory nerve comes directly off the L2-L3 roots, and runs under the inguinal ligament, where it may be injured or entrapped. The lateral femoral cutaneous nerve supplies sensation to a large oval area of skin over the lateral and anterior thigh. (Adapted from Haymaker W, Woodhal B. Peripheral nerve injuries. Philadelphia: W.B. Saunders; 1953; with permission.)
muscles in femoral neuropathy. Imaging with CT or MRI scan of the abdomen and pelvis generally is not recommended unless there is suspicion of a mass lesion, such as in a patient who has known or suspected cancer, or abdominal aortic aneurysm.

**How is meralgia paresthetica treated?**
In most patients, meralgia paresthetica is time-limited, and conservative therapy is warranted, including removing causative factors. Eliminating tight clothing may be sufficient. Symptoms in obese patients usually resolve with weight loss, and pregnant women after childbirth. In some patients, however, symptoms persist or become intolerable, and medical treatment should be initiated. This is especially true for diabetics, in whom improvement may take months to years. Because the area of discomfort is well demarcated, local treatment such as lidoderm patch, capsaicin, or lidocaine cream may be sufficient to alleviate the discomfort. If this fails, systemic treatment can be tried if there is no contraindication. Typical agents used to treat neuropathic pain include tricyclic or atypical antidepressants such as duloxetine, or anticonvulsants such as gabapentin, pregabalin, or carbamazepine, starting at a low dose and tapering up as tolerated. In refractory cases, patients may receive temporary benefit from local steroid injections, especially if surgical decompression is considered. It remains controversial whether neurolysis with transposition of the lateral femoral cutaneous nerve versus transection of the nerve (neurectomy) is more effective in those patients who ultimately require surgical management, although many believe that transection is the treatment of choice.

**Tarsal Tunnel Syndrome**

**Case vignette**
A 45-year-old woman described persistent foot pain after sustaining a nondisplaced fracture of the ankle 3 months previously, which required 4 weeks of casting. The pain worsened with walking. Examination showed mild atrophy of the right intrinsic foot muscles, with tenderness over the medial ankle. Toe and ankle plantar flexion and dorsiflexion were normal. Light touch and pinprick sensation were decreased over the medial sole of the right foot, and intact over the lateral foot and the dorsum of the foot. Deep tendon reflexes were intact and symmetric throughout, including the ankle jerks.

**What is tarsal tunnel syndrome, and what are its clinical manifestations?**
Most patients report the gradual onset of burning pain in the ankle and sole of the foot, with numbness and tingling in the heel or sole, depending on which branches are involved. Symptoms are generally unilateral, occur more commonly in women than men, and are worsened with weight bearing. Although there may be wasting of the intrinsic foot muscles, strength testing is usually normal, because proximal muscles also subserve these functions. There may be a Tinel’s sign at the tarsal tunnel, although this is a nonspecific sign.

The history and examination are most consistent with distal tibial neuropathy across the tarsal tunnel, also known as tarsal tunnel syndrome (TTS). TTS results from entrapment of the distal tibial nerve under the flexor retinaculum at the medial ankle posteriorly. TTS is probably quite rare, although its incidence is a matter of debate. Although some podiatrists feel that TTS is common, most neurologists believe that it is quite rare. As the distal tibial nerve runs under the flexor retinaculum through the tarsal tunnel at the medial malleolus, it divides into the calcaneal sensory
nerves and the medial and lateral plantar nerves (Fig. 8). The calcaneal nerves provide sensation to the heel of the sole. The medial and lateral plantar nerves are mixed nerves that innervate the intrinsic foot muscles. The medial plantar nerve supplies sensation to the first three toes and the medial fourth toe, while the lateral plantar nerve supplies the little toe and the lateral fourth toe. One or more of the three nerve branches (calcaneal, medial, and lateral plantar) may be involved in TTS.

Most cases of TTS are idiopathic, with no clear precipitating event. Trauma, such as ankle sprain or fracture, however, also accounts for a large proportion of TTS. Degenerative bone disease, connective tissue disorders such as rheumatoid arthritis, some systemic disorders such as diabetes mellitus and hypothyroidism, and some foot deformities, also may predispose to TTS. Rare cases are caused by varicose veins or other unusual mass lesions such as schwannoma or ganglion cyst in the tarsal tunnel. Occupations or activities that involve repetitive weight bearing, such as jogging, increase the risk for TTS.

**What is the differential diagnosis of tarsal tunnel syndrome?**

Local orthopedic problems such as plantar fasciitis or bursitis may mimic TTS, although sensory loss and foot weakness should not be seen. Other conditions that may cause numbness or burning of the sole with intrinsic foot muscle wasting include L5-S1 radiculopathy, lumbosacral plexopathy, sciatic neuropathy, proximal tibial neuropathy, or peripheral neuropathy. None of these conditions result in local foot pain,
however. In S1 radiculopathy, sciatic neuropathy, and proximal tibial neuropathy, the ankle jerk is reduced or absent, and there may be weakness of proximally innervated muscles, neither of which is seen in TTS. It may be difficult to distinguish early mild peripheral neuropathy from TTS. Peripheral polyneuropathy, however, usually begins in both feet simultaneously, while TTS is most often unilateral. Although TTS involves the sole of the foot, peripheral neuropathy often involves the sole and dorsum of the foot.

How is tarsal tunnel syndrome diagnosed?

The diagnosis of TTS often is made on the basis of typical symptoms and signs and response to treatment. Diagnosis in the EMG laboratory can be quite difficult, partly because of the absence of good evidence-based studies on the validity of electrodiagnostic techniques in TTS. Because the medial and lateral plantar sensory and mixed nerve potentials are quite small, they are often unobtainable even in normal subjects, especially in middle-aged or older subjects. Use of near-nerve recording may increase the yield of obtaining a potential. Unless a clear side-to-side difference is seen, however, an absent or low amplitude potential cannot be considered abnormal. Although tibial motor NCSs may reveal a prolonged distal latency if there is demyelination across the tarsal tunnel, this rarely is seen. In axonal loss lesions, the tibial motor amplitudes will be reduced, but this is a nonspecific finding that can be seen in proximal tibial or sciatic neuropathy, lumbosacral plexopathy, S1 radiculopathy, or peripheral neuropathy. Thus, other NCSs are done to exclude a proximal lesion, including peroneal motor and sural sensory NCSs. If the sural sensory response is abnormal, any abnormalities in the plantar nerves are likely secondary to either a polyneuropathy, sciatic neuropathy, or lumbosacral plexus lesion. The H reflex study may be helpful, being normal in TTS, but often abnormal in peripheral neuropathy, proximal tibial neuropathy, sciatic and lumbosacral plexus lesions, and S1 radiculopathy, all of which may cause sensory abnormalities over the sole of the foot.

Many patients find needle EMG of the intrinsic foot muscles painful and difficult to tolerate, and it is difficult to activate these muscles. Furthermore, these muscles often show increased insertional activity and occasionally denervation, even in normal patients. Thus, abnormalities seen in these muscles must be fairly marked, and asymmetric compared with the contralateral side. In addition to the intrinsic foot muscles, more proximal tibial and peroneal innervated muscles should be sampled to exclude a more proximal lesion.

If there are no precipitating factors such as ankle fracture or sprain, or connective tissue disorder, MRI scan of the ankle may be useful to identify a mass lesion such as ganglion cyst or tumor in the tarsal tunnel. If the diagnosis of TTS cannot be confirmed with EDX testing, which is often the case, then radiographs of the foot can be done to look for other causes of foot pain such as spurs, hairline fracture, or arthritis.

How is tarsal tunnel syndrome treated?

Conservative therapy is tried first, including eliminating exacerbating factors, such as tight shoes, and minimizing ankle swelling. The use of insoles and nonsteroidal anti-inflammatory medications may help reduce symptoms. Local corticosteroid injections also may be helpful in alleviating symptoms. If symptoms persist, or if a mass lesion is identified by imaging studies, surgical release of the flexor retinaculum is indicated. In one study, those patients with no history of trauma, or with a known mass such as tumor or ganglion cyst, or a short duration of symptoms, had the
best prognosis with surgical intervention. Some evidence indicates that combining neurovascular decompression with surgical release of the flexor retinaculum may be effective in idiopathic cases of TTS. In one small series, eight patients who had TTS diagnosed by classical clinical presentation and abnormal EDX studies and who had failed conservative therapy, underwent endoscopic tarsal tunnel release with good to excellent results. Other studies also have shown good results with endoscopic tarsal tunnel release.

**Piriformis Syndrome**

**Case vignette**

A 26-year-old woman complained of months of pain in the right buttock, radiating down the leg. Pain was worse with sitting, and relieved with standing or walking. She denied back pain. Neurologic examination was entirely normal, including strength testing, sensation, and deep tendon reflexes.

**What is piriformis syndrome, and what are its clinical manifestations?**

The typical presentation of piriformis syndrome consists of pain and tenderness in the buttock at the sciatic notch, often radiating down the leg, with the notable absence of back pain. It is more common in women than men, especially following minor trauma to the buttock. Some patients report paresthesias in the buttocks radiating down the back or side of the leg into the calf. Weakness of sciatic innervated muscles (gastrocnemius, hamstrings, tibialis anterior) is uncommon. Rarely, concurrent entrapment of the inferior gluteal nerve results in weakness of the gluteal muscles. Symptoms often are worsened during activities that require prolonged sitting, especially on a hard surface, or stooping or bending at the waist. Deep palpation of the sciatic notch may reproduce symptoms. Adduction and internal rotation of the hip may exacerbate symptoms, while standing or walking, or holding the leg externally rotated, may relieve symptoms.

**What are the causes and pathophysiology of piriformis syndrome?**

Sciatic-type symptoms in the absence of back pain, weakness, sensory loss, or reflex changes are consistent with proximal sciatic neuropathy. As the sciatic nerve leaves the pelvis, it runs under or through the piriformis muscle. Theoretically, a hypertrophied piriformis muscle or associated fibrous bands could compress the sciatic nerve at the pelvic outlet as it runs beneath or through the piriformis muscle in the buttock, resulting in signs and symptoms of proximal sciatic nerve dysfunction, also known as piriformis syndrome.

Robinson coined the term “Pyriformis Syndrome” in 1947, although it had been previously described by others as early as the late 1920s and 1930s. Since its original description, the syndrome has fallen in and out of favor as a genuine entity over the years. Indeed, most cases that comprised the original descriptions of piriformis syndrome in the 1930s probably would be diagnosed now as lumbosacral radiculopathy, with the advent of the MRI scan and CT/myelogram. Nevertheless, there remains a small group of patients who have buttock pain and tenderness, and sciatic type symptoms, with no demonstrable lesion on MRI scan or CT/myelogram of the lumbosacral spine, and no other discernible structural cause for sciatic symptoms. It is this group of patients for whom the diagnosis of piriformis syndrome is entertained. Criteria for a definitive diagnosis of piriformis syndrome include:

- Symptoms of sciatic neuropathy clinically, usually without weakness, sensory loss, or reflex changes
EDX evidence of sciatic neuropathy with no evidence of paraspinal involvement
Imaging studies of the lumbosacral nerve roots, spine, and pelvis that show no
evidence of radiculopathy, plexus infiltration or damage, or mass lesions
Surgical exploration showing entrapment of the sciatic nerve within a hypertrophied
piriformis muscle
Relief of symptoms following surgical decompression

What is the differential diagnosis of piriformis syndrome?
The major disorder commonly confused with piriformis syndrome is L5-S1 radiculopathy. Both may present with tenderness in the sciatic notch, and pain radiating down
the leg. In both disorders, symptoms may be exacerbated with sitting. Back pain, however, is common in radiculopathy, and is not seen in piriformis syndrome. Furthermore, the symptoms in radiculopathy are made worse, not better, with standing or walking. Other disorders confused with piriformis syndrome include lumbosacral plexopathy and sciatic neuropathy caused by a mass lesion, either in the pelvis or thigh, which may be difficult to differentiate from piriformis syndrome. Findings on the clinical examination and EDX testing should help differentiate these.

How is piriformis syndrome diagnosed?
The diagnosis of piriformis syndrome usually is based on a typical history and clinical
findings, in the absence of back pain or abnormalities of the lumbosacral nerve roots,
plexus, or sciatic nerve seen on imaging studies. EDX studies in piriformis syndrome
are invariably normal, or show only minor denervation or reinnervation in sciatic innervated muscles on needle EMG, with normal NCSs. Although sciatic nerve compres-
sion distal to the dorsal root ganglion theoretically should result in a low or absent
sural or superficial peroneal sensory potential, this generally is not seen in piriformis syndrome. MRI imaging of the buttocks may reveal a fibrous band or anomalous
vessel, or a hypertrophied piriformis muscle, compressing the sciatic nerve. This is
a nonspecific finding, however, seen with the same frequency in asymptomatic
patients, or on the asymptomatic side of symptomatic patients (personal
communication, Dr. Cheryl Petersilge). EDX and imaging studies are most useful in
excluding lumbosacral root lesions or mass lesions compressing sciatic innervated
fibers in the buttocks, pelvis, or thigh.

How is piriformis syndrome treated?
Conservative therapy is tried initially. This consists of physical therapy exercises,
including stretching of the piriformis muscle. If this is unsuccessful, then a sciatic nerve
block at the sciatic notch, or a local corticosteroid injection into the piriformis muscle,
can be done, usually under CT guidance. Indeed, some consider relief of symptoms
with sciatic nerve block to be diagnostic of piriformis syndrome, although it is equally
ture that patients who have lumbosacral radiculopathy also obtain relief with sciatic
nerve block. Botulinum toxin injection into the piriformis muscle, under EMG guidance,
produced clinical improvement in most patients in a few reported studies. Patients who fail conservative therapy, or who respond to local corticosteroid injection
initially but with return of symptoms, may be referred for surgical exploration of the
sciatic nerve. Surgery consists of sectioning of the piriformis muscle if hypertro-
phyed, or sectioning of fibrous bands or removal of vessels that may be compressing
the sciatic nerve in the buttock. The success of surgery is debated, although
those who have compressive fibrous bands or vessels, or abnormal EDX findings, may
have a better surgical outcome.
Interdigital Neuropathy (Morton’s Neuroma)

Case vignette
A 56-year-old woman described sharp, shooting pain in the ball of the right foot, in the web space between the third and fourth toes. The pain began approximately 6 months previously, and more recently she had noted numbness in the third and fourth toes. Standing and walking made her symptoms worse. Her pain radiated into the third and fourth toes, and occasionally posteriorly to the ankle. Neurologic examination revealed decreased light touch and pain sensation between the third and fourth toes of the right foot. Strength and deep tendon reflexes were intact throughout, and there was no wasting of the intrinsic foot muscles. There was tenderness over the third metatarsal head on the right, and compression of this area produced increased pain and paresthesias in the third and fourth toes.

What is interdigital neuropathy (Morton’s neuroma), and what are its clinical manifestations?
The finding of local pain in the ball of the foot between the third and fourth toes, worsened with weight bearing, and accompanied by numbness of the third and fourth toes, is a classic presentation of interdigital neuropathy, also known as Morton’s neuroma. It occurs most often between the third and fourth toes, although it can occur between the other toes. The pain often is accompanied by numbness of the third and fourth toes, although if the neuropathy occurs between other toes, then those toes may become numb. It is considerably more prevalent among women than men, especially middle-aged women.

What are the causes and pathophysiology of interdigital neuropathy (Morton’s neuroma)?
The likely etiology of Morton’s neuroma is chronic repetitive compression of the common plantar digital nerve between the third and fourth metatarsal heads, against the transverse metatarsal ligament. Hyperextension of the metatarsophalangeal joints, for example from wearing high-heeled or pointed shoes, exacerbates the compression. Other predisposing disorders include congenital foot deformities, foot dystonias, spasticity that results in hyperextension of the metatarsophalangeal joints, and rheumatoid arthritis.

What is the differential diagnosis of interdigital neuropathy (Morton’s neuroma)?
The differential diagnosis of Morton’s neuroma includes other causes of foot pain such as TTS, plantar fasciitis, or arthritis. TTS may be particularly difficult to differentiate, as the pain and numbness usually are limited to one side of the sole of the foot. In TTS, however, the numbness involves the sole of the foot, which distinguishes it from Morton’s neuroma. Although plantar fasciitis and arthritis can cause foot pain, especially in the ball of the foot, there is no numbness or sensory loss associated with these conditions unless there is an associated neuropathy. Furthermore, compression of the web space between the metatarsal heads usually does not reproduce the symptoms.

How is interdigital neuropathy (Morton’s neuroma) diagnosed?
The diagnosis of Morton’s neuroma generally is based on the typical clinical history of pain in the ball of the foot, usually between the third and fourth toes, worsened with weight bearing, and often accompanied by numbness of the corresponding toes. Compression of the web space between the metatarsal heads, by squeezing this area between the examiner’s thumb and index finger, often reproduces the pain, and is quite specific for Morton’s neuroma. A Tinel’s sign may be seen here on occasion.
EDX testing is most useful in excluding other peripheral nerve or nerve root disorders that may result in foot pain, especially TTS, sciatic neuropathy, or lumbosacral radiculopathy laboratory. Sensory NCSs of the interdigital nerves themselves can be done, using near-nerve needle electrodes, looking for a reduction in amplitude or slowing of conduction velocity in the involved interdigital nerve. This technique, however, is somewhat more painful to perform than standard sensory NCSs, and must be done in an EDX laboratory that is equipped to perform such studies. Thus, they are not generally part of the standard testing in the diagnosis of Morton’s neuroma.

Both MRI scan and ultrasound have been used in the evaluation of patients with suspected Morton’s neuroma, each with advantages and disadvantages. The sensitivity and specificity of the MRI scan is particularly dependent on the field of view, type of coil, and sequences used. Newer techniques have resulted in greater sensitivity and specificity. The MRI scan is particularly useful for preoperative localization and determining the size of the neuroma. The finding of a neuroma on MRI scan, however, does not necessarily imply a clinically relevant lesion, and clinical correlation is warranted, especially before consideration of surgical treatment. Although ultrasound is sensitive in detecting abnormalities in the intermetatarsal web space, is more widely available than MRI scan, and also predicts the size of the neuroma accurately, results differ concerning its specificity in distinguishing neuroma from other inflammatory or mass lesions.

How is interdigital neuropathy (Morton’s neuroma) treated?
The initial treatment consists of conservative measures, including physical therapy, foot orthotics, and eliminating offending factors such as high-heeled shoes. Local interdigital anesthetic block, often in conjunction with corticosteroids, may be very helpful in relieving symptoms in a significant number of patients. Repeated injections are often necessary, although a positive treatment response to the block does not necessarily predict a positive outcome if surgery is subsequently performed. Patients who fail conservative therapy, or have a return of symptoms after a combination of local blocks and conservative therapy such as physical therapy, orthotics, and removal of offending factors, are referred for surgery. In one study, those patients who had a presurgical MRI scan showing a neuroma measuring greater than 5 mm in transverse measurement had a significantly higher good surgical outcome with neurectomy than those who had a neuroma measuring 5 mm or less. Some debate exists as to the best surgical approach, neurolysis of the interdigital nerve versus surgical excision of the neuroma (neurectomy). Both approaches often afford complete relief of symptoms. Some favor neurolysis over neurectomy, because of the real potential of developing a recurrent neuroma in the nerve stump after neurectomy, or permanent numbness of the toes. Despite the multitude of published reports, however, there is incomplete evidence from well-designed randomized controlled trials to assess the long-term effectiveness of surgical versus nonsurgical treatments for Morton’s neuroma.

REFERENCES


