# Entrapment Syndromes

# 56

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## 56.1 Carpal Tunnel Syndrome

### 56.1.1 Synonyms

Median neuropathy at the wrist

### 56.1.2 ICD-10 Codes

G56.00

### 56.1.3 Description

Carpal tunnel syndrome (CTS) is a median nerve compression neuropathy in the carpal tunnel. It is the most common entrapment neuropathy of the upper extremity.

***Anatomy*** The median nerve arises from the C6–T1 nerve roots. Axons traverse all three trunks and then the medial and lateral cords. Sensation to the palmar lateral surface of the hand and the palmar thumb, index, middle, and lateral half of the ring finger is provided by the lateral cord. Axons from the medial cord provide innervation for median motor control of the hand with no sensory function; this includes the lumbricals 1 and 2, opponens pollicis, abductor pollicis brevis, and the flexor pollicis brevis (LOAF muscles). The carpal tunnel is bounded by the carpal bones and the transverse carpal ligament. The tunnel contains the four flexor digitorum superficialis tendons, the four flexor digitorum tendons, the flexor pollicis longus tendon, and the median nerve [2] (Fig. 56.1).

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*Carpal tunnel syndrome (CTS)* can be an idiopathic process or a result of increased volume and pressure within the tunnel caused by specific disorders such as repetitive motion injuries, thyroid disease, congestive heart failure, renal failure, or masses including hematomas or tumors [1, 2]. In the last trimester of pregnancy, total body fluid volume may increase leading to increased carpal tunnel pressures. CTS associated with pregnancy may subside after delivery [3]. Other conditions which can cause tunnel compromise include fracture, arthritis, and rheumatoid tenosynovitis. Underlying conditions which can cause generalized peripheral neuropathies make individuals more prone to focal neuropathies like CTS. Differential diagnosis of CTS is outlined in Table 56.1.

*Double crush syndrome* occurs when radiculopathies, brachial plexopathies, or thoracic outlet syndrome occur with CTS. C6 and C7 radiculopathies must be considered in the differential diagnosis when considering CTS due to overlap of symptoms [1, 2].

### 56.1.4 Clinical Presentation

Classic CTS complaints include numbness and tingling of the thumb, index, and middle fingers. At times, due to referred pain, symptoms can extend into the ring and small fingers. Nocturnal pain and numbness are common. Symptoms can radiate proximally into the forearm as well. People may attest to weakness in grip and poor dexterity. Holding a phone, grasping a steering wheel, or repetitive motions can engender symptoms. In general, any activity leading to prolonged wrist flexion can generate numbness, tingling, and pain in patients with CTS [2].

### 56.1.5 Physical Examination

If the median nerve is only irritated (no axonal loss), the sensory examination may be normal. If sufficient axon loss has occurred, then sensation will be decreased in the median nerve distribution including the thumb, index finger, middle



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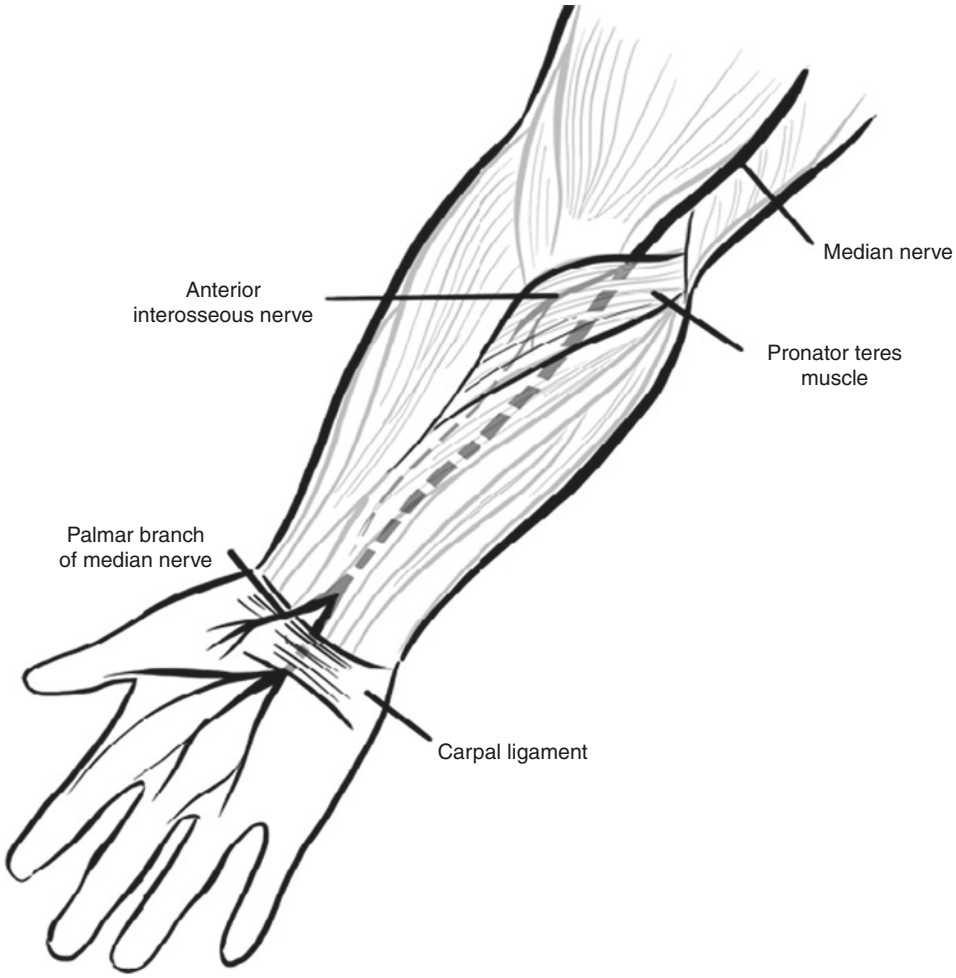


**Fig. 56.1** Course of median nerve in the forearm and carpal tunnel

**Table 56.1** Differential diagnosis of carpal tunnel syndrome

| | |
|---|---|
| Brachial plexopathy | Anterior interosseous neuropathy |
| Cervical radiculopathy | Musculoskeletal disorders of hand and wrist |
| Generalized peripheral neuropathy | Pronator teres syndrome |

finger, and the radially half of the ring finger. Motor function may be spared in mild to moderate cases. In more advanced CTS, thumb abduction weakness occurs, and atrophy of the abductor pollicis brevis may be seen.

As with all nerve entrapment cases, the examination including general inspection, palpation, range of motion, strength testing, deep tendon reflexes, and sensation of the upper limbs must take into account other possible causes for the presenting symptoms. *Tinel's sign* (mechanical tapping of the median nerve at the carpal tunnel) and *Phalen's test* (60-second maintained wrist flexion position eliciting median distribution paresthesias) are both provocative tests for CTS. Tinel's has about 65% sensitivity, and Phalen's test has about 85% sensitivity, and both have approximately 90% specificity for median nerve entrapment at the wrist [2, 24] (Fig. 56.2).

### 56.1.6 Diagnostic Workup

*NCS and EMG* are an extension of the physical examination. Sensory studies are more sensitive than motor studies. Median sensory distal latencies will be prolonged and should be compared to other nerves in the hand, either radial or ulnar. Antidromic studies will provide larger amplitudes. Axonal loss or conduction block will cause motor and sensory amplitude drop. Conduction studies of the median nerve through the forearm will help differentiate CTS from a more generalized peripheral neuropathy. EMG should sample C5–T1 selected muscles, the APB, and the cervical paraspinals [1].

*Ultrasound* is useful to assess the median nerve at the carpal tunnel proximal border (Fig. 56.3). At this site, the nerve will appear swollen (Fig. 56.4) and hypoechoic with a large cross-sectional area in comparison to the nerve more proximally [1]. Greater than 10mm$^2$ at the carpal tunnel inlet is diagnostic for CTS [22].

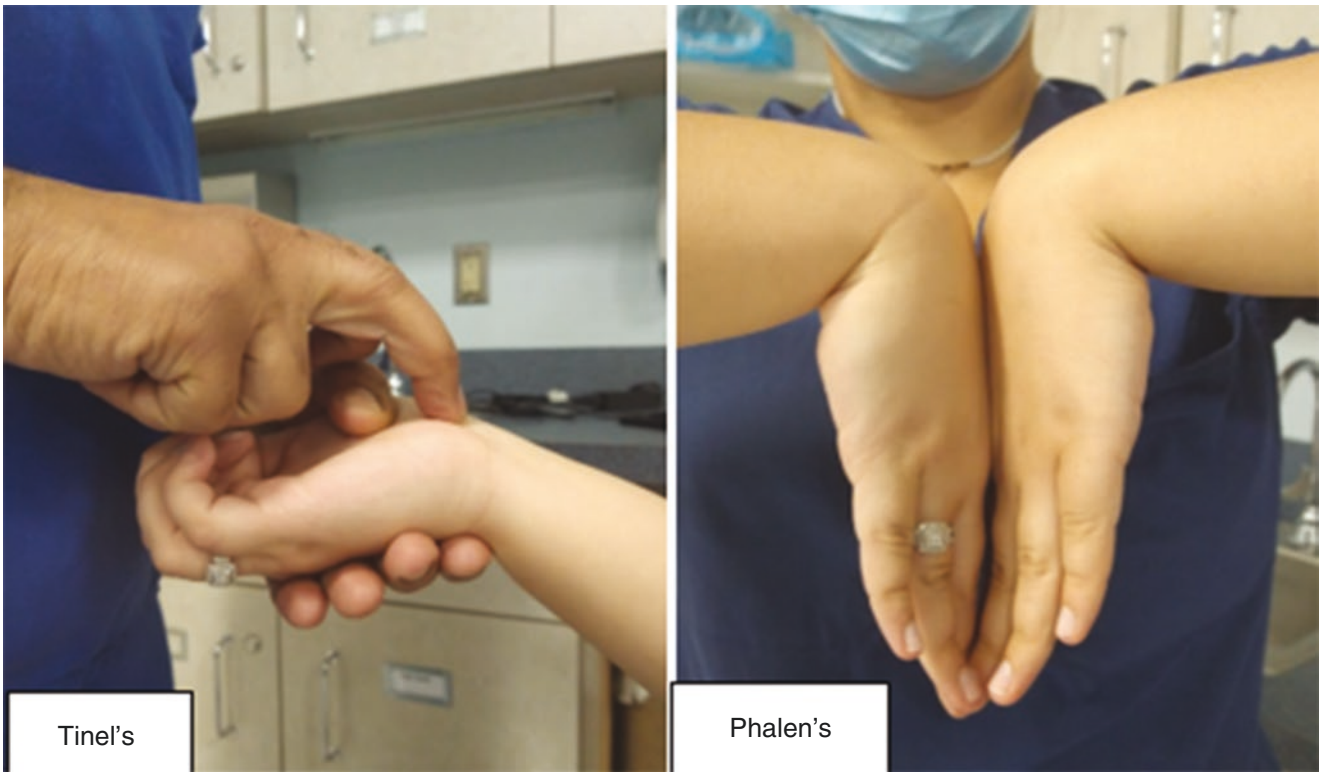


**Fig. 56.2** Tinel's test (left) and Phalen's test (right) are part of CTS examination. (Courtesy of S. Ali Mostoufi, MD, Boston Regenerative Medicine)

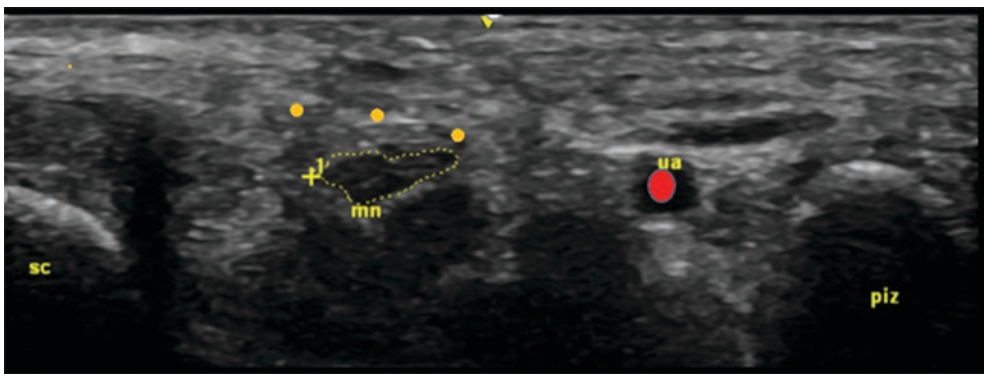


**Fig. 56.3** Median nerve at carpal tunnel inlet with pisiform and scaphoid bony landmarks and the ulnar artery (UA). Transverse carpal ligament is marked as yellow dots superficial to MN. Measurement of the median nerve can be done at this site for diagnostic purposes. (Figure courtesy of S. Ali Mostoufi, MD)

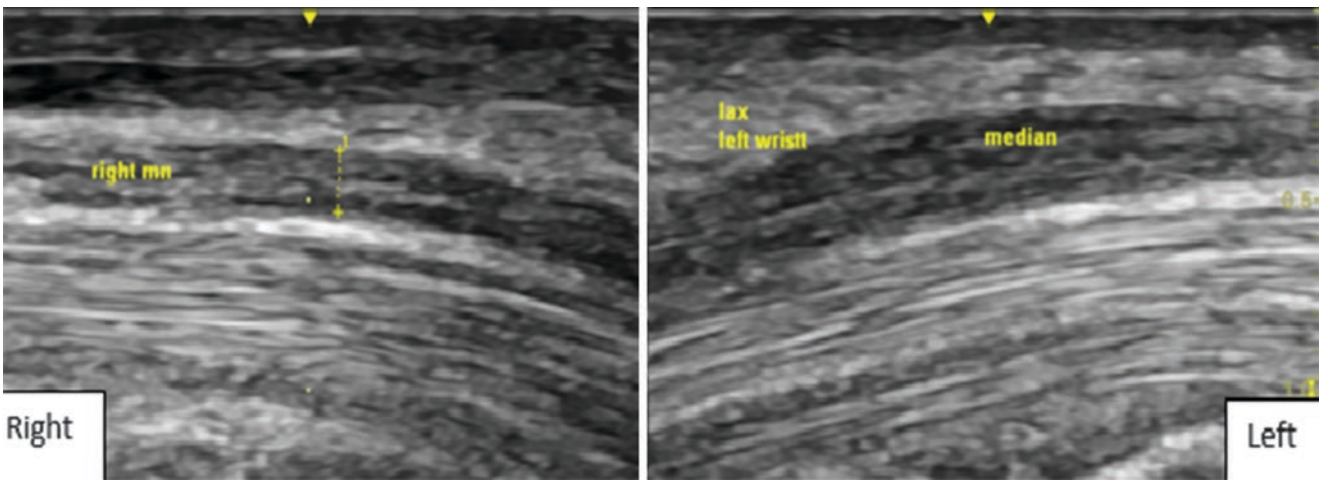


**Fig. 56.4** Median nerve in long axis. Side-to-side comparison demonstrating enlargement of the left median nerve. (Courtesy of S. Ali Mostoufi, MD, Boston Regenerative Medicine)

### 56.1.7 Treatment

Conservative treatment is indicated in mild cases with no weakness, atrophy, or denervation on EMG. Orthotics: carpal tunnel splints usually worn at night

- Medications such as NSAIDs and diuretics
- Ergonomic modifications
- Image-guided steroid injections may provide some relief but may not be a permanent solution (Fig. 56.5).
- Hydrodissection has been used with some success in mild to moderate CTS. For more details, please see Chap. 32.
- Platelet-rich plasma injection into the carpal tunnel has been used with some success in mild to moderate CTS. For more details, please see Chap. 32.
- Ultrasound-guided percutaneous release of the carpal ligament has proved successful in mild to moderate CTS (Fig. 56.6). For more details, please see Chap. 32.

Surgical release is indicated for persistent numbness and pain despite conservative treatment. In CTS with severe muscle atrophy, surgical release does not have a high rate of success due to the extent of axon damage [2]. Surgical care is discussed in more detail in Chap. 37.

## 56.2 Anterior Interosseous Nerve Entrapment

### 56.2.1 Synonyms

- Anterior interosseous neuropathy
- Anterior interosseous syndrome
- Pure median motor neuropathy of the forearm

### 56.2.2 ICD-10 Codes

G56.10

### 56.2.3 Description

*Anterior interosseous nerve (AIN) entrapment* is a pure median motor neuropathy in the forearm. It is a less common median neuropathy and can be mistaken for brachial plexopathy or other proximal median neuropathies. AIN results in weakness in thumb and finger pinch strength. Trauma to the volar forearm is one possible cause. True entrapment of the AIN is rare [1, 2]. Differential diagnosis of AIN is outlined in Table 56.2.

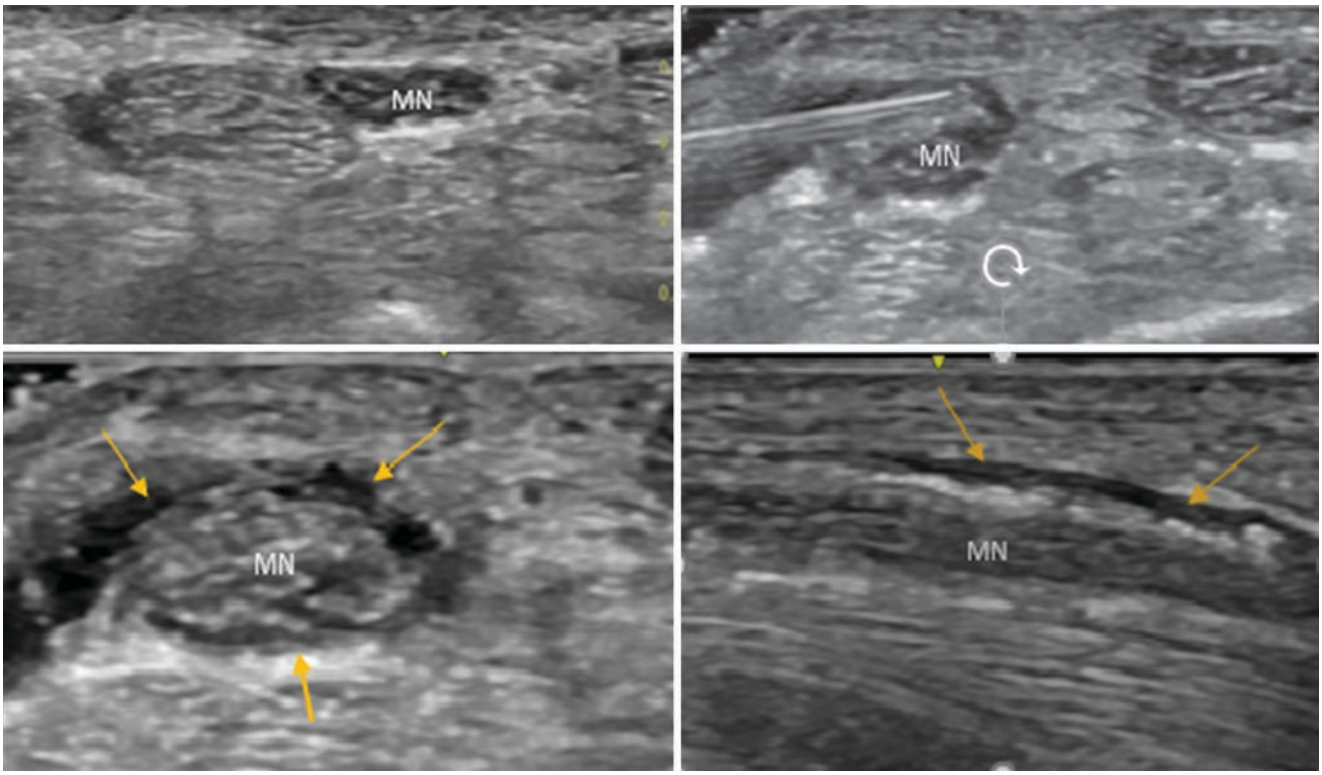


**Fig. 56.5** CTS injection with US. Needle tip is placed between the transcarpal ligament and the median nerve. The correct injection flow should be confirmed in both short- and long-axis images. (Courtesy of S. Ali Mostoufi, MD, Boston Regenerative Medicine)

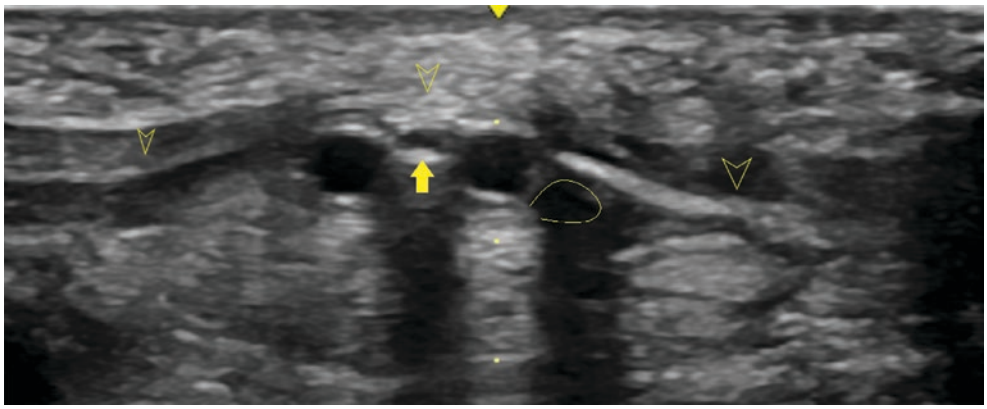

**Fig. 56.6** Percutaneous US-guided CTS release. Triangles point to the transverse carpal ligament, and the arrows demonstrate the blade engaged in the ligament, The median nerve (circle) is displaced laterally and protected by an air chamber to avoid injury. (Courtesy of S. Ali Mostoufi, Boston Regenerative Medicine)

**Table 56.2** Differential diagnosis of anterior interosseous syndrome

| | |
|---|---|
| Brachial plexopathy | Multifocal motor neuropathy |
| Cervical radiculopathy | Muscle or tendon injury in the forearm |
| Carpal tunnel syndrome | Pronator syndrome |

***Anatomy*** Refer to the "Carpal Tunnel Syndrome" section for full median nerve anatomy. The AIN branches in the proximal forearm as a pure median motor nerve to innervate the flexor digitorum profundus I and II, the flexor pollicis longus, and pronator quadratus.

### 56.2.4 Clinical Presentation

AIN entrapment presents with severe pain in the forearm with no sensory loss. Weakness is evident in forming the letter "O" with the thumb, index, and middle fingers, due to denervation of the muscles of distal flexion of these digits [1].

### 56.2.5 Physical Examination

Inspection of the forearm may or may not reveal volar forearm atrophy. The patient may feel pain on the volar aspect of the proximal third of the forearm. Sensory should be intact for the upper limb. Weakness should be evident as noted above in thumb, index, and middle finger in flexion. Holding onto a single sheet of paper with just the fingertips can be challenging for these patients [4]. Upper limb reflexes should be normal. A thorough examination of the cervical neck and upper limbs is needed to rule out other causes.

### 56.2.6 Diagnostic Workup

***Plain films*** Radiographs of the supracondylar humerus are indicated in AIN palsy, since 20% of fractures in this region will result in nerve palsy including AIN [23].

*Electrodiagnostic studies* are essential. Sensory and motor conduction studies of the median and ulnar nerves are needed to rule out other neuropathies and brachial plexopathy. EMG of the flexor digitorum I and II, flexor pollicis longus, and pronator quadratus are required and will show evidence of abnormalities. EMG of other muscles will rule in or out other nerve involvement.

*Diagnostic ultrasound* scans of the forearm may reveal nerve injury as swelling and increased heterogenicity. It can also show causes of entrapment including cysts, tumors, ischemia, hematomas, or fibrous bands [1].

### 56.2.7 Treatment

Conservative care involves stretching and strengthening of the forearm musculature. Surgical intervention may be needed for the release of fibrous bands or decompression of scar tissue or masses. Most patients improve without surgical intervention; recovery with conservative treatment may take up to 1 year after onset [4].

## 56.3 Pronator Teres Syndrome

### 56.3.1 Synonyms

- Median neuropathy at the pronator teres muscle
- Proximal median motor and sensory neuropathy of the forearm

### 56.3.2 ICD-10 Codes

G56.10

### 56.3.3 Description

*Pronator teres syndrome* is a median nerve entrapment at the pronator teres muscle affecting both motor and sensory function. The nerve is compressed at a fibrous arch connecting the two heads of the pronator teres muscle (Fig. 56.1). This

**Table 56.3** Differential diagnosis of pronator syndrome

| | |
|---|---|
| Anterior interosseous nerve syndrome | Generalized peripheral neuropathy |
| Brachial plexopathy | Forearm musculotendinous injury |
| Cervical radiculopathy | Multifocal motor neuropathy |

results in weakness in all median innervated muscles of the forearm and hand distal to the pronator teres and numbness in the median nerve distribution. This is an uncommon neuropathy. The entrapment may be caused by activities requiring a high frequency of pronation. It may result from direct trauma or the development of a fibrous band [1, 2, 4]. Full median nerve anatomy is discussed in the CTS section. Differential diagnosis of pronator syndrome is outlined in Table 56.3.

### 56.3.4 Clinical Presentation

Pain may be felt in the volar proximal forearm. Weakness will be apparent in flexion of the thumb, flexion of the index and middle fingers, flexion of the wrist, and abduction and opposition of the thumb. Numbness will involve the lateral palmar hand, the palmar thumb, and the index and middle fingers.

### 56.3.5 Physical Examination

Examination may find atrophy of the volar forearm and pain upon palpation in the proximal volar forearm. Percussion over the pronator teres may cause radiating dysesthesias to the lateral palmar hand. Symptoms may be replicated with resisted pronation.

Weakness as noted above is apparent in wrist flexion; thumb, index, and middle fingers; and abduction and opposition of the thumb. Sensory will be diminished in the lateral palmar hand; the thumb; and the index, middle, and lateral half of the ring finger.

Deep tendon reflexes are normal. A thorough examination of the arm and cervical region is necessary to rule out other pathology.

### 56.3.6 Diagnostic Workup

***EMG/NCS*** Electrodiagnostic studies are essential. Median nerve abnormalities will be present distal to the pronator teres. Axonal injury will result in low amplitudes for median motor and sensory function. EMG abnormalities will involve all median innervated muscles distal to the pronator teres. The median nerve function must be compared to other nerves to isolate abnormalities, in order to rule out other conditions.

*Diagnostic Ultrasound* of the forearm may help identify nerve swelling at the injury site or the presence of a fibrous band, mass, or hematoma [1].

### 56.3.7 Treatment

Conservative care includes stretching, soft tissue release techniques, and strengthening. Nonsurgical management with rest, nonsteroidal anti-inflammatory medications, and avoidance of troublesome activities are highly effective [4]. Corticosteroids can also be beneficial. Surgical release may be required to reduce scar tissue or fibrous bands, if conservative treatment fails, or if symptoms are caused by a mass [4].

## 56.4 Posterior Interosseous Nerve Syndrome

### 56.4.1 Synonyms

- Posterior interosseous neuropathy
- PIN syndrome

### 56.4.2 ICD-10 Codes

G56.80–G56.82

### 56.4.3 Descriptions

*Posterior interosseous nerve (PIN) syndrome* is a neuropathy at the forearm of the PIN. It occurs most frequently due to entrapment at the arcade of Frohse. Other potential etiologies, including the tendinous edge of the bordering tissues, adjacent muscles and vessels, ganglion cysts, tumor and other mass lesions, synovial pathologies, radial head fracture and Monteggia fracture-dislocation, and repetitive pronation/supination, may also cause compression of the PIN [1]. This is a rare diagnosis and can be difficult to distinguish from radial neuropathy occurring at the spiral groove [1, 5]. The differential diagnosis of PIN is listed in Table 56.4.

***Anatomy*** The radial nerve is essentially derived from the C5 and T1 roots; radial fibers then travel through the posterior division of all three trunks and posterior cords of the brachial plexus. The radial nerve travels through the arm,

**Table 56.4** Differential diagnosis of posterior interosseous neuropathy

| | |
|---|---|
| Brachial plexopathy | Radial nerve palsy at spiral groove |
| Cervical radiculopathy | Radial tunnel syndrome |
| Lesions in the contralateral motor cortex | Wartenberg syndrome |

wraps around the humerus in the spiral groove, descends into the lateral elbow, and divides into the superficial and deep branches proximal to the arcade of Frohse. The posterior interosseous nerve is the deep motor branch of the radial nerve. The radial nerve, after coursing anterior to the lateral epicondyle of the humerus, bifurcates into a superficial sensory and the PIN at the level of the radiocapitellar joint. After travelling through the radial tunnel, the PIN passes between the superficial and deep heads of the supinator muscle. After exiting the supinator canal, the PIN supplies the extensor compartment muscles of the forearm [1, 2, 5].

### 56.4.4 Clinical Presentation

Posterior interosseous neuropathy presents with paralysis, which can be partial or complete, involving some or all the posterior interosseous nerve-innervated muscle. In most instances, it is relatively acute over the course of several days to weeks. Pain in the elbow or proximal lateral extensor forearm is an inconsistent feature; when present, it is typically the initial symptom and short-lived, lasting for no more than 3 days.

### 56.4.5 Physical Examination

In posterior interosseous neuropathy, patients usually have weakness affecting the extensor carpi ulnaris, extensor digitorum communis, extensor digiti minimi, abductor pollicis longus, and extensor pollicis brevis. Typically, the extensor carpi radialis brevis and longus, supinator, brachioradialis, and triceps are not affected as they are innervated before the radial nerve divides.

Patients with PIN neuropathy have the characteristic presentation of finger drop with radial wrist deviation when attempting to extend the wrist and fingers, due to paralysis of the extensor carpi ulnaris and the unopposed contraction of the extensor carpi radialis longus and brevis. Patients should be able to weakly extend the wrist. Sensation should be intact, as the PIN has no sensory branches. Depending on the severity of injury, there may be a positive Tinel sign at the site of injury [1, 2, 5, 6]. As with all neuropathies, a full MSK/neuromuscular and cervical spine exam is required to rule out other etiologies.

### 56.4.6 Diagnostic Workup

*Imaging* studies can be useful if patients have bony structure abnormalities or mass lesions. *Electrodiagnostic studies* are key to this diagnosis. The study may show denervation signs in the muscles innervated by the posterior interosseous nerve, with sparing of muscles innervated by the radial nerve, including triceps, anconeus, brachioradialis, and extensor carpi radialis longus and brevis. Patients will have normal sensory nerve action potential of the superficial radial nerve [5].

Ultrasound may provide a convenient and complementary tool for diagnosis. Diagnostic neuromuscular ultrasound may demonstrate hypoechoic swelling of the PIN, while also providing direct visualization of the PIN throughout the entire supinator canal and adjacent anatomic structures. This enables the examiner to identify various potential causative lesions, as well as see possible secondary denervation atrophy of the affected muscles [6].

### 56.4.7 Treatment

Most patients without open trauma or mass lesions improve with conservative treatment, which can include splinting, NSAIDs, physical and occupational therapy, and activity modification [2]. Corticosteroid injections can also be beneficial [5]. For patients who fail 6 months of conservative treatment, surgical release of all five compression sites is recommended [5]. Rehabilitation should start soon after decompression with an early active range of motion. The patient may continue to see improvement for months after surgery [7].

## 56.5 Radial Tunnel Syndrome

### 56.5.1 Synonyms

- Radial neuropathy at the elbow
- Resistant tennis elbow

### 56.5.2 ICD-10 Codes

G56.30–G56.32

### 56.5.3 Descriptions

Radial tunnel syndrome is a radial nerve neuropathy at the elbow. It occurs as a result of chronic sporadic compression on the radial nerve by the radial head as it passes

**Table 56.5** Differential diagnosis of radial tunnel syndrome

| | |
|---|---|
| Biceps tendinopathy | Lesions in the contralateral motor cortex |
| Brachial plexopathy | Muscle tear of the extensor carpi radialis brevis |
| Cervical radiculopathy | Osteoarthritis of the radiocapitellar joint |
| Lateral epicondylitis | Posterior interosseous neuropathy |

under the supinator. This is a debatable diagnosis, as patients do not have any signs or symptoms other than pain, and they have normal electrodiagnostic studies [1, 5, 8]. Table 56.5 details the differential diagnosis of radial tunnel syndrome.

***Anatomy*** For complete anatomical overview of the radial nerve, please see the section "Posterior Interosseous Nerve Syndrome." The anatomical radial tunnel runs from the radial head to the inferior aspect of the supinator muscle. It is bordered by the supinator, extensor carpi radialis longus, extensor carpi radialis brevis, and brachioradialis muscle [8].

### 56.5.4 Clinical Presentation

Radial tunnel syndrome presents with pain over the radial proximal forearm, typically involving the dominant side. It is usually described as a dull ache, located deep in the extensor muscle mass, and may radiate proximally or distally into the arm and forearm respectively. Pain is usually worse at night or when the elbow is extended, forearm is pronated, or wrist is flexed [8].

### 56.5.5 Physical Examination

In radial tunnel syndrome, patients usually have tenderness over the radial nerve 3–5 cm distal to the lateral epicondyle and may be exacerbated with resisted forearm supination or wrist hyperextension [5, 8]. Pain while extending the middle finger against resistance has shown to be a strong clinical indicator of radial tunnel syndrome [8].

### 56.5.6 Diagnostic Workup

*Electrodiagnostic studies* are normal in radial tunnel syndrome but are still of value as clinically differentiating between radial tunnel syndrome and PIN syndrome is challenging. (PIN syndrome, as previously described, shows abnormalities on electrodiagnostic studies [5].)

*MRI* is usually also normal but could show muscle edema [8]. *Ultrasound* can be useful as it might demonstrate nerve edema; it can also help rule out other diagnoses such as lateral epicondylitis [8].

### 56.5.7 Treatment

Common conservative treatment methods include immobilization of the wrist with splinting, anti-inflammatory medication, ultrasound massage, and physical therapy. Activity modifications include avoiding prolonged elbow extension, forearm pronation, and wrist flexion. Nerve block has been beneficial in some patients. Surgical intervention is recommended if the symptoms do not improve with 3 months of conservative treatments [8]. Surgery usually also includes the release of the PIN [8].

## 56.6 Cubital Tunnel Syndrome

### 56.6.1 Synonyms

Ulnar neuropathy at the elbow

### 56.6.2 ICD-10 Codes

G56.21–G56.23

### 56.6.3 Description

*Cubital tunnel syndrome* is an ulnar neuropathy at the elbow. It is the second most common neuropathy affecting the upper extremity after carpal tunnel syndrome.

***Anatomy*** The ulnar nerve comes from the C8 and T1 roots, with a minor part from C7. The nerve fibers go through the lower trunk and medial cord of the brachial plexus. The ulnar nerve descends through the medial arm toward the elbow. Here, it enters the ulnar groove between the medial epicondyle and the olecranon process. After traversing the ulnar groove, the nerve dives into the cubital tunnel by passing under the flexor carpi ulnaris. The tunnel is sometimes known as the humeral-ulnar aponeurosis [1, 2]. This path of the ulnar nerve is seen in Fig. 56.7.

*Cubital tunnel syndrome* is usually caused by chronic compression due to repeated elbow flexion traumatizing the nerve [1, 2]. Ulnar neuropathy at the elbow is also common in patients immobilized after surgery and during general anesthesia or coma. Congenitally tight cubital tunnels can also lead to compression [1]. In Table 56.6, the differential diagnosis of cubital tunnel syndrome is listed.

### 56.6.4 Clinical Presentation

Cubital tunnel syndrome typically presents with paresthesias, numbness, and/or pain in the ulnar cutaneous distribution, particularly the ring and pinky finger. This can be worsened at night due to elbow flexion while sleeping, but other actions that cause elbow flexion can also trigger the symptoms [2, 9]. As the condition worsens, motor strength diminishes, and patients can report clumsy hand function. Further advanced cases might also report hand deformities [9].

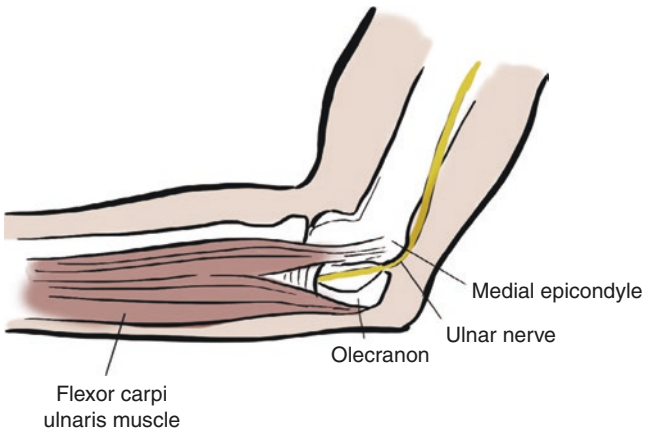


**Fig. 56.7** Path of ulnar nerve traveling through the cubital tunnel. After traversing the ulnar groove, the nerve dives into the cubital tunnel by passing under the flexor carpi ulnaris

**Table 56.6** Differential diagnosis of cubital tunnel syndrome

| | |
|---|---|
| Amyotrophic lateral sclerosis | Superior sulcus tumor (Pancoast's tumor) |
| Lower cervical radiculopathy | Thoracic outlet syndrome |
| Spinal cord diseases | Ulnar neuropathy distal to the elbow |

### 56.6.5 Physical Examination

Inspection of the hand can demonstrate atrophy of the intrinsic hand muscles. Strength testing can reveal weakness in the grip strength due to the involvement of the finger flexors. Flexion of the fifth digit at the distal interphalangeal joint is the best way to identify ulnar side involved muscle weakness [2]. Thumb adduction and flexion weakness can occur, but thumb abduction should not be involved [1]. Benediction posture, Wartenberg's sign, and Froment's sign are three examples of physical examination findings seen in more advanced cases (Fig. 56.8). Benediction hand, also known as claw hand deformity, is a hand positioned with metacarpophalangeal hyperextension and proximal and distal interphalangeal flexion of the fourth and fifth fingers [1, 9]. Wartenberg's sign is when the fifth finger on the involved hand is abducted at rest. Lastly, Froment's sign occurs when the patient is asked to pinch a piece of paper with their thumb and index finger. Due to intrinsic hand weakness, patients instead utilize the flexor pollicis longus and flexor digitorum profundus resulting in atypical flexion of the interphalangeal joints of those two fingers [1, 9]. Sensory exam can exhibit decrease in light touch over the ulnar half of the fourth finger and the complete fifth digit. No sensory changes in the forearm should be evident [1, 2, 9]. As with all suspected nerve entrapments, a complete neuromuscular examination of the involved extremity is vital to rule out other pathologies.

### 56.6.6 Diagnostic Workup

*X-rays* of the elbow to identify any bony abnormalities that might be causing a neuropathy can be helpful. If cervical radiculopathy or Pancoast tumor is of high concern, neck and chest radiographs, respectively, are recommended [9].

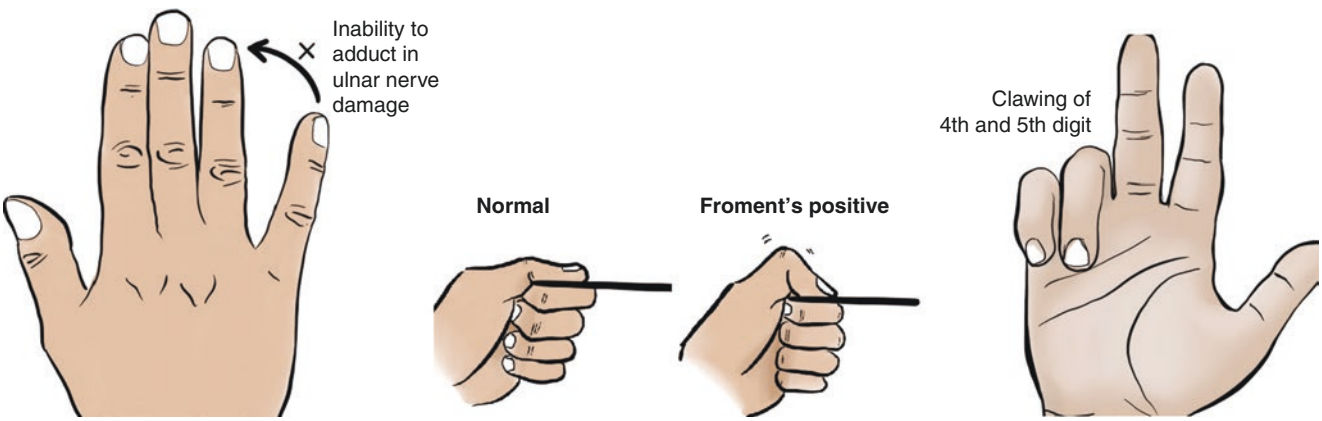


**Fig. 56.8** Wartenberg's sign (left) is when the fifth finger on the involved hand is abducted at rest (unable to adduct due to ulnar nerve injury). Froment test (middle) examines ulnar neuropathy at the cubital tunnel. Benediction sign or claw hand deformity (right) is a hand positioned with metacarpophalangeal hyperextension and proximal and distal interphalangeal flexion of the fourth and fifth fingers

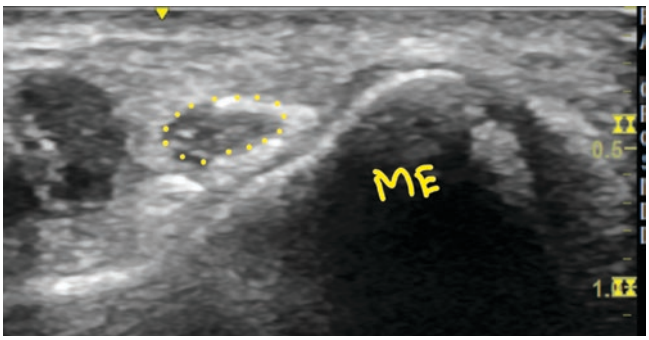


**Fig. 56.9** Ulnar nerve highlighted in the cubital tunnel. Medial Epicondyle bony landmark is seen (ME). In dynamic US examination, subluxation of the ulnar nerve over the medial epicondyle can be easily seen. (Image courtesy of S. Ali Mostoufi, MD, Boston Regenerative Medicine)

Electrodiagnostic studies are key in identifying cubital tunnel syndrome. Nerve conduction studies need to include motor studies above and below the elbow to try and identify any demyelinating and/or axonal involvement across the elbow. Slowed conduction velocities, decreased amplitudes, and prolonged distal latency can be seen. Electromyography of both the ulnar-innervated muscles distal to the elbow and the muscles involved in C8 radiculopathy is necessary [2].

*Ultrasound* is a useful tool to easily follow the ulnar nerve through the arm. Flexing the elbow while observing the nerve at the elbow is a useful tool for identifying subluxation over the medial epicondyle (Fig. 56.9). Cross-sectional areas taken in the short axis can objectively determine if the nerve is edematous and enlarged [1].

### 56.6.7 Treatment

Conservative treatment should be initially attempted, with 50–90% of cases showing improvement [9, 10]. The program would include therapy, night splints, and patient education. Avoiding positions that provide direct pressure on the nerve at the elbow is of the utmost importance. Nightly elbow braces avoid hyperflexion and allow for inflammation to decrease. NSAID and steroid injections are sometimes attempted, but the evidence supporting their use is limited [9]. An ultrasound-guided steroid injection is demonstrated in Fig. 56.10. Surgery is necessary in advanced cases or after failing conservative treatment. Commonly used procedures are cubital tunnel release, ulnar nerve anterior transposition, or medial epicondylectomy, with medial epicondylectomy less utilized recently. Postsurgical physical therapy is recommended to help the patient regain strength and range of motion [9].

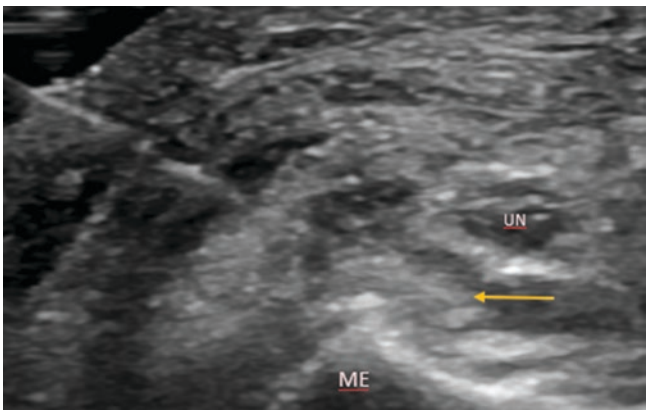


**Fig. 56.10** US-guided perineural steroid injection of the ulnar nerve (UN) at the cubital tunnel. Needle tip (arrow) seen deep to the UN and slight hydrodissection is seen. Medial epicondyle's (ME) bony landmark is noted. (Courtesy of S. Ali Mostoufi, MD Boston Regen)

## 56.7 Suprascapular Nerve Entrapment

### 56.7.1 Synonyms

- Suprascapular neuropathy
- Suprascapular nerve palsy

### 56.7.2 ICD-10 Codes

G56.80

### 56.7.3 Description

Suprascapular nerve entrapment is a neuropathy due to compression of the suprascapular nerve. The nerve is most frequently injured at the suprascapular notch, but it can also be compressed at the spinoglenoid notch. Suprascapular nerve entrapment is also caused by rotator cuff tears with significant retraction, labral tears with cysts, and other mass lesions. Overhead activities are associated with suprascapular nerve entrapment [1, 11]. Table 56.7 describes the various diagnoses on the differential of suprascapular nerve entrapment.

***Anatomy*** The suprascapular nerve arises from the upper trunk of the brachial plexus and receives innervation from the C5 and C6 roots. It travels posterior to the trapezius and clavicle on its path to the superior scapula. The nerve goes under the transverse suprascapular ligament at the suprascapular notch as it enters the scapular region. The transverse suprascapular ligament is the most likely site of entrapment. The suprascapular nerve then gives off motor

fibers to the supraspinatus before heading towards the more laterally located spinoglenoid notch. After traversing under the spinoglenoid ligament, the nerve supplies motor innervation to the infraspinatus. Along its path, deep sensory innervation is given to the glenohumeral joint, acromioclavicular joint, and coracoacromial ligament. It rarely provides cutaneous sensation to the lateral arm [1, 2, 11]. See Fig. 56.11 for further the path of the suprascapular nerve in the posterior shoulder.

### 56.7.4 Clinical Presentation

Pain is typically the presenting complaint. Due to the deep sensory nerve branches of the nerve, patients complain of the pain feeling deep and dull as opposed to a more superficial pain. It can start in the upper scapula region with radiation to the shoulder. Shoulder movements can worsen the pain, particularly overhead activities. Patients also frequently report weakness with an aspect of shoulder fatigability. Usually, no inciting event is reported, but some might report frequent overhead activities [1, 11].

**Table 56.7** Differential diagnosis of suprascapular nerve entrapment

| | |
|---|---|
| AC joint injury | Neuralgic amyotrophy |
| Cervical radiculopathy and brachial plexopathy | Rotator cuff injury |
| Glenohumeral osteoarthritis | Shoulder labral tear |

### 56.7.5 Physical Examination

With physical examination, inspection can discover atrophy of the supraspinatus and infraspinatus. Palpation over the suprascapular notch can yield tenderness. Patients can have weakness with shoulder abduction and external rotation due to the involvement of the supraspinatus and infraspinatus, respectively. Sensation should be intact as the suprascapular nerve does not have cutaneous involvement. Upper extremity reflexes should be normal. Site of entrapment changes the physical exam findings: entrapment after the supraspinatus is supplied (such as in entrapment at the spinoglenoid notch) would lead to normal shoulder abduction strength and no atrophy of the supraspinatus. A thorough exam of the cervical spine and shoulder is required to help narrow the diagnosis [11].

### 56.7.6 Diagnostic Workup

*Electrodiagnostic* studies are the gold standard for diagnosis. Despite there being no cutaneous distribution to test, the other sensory nerves in the arm should be tested to rule out brachial plexopathy. Motor conduction studies should be performed as well, with stimulation over Erb's point and recording the supraspinatus and infraspinatus. Motor studies may show decreased amplitudes and prolonged latencies. EMG of the supraspinatus and infraspinatus is key to diagnosing this disorder. Signs of denervation of those two muscles would point toward suprascapular neuropathy [1, 2].

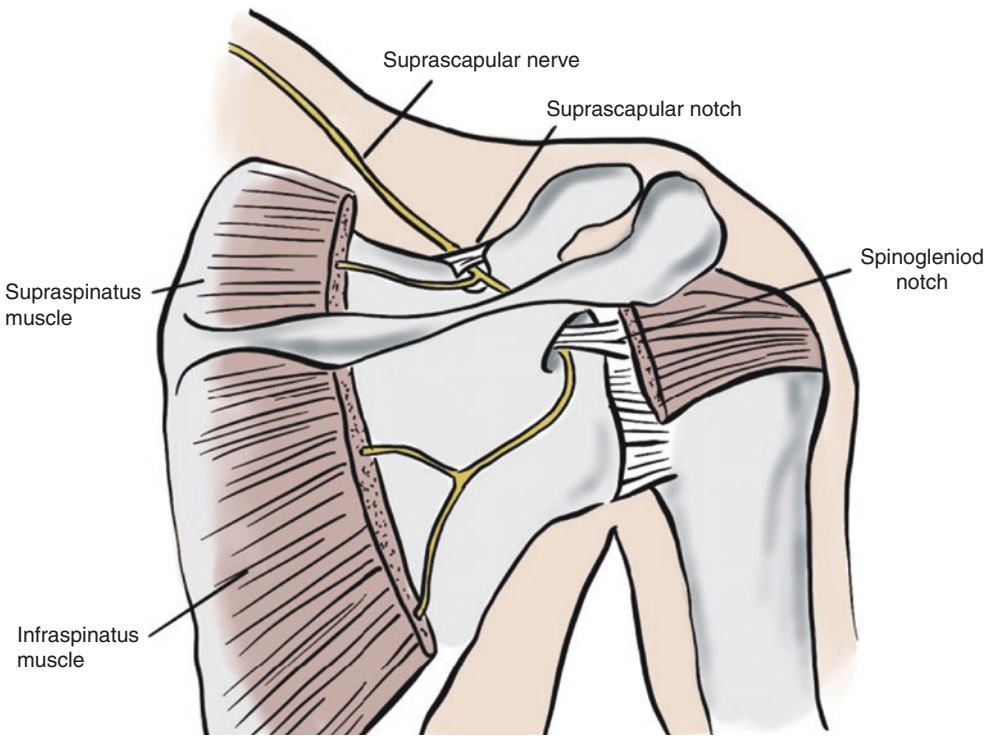


**Fig. 56.11** Suprascapular nerve path in the posterior shoulder

***Imaging*** Tests including radiographs, computed tomography, ultrasound, and magnetic resonance imaging can demonstrate the etiology of the entrapment, including fractures, ligament ossification, retracted rotator cuff tears, labral tears with cysts, and mass lesions [11].

### 56.7.7 Treatment

Conservative management involves avoiding overhead activities and utilizing physical therapy and nonsteroidal anti-inflammatory drugs. Physical therapy should focus on strengthening the shoulder and scapula musculature to improve motion and mechanics. If the patient fails conservative treatment or has a mass, then surgery is considered. These surgeries can decompress the entrapment and allow the nerve to heal. The surgical procedures can vary greatly based on the etiology of the problem and the location, but the release of the transverse scapular ligament is common [11]. If pain is a significant issue for the patient, then suprascapular nerve block or peripheral nerve stimulation are pain management treatment options [20] but they would not likely resolve any weakness.

## 56.8 Fibular Nerve Entrapment

### 56.8.1 Synonyms

- Peroneal neuropathy
- Foot drop
- Fibular neck entrapment

### 56.8.2 ICD-10 Codes

S84.10

### 56.8.3 Description

*Fibular nerve entrapment* is an injury to the fibular (peroneal) nerve at the fibular neck. The nerve travels superficially over the bone and is thus at risk at this location. Typically, both the deep and superficial peroneal nerves are involved [1]. Fibular nerve entrapment usually occurs after trauma or compression. Hospitalized patients with prolonged immobilization may develop it due to lack of positional changes and weight loss during the hospitalization. Frequent leg crossing has also been associated with peroneal nerve injury. While it is possible for the fibular nerve also to get impinged deep at the ankle, it is extremely rare and will therefore not be discussed in this section [1]. Table 56.8 details the differential diagnosis of fibular nerve entrapment.

**Table 56.8** Differential diagnosis of fibular nerve entrapment

| | |
|---|---|
| L5 radiculopathy | Peripheral neuropathy |
| Lumbosacral plexopathy | Sciatic neuropathy |
| Piriformis syndrome | Ischiofemoral impingement |

***Anatomy*** The common peroneal nerve runs within the sciatic nerve in the thigh after arising from the L4-S1 nerve roots and traversing the lumbosacral plexus. The sciatic nerves divide into the tibial nerve and common peroneal above the posterior knee. Sensory innervation is given off to the lateral knee via the lateral cutaneous nerve of the knee before the peroneal nerve passes over the fibular neck. At the neck, the future nerve fibers that become the deep peroneal nerve are closer to the bone, while the fibers for the superficial peroneal nerve are further away. The common fibular nerve splits into the superficial peroneal nerve and deep peroneal nerve after crossing the fibular neck. The superficial nerve gives sensation to the middle and lower lateral calf and also innervates the peroneus longus and brevis. It ends as cutaneous nerves giving sensation to the dorsal foot. The deep peroneal nerve innervates the tibialis anterior, extensor digitorum longus, extensor hallucis longus, and extensor digitorum brevis while also giving sensation to the area between the first and second toes [1]. Figure 56.12 demonstrates the path of the fibular nerve in the lower leg.

### 56.8.4 Clinical Presentation

Foot drop is a frequent presenting symptom. Patients report tripping while walking, ankle sprains, and foot slapping against the ground. About 80% of patients in one study reported sensory symptoms; pain was rare among the study group [12]. Loss of sensation occurs over the lateral distal leg and dorsal foot. Most present with acute onset of symptoms [12]. Recent significant weight loss causes the nerve to lose its protecting adipose tissue and could also be a part of the patient's history [1].

### 56.8.5 Physical Examination

Physical examination is a vital step in differentiating fibular nerve entrapment from L5 radiculopathy or other more global processes such as peripheral neuropathy. Peroneal neuropathy at the fibular neck leads to weakness in ankle dorsiflexion, foot eversion, and toe extension, but no weak-

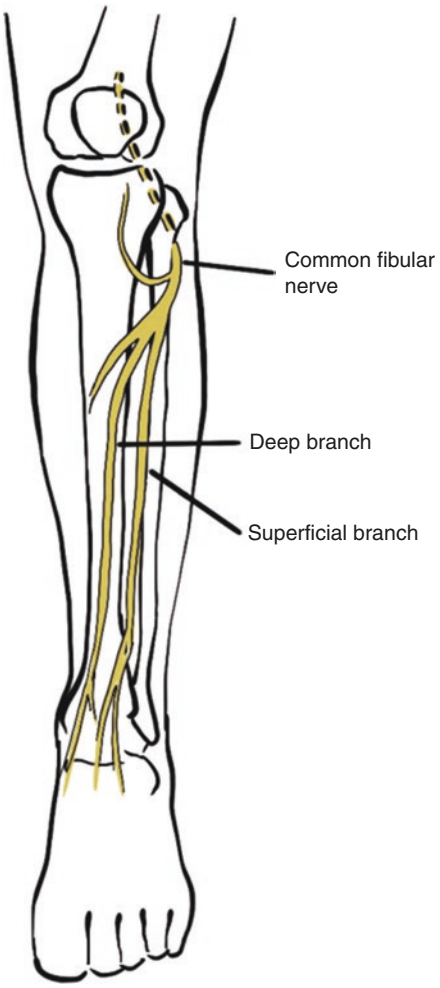


**Fig. 56.12** Fibular nerve path and its branching in the lower extremity distal to the knee

ness in foot inversion. Patients should not have proximal muscle weakness either, so evaluating strength at the hip and knee is an important part of the physical exam. Sensation should be normal in the upper thigh and over the lateral knee but can be diminished over the lateral calf and dorsal foot. Reflexes should be intact. Gait analysis can reveal methods to clear the dropped foot: hip hike, foot circumduction, steppage gait [1].

### 56.8.6 Diagnostic Workup

*Electrodiagnostic studies* (EMG/NCS) are the most important test for diagnosing fibular nerve entrapment. Slowing or conduction block across the fibular neck on peroneal motor nerve conduction studies (NCS) would signal neuropathy at this location. Decreased amplitudes can also be seen in axonal loss. Recording at the tibialis anterior rather than extensor digitorum brevis can be more revealing in peroneal motor NCS [1, 12]. The sural and tibial nerves should be evaluated as well to rule out a more widespread process. EMG should target the peroneal innervated muscles but must also test sufficient muscles to rule out L5 radiculopathy, lumbosacral plexopathy, and sciatic neuropathy.

*Ultrasound* can be used if EMG/NCS does not pinpoint the location of the lesion or if a mass is suspected. MRI can also identify a mass lesion resulting in fibular nerve compression.

### 56.8.7 Treatment

Conservative management typically leads to improvement in patients. Physical therapy focusing on strengthening of the lower extremities is key as chronic foot drop positioning otherwise leads to plantarflexion contracture [13, 17, 18]. Ankle-foot orthosis (AFO) should be considered in patients with significant foot drop. Two examples are pictured in Fig. 56.13. Functional electrical stimulation is an alternative to an AFO and generally leads to improved patient satisfaction [19]. Padding can be added over the fibular head in patients with significant weight loss. Surgery for foot drop is typically only utilized in slowly progressing patients or those with masses identified [13]. One surgical option in cases of contractures is tendon transfer [17, 18]. For those with poor prognosis or more complete injuries, tibial nerve partial transfer to tibialis anterior has been successful [18].

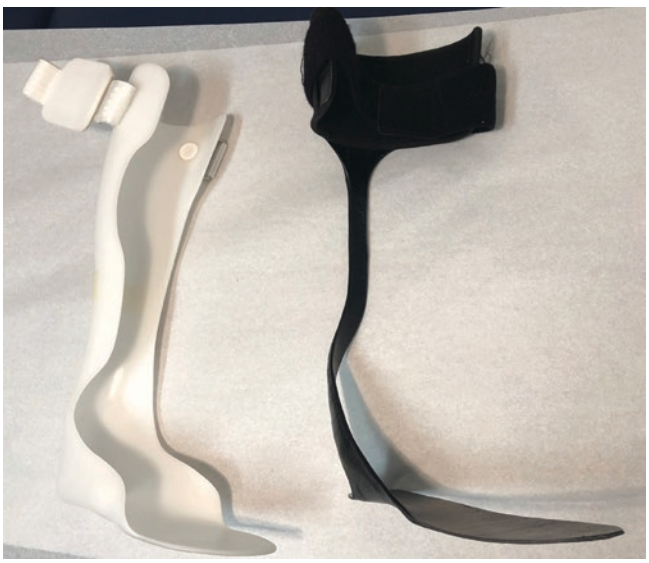

**Fig. 56.13** Ankle-foot orthoses. A solid plastic AFO and a carbon fiber AFO are two options for patients with foot drop

## 56.9 Tarsal Tunnel Syndrome

### 56.9.1 Synonyms

- Tibial neuropathy
- Plantar neuropathy

### 56.9.2 ICD-10 Codes

G57.50

### 56.9.3 Description

*Tarsal tunnel syndrome (TTS)* is a neuropathy due to entrapment of the tibial nerve at the medial ankle. The nerve gets compressed as it travels under the flexor retinaculum posterior to the medial malleolus. TTS is rare, with most cases of an idiopathic etiology. Trauma has been associated with this entrapment. Masses can also cause impingement of the nerve [1, 13]. In Table 56.9, the differential diagnoses of tarsal tunnel syndrome are listed.

***Anatomy*** The tibial nerve runs posterior to the medial malleolus and passes under the flexor retinaculum at the ankle, entering the tarsal tunnel. This tunnel is similar to the carpal tunnel in that it contains more than just the nerve; the tibial artery and vein pass through the tunnel along with the flexor hallucis longus tendon, flexor digitorum longus tendon, and tibialis posterior tendon. As the tibial nerve passes through the tunnel, it begins to separate into its three distal branches: the medial calcaneal sensory nerve, the lateral plantar nerve, and the medial plantar nerve. In some instances, the nerve does not divide until it exits the tunnel. The medial calcaneal sensory nerve is the sensory innervation of the heel. The lateral plantar nerve provides sensation to the lateral fourth toe and the fifth toe. It also has a motor component that innervates some of the intrinsic muscles of the foot such as the abductor digiti quinti pedis. The medial plantar nerve innervates the first three toes and the medial fourth toe while also innervating some of the intrinsic foot muscles including the abductor hallucis brevis and flexor hallucis brevis. The plantar nerves also provide sensory innervation to the sole of the foot [1, 13]. Figure 56.14 shows the relationship of the tibial nerve in the tarsal tunnel.

**Table 56.9** Differential diagnosis of tarsal tunnel syndrome

| | |
|---|---|
| Distal peripheral neuropathy | S1 radiculopathy |
| Lumbosacral plexopathy | Tendonitis of tibialis posterior |
| Plantar fasciitis | Calcaneal bone spur |
| Ankle osteoarthritis | Tendonitis of flexor hallucis longus, or flexor digitorum longus |

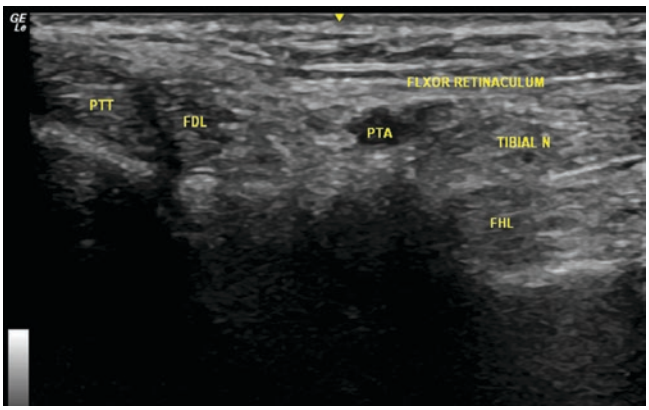


**Fig. 56.14** Short access US image of the tarsal tunnel. Content of the tunnel noted deep to flexor retinaculum which includes posterior tibial artery (PTA) and vein, tibial nerve, and flexor hallux longus tendon (FHL). Posterior tibial tendon (PTT) and flexor digitorum longus (FDL) are also seen more anterior in this cross section against the bony medial malleoli

### 56.9.4 Clinical Presentation

Patients often present with pain around the medial ankle, with some radiation into the sole. It is typically characterized as a burning sensation [14]. Some complain of loss of sensation over the heel or sole and might have wounds as a result. Patients could also report a history of ankle sprains or fractures or could have a history of connective tissue disorders. Weakness is another presenting symptom but could be difficult for patients to notice; atrophy might be more easily noticeable to the patient [1].

### 56.9.5 Physical Examination

Inspection can yield atrophy of the intrinsic foot muscles. Strength is difficult to assess as the bulk of foot and toe strength comes from the larger muscles in the leg, which would not be affected by TTS. Decreased sensation over the sole of the foot could be the only significant abnormality on exam [1]. Tinel's sign over the tarsal tunnel can be checked, but it is unreliable [14]. Achilles' reflex should be normal, as it is innervated prior to the tarsal tunnel.

### 56.9.6 Diagnostic Workup

*Electrodiagnostic* studies are frequently utilized in diagnosing TTS. However, it is a technically difficult study to perform and lacks evidence on the proper techniques. Side-to-side comparison must be performed of the medial and lateral plantar nerves, recording over the abductor hallucis brevis and abductor digiti quinti pedis, respectively. Mixed studies can also be of diagnostic value. However, all three of these tests are frequently not obtainable in normal

patients. Routine sural, tibial, and peroneal nerve studies should also be evaluated to rule out a more diffuse process. EMG of the foot muscles has many issues associated with it including patient tolerance, activation of the muscle, and infection risk [1, 13].

*Ultrasound* has been shown to be extremely useful in the TTS diagnosis process, as visualization of the tibial nerve in the tunnel is typically straightforward. Evaluating the nerve and its surroundings can demonstrate abnormalities with the nerve itself and nearby structures that might be impinging on the nerve. The plantar fascia can also be evaluated, as plantar fasciitis could be the actual cause of symptoms. MRI can also be used to observe mass lesions [1, 14].

### 56.9.7 Treatment

Conservative management with footwear modifications and nonsteroidal anti-inflammatory medications are the first steps. In some cases, tight-fitting shoes are the causative agent, and adjustment can relieve the symptoms.

US-guided tarsal tunnel corticosteroid injections with or without hydrodissection can provide symptomatic relief. Image guidance reduces the risk of tendon or neural injury during percutaneous injections [21]. Hydrodissection of the nerve can release it from scar tissue and relieve compression. Persistent symptoms or identified masses require surgical intervention [13]. Endoscopic release has been demonstrated as a successful technique that is simpler than more traditional open surgeries [15].

## 56.10 Femoral Nerve Entrapment

### 56.10.1 Synonyms

Femoral neuropathy

### 56.10.2 ICD-10 Codes

G57.20

### 56.10.3 Descriptions

*Femoral nerve entrapment* is a neuropathy that most typically causes knee extension weakness. When the nerve is injured more proximally, hip flexion will be weak as well. Femoral neuropathy can be caused by many conditions: retroperitoneal hematoma, compression at the inguinal ligament due to an iliopsoas bursal enlargement or hematoma, penetrating groin trauma, hip arthroplasty, poor lithotomy positioning, inadvertent clamping during femoral artery procedures, or pelvic surgery. Isolated femoral nerve entrapment is considered uncommon [1]. Table 56.10 lists other differential diagnoses that can present in a similar manner as femoral neuropathy.

***Anatomy*** The femoral nerve arises from the L2, L3, and L4 nerve roots. Axons traverse the lumbar plexus. In the retroperitoneal space, the nerve travels between the psoas and iliacus muscles, then under the iliacus fascia in the pelvis. It enters the thigh below the inguinal ligament and lateral to the femoral artery. The femoral nerve innervates the psoas and iliacus in the pelvis. In the thigh, the nerve innervates the four quadriceps muscles: the vastus lateralis, vastus medialis, vastus intermedius, and rectus femoris. It also innervates the pectineus and sartorius muscles. Sensory branches supply medial sensation to the thigh from the medial cutaneous and intermediate cutaneous nerves. The distal medial thigh and medial lower leg sensation is supplied by the saphenous nerve which is a terminal branch of the femoral nerve [1, 16]. Figure 56.15 details the path of the femoral nerve through the thigh.

**Table 56.10** Differential diagnosis of femoral neuropathy

| | |
|---|---|
| Femoral amyotrophy | Multifocal motor neuropathy |
| L3/L4 radiculopathies | Hip/knee/pelvis disorders associated with pain or weakness |
| Lumbar plexopathies | Myopathies |

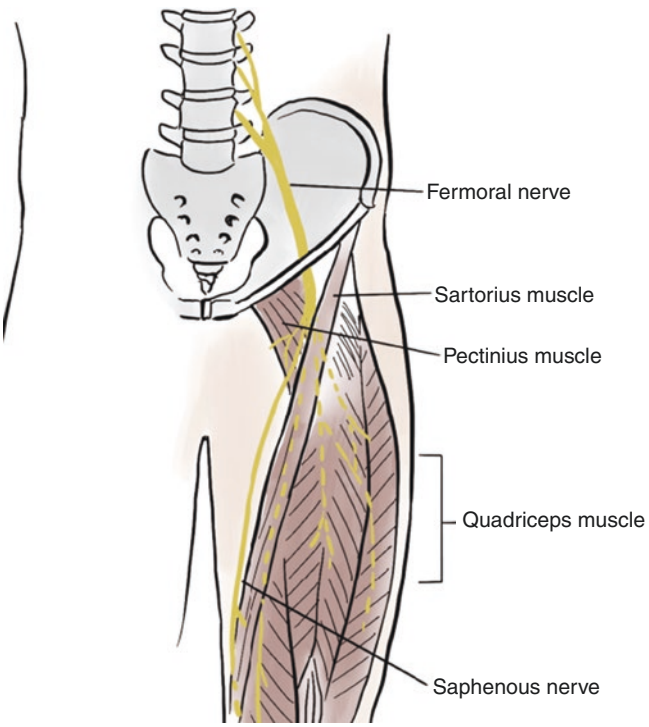


**Fig. 56.15** Path of the femoral nerve through the proximal thigh

### 56.10.4 Clinical Presentation

Femoral nerve entrapment presents with weakness in knee extension. Buckling of the knee is common. Dragging the leg and weakness of the hip in flexion can occur with proximal nerve involvement. Some sensory involvement in the medial thigh and medial lower leg may be present.

### 56.10.5 Physical Examination

Examination will find weakness in knee extension and perhaps hip flexion with an absent or diminished patellar reflex. Sensory may be impaired in the medial thigh and medial lower leg [1]. A thorough examination of the lumbar region and lower limbs is needed to rule out other causes of weakness.

### 56.10.6 Diagnostic Workup

*Electrodiagnostic* studies are useful to diagnose and exclude other causes of weakness. A comparative side-to-side femoral motor study may show significant amplitude differences. EMG must sample at least two femoral innervated muscles and also muscles innervated by nerves outside of the femoral nerve in order to isolate a femoral neuropathy. Saphenous sensory studies may be helpful in the diagnosis but are often difficult to obtain [1, 16].

*Ultrasound* may reveal a swollen and heterogeneous appearing femoral nerve at the site of pathology or disruption of the nerve. It can also reveal masses and hematomas at the site of pathology [1].

### 56.10.7 Treatment

Treatment is dependent on the cause of the neuropathy. Conservative care includes therapeutic modalities, strengthening, and gait training with devices. Bracing includes the application of a knee-ankle-foot orthosis. Crutches or canes may also be needed for stability. Conservative management usually leads to some improvement within 2 years [16]. Surgical intervention may be required for decompression of hematomas or masses or to relieve scar tissue as in a neurolysis.

## References


1. Preston DC, Shapiro BE. Electromyography and neuromuscular disorders E-book: clinical-electrophysiologic-ultrasound correlations. Amsterdam: Elsevier Health Sciences; 2020.
2. Amato AA, Russell JA. Neuromuscular disorders. 2nd ed. McGraw-Hill Education;2016:2–21. ISBN:9780071752503.
3. Padua L, Pasquale AD, Pazzaglia C, Liotta GA, Librante A, Mondelli M. Systematic review of pregnancy-related carpal tunnel syndrome. Muscle Nerve. 2010;42(5):697–702.
4. Dang AC, Rodner CM. Unusual compression neuropathies of the forearm, part II: median nerve. J Hand Surg Am. 2009;34(10):1915–20.
5. Bevelaqua AC, Hayter CL, Feinberg JH, Rodeo SA. Posterior interosseous neuropathy: electrodiagnostic evaluation. HSS J. 2012;8(2):184–9.
6. Djurdjevic T, Loizides A, Löscher W, Gruber H, Plaikner M, Peer S. High resolution ultrasound in posterior interosseous nerve syndrome. Muscle Nerve. 2014;49(1):35–9.
7. Kim DH, Murovic JA, Kim YY, Kline DG. Surgical treatment and outcomes in 45 cases of posterior interosseous nerve entrapments and injuries. J Neurosurg. 2006;104(5):766–77.
8. Moradi A, Ebrahimzadeh MH, Jupiter JB. Radial tunnel syndrome, diagnostic and treatment dilemma. Arch Bone Jt Surg. 2015;3(3):156.
9. Andrews K, Rowland A, Pranjal A, Ebraheim N. Cubital tunnel syndrome: anatomy, clinical presentation, and management. J Orthop. 2018;15(3):832–6.
10. Osei DA, Groves AP, Bommarito K, Ray WZ. Cubital tunnel syndrome: incidence and demographics in a national administrative database. Neurosurgery. 2017;80(3):417–20.
11. Boykin RE, Friedman DJ, Higgins LD, Warner JJ. Suprascapular neuropathy. JBJS. 2010;92(13):2348–64.
12. Katirji MB, Wilbourn AJ. Common peroneal mononeuropathy: a clinical and electrophysiologic study of 116 lesions. Neurology. 1988;38(11):1723–8.
13. Shapiro BE, Preston DC. Entrapment and compressive neuropathies. Med Clin N Am. 2009;93(2):285–315.
14. Samarawickrama D, Therimadasamy AK, Chan YC, Vijayan J, Wilder-Smith EP. Nerve ultrasound in electrophysiologically verified tarsal tunnel syndrome. Muscle Nerve. 2016;53(6):906–12.
15. Krishnan KG, Pinzer T, Schackert G. A novel endoscopic technique in treating single nerve entrapment syndromes with special attention to ulnar nerve transposition and tarsal tunnel release: clinical application. Oper Neurosurg. 2006;59(suppl_1):ONS-89.
16. Madani S, Doughty C. Lower extremity entrapment neuropathies. Best Pract Res Clin Rheumatol. 2020;34:101565.
17. Frontera WR, Silver JK, Rizzo TD. Essentials of physical medicine and rehabilitation e-book. Elsevier Health Sciences; 2018.
18. Giuffre JL, Bishop AT, Spinner RJ, Shin AY. Surgical technique of a partial tibial nerve transfer to the tibialis anterior motor branch for the treatment of peroneal nerve injury. Ann Plast Surg. 2012;69(1):48–53.
19. Bucklitsch J, Müller A, Weitner A, Filmann N, Patriciu A, Behmanesh B, Seifert V, Marquardt G, Quick-Weller J. Significant impact of implantable functional electrical stimulation on gait parameters: a kinetic analysis in foot drop patients. World Neurosurg. 2019;127:e236–41.
20. Gofeld M, Agur A. Peripheral nerve stimulation for chronic shoulder pain: a proof of concept anatomy study. Neuromodulation. 2018;21(3):284–9.
21. Lau JT, Daniels TR. Tarsal tunnel syndrome: a review of the literature. Foot Ankle Int. 1999;20(3):201–9.
22. Ziswiler HR, Reichenbach S, Vogelin E, Bachmann LM, Villiger PM, Juni P. Diagnostic value of sonography in patients with suspected carpal tunnel syndrome: a prospective study. Arthritis Rheum. 2005;52:304–11.
23. Vincelet Y, Journeau P, Popkov D, Haumont T, Lascombes P. The anatomical basis for anterior interosseous nerve palsy secondary to supracondylar humerus fractures in children. Orthop Traumatol Surg Res. 2013;99(5):543–7. https://doi.org/10.1016/j.otsr.2013.04.002. Epub 2013 Aug 2. PMID: 23916783.
24. Wiesman IM, Novak CB, Mackinnon SE, Winograd JM. Sensitivity and specificity of clinical testing for carpal tunnel syndrome. Can J Plast Surg. 2003;11(2):70–2. https://doi.org/10.1177/229255030301100205.