

Common Entrapment Neuropathies

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ABSTRACT

Purpose of Review: This article addresses relevant peripheral neuroanatomy, clinical presentations, and diagnostic findings in common entrapment neuropathies involving the median, ulnar, radial, and fibular (peroneal) nerves.

Recent Findings: Entrapment neuropathies are a common issue in general neurology practice. Early diagnosis and effective management of entrapment mononeuropathies are essential in preserving limb function and maintaining patient quality of life. Median neuropathy at the wrist (carpal tunnel syndrome), ulnar neuropathy at the elbow, radial neuropathy at the spiral groove, and fibular neuropathy at the fibular head are among the most frequently encountered entrapment mononeuropathies. Electrodiagnostic studies and peripheral nerve ultrasound are employed to help confirm the clinical diagnosis of nerve compression or entrapment and to provide precise localization for nerve injury. Peripheral nerve ultrasound demonstrates nerve enlargement at or near sites of compression.

Summary: Entrapment neuropathies are commonly encountered in clinical practice. Accurate diagnosis and effective management require knowledge of peripheral neuroanatomy and recognition of key clinical symptoms and findings. Clinical diagnoses may be confirmed by diagnostic testing with electrodiagnostic studies and peripheral nerve ultrasound.

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INTRODUCTION

This article focuses on peripheral mononeuropathies, indicating disease or dysfunction in a single peripheral nerve. Mononeuropathies may be caused by focal compression, inflammation, nerve tumors, trauma, or other etiologies. Compression (or entrapment) is the most common cause. While mononeuropathies may also be superimposed upon a background of polyneuropathy, a survey of polyneuropathies is beyond the scope of this article.

Entrapment mononeuropathies represent a common reason for visits to primary care and outpatient neurology practices. The most common of these, carpal tunnel syndrome, is related to chronic compression of the

median nerve. Carpal tunnel syndrome may be present in up to 42% of workers in certain occupations (eg, poultry processing) and has an annual incidence of 193 per 100,000 in all women.^{1,2} Its prevalence in the United States is estimated at 50 per 1000, with a cost of \$30,000 per affected individual.³ Morton neuroma, ulnar neuropathy, meralgia paresthetica, and radial neuropathy represent the other most common peripheral mononeuropathies.²

Knowing peripheral nerve anatomy and function allows clinical localization that can be further refined and confirmed with electrodiagnostic studies and peripheral nerve imaging. In patients with mononeuropathy, the etiology, severity, odds of spontaneous

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KEY POINT

■ Early diagnosis and effective management of mononeuropathies are essential in improving patient quality of life and reducing costs of care.

recovery, and patient preferences are important factors that guide treatment. Early diagnosis and effective management of mononeuropathies are essential in improving patient quality of life and reducing costs of care.

This article presents the most commonly encountered entrapment mononeuropathies, with a focus on relevant anatomy, clinical symptoms, methods of diagnosis, and recommended treatment. The pathophysiologic processes related to peripheral nerve trauma and compression are not covered in this discussion because of space limitations but are comprehensively reviewed by Stewart.⁴

MEDIAN NERVE

Median neuropathy at the wrist, specifically carpal tunnel syndrome, is the most common mononeuropathy of adults. A thorough understanding of the anatomy of the median nerve and adjacent structures and of associated diagnostic techniques is therefore invaluable in outpatient neurology.

Basic Anatomy

The median nerve forms from the terminal divisions of the medial and lateral cords of the brachial plexus, receiving contributions from the C5 to T1 nerve roots (Figure 7-1). It courses medial to the brachial artery throughout the upper arm. In the distal arm, the

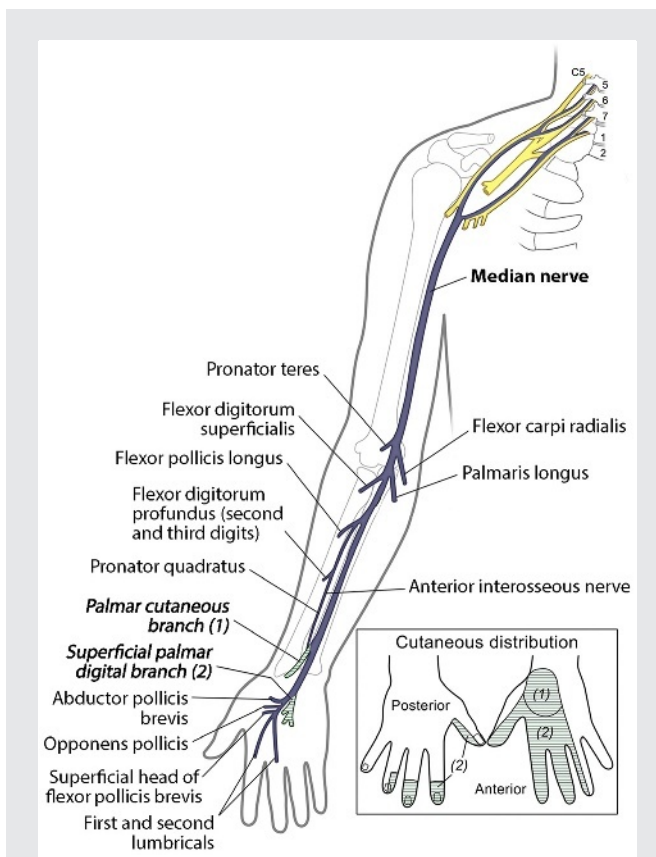


FIGURE 7-1 Median nerve. The nerve is labeled in bold, and sensory branches are labeled in italics.
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nerve may pass beneath the ligament of Struthers, which is present in some individuals (1% to 13%) and represents a rare cause of median nerve entrapment.^{5,6} The nerve then passes under the bicipital aponeurosis at the elbow.

Moving distally, the median nerve then travels between the two heads of the pronator teres, deep to the humeral head and superficial to the ulnar head. The nerve then continues distally between the flexor digitorum superficialis and flexor digitorum profundus muscles. Approximately 4 cm distal to the medial epicondyle, the anterior interosseus nerve branches from the main trunk of the median nerve.⁵ The anterior interosseus nerve innervates the flexor pollicis longus, flexor digitorum profundus to the second and third digits, and pronator quadratus.

Just proximal to the distal wrist crease, the palmar cutaneous branch of the median nerve leaves the main nerve trunk.⁵ It travels between the palmaris longus and flexor carpi radialis tendons and proceeds outside the carpal tunnel to provide sensation for the thenar eminence and proximal lateral palm.^{7,8}

The median nerve lies superficially at the volar distal wrist crease, an external landmark that roughly approximates the carpal tunnel inlet. The flexor retinaculum forms the roof of the carpal tunnel. Deep to and surrounding the nerve are the tendons of the flexor pollicis longus, flexor digitorum superficialis, and flexor digitorum profundus.⁹ The hook of the hamate, pyramidal, and pisiform bones form the medial (ulnar) border of the carpal tunnel, while the scaphoid bone, trapezoid bone, and tendon of the flexor carpi radialis muscle comprise the lateral (radial) aspect. The floor or inferior margin of the carpal tunnel formed by the carpal bones is known as the *carpal sulcus*.

In the distal carpal tunnel, the median nerve divides into the motor (thenar) branch and sensory branches to the digits and palm. The motor branch innervates the abductor pollicis brevis, opponens pollicis, and superficial head of flexor pollicis brevis.¹⁰ An accessory thenar branch may innervate the flexor pollicis brevis in nearly half of patients.¹⁰

Median nerve anatomic variants. Martin-Gruber anastomoses are the most common anatomic variants affecting the median nerve, with an estimated prevalence ranging from 20% to 40%.^{11,12} Martin-Gruber anastomoses typically leave the main median nerve or anterior interosseus nerve trunk near the elbow and cross over to join the ulnar nerve. There are four to six types of Martin-Gruber anastomoses based on the exact anatomy.¹² The main significance of a Martin-Gruber anastomosis is the confounding effect that it may cause in interpretation of electrodiagnostic studies, as discussed later in this article. Other rare anatomic variants of the median nerve include the Riche-Cannieu anastomosis (“the all-ular hand”), Berrettini anastomosis, and Marinacci anastomosis (the reverse Martin-Gruber anastomosis).¹¹

Clinical Presentation

The most common median mono-neuropathy results from median nerve compression at the wrist, causing carpal tunnel syndrome. Patients with carpal tunnel syndrome classically present with numbness, tingling, and other paresthesia affecting the first through third fingers and the lateral aspect of the fourth finger. It is not unusual for patients to report sensory symptoms affecting the entire hand or radiation from the hand to the proximal upper extremity. These sensory symptoms may be exacerbated

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by activities that require wrist flexion, including driving a car, and often wake the patient from sleep at night. Shaking or flicking the affected hand may alleviate the sensation, which can be quite painful. **Case 7-1** illustrates a classic case of early carpal tunnel syndrome.

Weakness may occur in more advanced carpal tunnel syndrome. Patients describe a generalized sense of

weakened grip and difficulty performing fine motor tasks, and they may drop items easily. It is often unclear how much of the disability is related to loss of muscle strength as opposed to loss of sensation. On physical examination, careful observation may reveal mild flattening of the thenar eminence or frank atrophy. A Tinel sign consists of paresthesia in

Case 7-1

A 24-year-old right-handed woman presented with numbness and tingling in her right hand that had begun about 3 months previously. Initially, the symptoms only occurred while carrying her infant son. She then began to wake at night with a sensation of painful numbness in the entire hand that radiated to her elbow. She tried wearing a neutral position wrist brace at night, but it provided only a minor degree of improvement. She had no history of wrist or upper limb injury.

On examination, she had mild weakness of thumb abduction and loss of sensation over the entire palmar surface of the right first through third fingers and the lateral half of the fourth finger. Percussion over the midvolar wrist sent shooting electrical pains into her hand.

Nerve conduction studies were performed and showed slowing of the median mixed and sensory nerve action potentials across the wrist with normal motor responses. Nerve ultrasound demonstrated enlargement of the median nerve at the carpal tunnel inlet (**Figure 7-2**). She was diagnosed with carpal tunnel syndrome and underwent a local corticosteroid injection performed by an orthopedic surgeon, resulting in relief of symptoms.

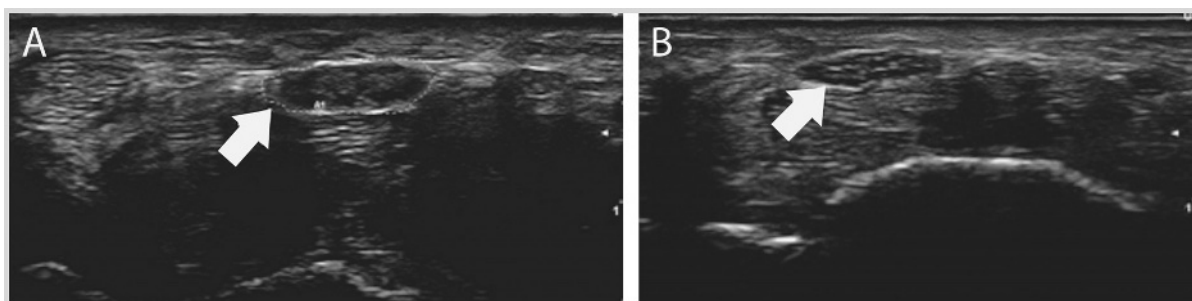


FIGURE 7-2 Ultrasonography showing the median nerve at the carpal tunnel inlet at the level of the distal wrist crease. *A*, Ultrasonography of the patient in **Case 7-1** showing enlarged cross-sectional area (19 mm²) of the median nerve (*arrow*) associated with loss of nerve signal. *B*, Ultrasonography of a healthy control showing normal median nerve (*arrow*) measuring 8 mm² at the distal wrist crease with normal signal.

Comment. This case is a classic presentation of carpal tunnel syndrome, confirmed by nerve conduction studies and nerve ultrasound. Although the median nerve sensory territory does not extend proximal to the wrist, patients often describe radiation of paresthesia into the arm, particularly upon waking. As an initial treatment option, nocturnal wrist splinting may relieve symptoms, although the response may be incomplete in more severe cases of carpal tunnel syndrome. In patients without axonal loss or weakness, corticosteroid injections of the carpal tunnel are a reasonable treatment option that may alleviate symptoms short of surgical decompression.

the distal distribution of a sensory nerve when it is percussed. In carpal tunnel syndrome, a Tinel sign may be present at the distal wrist crease, but the sensitivity may be as low as 30% to 43% with specificity up to 65%.^{13,14} The Phalen test is performed by having the patient flex the wrists and press the dorsum of both hands together for 30 to 60 seconds. A false-positive result may occur when interpreting musculoskeletal wrist pain as a positive result, and specificity is only 15% to 17% range. Sensitivity of Phalen testing is better, but only around 50% to 67%.^{14–16} Patients with positive Tinel and Phalen testing have lower nerve conduction velocities than those with negative testing.¹⁷ A negative Phalen test is a strong predictor of normal nerve conduction studies.¹⁸

Strength testing for suspected carpal tunnel syndrome should include upper limb and especially forearm and intrinsic hand muscles with special focus on the abductor pollicis brevis, opponens pollicis, and flexor pollicis longus muscles. The finding of weakness in the abductor pollicis brevis and opponens pollicis with normal strength in the flexor pollicis longus helps localize the pathology to the carpal tunnel. More proximal lesions of the median nerve would involve the flexor pollicis longus. Detailed sensory examination will reveal loss of sensation in the palmar first through third digits and lateral fourth digit. Sensation of the proximal lateral palm may be spared in carpal tunnel syndrome because of its innervation by the palmar cutaneous branch of the median nerve.

More proximal lesions of the median nerve have the same distribution of sensory loss as seen in carpal tunnel syndrome with the addition of the palm, but lack a Tinel sign at the

wrist and positive Phalen testing. Depending upon the exact level of the proximal median nerve lesion, weakness in the flexor pollicis longus, flexor digitorum profundus to the second and third digits, flexor digitorum superficialis, and pronator teres muscles may be present. Isolated lesions of the anterior interosseus nerve may lead to weakness in the flexor pollicis longus, flexor digitorum profundus to the second and third digits, and the pronator quadratus. Unlike carpal tunnel syndrome, anterior interosseus neuropathies are generally due to noncompressive causes such as idiopathic brachial neuritis/neuralgic amyotrophy.¹⁹

Electrodiagnostic Testing in Carpal Tunnel Syndrome

Most neurologists consider electrodiagnostic testing to be the gold standard in diagnosing carpal tunnel syndrome. Considerable debate exists regarding the optimal set of tests for making a diagnosis. The American Association of Electrodiagnostic Medicine, now known as the American Association of Neuromuscular and Electrodiagnostic Medicine (AANEM), has published basic guidelines for recommended electrodiagnostic testing in patients with suspected carpal tunnel syndrome. The recommendations include performing an initial median sensory nerve conduction study across the wrist at a distance of 13 cm or 14 cm. If normal, a shorter segment study (7 cm to 8 cm) of the median mixed nerve is recommended. These studies should be performed with a radial or ulnar sensory or mixed response across the same distance in the same limb for comparison of interlatency differences. A median motor response recording over the abductor pollicis brevis is also recommended, along

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- When performing nerve conduction studies, clinicians should be aware of the changes caused by a Martin-Gruber anastomosis.
- Peripheral nerve ultrasound is a useful tool in diagnosing entrapment neuropathies, with nerves consistently enlarged at or near sites of compression.

with measurement of another motor response in the same limb. Optional testing includes needle EMG of C5 to T1 muscles (to exclude cervical radiculopathy as a contributing factor) and supplementary nerve conduction studies.²⁰

Electrodiagnostic testing in Martin-Gruber anastomosis. When performing median nerve conduction studies, clinicians should be aware of the changes caused by a Martin-Gruber anastomosis. A Martin-Gruber anastomosis may result in low distal amplitude of the median nerve compound muscle action potential (CMAP) when recording over the abductor pollicis brevis and stimulating at the wrist. With proximal nerve stimulation, the median CMAP amplitude will be higher. This can create confusion and concern for overstimulation of the median nerve at the elbow, with costimulation of the ulnar nerve. The presence of a Martin-Gruber anastomosis can be confirmed by stimulating the median nerve at the elbow and recording a CMAP in an ulnar-innervated hand muscle, typically the first dorsal interosseus or abductor digiti minimi.²¹ Identifying a Martin-Gruber anastomosis by recording over ulnar-innervated thenar muscles is discouraged, as the abductor pollicis brevis CMAP may confound interpretation. A drop in amplitude between distal and proximal stimulation of the ulnar nerve may also be present in a Martin-Gruber anastomosis and be mistaken for conduction block.²¹

Ultrasonographic Testing in Carpal Tunnel Syndrome

Neuromuscular ultrasonography has recently emerged as a novel diagnostic tool for carpal tunnel syndrome. The exact role for neuromuscular ultrasonography in patient care is still under investigation, but substantial

evidence exists to support its use.²² In neuromuscular ultrasonography, high-frequency ultrasound probes provide detailed peripheral nerve anatomic information. Nerves are consistently enlarged at sites of compression. In carpal tunnel syndrome, the median nerve typically has a maximal point of enlargement at or near the carpal tunnel inlet corresponding to the surface anatomic marker of the distal wrist crease (**Figure 7-2**). Reference values are varied, but a cross-sectional area of the median nerve of greater than 12 mm² is generally considered abnormal. A 2014 study found a cross-sectional area of greater than 10 mm² to have 89% sensitivity and 90% specificity for diagnosing carpal tunnel syndrome, as compared to a validated clinical diagnostic tool.²³

Peripheral nerve ultrasound is a useful tool in diagnosing entrapment neuropathies, with nerves consistently enlarged at or near sites of compression. Substantial literature supports the use of ultrasound in diagnosing carpal tunnel syndrome.^{22,23} Neuromuscular ultrasonography also provides added value in understanding the pathogenesis of carpal tunnel syndrome. Routine ultrasound studies may reveal contributing factors or issues that confound the diagnosis of carpal tunnel syndrome. These may range from anatomic variants and intraneural tumors to the relatively common bifid median nerve and the persistent median artery. The persistent median artery is an accessory artery that arises from the ulnar artery in the proximal forearm as an embryologic remnant. When present, it courses with the median nerve through the forearm and carpal tunnel. Other non-nerve findings may include coexistent tenosynovitis or accessory musculature.²²

Electrodiagnostic and Ultrasonographic Testing in Other Median Neuropathies

Testing for proximal median neuropathies involves the same techniques used for the assessment of carpal tunnel syndrome, with less need for focused testing across the carpal tunnel and increased need for needle EMG to help determine the level and severity of involvement. Neuromuscular ultrasonography can be helpful in identifying peripheral nerve trauma or other abnormalities of the nerve, including focal enlargement of proximal segments.

Treatment of Carpal Tunnel Syndrome

Clinical practice guidelines are available for the treatment of carpal tunnel syndrome, although adherence to these published standards varies widely in practice.²⁴ The American Academy of Orthopaedic Surgeons (AAOS) states that nonsurgical treatments (splinting of the wrist to a neutral position and local corticosteroid injections) are a reasonable option for those early in the course of symptoms without evidence of median nerve denervation. A trial period of 2 to 7 weeks is recommended to observe for improvement. Carpal tunnel release is strongly recommended (Grade A, Level I evidence) either as a first option for treatment or for those for whom nonsurgical therapies have failed. The AAOS made no recommendations for patients with carpal tunnel syndrome and coexistent diabetes mellitus, cervical radiculopathy, or polyneuropathy or for carpal tunnel syndrome in the workplace.²⁵ Heat therapy, electrical stimulation, iontophoresis, and laser therapies were among the treatments with no evidence to support their use.²⁵ The American Congress of Rehabilitative Medicine (ACRM) also has published guidelines on carpal tunnel syndrome

treatment.²⁶ These recommendations largely mirror those of the AAOS.

The authors individualize treatment for carpal tunnel syndrome based on the severity of nerve compression as determined by the electrodiagnostic findings. For patients with mild carpal tunnel syndrome involving abnormal findings isolated to the median mixed or sensory responses, the authors begin a trial of neutral position wrist splinting and nonsteroidal anti-inflammatory medications where appropriate. Patients who do not experience satisfactory symptom relief may subsequently be referred for a trial of local corticosteroid injections. Decompressive surgery to transect the transverse carpal ligament may subsequently be considered in patients with significant residual symptoms. For patients with severe carpal tunnel syndrome with prolonged, low-amplitude CMAPs and denervated median-innervated thenar muscles by needle EMG, the authors may employ the same treatments acutely but also refer patients for expeditious surgical consultation with a hand surgery specialist in an effort to salvage the maximum degree of sensorimotor function in the hand.

ULNAR NERVE

Ulnar neuropathy, often occurring at or near the elbow, is the second most common mononeuropathy seen in outpatient neurology settings.

Basic Anatomy

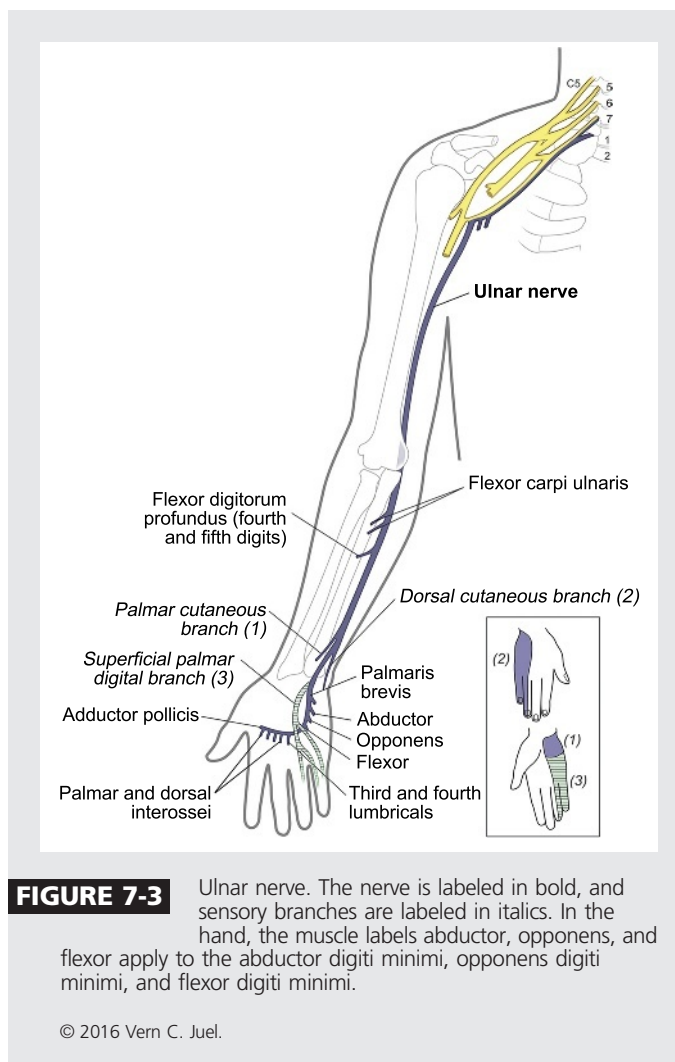
The ulnar nerve arises from the C8 to T1 (and occasionally from C7 to T1) nerve roots and the medial cord of the brachial plexus and descends through the posteromedial arm, anterior to the medial head of the triceps brachii (Figure 7-3). The first branch off the ulnar nerve is typically a sensory branch to the elbow, followed by a

KEY POINT

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KEY POINT

■ The medial collateral ligament of the elbow defines the floor of the cubital tunnel, while the roof consists of the thickened fascia known as the Osborne (arcuate) ligament. This ligament lies between the medial and ulnar heads of the flexor carpi ulnaris.



motor branch to the flexor carpi ulnaris.²⁷ Compression in the arm is rare but may occur at the arcade of Struthers, a fibrous band running from the medial head of the triceps to the medial intermuscular septum. Considerable debate exists over the prevalence of this anatomic variant and its clinical significance.²⁸

The ulnar nerve then passes through the epicondylar (ulnar) groove, located between the medial epicondyle and the olecranon. An anomalous anconeus epitrochlearis muscle overlying the ulnar nerve and extending between the olecranon and medial epicondyle may be a source of compression in some individuals.²⁷ The

cubital tunnel is located just distal to the elbow. The medial collateral ligament of the elbow defines the floor of the cubital tunnel, while the roof consists of the thickened fascia known as the Osborne (arcuate) ligament. This ligament lies between the medial and ulnar heads of the flexor carpi ulnaris.

Within the proximal forearm, the ulnar nerve is located medial and superior to the flexor digitorum profundus and deep to the flexor carpi ulnaris and gives branches to innervate these muscles along its course. The ulnar nerve lies lateral to the flexor carpi ulnaris and medial to the ulnar artery in the distal forearm.

Compression of the ulnar nerve in the forearm is rare.

The Guyon canal, or ulnar tunnel, is a site of potential ulnar nerve entrapment at the wrist. The canal extends from the proximal end of the pisiform to the hook of the hamate. The flexor retinaculum and hypothenar muscles define the floor, while the roof consists of the volar carpal ligament. The lateral (radial) border is defined by the hook of the hamate, while the pisiform, pisohamate ligament, and abductor digiti minimi muscle belly compose the medial (ulnar) border. The ulnar nerve passes through the Guyon canal with the ulnar artery.²⁹

The Guyon canal can be further subdivided into zones. Zone 1 extends from the proximal volar carpal ligament to the bifurcation of the ulnar nerve. Pathology in this region affects both the motor and sensory functions of the nerve. Zone 2 extends laterally beneath the palmaris brevis to the fibrous arch of the hypothenar muscles and contains only motor fibers of the ulnar nerve. Zone 3 extends medially from the ulnar nerve bifurcation and contains both motor and sensory fibers. The motor fibers form a superficial branch supplying the palmaris brevis muscle, so zone 3 pathology is generally considered to affect only sensory function.³⁰

Clinical Presentation and Examination Findings

The ulnar nerve provides sensory innervation to the fourth and fifth fingers, the medial palm, and the dorsomedial hand. Patients with ulnar neuropathy can present with paresthesia that may range from a feeling of deadness to shooting electriclike pains that are most prominent in the fourth and fifth digits. It is not unusual for patients to note pain radiation between the elbow and hand, although

clinical assessment should not reveal an objective sensory deficit in the medial forearm. Patients may have pain and tenderness near the epicondylar groove in cases of ulnar neuropathy at the elbow, although this is not always present.

Ulnar motor dysfunction may be perceived as clumsiness with mild or early ulnar neuropathy at the elbow. Patients may note difficulty with fine motor tasks or drop things easily. Frequently, patients experience weakness of the fifth digit and may struggle to place it into a jacket or pants pocket with the rest of the hand. Grip weakness is also a frequent symptom with more advanced motor involvement. Observant patients may notice atrophy of the intrinsic hand muscles, particularly the first dorsal interosseous.

On clinical examination, the Froment sign may be present. To test for the Froment sign, the patient is asked to hold a thin object (eg, a sheet of paper) between the thumb and index finger and an examiner attempts to remove it. In patients with weakness of the ulnar-innervated adductor pollicis, the object may be easily removed or the patient may attempt to compensate for the weakness by activating the median-innervated flexor pollicis longus and pinching with the distal phalanx of the thumb. Motor testing of the ulnar-innervated flexor digitorum profundus to the fourth and fifth digits along with the flexor carpi ulnaris should be performed as part of the standard examination.

A Tinel sign may be present over the ulnar nerve at sites of entrapment. Gentle palpation of the epicondylar groove during elbow flexion and extension can be helpful in diagnosing ulnar nerve subluxation, which is also present in a proportion of asymptomatic individuals.³¹ Although its presence is not diagnostic of ulnar

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KEY POINT

- Needle EMG is essential in assessing ulnar neuropathy at the elbow.

neuropathy and it is not an established risk factor, identifying subluxation may be helpful in selecting the appropriate surgical intervention in patients with ulnar neuropathy at the elbow. Subluxation can be detected by palpation of the ulnar nerve moving over the medial epicondyle with flexion or extension at the elbow.

Although rarely used by neurologists, the surgical literature makes frequent use of the McGowan grading system in the assessment of ulnar neuropathy at the elbow. It is helpful to know this system when reviewing the literature on the topic and communicating with surgical colleagues. Grade 1 consists of paresthesia in the fourth and fifth digits and a feeling of hand clumsiness. Grade 2 includes weakness and impaired sensation, sometimes with mild atrophy of the intrinsic hand muscles. In McGowan grade 3, the sensory and motor deficits are severe and marked muscle atrophy is present.³²

Electrodiagnostic Testing

Electrodiagnostic testing is considered the gold standard for the diagnosis of ulnar neuropathy, but the recommended testing and definition of abnormality is debated. The majority of work published has centered on ulnar neuropathy at the elbow, the most common variant of ulnar neuropathy. AANEM guidelines are frequently cited in most studies of ulnar neuropathy at the elbow.³³ The AANEM guidelines for the diagnosis of ulnar neuropathy at the elbow recommend performing ulnar sensory nerve conduction studies and motor nerve conduction studies to the abductor digiti minimi. Elbow position during testing (with flexion between 70 degrees and 90 degrees recommended) should be recorded and adequate warming maintained. A

minimum of 10 cm should be present between the above-elbow and below-elbow sites of stimulation. The diagnosis of ulnar neuropathy at the elbow can be made when the conduction velocity across the elbow is greater than or equal to 10 m/s slower than the wrist to below-elbow segment. Other supportive findings include a drop in CMAP amplitude of greater than 20% across the elbow.³³

Criticism of the initial AANEM guidelines center on their lack of sensitivity for precise localization in some patients with ulnar neuropathy at the elbow. Sensitivity has ranged from 37% to 86%, while specificity has been greater than 95%.³⁴ Techniques for improving sensitivity reduce specificity, and this should be considered when performing additional testing. Inching is a popular technique often used to identify sites of pathology in ulnar neuropathy at the elbow. The 10-cm segment between the above-elbow and below-elbow sites is divided into 2-cm segments. Stimulation is applied at each point, with attention to the latency differences and CMAP waveforms. Using this approach, the sensitivity of nerve conduction studies is improved, detecting up to 90% of patients with clinical evidence of ulnar neuropathy at the elbow.³⁵ Although this technique offers improved sensitivity and more precise localization, it requires attention to technical detail for accurate results.

Needle EMG is essential in assessing ulnar neuropathy at the elbow, as outlined in **Case 7-2**. Examination of ulnar-innervated muscles is helpful for both localization and assessment of severity. Other muscles with C7 and C8 innervation should also be sampled to assess for the presence of coexistent cervical radiculopathy or brachial plexopathy. Electromyographers should be aware that little evidence

Case 7-2

A 52-year-old left-handed man noted progressive numbness and tingling of his right fourth and fifth fingers. His hand was clumsy at times, and he began having difficulty buttoning his shirts. He reported a “funny bone” sensation that radiated into his right hand, particularly after talking on the phone for extended periods of time. Examination demonstrated mild atrophy of the right first dorsal interosseus muscle. A Tinel sign was present over the right epicondylar (ulnar) groove. He had moderate weakness of right finger abduction and deep finger flexion of the fourth and fifth digits with minimal weakness of thenar and finger extensor muscles. He also had sensory loss over the right medial palm, the medial aspect of the fourth finger and the entire fifth finger, and the dorsal medial hand. Ulnar neuropathy at the elbow was suspected and classified as a McGowan grade 2 based upon the clinical assessment.

The patient’s physician chose not to perform electrodiagnostic testing at that time. An evaluating surgeon recommended ulnar nerve transposition, as weakness and atrophy were already present. Following surgery, the patient continued to have progressive symptoms and was then referred for electrodiagnostic studies. Testing performed 6 months after surgery revealed mild slowing of the ulnar motor conduction velocity at the elbow with normal sensory responses. Ultrasonography of the ulnar nerve was normal. Detailed needle EMG found evidence of denervation and reinnervation in the right extensor indicis proprius, first dorsal interosseus, abductor pollicis brevis, and C8 to T1 paraspinals. Along with a mild right ulnar neuropathy at the elbow, a coexistent right C8 radiculopathy was diagnosed and confirmed by imaging of the cervical spine.

Comment. Although the patient had symptoms compatible with ulnar neuropathy at the elbow, mild weakness of thenar (median) and finger extensor (radial) muscles was overlooked, and he was referred for surgical intervention before the localizations for the deficits were confirmed. When he failed to improve, electrodiagnostic studies subsequently performed showed that a C8 radiculopathy was also present. This is an example of double crush syndrome, in which two separate peripheral nerve lesions occur along the course of the same nerve with both lesions contributing to the clinical findings. This case illustrates the importance of careful clinical examination and electrodiagnostic testing to establish accurate localization prior to surgery.

KEY POINT

- As with carpal tunnel syndrome, neuromuscular ultrasonography has emerged as an alternative means of assessing ulnar neuropathy at the elbow. The normal ulnar nerve cross-sectional area is 8 mm² to 11 mm² at the elbow, with larger values suggesting focal compression.

exists regarding the utility of needle EMG in determining prognosis.³⁶

Ultrasonographic Testing

As with carpal tunnel syndrome, neuromuscular ultrasonography has emerged as an alternative means of assessing ulnar neuropathy at the elbow. The normal ulnar nerve cross-sectional area is 8 mm² to 11 mm² at the elbow, with larger values suggesting focal compression.³⁷ Neuromuscular

ultrasonography is not believed to be a superior means of diagnosing ulnar neuropathy at the elbow for all patients, although limited evidence indicates that it may be more sensitive than nerve conduction studies early in the disease.^{38,39} Neuromuscular ultrasonography is best used as an adjunct to clinical and electrodiagnostic data to aid in localization.⁴⁰ It is helpful in pinpointing the area of pathology and may identify alternative etiologies

KEY POINTS

- Testing of the dorsal ulnar cutaneous sensory nerve response can be added to EMG and ultrasonography when assessing lesions distal to the elbow, assisting in localization of an ulnar neuropathy at or proximal to the wrist.
- Three months of conservative treatment with the use of elbow pads and avoidance of prolonged elbow flexion is recommended as first-line treatment in those patients with ulnar neuropathy at the elbow with mild symptoms and less severe electrodiagnostic findings. Up to 50% of patients will have resolution of symptoms with this approach.
- Unlike in carpal tunnel syndrome, corticosteroid injections have not shown efficacy in ulnar neuropathy at the elbow.

for ulnar neuropathy at the elbow. These include anatomic variants (eg, anomalous anconeus epitrochlearis muscle), ganglion cysts, and other mass lesions.

The evaluation of ulnar neuropathy outside the elbow region follows many of the previously mentioned principles. Whether evaluating proximal or distal lesions, ulnar motor and sensory nerve conduction studies are essential. Ultrasonography and needle EMG also provide helpful additive information. Testing of the dorsal ulnar cutaneous sensory nerve response can be added when assessing lesions distal to the elbow, assisting in localization of ulnar nerve lesions at or proximal to the wrist. The dorsal ulnar cutaneous sensory nerve response will be normal with the lesions at the wrist, but abnormal with lesions in the region of the proximal forearm or elbow.

Treatment of Ulnar Neuropathy at the Elbow

The best approach to treating ulnar neuropathy at the elbow remains controversial. Three months of conservative treatment with the use of elbow pads and avoidance of prolonged elbow flexion is recommended as first-line treatment in those with mild symptoms and less severe electrodiagnostic findings. Up to 50% of patients will have resolution of symptoms with this approach.⁴¹ For patients for whom conservative measures fail or who have evidence of significant axonal loss on initial electrodiagnostic examination, surgical management is indicated. Three general approaches are used: simple decompression through excision of the arcuate ligament, medial epicondylectomy, and ulnar nerve transposition.⁴² An early analysis of published data demonstrated that patients with more severe disease (McGowan grade 3) have the best

outcomes with anterior ulnar nerve transposition.⁴³ Other studies have favored simple decompression over transposition, unless difficulties with painful ulnar nerve luxation over the medial epicondyle are present.⁴³

No consensus exists on when ulnar neuropathy at the elbow becomes severe enough to warrant surgery. Some have suggested that surgical treatment is warranted in patients with mild (McGowan grade 1) ulnar neuropathy at the elbow. Excellent outcomes have been reported in this population using simple decompression and medial epicondylectomy.⁴⁴ Further supporting the case for early intervention are data demonstrating that patients with McGowan grade 3 ulnar neuropathy at the elbow are more likely to have fair or poor postsurgical outcomes.⁴⁵

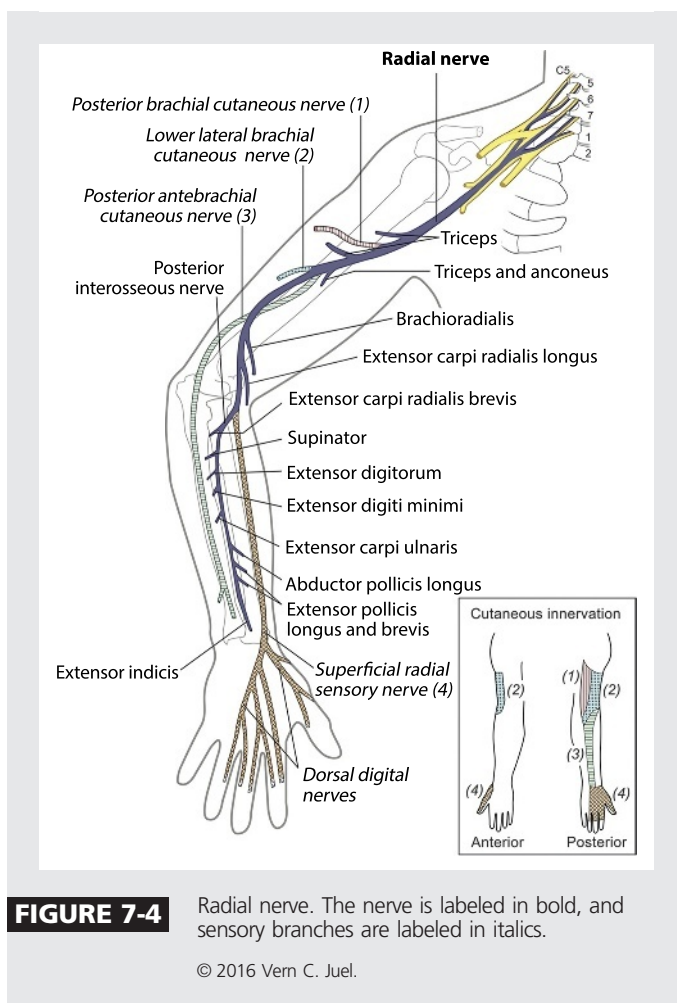
Few alternative approaches to the treatment of ulnar neuropathy at the elbow are available. Unlike in carpal tunnel syndrome, corticosteroid injections have not shown efficacy in ulnar neuropathy at the elbow.^{46,47} Ultrasound and low-level laser therapy have shown some promise but are neither widely accepted nor available.⁴⁸ Additional research is needed to understand how clinical, electrodiagnostic, and sonographic data might be combined to predict and optimize patient outcomes in ulnar neuropathy at the elbow.

RADIAL NERVE

The radial nerve is less likely to be affected by chronic compression than the median or ulnar nerves but remains a frequent mononeuropathy because of acute compressive lesions.

Basic Anatomy

The radial nerve is the terminal branch of the posterior cord of the brachial plexus (**Figure 7-4**). In the axilla, the



radial nerve yields three branches, including the posterior brachial cutaneous nerve and branches innervating the long and medial heads of the triceps brachii. It then travels with the deep brachial artery between the long head of the triceps and the humerus and courses through the spiral groove between the lateral and medial heads of the triceps. Two sensory branches are present in this region, including the lower lateral brachial cutaneous nerve, the posterior antebrachial cutaneous nerve, and motor branches to the lateral and medial heads of the triceps. After passing through the lateral intermuscular septa, the radial nerve travels between the brachialis and brachioradialis muscles just anterior to

the lateral epicondyle, with branches to the brachioradialis and the extensor carpi radialis longus and brevis. The nerve then bifurcates into two terminal branches, a superficial sensory branch and a deep branch called the posterior interosseous nerve.

The posterior interosseous nerve passes through the arcade of Fröhse, formed by a fibrous arch arising from the superficial head of the supinator muscle at its attachment to the lateral epicondyle. The posterior interosseous nerve travels between and innervates the superficial and deep heads of the supinator muscle to supply the wrist and finger extensor muscles.⁴⁹ The superficial sensory branch supplies sensation to the dorsolateral hand. It lies beneath the

KEY POINTS

- Slowly progressive radial neuropathy may occur with humeral fracture healing due to callus formation creating nerve entrapment.
- Posterior interosseous syndrome is a pure motor syndrome without associated sensory loss.
- Electrodiagnostic testing of the radial nerve requires nerve conduction studies and needle EMG.

brachioradialis muscle at the elbow and in the proximal forearm as it travels with the radial artery. In the distal third of the forearm, the nerve separates from the artery and travels superficially beneath the brachioradialis tendon. The nerve then travels between the brachioradialis and extensor carpi radialis longus tendons. Distally, the nerve pierces the overlying forearm fascia and divides into lateral and medial divisions and ultimately into dorsal digital nerves.⁵⁰

Clinical Presentation and Examination Findings

The most common presentation of radial neuropathy is that of acute compression at the level of the spiral groove, commonly known as Saturday night palsy. In this syndrome, patients compress the medial arm against a firm surface (eg, arm draped over a chair back) during prolonged sleep, deep sedation, or intoxication. They may awaken unable to extend the fingers or wrist. Numbness or paresthesia is present over the dorsolateral hand. Pain at the spiral groove is relatively uncommon. The symptoms typically resolve over 2 to 3 months.

Because of the close association between the radial nerve and the humerus, fractures of the humeral shaft are also a common cause of proximal radial neuropathies.^{51,52} Examination findings are dependent upon the level of the fracture. Acute presentations are readily identified in most cases, but delayed involvement may be overlooked. Slowly progressive radial neuropathy may occur with fracture healing due to callus formation creating nerve entrapment⁵³ or may be related to the surgical hardware used to repair the fracture itself.^{54,55}

Posterior interosseous syndrome is a pure motor syndrome without associated sensory loss. It is much less common than radial compression at the spiral groove and results from posterior interosseous nerve compression within the arcade of Fröhse related to repetitive supination, space-occupying lesions, and trauma. It presents with marked weakness of finger extension and a lesser degree of wrist extension weakness. Wrist extension may be relatively spared due to extensor carpi radialis longus innervation occurring proximal to the division of the common radial nerve (Figure 7-4). As the posterior interosseous nerve is a purely motor nerve, patients experience no sensory symptoms. Patients sometimes report vague discomfort over the dorsal forearm, worsened by activity involving supination of the arm. Pain with resisted supination of the forearm can be used as a provocative test, but its specificity and sensitivity are poorly defined.

Isolated lesions of the superficial radial sensory nerve may also occur. These may result from focal compression or trauma, as in cheiralgia paresthetica (compression of the superficial radial nerve at the wrist), or as a rare type of diabetic mono-neuropathy.⁵⁶⁻⁵⁸ Patients experience sensory loss, sometimes associated with uncomfortable paresthesia over the dorsolateral hand. Sensory testing of the affected region and the absence of associated weakness are essential in making the clinical diagnosis.

Electrodiagnostic Testing

Electrodiagnostic testing of the radial nerve requires nerve conduction studies and needle EMG. Typically, the radial motor response is recorded over the extensor indicis proprius following stimulation at the forearm, lateral elbow, and spiral groove. This

permits assessment of potential areas of entrapment, and special attention to CMAP amplitudes and dispersion is necessary. Stimulation at Erb point is helpful in localizing proximal humeral lesions, although costimulation of the brachial plexus may contaminate the recordings.⁵⁹ A superficial radial sensory response is valuable for differentiating between common radial and posterior interosseous nerve pathology. As with any mononeuropathy, nerve conduction studies of other upper extremity nerves may be needed to confirm isolated involvement of the radial nerve.

Needle EMG can assist in localization and prognosis. The triceps brachii, brachioradialis, and long head of extensor carpi radialis are spared with isolated posterior interosseous nerve pathology. Distally, the extensor digitorum communis and extensor indicis proprius muscles are sampled. Additional examination of nonradial muscles is helpful and often necessary in excluding brachial plexopathy or cervical radiculopathy as a cause of symptoms.

Ultrasonographic Testing

Neuromuscular ultrasonography may also be used in the diagnosis of radial neuropathies. Focal enlargement has been documented at sites of compression and can be helpful in localization. Radial nerve cross-sectional area is typically less than 10 mm² in the upper arm and antecubital fossa, with the superficial radial sensory nerve measuring only 1 mm² to 3 mm².^{60,61} The posterior interosseous nerve cross-sectional area measures approximately 2 mm².⁶² Interestingly, distal enlargement of the posterior interosseous nerve has been documented in cases of proximal radial nerve lesions, suggesting a double crush syndrome.^{63,64} Additional investi-

gation is needed to determine how finding these secondary lesions might impact prognosis and treatment. Although peripheral nerve trauma is not covered in this article, neuromuscular ultrasonography can be used to determine nerve continuity within hours or days of peripheral nerve trauma, expediting treatment decisions.

Treatment of Radial Neuropathies

The most common of the radial neuropathies, acute compression at the spiral groove (Saturday night palsy), typically resolves without additional intervention. These acute compressions cause focal demyelination that will resolve within 2 to 3 months. Avoidance of further compression is recommended during this time. If secondary axonal damage has occurred, recovery may take longer and be incomplete.

Aside from repair of traumatic nerve injuries and compression due to mass lesions, surgical interventions for radial neuropathy are uncommon, and no standard procedures exist for routine release of the radial nerve.

In patients with posterior interosseous syndrome, anti-inflammatory medications, rest, and corticosteroid injections are common first-line treatment recommendations.⁶³ Surgical release of the superficial head of the supinator muscle may be performed in patients for whom conservative management has failed, but this is rarely performed at the authors' institution. Although the surgical literature reports good response to treatment in most cases,^{65,66} patients involved in workers' compensation claims are known to have less favorable outcomes.⁶⁷ Given the lack of large prospective trials and potential for surgical complications,⁶⁸ posterior

KEY POINTS

- Needle EMG can assist in localization and prognosis of radial nerve entrapment. The triceps brachii, brachioradialis, and long head of extensor carpi radialis are spared with isolated posterior interosseous nerve pathology.
- The most common of the radial neuropathies, acute compression at the spiral groove (Saturday night palsy), typically resolves without additional intervention. These acute compressions cause focal demyelination that will resolve within 2 to 3 months.

KEY POINT

■ The common fibular (peroneal) nerve is the lateral division of the sciatic nerve and formed by the L4 to S1 spinal roots.

interosseous nerve release surgery is not routinely advised.

FIBULAR (PERONEAL) NERVE

Fibular neuropathy is the most frequent entrapment neuropathy of the lower extremity. An organized diagnostic approach is necessary in the evaluation of affected patients.

Basic Anatomy

The common fibular (peroneal) nerve is the lateral division of the sciatic nerve and formed by the L4 to S1 spinal roots. It branches from the sciatic nerve in the distal thigh and wraps around the biceps femoris tendon and fibular head on its course to the anterolateral leg.⁶⁹ The fibular nerve gives off a branch to

the short head of the biceps femoris muscle before separating from the sciatic nerve in the popliteal fossa. The nerve branches within the fossa to make a small contribution to the sural nerve, although considerable variability exists. The fibular tunnel, a potential site of nerve entrapment, is composed of the arch made by the peroneus longus, the soleus tendon, and the proximal fibula.⁶⁹

At the level of the fibular head, the common fibular nerve divides into deep and superficial divisions (**Figure 7-5** and **Figure 7-6**). The deep branch of the fibular nerve provides innervation to the muscles of the anterior compartment (tibialis anterior, extensor digitorum longus, extensor hallucis longus, extensor digitorum longus, extensor hallucis longus, peroneus tertius, extensor digitorum brevis, first dorsal interosseous, and dorsal digital cutaneous nerve).

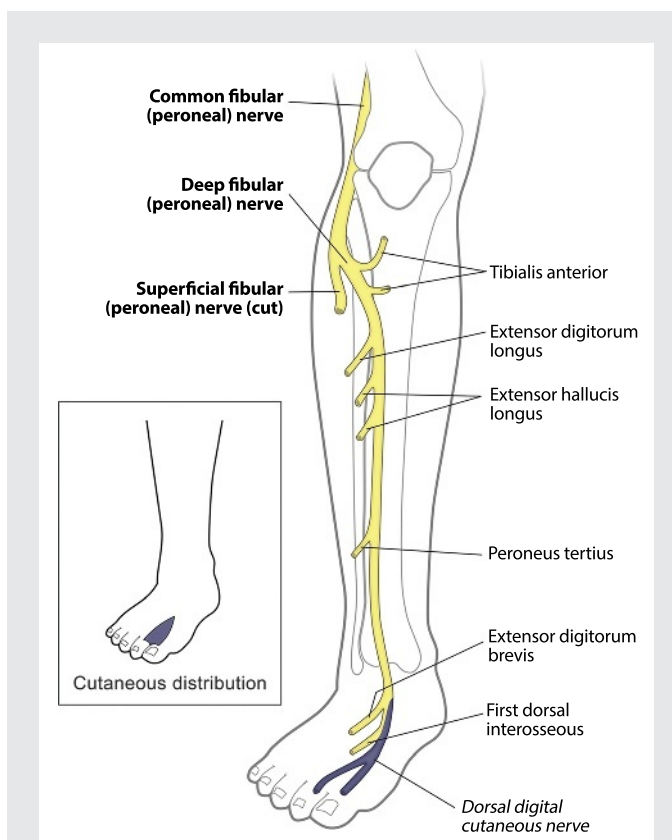


FIGURE 7-5 Common and deep fibular (peroneal) nerves. The nerves are labeled in bold, and sensory branches are labeled in italics.

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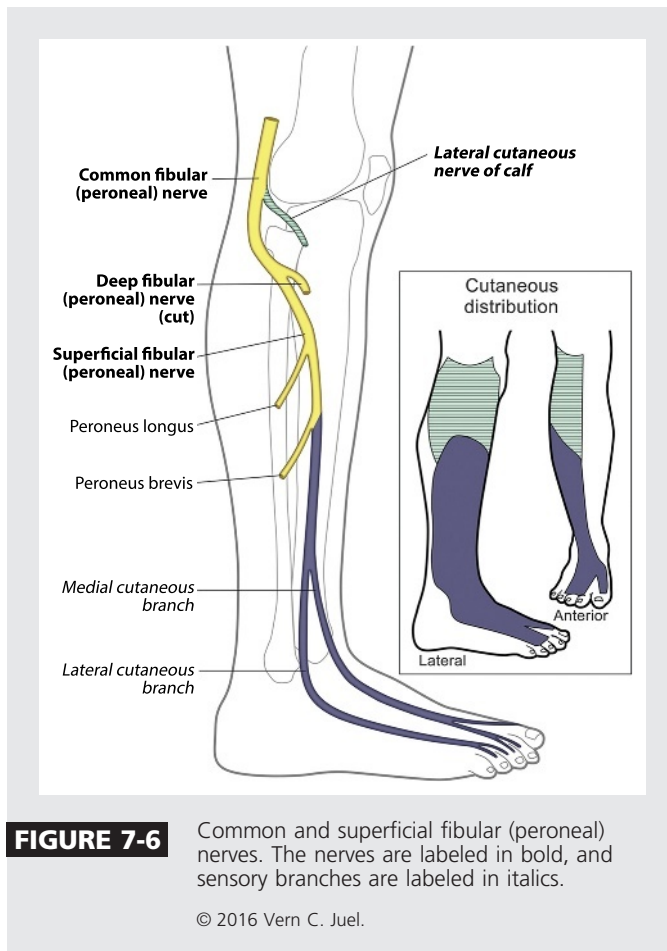


FIGURE 7-6 Common and superficial fibular (peroneal) nerves. The nerves are labeled in bold, and sensory branches are labeled in italics.

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KEY POINT

■ Compression of the common fibular nerve at the level of the fibular head is the most common lower extremity mononeuropathy. Common causes include weight loss, prolonged immobility, and frequent crossing of the legs.

longus, peroneus tertius, extensor digitorum brevis) and sensation to the dorsal web space between the first and second toes. The superficial fibular nerve innervates the lateral compartment muscles (peroneus longus and peroneus brevis) and supplies sensation to the distal lateral leg and dorsal foot.⁷⁰

Clinical Presentation and Examination Findings

Compression of the common fibular nerve at the level of the fibular head is the most common lower extremity mononeuropathy.⁷¹ Common causes include weight loss, prolonged immobility, and frequent crossing of the legs. Other etiologies include prolonged squatting, knee dislocation, ankle sprains, intraneural ganglion cysts, fibu-

lar neck exostosis, and acute compression after wearing tight-fitting pants (ie, skinny jeans).⁷²⁻⁷⁴ Patients with common fibular neuropathy typically present with difficulty walking. While some patients describe overt footdrop, many will report a “toe dragging,” “foot slapping,” or frequent tripping. Paresthesia is sometimes noted in the lower lateral leg and dorsal foot but is often overlooked in the setting of significant weakness. Pain may also be present at the site of compression.

Detailed physical examination may reveal a Tinel sign over the fibular nerve near the knee. Observation may demonstrate frank footdrop when walking or steppage gait. In milder cases, the gait may appear unaffected, with weakness noted only when the patient is asked to walk on his or her heels. Motor

KEY POINTS

- Any weakness of ankle inversion, toe flexion, or hip abduction suggests an L5 radiculopathy as opposed to an isolated fibular neuropathy.
- Superficial fibular neuropathies spare sensation in the first dorsal web space, as the deep fibular nerve innervates this sensory field.

testing will reveal weakness in the ankle dorsiflexor, evertor, and toe extensor muscles. Sensory loss can be variable but typically spares the dorsal fifth toe. Any weakness of ankle inversion, toe flexion, or hip abduction suggests an L5 radiculopathy as opposed to an isolated fibular neuropathy.

Cases of isolated deep fibular neuropathy are less common. Etiologies include trauma, compression, and ganglion cysts.⁷⁵ Involvement spares the ankle evertors and sensation of the lateral leg and foot. Sensory loss of the web space between the first and second toes is present. Superficial fibular neuropathy is also relatively rare. Depending upon the level of involvement, it can present with sensorimotor or isolated sensory symptoms. Weakness of ankle eversion is detected in more proximal disease, while loss of sensation over the lateral leg and dorsolateral foot can occur with either proximal or distal lesions. The web space between the first and second toes will be spared any loss of sensation, as the deep fibular nerve supplies this area.

Electrodiagnostic Testing

Motor nerve conduction studies of the fibular nerve are performed with the recording electrode placed over the extensor digitorum brevis with stimulation at the anterior ankle, fibular head, and popliteal fossa. If the motor response is absent, the recording electrode can be placed over the tibialis anterior with stimulation at the fibular head and popliteal fossa. The superficial fibular sensory response can also be performed to assist in localization.

Needle EMG is necessary to assess severity and assist in prognosis. A minimum examination consists of the tibialis anterior, peroneus longus, and short head of the biceps femoris.

Other L4 to L5 and sciatic muscles should be sampled to differentiate isolated fibular mononeuropathy from sciatic neuropathy, lumbosacral plexopathy, and lumbar radiculopathy. For example, abnormalities on needle EMG of the tibialis posterior can assist in differentiating L5 radiculopathy from fibular neuropathy.

Ultrasonographic Testing

On neuromuscular ultrasonography, increased cross-sectional area is present at sites of compression; early studies suggest that larger nerve cross-sectional area is associated with axonal loss.⁷⁶ The nerve is usually smaller than 12 mm² at the level of the fibular head.⁶⁰ Neuromuscular ultrasonography in fibular neuropathy at the fibular head may reveal anatomic causes, particularly in patients without risk factors for nerve compression. In one study, neuromuscular ultrasonography demonstrated compressive intraneural ganglion cysts at the fibular head in 18% of patients.⁷² A 2015 analysis found abnormal biceps femoris anatomy, ganglion cysts, and lipomas in a series of only 21 patients with footdrop due to common fibular neuropathy.⁷⁷ This type of information cannot be obtained from electrodiagnostic testing alone and is essential in guiding appropriate treatment. **Case 7-3** describes how electrodiagnostic studies and neuromuscular ultrasonography can be used together in clarifying complicated cases of fibular nerve entrapment.

Fibular Neuropathy Treatment

Treatment of fibular neuropathy is guided by etiology. Surgical repair is often necessary after traumatic injury. Compressive intraneural ganglion cysts must be excised to prevent recurrence and further nerve injury. For unremarkable cases of compression

Case 7-3

A 79-year-old man with lung cancer noted left foot weakness after entering a rehabilitation program following a 2-month hospital stay. He was persistently tripping over his left foot, and his wife stated that his walking was “too loud.” He denied any trauma to the leg and had not noticed any pain but did describe an intermittent tingling sensation over his dorsal left foot. He had no low back pain.

Examination was remarkable for cachexia and mild diffuse weakness of the bilateral upper and lower extremities. He also had superimposed weakness with only movement against gravity for left ankle dorsiflexion, ankle eversion, and extension of all toes. Bilateral lower extremity reflexes were absent, and he had reduced sensation in a stocking-glove distribution bilaterally.

Electrodiagnostic testing demonstrated evidence of a widespread sensorimotor axonal polyneuropathy. No fibular motor response was present with recording over the extensor digitorum brevis on either side. Bilateral superficial fibular sensory responses were absent bilaterally, as were sural sensory responses. Needle EMG exhibited denervation and reinnervation in the bilateral tibialis anterior and medial gastrocnemius, but the changes were most prominent in the left tibialis anterior. A fibular motor response recording over the left tibialis anterior demonstrated a drop in amplitude and slowing across the fibular head. Ultrasound of the left fibular nerve at this site showed marked focal enlargement of the nerve. The patient was diagnosed with left fibular neuropathy at the fibular head superimposed on a background of sensorimotor polyneuropathy.

Comment. The patient in this case had a preexisting polyneuropathy complicating assessment of superimposed mononeuropathies. Fibular neuropathy at the fibular head was strongly suspected given the patient’s presentation and risk factors of immobilization and apparent weight loss. The use of needle EMG, alternate recording sites for fibular motor responses, and ultrasonography permitted an accurate diagnosis.

at the fibular head with no or minor changes on electrodiagnostic testing, time and avoidance or correction of risk factors may suffice. For more severe cases involving axonal loss, surgery is indicated. This is complicated by lack of a standard approach and the existence of differing sites of potential entrapment. Most patients have improvement in function and pain following intervention, but the published studies are small and lack standardization.^{78–80} Microsurgical decompression, with dissection of only the fibrous band between the superficial head of the peroneus longus and the soleus, may be an equally effective approach as compared to a more invasive surgical decompression.⁸¹

OTHER LOWER EXTREMITY ENTRAPMENT NEUROPATHIES

Any peripheral nerve can be affected by entrapment. A few of the lesser known peripheral nerve entrapment syndromes are reviewed here.

Tarsal Tunnel Syndrome

The tibial nerve derives from the sciatic nerve and receives contributions from the L4 to S1 nerve roots. The tibial nerve branches from the sciatic nerve at the level of the popliteal fossa and travels distally beneath the arch of the soleus. Its branches provide innervation to muscles of ankle plantar flexion and to the sural nerve. At the level of the ankle, it passes through the tarsal tunnel before dividing into its terminal

KEY POINTS

- The tarsal tunnel represents the space between the flexor retinaculum and medial malleolus, containing the tibial nerve, posterior tibial artery, posterior tibial vein, flexor hallucis longus, tibialis posterior, and flexor digitorum longus.
- Idiopathic tarsal tunnel syndrome is rare. Potential etiologies of tibial neuropathy at the ankle include trauma, masses, accessory muscles, bony malformations, vascular anomalies, and iatrogenic causes.
- The most common location for Morton neuroma is the third or fourth interdigital nerves.

branches, the medial and lateral plantar nerves. The tarsal tunnel represents the space between the flexor retinaculum and medial malleolus, containing the tibial nerve, posterior tibial artery, posterior tibial vein, flexor hallucis longus, tibialis posterior, and flexor digitorum longus.⁸² The medial and lateral plantar nerves supply cutaneous sensation over the plantar surface of the foot, along with innervation of intrinsic foot muscles. The tarsal tunnel is a potential site of tibial nerve entrapment.

Tarsal tunnel syndrome, also known as tibial neuropathy at the ankle, is a controversial topic in neurology. In most cases, the diagnosis is established by clinical presentation, although diagnostic criteria are ill defined. Symptoms consist of numbness and painful paresthesia involving the heel, medial ankle, and the sole of the foot, although some definitions include ankle pain with weight bearing.⁸² A Tinel sign may be present over the tibial nerve at the ankle. In the authors' experience, idiopathic tarsal tunnel syndrome is rare. Potential etiologies of tibial neuropathy at the ankle include trauma, masses,⁸³ accessory muscles,^{84,85} bony malformations,⁸⁶ vascular anomalies,⁸⁷ and iatrogenic causes.⁸⁸

Electrodiagnostic testing can be helpful in excluding other causes of tarsal tunnel syndrome symptoms, but its exact utility in diagnosis is unknown.⁸⁹ No single set of characteristic electrodiagnostic findings exists, as it is often stated that plantar nerve responses are frequently unobtainable in individuals who are asymptomatic, particularly with advanced age. A systematic review of the literature from 1965 to 2002 yielded only four studies meeting review criteria, and these met only Class III level of evidence. The review concluded that nerve conduction studies might be helpful in

diagnosis.⁸⁹ Neuromuscular ultrasonography may aid in assessment of structural causes of tarsal tunnel syndrome symptoms, and some reports⁹⁰ establish cross-sectional area cutoff values to be used in the diagnosis of idiopathic tarsal tunnel syndrome.

In the absence of a structural cause of compression, the exact value of surgical decompression is unknown. A 2015 review of 31 patients undergoing surgery for tarsal tunnel syndrome demonstrated good outcomes in 71% but found that only 6 of 11 patients with idiopathic tarsal tunnel syndrome improved. The best surgical outcomes occurred in those with a determined cause of tarsal tunnel syndrome, including trauma and masses.⁹¹ This should be considered when designing a treatment plan.

Morton Neuroma

Although a separate entity from tarsal tunnel syndrome, Morton neuroma is a distal tibial mononeuropathy presenting with shooting pains into the toes and other associated paresthesia of the forefoot provoked by weight bearing. It results from chronic repetitive mechanical trauma of a plantar digital nerve and is considered to represent a degenerative perineural fibrous enlargement of the nerve, as opposed to a true neuroma. The most common location is the third or fourth interdigital nerves. Diagnosis is through clinical examination and, more recently, ultrasound imaging.⁹²⁻⁹⁴

Meralgia Paresthetica

Meralgia paresthetica is the commonly used term describing pathology of the lateral femoral cutaneous nerve, also known as the lateral cutaneous nerve of the thigh. The lateral femoral cutaneous nerve arises from the L1 to L3 nerve roots and lumbar plexus and is a pure sensory nerve. Its path

over the iliacus and under the inguinal ligament make it prone to injury and compression.⁹⁵ The lateral femoral cutaneous nerve may have a variable course between individuals, with five distinct patterns identified, making diagnostic testing and treatment challenging.⁹⁶

Meralgia paresthetica is associated with obesity, diabetes mellitus, and wearing tight clothing.⁹⁵ Other causes include trauma and surgical injury. Typical symptoms of meralgia paresthetica include lateral or anterolateral thigh paresthesia and sensory loss. Pain can be severe and described as stabbing, shooting, or burning. Physical examination should reveal sensory loss in the distribution of the lateral femoral cutaneous nerve without associated findings suggestive of lumbar radiculopathy or lumbosacral plexopathy. Electrodiagnostic testing is useful not only in diagnosis but also for exclusion of other causes. Neuromuscular ultrasonography can be helpful in guiding electrode placement for testing the lateral femoral cutaneous nerve and for identifying areas of focal nerve enlargement.^{97,98}

Treatment of meralgia paresthetica is most often conservative, including behavioral modifications (eg, wearing looser clothing), symptomatic relief of painful paresthesia with nonsteroidal anti-inflammatory drugs, and nerve blocks.⁹⁵ Surgical exploration and treatment should be considered a last resort in the absence of a mass lesion.

CONCLUSION

Entrapment neuropathies are a common indication for outpatient neurologic consultation. While some are quite common and easily diagnosed, such as carpal tunnel syndrome and ulnar neuropathy at the elbow, others may result from more obscure etiolo-

gies and require special investigation. Entrapment neuropathies may present in the context of other peripheral neuropathic processes and be superimposed on a background of polyneuropathy or occur in parallel with other mononeuropathies or radiculopathies. Clinical assessment and localization may be confirmed and refined with electrodiagnostic studies and neuromuscular ultrasonography. Electrodiagnostic studies provide physiologic information regarding peripheral nerve and muscle function, and neuromuscular ultrasonography provides complementary anatomic information. Early diagnosis and expeditious treatment seek to minimize morbidity and maximize function to achieve optimal patient outcomes.

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KEY POINT

- Treatment of meralgia paresthetica is most often conservative, including behavioral modifications (eg, wearing looser clothing), symptomatic relief of painful paresthesia with nonsteroidal anti-inflammatory drugs, and nerve blocks.

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