Common Entrapment Neuropathies

Elia Malek, MD[1] Johnny S. Salameh, MD, FANA[1]

[1] Department of Neurology, American University of Beirut Medical Center, Riad El Solh, Beirut, Lebanon

Semin Neurol 2019;39:549–559.

Address for correspondence Johnny S. Salameh, MD, FANA, Department of Neurology, American University of Beirut Medical Center, P.O. Box: 11-0236, Riad El Solh, Beirut, 1107 2020, Lebanon (e-mail: js73@aub.edu.lb).

Abstract

Entrapment neuropathies are defined as compression of peripheral nerves due to known or unknown causes. The high incidence and variety of presentations require a comprehensive knowledge of these conditions, especially in neurology and orthopedic surgery clinical practices. Detailed knowledge of topographic anatomy, clinical manifestations, and appropriate use of electrophysiological studies with selective addition of neuromuscular ultrasonography are needed to establish an early and accurate diagnosis to advice patients and provide them with a comprehensive treatment plan. In this article, we discuss the most common forms of entrapment neuropathies in the upper and lower extremities.

Keywords
- entrapment neuropathies
- compression neuropathies
- mononeuropathies

Entrapment or compression neuropathies are a group of mononeuropathy syndromes occurring due to impingement of a single peripheral nerve along the upper or lower extremities. By definition, compression occurs in certain regions where a nerve passes through a tight path or canal formed by rigid or soft tissues (bone, tendon, or membrane). The true incidence and prevalence of entrapment neuropathies is unknown since it is an underrecognized entity. Carpal tunnel syndrome (CTS) is the most common presentation with a variable prevalence ranging between 3.72 and 5.8%, and an incidence of at least 81 per 100,000.[1–3] This makes entrapment neuropathies one of the most common causes of neurological consultations in clinical practice. It is essential to learn the anatomy surrounding the pathway of the peripheral nerves with the highest risk of compression, in addition to the typical clinical manifestations to make an accurate diagnosis. Electrophysiological studies combined with the recently implemented ultrasonographic visualization of the nerve are the cornerstone in confirming the diagnosis, in addition to exploring the possible cause and nature of the lesion.[4] Entrapments due to traumatic causes are beyond the scope of this review.

Median Nerve Entrapment

Anatomy

The median nerve receives contributions from C5, C6, C7, C8, and T1 roots, through the end terminals of the lateral and medial cords. It travels along the arm medially down to the brachial artery without giving any branches. In the proximal forearm, it innervates the pronator teres (PT), flexor carpi radialis, and flexor digitorum sublimis (FDS). Before reaching this level, the median nerve may be compressed under the ligament of Struthers which lies between the supracondylar process and the medial epicondyle of the humerus in some cases.[5,6] In the antecubital region, the median nerve runs under the bicipital aponeurosis (lacertus fibrosus) which can be either part of the PT or present as a separate entity attached to the ulna.[7,8] After passing under the PT, which can be another site of nerve compression by the muscle itself, the anterior interosseous nerve (AIN) branches off to innervate the flexor pollicis longus (FPL), flexor digitorum profundus (FDP) to the 2nd and 3rd digits, and pronator quadratus (PQ) muscles. More distally, the median nerve can be compressed between the FDP and FDS muscles before reaching the most common site of compression, the carpal tunnel at the wrist. This tunnel is located in the middle third of the wrist. The floor and sides of the tunnel are formed by the trapezium, trapezoid, capitate, and hamate bones, while the roof (palmar side) is formed by the flexor retinaculum (transverse carpal ligament). Tendons of the FDP to 2nd and 3rd digits, FDS, and FPL run beside the median nerve, which makes the area even tighter. It is important to note that sensation of the thenar eminence is mediated by the palmar sensory cutaneous branch that branches off the median

Issue Theme Peripheral Neuropathies; Guest Editors, Michelle Kaku, MD, and Peter Siao, MD

DOI https://doi.org/10.1055/s-0039-1693004.
ISSN 0271-8235.

nerve before the nerve enters the carpal tunnel. Another important normal variant to keep in mind is the high incidence of Martin–Gruber anastomosis (connecting fibers between the median and ulnar nerves) reaching up to 39% in the general population and may result in misinterpretation of the electrophysiological findings.[9]

Clinical

Clinical manifestation of median nerve compression varies according to the site of entrapment.

As mentioned above, CTS is the most common entrapment syndrome. It manifests with intermittent or persistent numbness, tingling, and paresthesia with or without pain in the palmar aspect of the first three digits that may be reported by patients as being all over the palm, radiating up to the forearm (and even shoulder), and frequently awakening patients from sleep. Motor involvement may manifest in advanced conditions as weakness in thumb opposition and thumb abduction (abductor pollicis brevis muscle) along with atrophy in the thenar muscles. Patients complain of reduced hand dexterity as well as hand grip weakness, especially in performing actions like turning doorknobs, buttoning shirts, or using smartphones. Physicians should always consider an alternate diagnosis in the presence of neck pain. Tinel's and Phalen's bedside testing may help with diagnosis but have low sensitivity (43 and 67%, respectively),[10] which can be subjective due to significant intra- and interexaminer difference in technique application.[11]

The most proximal nontraumatic median nerve entrapment is due to the presence of ligament of Struthers in the supracondylar area. Patients complain of local pain in the area of the ligament in addition to symptoms occurring in the area of distribution of median nerve such as paresthesias in the palmar aspect of the first three digits and the radial side of the 4th digit, and weakness in PT as well as median-innervated intrinsic hand muscles. In addition, pronation and/or supination with extended elbow may aggravate the symptoms and even reduce the radial pulse.[12,13]

Moving distally, the median nerve can be entrapped by either the FDS, bicipital aponeurosis, or PT muscle itself. This will lead to a pronator syndrome (PS). The onset of symptoms is usually insidious, similar to the entrapment caused by the ligament of Struthers; however, the pain is more localized to the proximal forearm, namely, the PT muscle, while sparing its function. The motor deficit in the median innervated intrinsic hand muscles is usually mild, while paresthesias in the digits are more prominent with repetitive pronation without the nocturnal onset typically seen in CTS.[14] Anterior interosseous syndrome (AIS) is another acquired entrapment due to compression of the AIN by the same tissues as in the PS. The clinical manifestation may be complete or incomplete paresis of FPL, FDP of the 2nd and 3rd digits, and PQ muscles. Patients usually present with dull pain in the forearm, inability to flex the distal phalanx of the thumb, index, and middle fingers, and an inability to pronate in the flexed elbow position. The inability to make a round circle using the thumb and index/middle fingers (OK sign) is a typical characteristic (►**Fig. 1**). However, no sensory deficit or paresthesias are typically seen in this syndrome.[15]

Fig. 1 Anterior interosseous syndrome. The patient is asked to perform the "OK" sign. Weakness in flexor pollicis longus (FPL) and 2nd flexor digitorum profundus (FDP) can be noted.

Electrophysiological Studies

For patients with CTS, the American Association of Neuromuscular and Electrodiagnostic Medicine (AANEM) guidelines recommend performing median sensory nerve conduction study (NCS) across the wrist recording at a distance of 13 to 14 cm. In case of a normal result, it is recommended to perform comparative study of either the median mixed NCS across the wrist with ulnar mixed NCS across the wrist using the same recording distance (7–8 cm), or median sensory with ulnar/radial sensory NCS across the wrist of the same hand, or sensory/mixed median NCS across the carpal tunnel with sensory/mixed NCS of proximal or distal parts of the median nerve in the same limb. Median motor NCS should also include the recording of another motor NCS of another nerve/muscle in the same limb (►**Fig. 2**). Other techniques such as comparative studies of median motor distal latency (DL) to ulnar motor DL, median motor terminal latency index, and others are considered optional. Needle electromyography (EMG) is also considered optional. Muscles innervated by C8-T1 roots including thenar muscles innervated by the median nerve can be performed to rule out root disease.[16] A drop in amplitude of median compound muscle action potential (CMAP) from the elbow stimulation site to the wrist stimulation site while recording in the median-innervated thenar muscles should raise the possibility of the presence of a Martin–Gruber anastomosis.

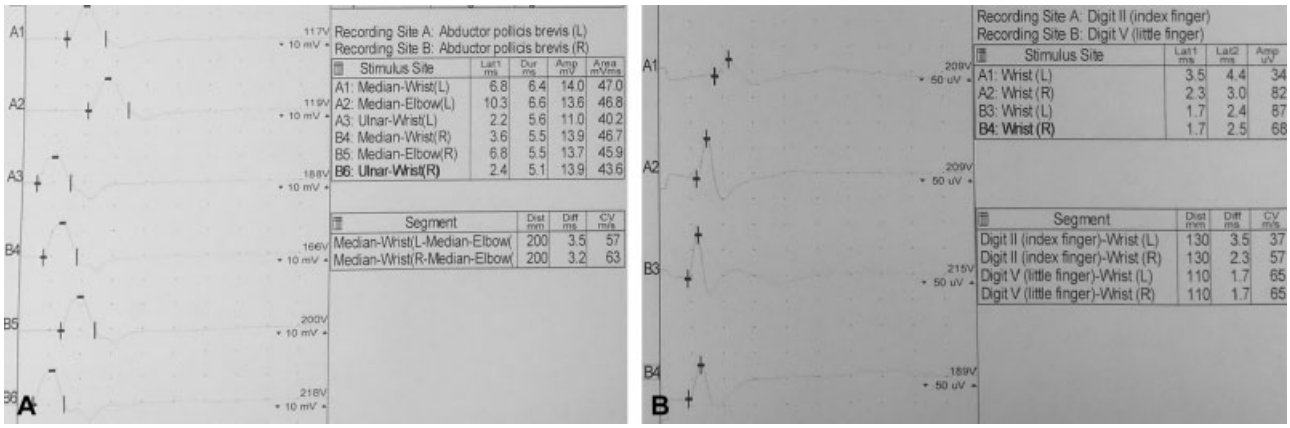

Fig. 2 Left median neuropathy at the wrist. A 65-year-old female presenting with left hand numbness. (**A**) Left median nerve motor response showed a moderately prolonged distal motor latency with normal compound muscle action potential (CMAP) amplitude and normal forearm conduction velocity. (**B**) Left median nerve sensory response showed moderately reduced sensory nerve action potential (SNAP) amplitude with a moderately prolonged peak latency and slow conduction velocity across the wrist.

In AIS, motor NCS of the AIN using surface electrodes may be of limited value because the prolonged motor latency to the FPL may be masked by the normal motor latency to the other median-innervated muscles of the forearm. Needle EMG may show neuropathic changes in the PQ, FPL, and FDP muscles.[17] NCS and needle EMG are of limited value in confirming the diagnosis of PS.[18]

Ultrasonography

AANEM guidelines recommend performing diagnostic ultrasound in suspected CTS when the technique is available.[19] Studies have shown that the cross-sectional area (CSA) of the median nerve greater than 10 to 12 mm^2 has a sensitivity of 89% and a specificity of 90% in diagnosing CTS.[20] However, electrophysiological studies remain the gold standard for the diagnosis of CTS.

Treatment

The American Academy of Orthopedic Surgeons' guidelines suggest that conservative treatment such as wrist immobilization and steroid injection into the carpal tunnel have good evidence in improving patient outcomes in CTS. There is less evidence for oral steroids, laser therapy, ketoprofen phonophoresis, and therapeutic ultrasound. Surgical release of the median nerve showed a greater benefit in comparison to conservative treatment in the long term.[21] Selecting treatment options should be individualized according to the severity of the condition and coexisting comorbidities. A trial of immobilization with wrist splinting (mainly at night) and oral anti-inflammatory agents can be recommended in mild CTS before surgery, while patients with severe CTS should be offered the surgical option directly.

Evidence for treatment of PS and AIS are lacking. Several reports suggested that conservative treatment alone is enough and can lead to recovery after 12 to 18 months in PS and AIS.[22,23] Surgical decompression might be attempted, although published small series reported controversial results.[22]

Ulnar Nerve Entrapment

Anatomy

The ulnar nerve is formed mainly by the C8-T1 nerve roots through the lower trunk and via the medial cord of the brachial plexus. It descends through the medial arm, piercing the medial intermuscular septum, and passes through the arcade of Struthers. At the level of the elbow, the ulnar nerve passes superficially between the medial epicondyle and olecranon, the so-called ulnar groove. Anconeus epitrochlearis muscle, an anomalous muscle that lies between the medial epicondyle and olecranon, may be found in up to 34% of individuals and may cause compression of the ulnar nerve.[24] Distal to the elbow, the ulnar nerve passes through the cubital tunnel. The medial wall of the cubital tunnel is formed by the medial epicondyle, while the lateral border is formed by the olecranon, medial collateral ligament, and thickened fascia (arcuate ligament) between the two heads of the flexor carpi ulnaris muscle (FCU). The roof is formed by the epicondyle-olecranon ligament (Osborne ligament), while the floor is formed by the elbow capsule. The ulnar nerve innervates the FCU and FDP muscles to digits 4 and 5 in this area. In the forearm, the ulnar nerve travels medial to the ulnar artery. At the distal forearm, the dorsal ulnar sensory cutaneous branch takes off proximal to the wrist and innervates the dorsal medial hand. At the level of the wrist, the ulnar nerve enters the Guyon's canal, which is formed by the wrist bones (pisiform, hamate, and triquetrum bones) and the transverse carpal ligament. Guyon's canal is divided according to a three-zone classification. Zone 1 contains the ulnar nerve trunk proximal to its superficial and deep branches, zone 2 contains the deep motor branch of the ulnar nerve, and zone 3 includes the superficial sensory branch.[25] The deep motor branch innervates the flexor digiti minimi, opponens digiti minimi, and abductor minimi digiti (ADM) muscles, then terminates by innervating the interosseous, 3rd and 4th lumbricals, adductor pollicis muscles, and part of the flexor pollicis brevis muscle. The superficial branch of the

ulnar nerve gives sensory innervation to the medial part of the 4th and 5th finger, as well as the space between them.

Clinical

Compression of the ulnar nerve may occur in the forearm at the level of the ligament of Struthers, anconeus epitrochlearis muscle (if present), ulnar groove or cubital canal due to stretching, and elbow flexion/extension leading to either compression or subluxation of the nerve from the groove. Less commonly, the ulnar nerve can be entrapped at the level of the Guyon's canal by a bony spur, ulnar artery, aneurysm, or cyst.

Ulnar nerve entrapment at the elbow is the second most common form of entrapment after CTS. Ulnar nerve entrapment at the elbow can cause sensory and/or motor symptoms. Patients may complain of numbness/tingling involving the medial hand and 4th and 5th fingers. Pain may be present in the ulnar groove, and may be precipitated by palpation with simultaneous flexion and extension of the elbow. Patients may complain of reduced hand dexterity and weakness of the intrinsic hand muscles. They may also report inability to form a full grip (weakness of ulnar FDP muscles), or may note that their 5th finger is getting trapped outside while trying to insert their hand in their pocket. Upon inspection and in severe cases, atrophy of intrinsic hand muscles and appearance of the Benediction posture may be present. In this case, there is a resting extension in the 4th and 5th metacarpal joints, with flexion in the 4th and 5th proximal and distal interphalangeal joints secondary to weakness in the 3rd and 4th lumbricals. The "Froment's sign" may be also evident, with the patient try to hold a piece of paper with their thumb and index fingers. In this case, there will be compensatory flexion of these two fingers (median innervation) due to weakness of the intrinsic muscles. Absence of sensory symptoms in the dorsal medial hand may indicate ulnar nerve compression at the level of the wrist, thus sparing the dorsal ulnar sensory cutaneous branch.

Electrophysiological Studies

Electrophysiological testing is considered the gold standard for diagnosing ulnar neuropathy at the elbow. The AANEM has released a practice parameter for the correct use of this diagnostic tool.[26] It is recommended that NCS should be performed with the elbow being in a moderately flexed position (70–90 degrees) to get a true ulnar nerve length. The routine motor NCS of the ulnar nerve should include stimulation at the wrist, below the elbow, and above the elbow while placing the recording electrodes over the ADM (►**Fig. 3**). A 10-cm distance between the two stimulation sites across the elbow has the highest yield in finding the entrapment. The distance from the elbow (medial epicondyle) to the distal stimulation point should not be greater than 3 to 4 cm. Several abnormal findings may suggest a focal lesion across the elbow: absolute motor nerve conduction velocity (NCV) from above to below elbow of less than 50 m/s, an above to below elbow NCV slowing of at least 10 m/s in comparison the wrist-below elbow segment NCV, or a 20%

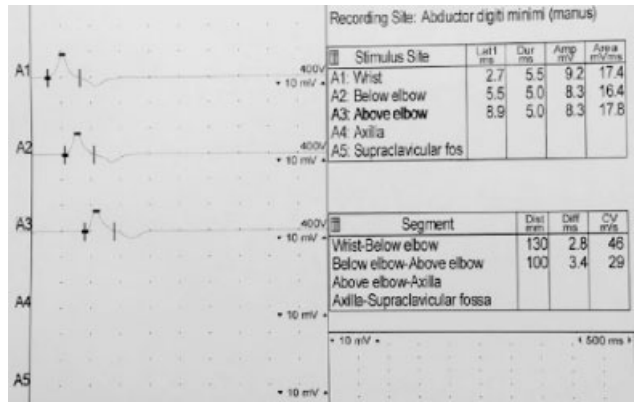

Fig. 3 Ulnar neuropathy at the elbow. A 43-year-old man presenting with numbness in the right ulnar nerve sensory distribution. Nerve conduction study revealed a significantly slow conduction velocity across the elbow segment.

reduction in CMAP amplitude and/or area from below to above elbow stimulation sites (conduction block or temporal dispersion). As for sensory NCS, side-to-side comparison is important specially when the ulnar sensory response is within normal limits but clinical suspicion is high. Motor NCS recording from the first dorsal interosseous muscle (FDI) may help in fascicular involvement where the fascicles to ADM are spared (►**Fig. 4**). A 1- or 2.5-cm incremental inching study between the below to above elbow stimulation sites looking for abnormal changes in CMAP amplitude, area, or latency may further aid in diagnosis and localization. Needle EMG examination of ulnar innervated muscles including the ADM or FDI and other nonulnar innervated C8-T1 muscles, lower trunk, and medial cord muscles may help in excluding other lesions.

There is no consensus to diagnose ulnar neuropathy at the wrist. In addition to the routine motor and sensory studies that are used to evaluate the ulnar nerve at the elbow, other less commonly used techniques are recommended and include ulnar motor NCS with stimulation across the wrist while recording from the FDI. The resulting DL can be

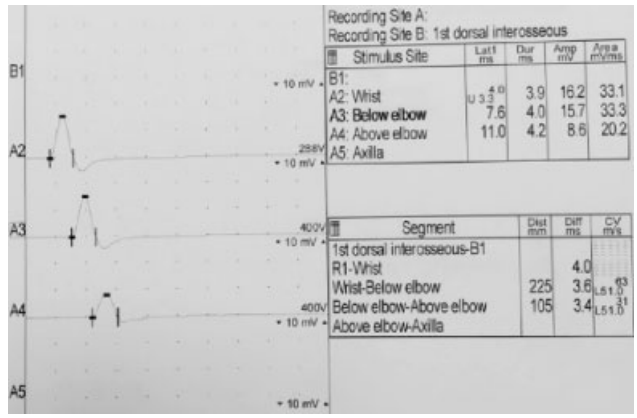

Fig. 4 A 34-year-old man presenting with numbness in the medial aspect of the left hand. Left ulnar motor nerve conduction studies recording over the first dorsal interosseous muscle reveal a partial conduction block with slowing of conduction velocity across the elbow segment.

compared with the recorded DL from the contralateral FDI and ipsilateral ADM.[27] A normal sensory NCS of the dorsal ulnar cutaneous nerve may further support a diagnosis of ulnar neuropathy at the wrist.[28] A conduction block found on inching study across the wrist while recording from the FDI may increase the sensitivity of the study and give a more precise localization.[29] Another technique is to stimulate proximally and distally (palmar) to the wrist while recording from the FDI, looking for conduction block or focal slowing across the wrist.[30]

Ultrasonography

As in CTS, electrophysiological studies remain the gold standard for diagnosis. Ultrasonography may aid in confirming the diagnosis and visualization of the injured ulnar nerve. The CSA of a normal ulnar nerve at the elbow is 8 to 11 mm^2, and an increase in these values may suggest an injury possibly due to entrapment.[31] The sensitivity and specificity of diagnosing ulnar neuropathy at the elbow by ultrasound reaches up to 93 and 50%, respectively.[32] Ultrasonographic examination of the ulnar nerve at the wrist has been less frequently studied. One study reported a normal CSA at the wrist crease at around 5.9 mm^2,[33] while another study reported a normative CSA at the level of Guyon's canal around 7 mm^2.[34]

Treatment

There is no consensus or guidelines concerning the best treatment modalities for ulnar nerve entrapment at the elbow. Conservative treatment is recommended as a start in mild to moderate conditions. A mean period of 22-month follow-up of patients with mild cubital tunnel syndrome resulted in symptom resolution in 90% of cases.[35] Another study involved the application of night splinting, nerve gliding, or just informing patients about the diagnosis resulted in subjective and objective improvement of symptoms in 89.5% of patients in mild to moderate cubital tunnel syndrome after a 6-month follow-up.[36] Local injection of corticosteroids was found to be ineffective when compared with placebo.[37]

Surgical treatment is usually conserved for severe cases, or failure of conservative treatment after 3 or more months. The two proposed surgical modalities are simple nerve decompression or nerve transposition surgeries. A Cochrane review of 9 randomized controlled trials involving 587 participants with ulnar neuropathy at the elbow showed that both surgical options were equally effective, but the transposition was associated with higher possibilities of complications.[38]

Treatment of ulnar nerve entrapment at the wrist may include conservative and/or surgical options. The approach is the same as in ulnar entrapment at the elbow, where it is recommended to start with conservative treatment in mild to moderate cases, which consists of patient education, avoiding mechanical overload on the wrist, and splinting for up to 12 weeks. In severe conditions or in case of failure of conservative treatment, surgical exploration and decompression of the nerve is recommended.[39,40]

Radial Nerve Entrapment

Anatomy

The radial nerve receives innervation from C5 to T1 roots through the posterior divisions of the three trunks, and is actually the continuation of the posterior cord. At the proximal part of the arm, the radial nerve gives off three branches: the posterior cutaneous nerve of the arm, the lower lateral cutaneous nerve of the arm, and the posterior cutaneous nerve of the forearm. This is followed by branches innervating the triceps brachii muscle (all three heads), and the anconeus. In the middle arm, the radial nerve wraps around the posterior humerus bone at the spiral groove. At the elbow level and anterior to the lateral epicondyle, the radial nerve gives branches to the brachioradialis and extensor carpi radialis muscles, then divides into two branches (superficial and deep) just after passing the lateral epicondyle. The superficial branch moves over the radial bone without giving any branches in the forearm. After passing the wrist, the superficial branch gives sensory innervation to the lateral part of the dorsal aspect of the hand, as well as to the dorsal aspect of the thumb and dorsal aspects of the proximal phalanges of the 2nd, 3rd, and 4th digits. The deep branch innervates the supinator and the extensor carpi radialis brevis muscles, before piercing through the arcade of Frohse. The arcade of Frohse is a membranous structure which forms the superficial layer of the supinator muscle. The mean distance from the lateral epicondyle to the arcade of Frohse is about one-fifth of the distance from the lateral epicondyle to the styloid process of the radial bone, while its length is approximately 0.8 cm.[41] After entering this structure, the deep branch becomes the posterior interosseous nerve (PIN). The PIN gives several motor branches to innervate wrist, thumb, and finger extensors, including the extensor digitorum communis (EDC), extensor carpi ulnaris, extensor indicis proprius (EIP), abductor pollicis longus, and extensor pollicis longus and brevis muscles.

Clinical

The radial nerve is less commonly entrapped compared with the ulnar and median nerves. It can be compressed at several levels along its path. Compression can occur at the axilla, spiral groove, along the PIN course, or along the superficial branch of the nerve.

Radial nerve entrapment at the level of the spiral groove of the humerus is the most common site of entrapment. It can occur due to prolonged immobilization or direct compression of the nerve over the humeral bone in case of prolonged bending of the arm over a chair while being under the effect of an intoxicant (Saturday night palsy). Humeral fracture is the most common secondary cause of radial nerve entrapment at this level. Patients usually present with an extended limb in the pronated position (weak brachioradialis and supinator muscles), and a wrist drop (weakness of wrist and finger extensor muscles). Sensory deficit may involve the lateral part of the dorsal aspect of the hand and the dorsal aspect of the first three digits (superficial sensory radial branch). Physical examination reveals spared triceps muscle function.

Radial nerve compression may also occur in the axilla, usually from an inappropriate use of crutches. The clinical picture is the same as in the case of entrapment at the spiral groove with the additional involvement of the triceps muscle (weakness in elbow extension) and sensory deficit in the lateral arm and posterior aspect of the forearm. Physical examination should include the deltoid and latissimus dorsi muscles to exclude a more proximal lesion (posterior cord).

A form of radial nerve entrapment is compression of the PIN by the arcade of Frohse in the proximal forearm. This is caused by repetitive supination or by external compression. Patients usually complain of local pain precipitated by supination with weakness of finger extension but relative sparing of wrist extension with no sensory deficit.

Cheiralgia paresthetica is a radial neuropathy syndrome that involves the superficial sensory radial nerve branch due to external compression of tight objects over the wrist (superficial branch passes over the radial bone). Patients usually complain of numbness and paresthesias in the radial nerve sensory territory over the dorsal aspect of the hand and fingers with an absence of weakness.

Electrophysiological Studies

There are no guidelines on electrodiagnostic studies of the radial nerve. Radial motor NCS should be performed on both limbs and should include recording of EIP muscle (recording electrode 2–3 cm proximal to the ulnar styloid with reference electrode over the ulnar styloid) while stimulating over the forearm, lateral elbow, below the spiral groove (area between biceps and triceps muscles), and above the spiral groove. A drop in CMAP amplitude and/or area, presence of temporal dispersion, conduction block, or a significant difference in amplitude between the two limbs (50% side-to-side comparison) is more important than conduction velocity values, due to technical difficulty of measuring the true length of the radial nerve. Radial sensory NCS involves recording from the superficial sensory branch where the recording electrodes are placed over the extensor tendons of the thumb while stimulating proximally at the forearm over the radial bone approximately 10 cm from the recording electrode. Needle EMG should include the triceps, brachioradialis, EDC, and EIP muscles. Triceps muscle is typically spared in case of radial nerve compression at the level of the spiral groove, while both triceps and brachioradialis muscles are spared in PIN involvement. Additional examination of the deltoid, cervical paraspinals, and other nonradial innervated muscles receiving contribution from C7 nerve roots are important to exclude a more proximal lesion.[42]

Ultrasonography

Ultrasonography of the radial nerve can aid in diagnosis and localization, and can reveal possible causes of nerve entrapment. The mean CSA of the radial nerve at the spiral groove is around 10 mm^2,[43] while the mean CSA of the nerve at the arcade of Frohse is around 0.5 to 1.42 mm^2.[44] The CSA of the superficial sensory branch of the radial nerve ranges from 1 to 3 mm^2.[45] More studies are needed to increase the diagnostic value of this testing modality.

Treatment

There is no consensus concerning the treatment of radial nerve entrapment. Conservative care is the mainstay for management in the absence of secondary causes. This includes rest, physical therapy, and patient education to avoid possible postures or positions leading to further compression. A retrospective review revealed that most patients show a complete recovery in 3 to 4 months.[46] Steroid injection may relieve local pain symptoms.[47] Surgical treatment may be considered in the presence of secondary causes (external compression) as mentioned earlier, or in case of failure of conservative treatment. Variable success rates have been reported with decompression of the radial nerve.[48,49]

Fibular (Peroneal) Nerve Entrapment

Anatomy

The fibular nerve receives innervation from L4 to S1 (mainly L5) nerve roots through the sciatic nerve. The common fibular nerve runs in a separate fascicle within the sciatic nerve in the posterior thigh, where its fibers innervate the short head of the biceps femoris muscle. The common fibular nerve separates completely as an independent nerve before reaching the popliteal fossa, where the other part of the sciatic nerve becomes the tibial nerve. At the level of the popliteal fossa, the lateral cutaneous nerve of the knee branches off. After that, the fibular nerve passes around the fibular neck and through the fibular tunnel. The fibular tunnel is formed by the fibular bone and peroneus longus muscle. At that level, the fibers that will eventually form the deep fibular nerve cross more medially (near the fibular tunnel) in comparison to those that will form the superficial fibular nerve. After bifurcation, the deep fibular nerve gives off branches to the anterior compartment which includes peroneus tertius, tibialis anterior (TA), extensor digitorum longus, and extensor digitorum brevis (EDB) muscles. At the level of the ankle, the deep fibular nerve enters the anterior tarsal tunnel which is formed by the inferior extensor retinaculum (roof) and fascia overlying the talus and navicular bones (floor). After passing through this tunnel, the nerve gives sensory innervation to the web space between the first and second toes. The superficial fibular nerve gives off branches to innervate the muscles of the lateral compartment, including the peroneus longus and brevis (ankle evertors), as well as sensory innervation to the lateral two-thirds of the calf. At the foot dorsum, it divides into medial and intermediate dorsal cutaneous nerves, which give sensory innervation over the dorsal and dorsomedial parts of the foot. In up to 40% of the population, an accessory deep fibular nerve that arises from the superficial fibular nerve can be identified.[50] It travels posterior to the lateral malleolus and innervates the lateral EDB muscle.

Clinical

The fibular neck is the most common site of entrapment of the common fibular nerve, and is considered the most common type of entrapment in the lower limb.[51] It can present as an acute neuropathy caused by sedation, prolonged immobilization, force stretch, prolonged squatting, or

leg crossing. It can also present in an insidious, progressive, and chronic manner after weight loss, repetitive traumatic maneuvers (e.g., squatting, leg crossing), or after an external compression of a slowly growing tumor. Patients usually complain of motor deficit such as gait abnormality, frequent tripping, or needing to raise the limb higher than usual to avoid toe or foot dragging (steppage gait). Less commonly, patients complain of decreased sensation over the dorsum of the foot and lower lateral calf, and/or local pain in the lateral knee. Examination may reveal Tinel's sign over the fibular neck. Clinicians should pay attention to differentiating fibular neuropathy from a higher site of pathology such as sciatic nerve lesions, lumbosacral plexus lesions, or an L5 radiculopathy. All of these pathologies may present with a foot drop, weakness of foot eversion and dorsiflexion, and sensory deficits involving the same dermatomes. However, weakness of foot inversion, decreased ankle jerk reflex, or sensory deficit in the sole of the foot and lateral knee area should raise suspicion for a sciatic nerve lesion. Additional weakness in thigh abduction or sensory disturbances in the thighs may indicate an L5 radiculopathy. An objective examination of foot inversion and eversion in cases of foot drop should involve holding the foot back in its normal position before attempting to examine the motor power.

Isolated entrapment of the distal deep fibular nerve (anterior tarsal tunnel syndrome) can be either caused by an external pressure and/or local changes in the structures forming the tunnel. Several factors contributing to these causes include trauma (sprain or fracture), wearing tight shoes or high heels, osteoarticular changes in bones, abnormal postures, and local edema. Patients usually complain of numbness and paresthesia locally or in the first interdigital space. These symptoms could occur in certain foot positions or may be related to footwear. They can also occur at night. In severe cases, mild weakness in toe extension and atrophy of the EDB muscle can occur, but weakness should not become severe since other toe extensors compensate for this function.[52,53]

Superficial fibular nerve entrapment is rare and difficult to diagnose. It may be entrapped proximally between the fascia of the lateral compartment of the leg. In this case, the patient may present with weakness in foot eversion and dorsiflexion (weakness in the peroneus longus and brevis) as well as pain and a sensory deficit in the lower lateral and dorsum of the foot excluding the area innervated by the deep fibular nerve. The superficial fibular nerve can also be entrapped distally at the level of the ankle as it emerges from the deep fascia subcutaneously. In this condition, the symptoms are limited to local pain and sensory symptoms without motor deficit.[54–56]

Electrophysiological Studies

Nerve conduction studies and needle EMG are recommended to localize and diagnose patients presenting with foot drop or other presentations of a possible fibular nerve entrapment. The AANEM recommends performing fibular nerve motor NCS recording at the EDB and TA, stimulating at the ankle as well as proximally below and above the fibular head (►**Fig. 5**). It is also recommended to perform a fibular nerve sensory NCS. Additional motor and sensory NCS of other healthy nerves of the same and contralateral limb are recommended to exclude a higher lesion or a systemic pathology.[57] Fibular as well as tibial nerve F-wave latencies can be performed. Sensory NCS of the deep and superficial fibular nerve branches can further support or exclude the involvement of one or both of these nerves. Needle EMG is highly recommended to be performed in all cases, since NCS solely cannot exclude a higher lesion (sciatic nerve, lumbosacral plexus, or L5 root). This part of the examination may start with sampling of fibular innervated muscles like the TA (deep fibular nerve) and peroneus longus (superficial fibular nerve). The EDB muscle is usually avoided due to a higher rate of false positive results.[58] Muscles innervated by the tibial nerve may be sampled next, especially the posterior tibialis muscle as it derives its innervation from L5 root. Sciatic nerve involvement can be excluded by sampling hamstring muscles, especially the short head of the biceps femoris muscle since it is a part of the fibular fascicle in the sciatic nerve and its branch takes off above the fibular head. In case of positive findings in all of the above-mentioned muscles, examination of gluteal and/or lumbar paraspinal muscles is recommended.

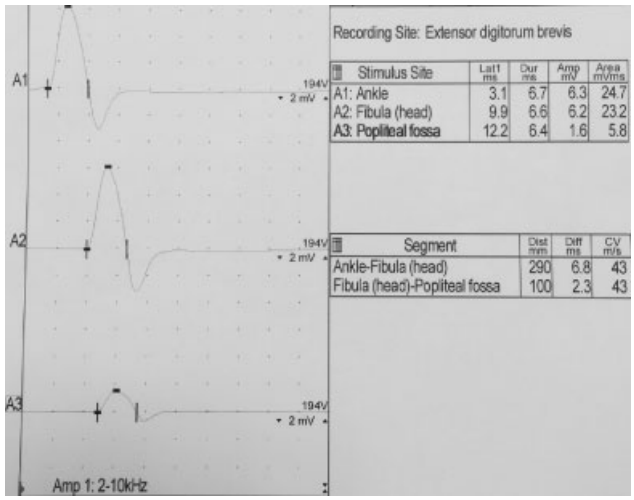

Fig. 5 A 27-year-old man presenting with right foot drop of several weeks' duration. He also reports rapid weight loss after undergoing a gastric sleeve surgery. Motor nerve conduction studies of the right fibular (peroneal) nerve showed a partial conduction block across the fibular head.

Ultrasonography

Ultrasonography may be used in fibular neuropathy. It can add valuable information to electrophysiological studies regarding the cause, exact area, and severity of entrapment, where the latter can have a prognostic value. The mean CSA of the fibular nerve at the popliteal fossa and the fibular head are around 12 and 11 mm^2, respectively.[59] A study done on patients with fibular nerve entrapment at the fibular head showed that the CSA is normal in patients with conduction block, while the CSA is increased in patients with axonal damage. This is important, as a possible diagnostic/

prognostic value in cases of fibular nerve conduction block as normal CSA might indicate a better prognosis.[60] Furthermore, it was shown that the damaged common fibular nerve fascicle can be visualized and accurately delineated.[61] Similarly, ultrasonography may aid in localizing any entrapment in the superficial and deep branches of the fibular nerve.[62]

Treatment

There is no consensus concerning the management of common fibular nerve entrapment. The initial management usually starts with patient education to avoid certain positions such as squatting, leg crossing, and others. A special pad may be worn over the fibular head area to avoid spontaneous nerve compression especially at night. Ankle-foot orthoses can be used in severe cases to maintain a normal posture while walking. Physical therapy focusing on strengthening of fibular-innervated muscles is the mainstay of conservative therapy.

There are no guidelines concerning when and where the surgical approach should be entertained. Surgical exploration and repair is usually considered in cases of traumatic lesions (especially in the absence of nerve continuity on electrophysiological examination), any sudden deterioration of the clinical condition, or when there is no improvement within 3 months. Reports on surgical exploration using either open technique or microsurgery have revealed variable results; however, there are increased rates of improvement in motor and sensory deficits especially in cases of idiopathic entrapment.[63,64]

Superficial fibular nerve entrapment, once diagnosed, is usually treated conservatively with physical therapy.[65] Surgical decompression may help reduce pain in severe cases.[66] There is a lack of guidelines concerning the treatment of anterior tarsal syndrome. Currently, the approach is guided by the clinical condition and starts with conservative measures such as shoe modification and physical therapy. Surgical treatment may be attempted in selected cases.[67,68]

Tibial Nerve Entrapment

Anatomy

The tibial nerve receives a similar contribution from L4 to S1 (mainly S1) nerve roots through the sciatic nerve and becomes a completely independent nerve at the level of the popliteal fossa. The tibial nerve innervates all muscles of the posterior compartment of the leg including the superficial compartment (plantaris, soleus, and gastrocnemius) and deep compartment (popliteus, flexor hallucis longus, flexor digitorum longus, and tibialis posterior). The sensory contribution of the tibial nerve in the leg is limited to the cutaneous branch at the level of the popliteal fossa that eventually joins another branch off the common fibular nerve to form the sural nerve. At the level of the ankle, the tibial nerve travels beneath the medial malleolus and passes through the tarsal tunnel. This tunnel is formed by the medial malleolus, calcaneus, and talus bones. The tunnel's fibrous roof is formed by the flexor retinaculum (band extending from medial malleolus to medial calcaneus). In addition to the tibial nerve, this tunnel contains the tibial artery and vein, and four foot intrinsic muscle tendons. Thereafter, the tibial nerve gives one or two sensory branches to the calcaneal area of the foot (medial and lateral calcaneal nerves). Distally in the foot, the tibial nerve divides into the medial and lateral plantar nerves, which are mixed nerves. The medial plantar nerve gives motor innervation to the abductor hallucis, flexor digitorum brevis, flexor hallucis brevis, and first lumbricals, while the sensory innervation covers the medial part of the sole, the first three toes, and the medial part of the 4th toe. The lateral plantar nerve gives motor innervation to the flexor digitorum accessorius, abductor digiti minimi quinti pedis, flexor digiti minimi, 2nd, 3rd, and 4th lumbricals, and 2nd, 3rd, and 4th interosseous muscles. The sensory innervation of this nerve covers the lateral part of the sole, the 5th toe, and the lateral part of the 4th toe.

Clinical

Tarsal tunnel syndrome (compression of tibial nerve in the tarsal tunnel) is still a controversial topic. It clinically manifests with sensory symptoms including pain, numbness, burning, or tingling in the area of compression with radiation of the symptoms toward the sole or heel depending on the level of compression. Symptoms may be exacerbated by weight bearing and maybe present at night. Tinel's sign may be detected near the medial malleolus, in addition to atrophy of intrinsic foot muscles (a nonspecific finding). Idiopathic entrapment is rare. External compression due to space occupying lesions, edema, or joint/bone degenerative changes (athletes, prolonged weight bearing, inflammatory disease) are common causes.[69,70]

Morton's neuroma (intermetatarsal neuroma) is a term referred to pathological thickening of one of the common digital nerves (branches of medial or lateral plantar nerves) laying near the metatarsal heads.[71] It is still debatable whether this condition is caused by direct compression of the distal metacarpal transverse ligament or other factors such as trauma or high heels.[72] This condition affects mostly females, may be bilateral (one-fifth of cases), and is mostly located in the third interdigital space.[72] It manifests as burning, shooting, or sharp pain in the forefoot, mainly in the interdigital space radiating to the corresponding toe and proximally to the whole foot.[73]

Electrophysiological Studies

The electrophysiological testing in tarsal tunnel syndrome is difficult and may be misleading. Motor, sensory, or mixed NCS may reveal absent responses even in healthy subjects. In addition to the small obtained responses, age and effect of temperature are important factors. According to the AANEM recommendations,[74] these studies may include mixed or sensory NCS of medial and lateral plantar nerves, in addition to NCS of fibular, tibial (including F-wave latencies), and sural nerves. The latter is done to exclude plexopathy and polyneuropathy. Needle EMG of the intrinsic foot muscles of both plantar nerves (e.g., abductor hallucis brevis and abductor digiti quinti pedis) is rarely performed due to patients' pain

intolerance and presence of pathologic changes (spontaneous activity and large motor unit action potentials) in these muscles even in normal subjects.

Electrodiagnostic studies are of limited value in a Morton's neuroma. One recent study suggested that sensory NCS stimulating the medial and lateral surfaces of the two affected toes (digital nerves) while recording at the medial malleolus level has a sensitivity that may reach 57%.[75] A higher sensitivity reaching 90% was reported performing the same NCS while using subdermal electrodes.[76]

Ultrasonography

Ultrasound testing in patients with tarsal tunnel syndrome and Morton's neuroma may be of greater diagnostic value than electrophysiological testing. Ultrasonography can show enlargement of the tibial nerve distal to the tarsal tunnel compared with a level proximal to the tunnel with a sensitivity of 61% and specificity of 74%.[77,78] Ultrasonography and magnetic resonance imaging are of similar accuracy in diagnosing Morton's neuroma.[79,80]

Treatment

There are no trials to guide treatment strategies in tarsal tunnel syndrome. Choosing a treatment modality should be according to the cause and severity of each case. Conservative treatment consists of patient education and physical therapy. Decompressive surgery may be attempted in case of treatment failure or in severe cases, with up to 71% reported improvement in symptoms.[81]

Conservative treatment of Morton's neuroma starts with application of a plantar orthosis. Treatment with alcohol injections, steroid injections, botulinum injections, and extracorporeal wave therapy may lead to partial relief of symptoms.[82–85] Surgical excision yields a good to excellent outcome in more than 70% of reported cases.[86,87]

Lateral Femoral Cutaneous Nerve

Anatomy

The lateral femoral cutaneous nerve is directly derived from L2 to L3 roots. It passes over the iliac muscles toward the anterior superior iliac spine and crosses under the inguinal ligament. It becomes superficial at approximately 10 cm below the ligament and provides sensory innervation over the lateral part of the thigh. Different anatomical variations of the nerve path impose a challenge in diagnosis and surgical treatment.[88,89]

Clinical

Lateral femoral cutaneous neuropathy, or meralgia paresthetica, most commonly affects adult males. Risk factors include obesity, diabetes mellitus, occupation-related risk factors, pregnancy, local surgery, use of corticosteroids, and wearing tight fitting clothes.[90,91] Clinical manifestations include numbness, tingling, burning, or stabbing sensation in the lateral aspect of the thigh. Patients are usually able to localize the exactly affected dermatome, but symptoms may change with changes in posture. Examination may reveal tenderness over the lateral part of the inguinal ligament.

Electrophysiological Studies and Other Diagnostic Utilities

NCS of the lateral femoral cutaneous nerve should be always interpreted with caution, especially in obese patients or in case of absent responses.[42] Side-to-side amplitude ratio > 2.3 yields a higher sensitivity than sensory nerve action potential amplitude.[92] NCS of other nerves and needle EMG of selected muscles should be always performed to rule out other causes. Ultrasonographic examination may show focal enlargement in nerve diameter and reveal the possible underlying cause.[93,94]

Treatment

Most cases of meralgia paresthetica resolve with conservative treatment, which consists of avoiding tight clothes or belts, weight loss, administration of medications used in neuropathic pain, and local anesthetics. Local injection of corticosteroids, anesthetics, and radiofrequency nerve ablation may be attempted.[95,96] Surgical treatment consists of neurolysis or neurectomy. Neurectomy possibly yields more favorable outcomes according to one report.[97]

Conclusion

Entrapment neuropathies are a common group of conditions encountered in general practice and a frequent reason for neurological and orthopedic surgery referral. A detailed history, clinical examination, and electrophysiological studies done by a well-trained physician are needed to reach a definite diagnosis and exclude other causes. Recently, neuromuscular ultrasonography has emerged and has been demonstrated to be of diagnostic value, especially in detecting the etiology and precise localization, and may assist in treatment. A timely diagnosis is always needed to reach better clinical outcomes.

Conflict of Interest
None.

Acknowledgments
None.

References

1 Papanicolaou GD, McCabe SJ, Firrell J. The prevalence and characteristics of nerve compression symptoms in the general population. J Hand Surg Am 2001;26(03):460–466
2 Latinovic R, Gulliford MC, Hughes RA. Incidence of common compressive neuropathies in primary care. J Neurol Neurosurg Psychiatry 2006;77(02):263–265
3 de Krom MC, Knipschild PG, Kester AD, Thijs CT, Boekkooi PF, Spaans F. Carpal tunnel syndrome: prevalence in the general population. J Clin Epidemiol 1992;45(04):373–376
4 Heinemeyer O, Reimers CD. Ultrasound of radial, ulnar, median, and sciatic nerves in healthy subjects and patients with hereditary motor and sensory neuropathies. Ultrasound Med Biol 1999; 25(03):481–485
5 Smith RV, Fisher RG. Struthers ligament: a source of median nerve compression above the elbow. Case report. J Neurosurg 1973;38 (06):778–779
6 Gessini L, Jandolo B, Pietrangeli A. Entrapment neuropathies of the median nerve at and above the elbow. Surg Neurol 1983;19 (02):112–116

7 Johnson RK, Spinner M, Shrewsbury MM. Median nerve entrapment syndrome in the proximal forearm. J Hand Surg Am 1979;4 (01):48–51
8 Caetano EB, Vieira LA, Almeida TA, Gonzales LAM, Bona JE, Simonatto TM. Bicipital aponeurosis. Anatomical study and clinical implications. Rev Bras Ortop 2017;53(01):75–81
9 Lee KS, Oh CS, Chung IH, Sunwoo IN. An anatomic study of the Martin-Gruber anastomosis: electrodiagnostic implications. Muscle Nerve 2005;31(01):95–97
10 Makanji HS, Becker SJ, Mudgal CS, Jupiter JB, Ring D. Evaluation of the scratch collapse test for the diagnosis of carpal tunnel syndrome. J Hand Surg Eur Vol 2014;39(02):181–186
11 Boland RA, Kiernan MC. Assessing the accuracy of a combination of clinical tests for identifying carpal tunnel syndrome. J Clin Neurosci 2009;16(07):929–933
12 Suranyi L. Median nerve compression by Struthers ligament. J Neurol Neurosurg Psychiatry 1983;46(11):1047–1049
13 Caetano EB, Sabongi JJ, Vieira LA, Caetano MF, de Bona JE, Simonatto TM. Struthers' ligament and supracondilar humeral process: an anatomical study and clinical implications. Acta Ortop Bras 2017;25(04):137–142
14 Hartz CR, Linscheid RL, Gramse RR, Daube JR. The pronator teres syndrome: compressive neuropathy of the median nerve. J Bone Joint Surg Am 1981;63(06):885–890
15 Hill NA, Howard FM, Huffer BR. The incomplete anterior interosseous nerve syndrome. J Hand Surg Am 1985;10(01):4–16
16 American Association of Electrodiagnostic Medicine, American Academy of Neurology, and American Academy of Physical Medicine and Rehabilitation. Practice parameter for electrodiagnostic studies in carpal tunnel syndrome: summary statement. Muscle Nerve 2002;25(06):918–922
17 Seror P. Anterior interosseous nerve lesions. Clinical and electrophysiological features. J Bone Joint Surg Br 1996;78(02):238–241
18 Olehnik WK, Manske PR, Szerzinski J. Median nerve compression in the proximal forearm. J Hand Surg Am 1994;19(01):121–126
19 Cartwright MS, Hobson-Webb LD, Boon AJ, et al; American Association of Neuromuscular and Electrodiagnostic Medicine. Evidence-based guideline: neuromuscular ultrasound for the diagnosis of carpal tunnel syndrome. Muscle Nerve 2012;46(02):287–293
20 Fowler JR, Munsch M, Tosti R, Hagberg WC, Imbriglia JE. Comparison of ultrasound and electrodiagnostic testing for diagnosis of carpal tunnel syndrome: study using a validated clinical tool as the reference standard. J Bone Joint Surg Am 2014;96(17):e148
21 Graham B, Peljovich AE, Afra R, et al. The American Academy of Orthopaedic Surgeons Evidence-Based Clinical Practice Guideline on: management of carpal tunnel syndrome. J Bone Joint Surg Am 2016;98(20):1750–1754
22 Miller-Breslow A, Terrono A, Millender LH. Nonoperative treatment of anterior interosseous nerve paralysis. J Hand Surg Am 1990;15(03):493–496
23 Nakano KK, Lundergran C, Okihiro MM. Anterior interosseous nerve syndromes. Diagnostic methods and alternative treatments. Arch Neurol 1977;34(08):477–480
24 Gruber W. The anomalous course of the ulnar nerve in front of the medial epicondyle [In German]. Arch Anat Physiol Wissen Med 1867:560–564
25 Gross MS, Gelberman RH. The anatomy of the distal ulnar tunnel. Clin Orthop Relat Res 1985;196:238–247
26 Campbell WW; American Association of Electrodiagnostic Medicine. Guidelines in electrodiagnostic medicine. Practice parameter for electrodiagnostic studies in ulnar neuropathy at the elbow. Muscle Nerve Suppl 1999;8:S171–S205
27 Olney RK, Wilbourn AJ. Ulnar nerve conduction study of the first dorsal interosseous muscle. Arch Phys Med Rehabil 1985;66(01): 16–18
28 Jabre JF. Ulnar nerve lesions at the wrist: new technique for recording from the sensory dorsal branch of the ulnar nerve. Neurology 1980;30(08):873–876
29 McIntosh KA, Preston DC, Logigian EL. Short-segment incremental studies to localize ulnar nerve entrapment at the wrist. Neurology 1998;50(01):303–306
30 Cowdery SR, Preston DC, Herrmann DN, Logigian EL. Electrodiagnosis of ulnar neuropathy at the wrist: conduction block versus traditional tests. Neurology 2002;59(03):420–427
31 Beekman R, Visser LH, Verhagen WI. Ultrasonography in ulnar neuropathy at the elbow: a critical review. Muscle Nerve 2011;43 (05):627–635
32 Ellegaard HR, Fuglsang-Frederiksen A, Hess A, Johnsen B, Qerama E. High-resolution ultrasound in ulnar neuropathy at the elbow: a prospective study. Muscle Nerve 2015;52(05):759–766
33 Cartwright MS, Shin HW, Passmore LV, Walker FO. Ultrasonographic findings of the normal ulnar nerve in adults. Arch Phys Med Rehabil 2007;88(03):394–396
34 Peeters EY, Nieboer KH, Osteaux MM. Sonography of the normal ulnar nerve at Guyon's canal and of the common peroneal nerve dorsal to the fibular head. J Clin Ultrasound 2004;32(08):375–380
35 Eisen A, Danon J. The mild cubital tunnel syndrome. Its natural history and indications for surgical intervention. Neurology 1974; 24(07):608–613
36 Svernlöv B, Larsson M, Rehn K, Adolfsson L. Conservative treatment of the cubital tunnel syndrome. J Hand Surg Eur Vol 2009;34 (02):201–207
37 vanVeen KE, Alblas KC, Alons IM, et al. Corticosteroid injection in patients with ulnar neuropathy at the elbow: a randomized, double-blind, placebo-controlled trial. Muscle Nerve 2015;52(03):380–385
38 Caliandro P, La Torre G, Padua R, Giannini F, Padua L. Treatment for ulnar neuropathy at the elbow. Cochrane Database Syst Rev 2016; 11:CD006839
39 Hoogvliet P, Coert JH, Fridén J, Huisstede BM; European HANDGUIDE group. How to treat Guyon's canal syndrome? Results from the European HANDGUIDE study: a multidisciplinary treatment guideline. Br J Sports Med 2013;47(17):1063–1070
40 Coraci D, Loreti C, Piccinini G, Doneddu PE, Biscotti S, Padua L. Ulnar neuropathy at wrist: entrapment at a very "congested" site. Neurol Sci 2018;39(08):1325–1331
41 Ozturk A, Kutlu C, Taskara N, Kale AC, Bayraktar B, Cecen A. Anatomic and morphometric study of the arcade of Frohse in cadavers. Surg Radiol Anat 2005;27(03):171–175
42 Preston DC, Shapiro BE. Electromyography and Neuromuscular Disorders: Clinical-Electrophysiologic Correlations. Boston: Butterworth-Heinemann; 2013:336–340
43 Cartwright MS, Mayans DR, Gillson NA, Griffin LP, Walker FO. Nerve cross-sectional area in extremes of age. Muscle Nerve 2013;47(06):890–893
44 Babaei-Ghazani A, Roomizadeh P, Sanaei G, et al. Ultrasonographic reference values for the deep branch of the radial nerve at the arcade of Frohse. J Ultrasound 2018;21(03):225–231
45 Visser LH. High-resolution sonography of the superficial radial nerve with two case reports. Muscle Nerve 2009;39(03):392–395
46 Arnold WD, Krishna VR, Freimer M, Kissel JT, Elsheikh B. Prognosis of acute compressive radial neuropathy. Muscle Nerve 2012;45 (06):893–895
47 Sarhadi NS, Korday SN, Bainbridge LC. Radial tunnel syndrome: diagnosis and management. J Hand Surg [Br] 1998;23(05):617–619
48 Bolster MA, Bakker XR. Radial tunnel syndrome: emphasis on the superficial branch of the radial nerve. J Hand Surg Eur Vol 2009;34 (03):343–347
49 Simon Perez C, García Medrano B, Rodriguez Mateos JI, Coco Martin B, Faour Martin O, Martin Ferrero MA. Radial tunnel syndrome: results of surgical decompression by a postero-lateral approach. Int Orthop 2014;38(10):2129–2135
50 Tomaszewski KA, Roy J, Vikse J, Pękala PA, Kopacz P, Henry BM. Prevalence of the accessory deep peroneal nerve: a cadaveric study and meta-analysis. Clin Neurol Neurosurg 2016;144:105–111
51 Craig A. Entrapment neuropathies of the lower extremity. PM R 2013;5(5, Suppl):S31–S40

52 Gessini L, Jandolo B, Pietrangeli A. The anterior tarsal syndrome. Report of four cases. J Bone Joint Surg Am 1984;66(05):786–787
53 Logullo F, Ganino C, Lupidi F, Perozzi C, Di Bella P, Provinciali L. Anterior tarsal tunnel syndrome: a misunderstood and a misleading entrapment neuropathy. Neurol Sci 2014;35(05):773–775
54 Henry AK. Extensile Exposure. Edinburgh, UK: E&S Livingstone; 1945
55 Flanigan RM, DiGiovanni BF. Peripheral nerve entrapments of the lower leg, ankle, and foot. Foot Ankle Clin 2011;16(02):255–274
56 Styf J. Entrapment of the superficial peroneal nerve. Diagnosis and results of decompression. J Bone Joint Surg Br 1989;71(01):131–135
57 Marciniak C, Armon C, Wilson J, Miller R. Practice parameter: utility of electrodiagnostic techniques in evaluating patients with suspected peroneal neuropathy: an evidence-based review. Muscle Nerve 2005;31(04):520–527
58 Morgenlander JC, Sanders DB. Spontaneous EMG activity in the extensor digitorum brevis and abductor hallucis muscles in normal subjects. Muscle Nerve 1994;17(11):1346–1347
59 Cartwright MS, Passmore LV, Yoon JS, Brown ME, Caress JB, Walker FO. Cross-sectional area reference values for nerve ultrasonography. Muscle Nerve 2008;37(05):566–571
60 Tsukamoto H, Granata G, Coraci D, Paolasso I, Padua L. Ultrasound and neurophysiological correlation in common fibular nerve conduction block at fibular head. Clin Neurophysiol 2014;125 (07):1491–1495
61 Bignotti B, Cadoni A, Assini A, Martinoli C, Tagliafico A. Fascicular involvement in common fibular neuropathy: evaluation with ultrasound. Muscle Nerve 2016;53(04):532–537
62 Luz J, Johnson AH, Kohler MJ. Point-of-care ultrasonography in the diagnosis and management of superficial peroneal nerve entrapment: case series. Foot Ankle Int 2014;35(12):1362–1366
63 Maalla R, Youssef M, Ben Lassoued N, Sebai MA, Essadam H. Peroneal nerve entrapment at the fibular head: outcomes of neurolysis. Orthop Traumatol Surg Res 2013;99(06):719–722
64 Souter J, Swong K, McCoyd M, Balasubramanian N, Nielsen M, Prabhu VC. Surgical Results of Common Peroneal Nerve Neuroplasty at Lateral Fibular Neck. World Neurosurg 2018;112:e465–e472
65 Anandkumar S. Physical therapy management of entrapment of the superficial peroneal nerve in the lower leg: a case report. Physiother Theory Pract 2012;28(07):552–561
66 Franco MJ, Phillips BZ, Lalchandani GR, Mackinnon SE. Decompression of the superficial peroneal nerve: clinical outcomes and anatomical study. J Neurosurg 2017;126(01):330–335
67 Lui TH. Endoscopic anterior tarsal tunnel release: a case report. J Foot Ankle Surg 2014;53(02):186–188
68 Yassin M, Garti A, Weissbrot M, Heller E, Robinson D. Treatment of anterior tarsal tunnel syndrome through an endoscopic or open technique. Foot 2015;25(03):148–151
69 McSweeney SC, Cichero M. Tarsal tunnel syndrome-a narrative literature review. Foot 2015;25(04):244–250
70 Hong CH, Lee YK, Won SH, Lee DW, Moon SI, Kim WJ. Tarsal tunnel syndrome caused by an uncommon ossicle of the talus: a case report. Medicine (Baltimore) 2018;97(25):e11008
71 Morscher E, Ulrich J, Dick W. Morton's intermetatarsal neuroma: morphology and histological substrate. Foot Ankle Int 2000;21 (07):558–562
72 Alexander IJ, Johnson KA, Parr JW. Morton's neuroma: a review of recent concepts. Orthopedics 1987;10(01):103–106
73 Di Caprio F, Meringolo R, Shehab Eddine M, Ponziani L. Morton's interdigital neuroma of the foot: a literature review. Foot Ankle Surg 2018;24(02):92–98
74 Patel AT, Gaines K, Malamut R, Park TA, Toro DR, Holland N; American Association of Neuromuscular and Electrodiagnostic Medicine. Usefulness of electrodiagnostic techniques in the evaluation of suspected tarsal tunnel syndrome: an evidence-based review. Muscle Nerve 2005;32(02):236–240
75 Aydinlar EI, Uzun M, Beksac B, Ozden VE, Karaarslan E, Oge AE. Simple electrodiagnostic method for Morton neuroma. Muscle Nerve 2014;49(02):193–197
76 Pardal-Fernández JM, Palazón-García E, Hernández-Fernández F, de Cabo C. Contribution of a new electrophysiologic test to Morton's neuroma diagnosis. Foot Ankle Surg 2014;20(02):109–114
77 Samarawickrama D, Therimadasamy AK, Chan YC, Vijayan J, Wilder-Smith EP. Nerve ultrasound in electrophysiologically verified tarsal tunnel syndrome. Muscle Nerve 2016;53(06):906–912
78 Tawfik EA, El Zohiery AK, Abouelela AA. Proposed sonographic criteria for the diagnosis of idiopathic tarsal tunnel syndrome. Arch Phys Med Rehabil 2016;97(07):1093–1099
79 Bignotti B, Signori A, Sormani MP, Molfetta L, Martinoli C, Tagliafico A. Ultrasound versus magnetic resonance imaging for Morton neuroma: systematic review and meta-analysis. Eur Radiol 2015; 25(08):2254–2262
80 Torres-Claramunt R, Ginés A, Pidemunt G, Puig L, de Zabala S. MRI and ultrasonography in Morton's neuroma: diagnostic accuracy and correlation. Indian J Orthop 2012;46(03):321–325
81 Reichert P, Zimmer K, Wnukiewicz W, Kuliński S, Mazurek P, Gosk J. Results of surgical treatment of tarsal tunnel syndrome. Foot Ankle Surg 2015;21(01):26–29
82 Gurdezi S, White T, Ramesh P. Alcohol injection for Morton's neuroma: a five-year follow-up. Foot Ankle Int 2013;34(08):1064–1067
83 Thomson CE, Beggs I, Martin DJ, et al. Methylprednisolone injections for the treatment of Morton neuroma: a patient-blinded randomized trial. J Bone Joint Surg Am 2013;95(09):790–798, S1
84 Climent JM, Mondéjar-Gómez F, Rodríguez-Ruiz C, Díaz-Llopis I, Gómez-Gallego D, Martín-Medina P. Treatment of Morton neuroma with botulinum toxin A: a pilot study. Clin Drug Investig 2013;33(07):497–503
85 Seok H, Kim SH, Lee SY, Park SW. Extracorporeal shockwave therapy in patients with Morton's neuroma: a randomized, placebo-controlled trial. J Am Podiatr Med Assoc 2016;106(02):93–99
86 Kasparek M, Schneider W. Surgical treatment of Morton's neuroma: clinical results after open excision. Int Orthop 2013;37(09): 1857–1861
87 Bucknall V, Rutherford D, MacDonald D, Shalaby H, McKinley J, Breusch SJ. Outcomes following excision of Morton's interdigital neuroma: a prospective study. Bone Joint J 2016;98-B(10): 1376–1381
88 de Ridder VA, de Lange S, Popta JV. Anatomical variations of the lateral femoral cutaneous nerve and the consequences for surgery. J Orthop Trauma 1999;13(03):207–211
89 Lee SH, Shin KJ, Gil YC, Ha TJ, Koh KS, Song WC. Anatomy of the lateral femoral cutaneous nerve relevant to clinical findings in meralgia paresthetica. Muscle Nerve 2017;55(05):646–650
90 van Slobbe AM, Bohnen AM, Bernsen RM, Koes BW, Bierma-Zeinstra SM. Incidence rates and determinants in meralgia paresthetica in general practice. J Neurol 2004;251(03):294–297
91 Weng WC, Wei YC, Huang WY, Chien YY, Peng TI, Wu CL. Risk factor analysis for meralgia paresthetica: a hospital-based study in Taiwan. J Clin Neurosci 2017;43:192–195
92 Seror P, Seror R. Meralgia paresthetica: clinical and electrophysiological diagnosis in 120 cases. Muscle Nerve 2006;33(05):650–654
93 Onat SS, Ata AM, Ozcakar L. Ultrasound-guided diagnosis and treatment of meralgia paresthetica. Pain Physician 2016;19(04): E667–E669
94 Aravindakannan T, Wilder-Smith EP. High-resolution ultrasonography in the assessment of meralgia paresthetica. Muscle Nerve 2012;45(03):434–435
95 Tumber PS, Bhatia A, Chan VW. Ultrasound-guided lateral femoral cutaneous nerve block for meralgia paresthetica. Anesth Analg 2008;106(03):1021–1022
96 Fowler IM, Tucker AA, Mendez RJ. Treatment of meralgia paresthetica with ultrasound-guided pulsed radiofrequency ablation of the lateral femoral cutaneous nerve. Pain Pract 2012;12(05):394–398
97 Payne R, Seaman S, Sieg E, Langan S, Harbaugh K, Rizk E. Evaluating the evidence: is neurolysis or neurectomy a better treatment for meralgia paresthetica? Acta Neurochir (Wien) 2017;159(05): 931–936