

ENTRAPMENT NEUROPATHIES

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This presentation will use a case-based format to highlight the clinical presentation, diagnosis, and treatment of some of the most common entrapment neuropathies of the upper and lower limbs. The primary focus will be entrapment neuropathies related to chronic compression with less attention given to focal neuropathies related to acute compression, such as radial neuropathy at the spiral groove (Saturday Night's palsy).

General Concepts

Focal neuropathies may occur in the setting of acute or chronic mechanical compression, ischemia, inflammation, infection, tumor infiltration, as well as other conditions. Entrapment neuropathy describes a subset of focal neuropathies related to chronic compression of a peripheral nerve resulting in associated pain, paresthesia, and loss of sensory and/or motor function of that nerve. Entrapment may occur with essentially any peripheral nerve, but certain nerves are particularly vulnerable due to anatomic considerations such as a narrow anatomic course, superficial location, nearby osseous structures, or fibro-osseous tunnels through which a nerve passes. The clinical presentation of specific entrapment syndromes depends on several factors including the chronicity, location, severity, and mechanism of involvement.

The pathophysiology of entrapment neuropathy has been attributed to chronic mechanical pressure and associated ischemia and edema. The acute changes of entrapment neuropathy include focal demyelination and with prolonged entrapment Wallerian-like degeneration may be seen. Certain systemic or generalized conditions may predispose an individual to the development of entrapment neuropathy. Additionally, there are genetic factors that may predispose to certain nerve entrapment syndromes. A classic but infrequent example is hereditary neuropathy with predisposition to pressure palsies (HNPP). Individuals with HNPP may present with multiple focal neuropathies typically at common sites of entrapment. HNPP is an autosomal dominant disorder that leads to increased susceptibility to nerve compression and is related to a PMP22 deletion, or rarely a point mutation, on chromosome 17p11.2-12. Coexistent medical conditions may increase an individual's risk for the development of peripheral nerve entrapment including endocrinologic disorders (diabetes, hypothyroidism, and acromegaly), the presence of a generalized neuropathy, rapid weight loss, as well as focal or generalized edema.

Diagnosis of entrapment neuropathy can often be made on clinical grounds alone, but testing with electromyography and nerve conduction studies (EMG and NCS), imaging modalities such as ultrasound and MRI, as well as laboratory studies help confirm the diagnosis and exclude other mimicking or coexistent disorders. The goal of the evaluation of entrapment neuropathies should be to localize precisely, determine the severity, and, if possible, determine the underlying etiology of the entrapment. As always the examination should localize the lesion, but factors such as fascicular involvement are not uncommon and may at times be misleading. EMG and NCS remains the primary mode of evaluation for most patients with suspected entrapment neuropathy. The findings in entrapment neuropathy were initially described in 1956.¹ Characteristic findings include slowed nerve action potential propagation as measured by prolonged distal latency or reduced conduction velocity across sites of chronic compression supporting an underlying pathophysiology of demyelination. With increasing severity, amplitude loss may be seen on sensory and motor nerve conduction studies consistent with axon dysfunction and loss.

Entrapment neuropathy treatment strategies are designed around the core concept that chronic mechanical compression underlies the development focal demyelination and subsequent axonal dysfunction and loss. In some instances simply avoiding provoking postures, such as leaning on one's elbow in ulnar neuropathy at the elbow, may be sufficient. In select cases, surgery may be required to prevent further progression and associated nerve dysfunction. Understanding the underlying mechanism of injury and the associated natural history is a fundamental aspect of designing a treatment plan. The management of peripheral nerve entrapment syndromes depends on multiple factors including but not limited to the chronicity and severity of symptoms, underlying mechanism, and associated predisposing factors. Certain individuals with predisposing conditions such as

anatomic variations or systemic conditions may be less likely to respond to conservative treatments. Understandably, treatment considerations are more well defined in common conditions such as carpal tunnel syndrome.

Upper Limb Entrapment Neuropathy Syndromes

Median Neuropathies

Anatomy: The median nerve is formed by fibers from C5-C7 cervical roots traveling within the lateral cord merged with fibers from C8-T1 nerve roots traveling within the medial cord. The median nerve travels through the medial arm along with the brachial artery and vein and crosses anterior to the elbow joint. Entering into the forearm, the median nerve typically passes between the two heads of the pronator teres. Within the forearm the nerve passes deep to the sublimis bridge and travels distally between the flexor digitorum superficialis and flexor digitorum profundus muscles and passes under the transverse carpal ligament.

There are no important sensory or motor branches supplied above the elbow. Within the forearm, motor branches of the median nerve innervate pronator teres, flexor carpi radialis, and flexor digitorum sublimis. The anterior interosseous is a major branch of the median nerve within the forearm supplying motor innervation to flexor pollicis longus, flexor digitorum profundus to digits 2 and 3, and pronator quadratus. The palmar cutaneous nerve branches from the median nerve prior to the median nerve passing within the carpal tunnel. It supplies sensation over the thenar eminence. After passing through the carpal tunnel, the median nerve innervates abductor pollicis brevis, opponens pollicis, superficial head of the flexor pollicis brevis, and the first and second lumbrical muscles by C8-T1 motor fibers. The thenar compartment muscles including abductor pollicis brevis, opponens pollicis, and the superficial head of flexor pollicis brevis are innervated by the recurrent motor branch. Typically the recurrent motor branch divides from the median nerve and travels along with the median nerve under the transcarpal ligament.² The course is variable which may rarely predispose to injury during carpal tunnel release and thus its nickname "the million-dollar nerve". The median nerve within the hand supplies sensation of the palmar surfaces of the thumb, digits 2, 3, and lateral half of digit 4.

Median Neuropathy at the Wrist

Entrapment of the median nerve at the carpal tunnel better known as carpal tunnel syndrome is the most common and best characterized of all the entrapment neuropathies. Previous studies have estimated the prevalence of carpal tunnel syndrome in the general population to be 2% in men and 3% in women.³ Carpal tunnel syndrome (CTS) is a clinical diagnosis that describes the constellation of symptoms that occur in the setting of chronic median nerve compression at the wrist as the nerve passes posterior to the transverse carpal ligament. Compression inhibits axonal transport and epidural blood flow leading to intraneural edema, myelin thinning, nerve fiber degeneration, and fibrosis.⁴⁻⁶ The available literature does not support an inflammatory component in the pathogenesis of CTS.⁷ Uncommonly the median nerve may be compressed acutely within the carpal tunnel causing an abrupt onset of symptoms within hours. These cases are usually termed acute CTS.⁸

Multiple factors may predispose the development of median neuropathy at the wrist. These factors likely play a role by either increasing the pressure within the carpal tunnel or increasing the susceptibility of the median nerve to pressure or ischemia. The existing data is mixed but suggests that an underlying peripheral neuropathy may increase susceptibility of the median to compression at the transverse carpal ligament. By and large most CTS is idiopathic, and no specific underlying predisposing condition is identified. Systemic predisposing conditions include pregnancy, thyroid disorders, chronic kidney disease, acromegaly, diabetes, hereditary neuropathy with predisposition to pressure palsies, and familial amyloidosis related to transthyretin mutation. Genetic factors contributing to idiopathic CTS were first described in 1969 and are recognized in about 30% of patients.⁹⁻¹⁰ There are numerous studies suggesting that CTS is a cumulative trauma disorder, but studies also argue that body habitus, diabetes, age, as well as other factors influence work related CTS.^{8, 11-12} There does not seem to be compelling evidence in favor of a clear association between keyboard or computer use and the development of CTS.¹³

Clinical Features: Patients with carpal tunnel syndrome present with symptoms in the distribution of the distal median nerve (i.e. sparing the palmar cutaneous sensory branch distribution) with pain, numbness, and tingling affecting the thumb, index, and middle fingers. In early or mild cases, symptoms are intermittent, frequently during sleep or with certain exacerbating activities. Generalized symptoms of numbness affecting the whole hand are frequently reported. Pain is often poorly localized involving the upper limb from hand and to as proximal as

the shoulder in select cases. Interestingly only about one half of patients can reliably localize the symptoms to the distribution of the distal median nerve.¹⁴ Neck pain is not a feature of carpal tunnel syndrome. Wrist pain without any sensory symptoms of the median innervated digits is less likely to be related to CTS. Weakness is uncommonly seen, but in severe or very chronic cases, thenar muscle weakness and atrophy may be present. Very rarely patients will present with weakness of the thenar compartment muscles and minimal to no sensory signs or symptoms related to isolated involvement of the recurrent motor branch. Other motor predominant processes such as motor neuron disease, multifocal motor neuropathy or Hirayama disease may present with predominantly hand involvement and should be carefully excluded. Additionally structural lesions, such as a ganglion cyst, selectively compressing the recurrent motor branch should be considered.

Early in the course of CTS, the physical examination may be normal. With detailed clinical testing, many patients will have a normal sensory examination, and even with quantitative sensory testing around 20% will have a normal sensory examination.¹⁵ With ongoing nerve involvement, loss of sensation is seen in the distribution of the distal median nerve with sparing of the thenar eminence and palm (palmar cutaneous sensory branch). Sensory examination with light touch and pinprick sensation is more useful compared with vibration and 2 point discrimination. With increasing severity and chronicity of involvement, motor weakness may become apparent. Thenar atrophy is only seen in very severe cases. Examination must include strength testing of other hand and forearm muscles to exclude mimicking conditions. Provocative maneuvers are may be used to support the diagnosis of carpal tunnel syndrome. Tinel's sign involves eliciting paresthesias in the distribution of a nerve with gentle tapping at the entrapment site of consideration (i.e. the wrist in carpal tunnel syndrome). This sign has been demonstrated to be of limited sensitivity and specificity and may be present in individuals without entrapment neuropathy.¹⁶ The wrist flexion test, originally described by Phalen in 1951, is another bedside maneuver commonly used in the assessment of carpal tunnel syndrome. The Phalen test involves sustained light, unforced wrist flexion for 30-60 seconds. The reproduction or exacerbation of symptoms suggests the diagnosis of carpal tunnel syndrome. This test has a demonstrated sensitivity of 75% and specificity of 47%.¹⁶

Diagnosis: The differential of median neuropathy at the wrist may include disorders of the musculoskeletal system as well as other peripheral nerve disorders. Musculoskeletal conditions such as Dequervain's tenosynovitis and thumb joint arthropathy are common causes of hand and wrist pain, but sensory symptoms are characteristically absent. C6 and C7 radiculopathies are common mimics due to a sensory territory overlapping with that of the median nerve. Neck pain is not associated with carpal tunnel syndrome and should be a clue to help guide the evaluation to an alternate diagnosis. Proximal median nerve injury may produce similar symptoms, but associated uncharacteristic findings of thenar and palm sensation changes should help to exclude CTS.

Confirmation of median neuropathy at the wrist is best obtained through the use of nerve conduction studies and electromyography. Numerous nerve conduction study techniques have been used to confirm demyelination of the median nerve at the wrist/carpal tunnel space with varying sensitivities and specificities. Electrodiagnostic study results have been estimated to have a sensitivity of approximately 85% and a specificity of approximately 95%, but these values vary by choice of testing.¹⁷ Increasingly ultrasound is being used for diagnosis, but electrodiagnostic studies remain superior in the majority of cases.

Treatment: The treatment of median neuropathy at the wrist is the best studied of all the entrapment neuropathy syndromes, but a full understanding of the natural history of CTS continues to be lacking. In one multicenter natural history study, approximately 20% of hands improved with no intervention, although two thirds of the patients reduced occupational or recreational hand activities.¹⁸ When determining the best treatment option for a particular patient it is important to consider several factors including the duration of symptoms, whether the symptoms occur intermittently or only with certain activities, the clinical severity on the basis of examination and electrodiagnostic testing, and coexistent factors. If a patient has only mild or intermittent symptoms conservative management is most appropriate. This may include activity modification, wrist splints, oral medications, and ultrasound therapy (see Table 1). Wrist splints are a mainstay in conservative management. This strategy is inexpensive, readily available, and effective. Wearing splints has been demonstrated to improve symptoms in over 50% of patients.¹⁹ Patients with predominantly nocturnal symptoms or a shorter duration symptoms are more likely to respond to wrist splinting. Splints should maintain the wrist in a neutral position as this is associated with lower pressures within the carpal tunnel. There is no clear data to support or refute whether activity modification will modify the natural history of CTS, and many patients have no history of repetitive activity or heavy manual labor. Even so, when feasible, avoiding aggravating activities is reasonable and should be recommended. Other effective nonsurgical options may include oral or injected corticosteroids. One of the most important but difficult questions to answer is when to consider referral for possible surgical intervention. Surgical release is safe, economical, and associated with satisfactory results in over 70% of individuals. Patient preference

should weigh heavily on the decision to pursue surgery, and in patients with persistent or severe CTS, surgical intervention using open or endoscopic techniques should be considered.²⁰⁻²¹ Failure of improvement following surgery may be attributable to incomplete correction of compressive lesion, incorrect diagnosis, and persistent median nerve injury not reversible by decompression.

Certain clinical situations require special consideration in the management of CTS. In transient predisposing conditions such as pregnancy, thyroid disease, or acromegaly, CTS should be managed conservatively as patients may experience complete resolution of symptoms.²²⁻²⁴ CTS superimposed on peripheral neuropathy, including diabetic neuropathy, should be managed in a similar manner as compared to isolated CTS.²⁵ This being said, it may be difficult, if not impossible, to confirm the diagnosis with confidence in patients with predominantly demyelinating peripheral neuropathy. As previously mentioned hereditary neuropathy with predisposition to pressure palsy or HNPP may contribute to the development of median neuropathy at the wrist. Successful surgical intervention has been reported in HNPP, but caution should be used when considering surgical intervention in CTS, as well as other entrapment syndromes, due to increase susceptibility to nerve injury.²⁶

Carpal Tunnel Syndrome: Potential Treatments			
Non-surgical			
Type of intervention	Theoretical Mechanism	Benefit	Risks
Splinting	Limit increased pressure within the carpal tunnel	Short term benefit	Minimal
Injected Corticosteroids	Reduced inflammation or swelling within the carpal tunnel	Short-term benefits possibly better than surgery; may suggest successful surgical intervention	Worsening symptoms, inadvertent median nerve injection, infection, etc
Oral Corticosteroids	Reduced inflammation or swelling within the carpal tunnel	Short term benefits; less effective than injection	Systemic side effects
Ultrasound	Increased cell permeability to promote healing	Mixed results; sustained therapy potentially more beneficial	Minimal
NSAIDS	Reduced inflammation or swelling within the carpal tunnel	None	Systemic side effects
Nerve Gliding	Eliminate nerve tethering and restore normal movement	No clear benefit	Minimal
Surgical			
Type of intervention	Theoretical Mechanism	Benefit	Risk
Open or Endoscopic	Reduced pressure within the carpal tunnel	Definitive treatment; more effective long-term; No clear difference between endoscopic and open	Injury to vital structures, infection, persistent or worsening pain, etc

Table 1: Potential treatments of CTS

Other focal median nerve syndromes

Median neuropathy at the wrist explains the majority of patients who present with median neuropathy. Rarely proximal median neuropathy may mimic carpal tunnel syndrome. Typically these cases can be clearly distinguished on the basis of weakness of proximal median-innervated muscles as well as the presence of sensory loss in the palmar aspect of the hand. Other entrapment syndromes of the median nerve have been described related to compression by the ligament of Struthers, biceps brachii aponeurosis, pronator teres muscle, and flexor digitorum superficialis muscle.

Pronator Syndrome: Pronator syndrome is an ill-defined entrapment syndrome involving compression of the median nerve as it passes between the pronator teres muscles. Symptoms may include aching pain in the forearm associated with sensory and or motor deficits in the distribution of the proximal median nerve. Pronator syndrome has been described using two terms to clarify discrepancies in the literature. True neurogenic pronator syndrome describes the syndrome with the presence of sensory and motor signs median neuropathy confirmed by electrodiagnostic studies. Conversely nonspecific pronator syndrome is more appropriate when median

neuropathy cannot be confirmed.²⁷ Conservative management includes avoidance of exacerbating activities and injection of the pronator teres muscle with local anesthetic agent and/or corticosteroids. Surgical intervention with a pronator release should only be considered for severe, persistent symptoms in patients with a definite diagnosis.

Anterior Interosseous Syndrome: The anterior interosseous nerve innervates the pronator quadratus, flexor pollicis longus, and flexor digitorum profundus to digits 2 and 3. Anterior interosseous syndrome describes symptoms of deep aching pain in the forearm followed by weakness in all or select anterior interosseous innervated muscles. Causes attributed to anterior interosseous syndrome are varied including idiopathic, trauma, external compression, and compression by the ulnar head of the pronator teres and flexor digitorum superficialis. There is significant overlap between anterior interosseous syndrome related to chronic compression and neuralgic amyotrophy (also known as Parsonage Turner Syndrome or idiopathic brachial plexus neuropathy).²⁸ Standard nerve conduction studies are typically normal. Thus distinguishing between these conditions may be difficult. Symptoms of slowly progressive pain localized to only the forearm associated with deficits in only the distribution of the anterior interosseous nerve may suggest chronic compression. Conversely, an abrupt onset of severe pain or weakness is usually less likely related to anterior interosseous nerve entrapment.

Ulnar Neuropathies

Anatomy: The ulnar nerve is formed by sensory and motor axon contributions from the C8 and T1 cervical roots which pass through the lower trunk and medial cord of the brachial plexus. There are no ulnar innervated muscles above the elbow. Within the forearm the ulnar nerve innervates flexor carpi ulnaris and flexor digitorum profundus to digits 4 and 5. Two sensory branches occur within the forearm. The palmar cutaneous nerve branches mid forearm and passes superficial to Guyon's canal to supply sensation of the hypothenar region. The dorsal ulnar cutaneous sensory nerve branches in the distal forearm to supply the dorsal aspect of the medial hand, medial aspect of the fourth digit finger, and the dorsal aspect of the fifth digit. The ulnar nerve enters the hand through Guyon's canal splitting into two terminal branches. The superficial terminal branch innervates palmaris brevis and provides sensation of the distal medial palm and the palmar surfaces of the fifth digit and medial half of the fourth digit. The deep terminal branch supplies the hypothenar, all the interossei, the third and fourth lumbrical, and adductor pollicis muscles.

Ulnar Neuropathy at the Elbow

The presentation of ulnar nerve entrapment depends on the location and severity of the entrapment. Ulnar neuropathy at the elbow (UNE) is the second most common entrapment neuropathy. The most common initial presentation of UNE will be numbness of the medial hand, dorsal and palmar aspects, and the medial half of the fourth and the fifth digit. These symptoms are frequently associated with vague pain about the elbow. If the severity is sufficient to involve motor axons, weakness may be present in all ulnar innervated muscles, although, the weakness is usually more prominent distally. In patients with numbness sparing of the dorsal aspect of the medial hand, a more distal lesion at the wrist should be considered. This is explained by the fact that the dorsal ulnar cutaneous sensory branch leaves the main ulnar nerve within the forearm to supply the dorsal aspect of the hand. Occasional patients with UNE will present with significant weakness and intrinsic muscle atrophy and less prominent sensory complaints.

Ulnar compression at the elbow usually occurs at the condylar groove or in the cubital tunnel, but multiple other sites have been described. The roof of the cubital tunnel is defined by the aponeurosis of the two heads of flexor carpi ulnaris (also called Osborne's ligament) which travels between the medial epicondyle and the olecranon. In cubital tunnel syndrome, compression of the ulnar nerve usually occurs 1.5 to 3 cm distal to the epicondyle. One well-known syndrome involving the ulnar nerve at the elbow is the tardy ulnar palsy. This syndrome of ulnar neuropathy following a remote fracture of the distal humerus was first described most definitively in 1878 by Panas.²⁹ In this syndrome, after the fracture is healed, the ulnar nerve is more susceptible to trauma related to structural changes at the elbow.

Clinical Features: With mild or early UNE, the sensory examination may be normal. As the symptoms progress, patients may demonstrate sensory loss of the volar and dorsal aspects of the medial hand and the fourth and fifth digits. Occasional patients will present with varying sensory distribution such as sparing of the dorsal hand which may be related to relative sparing of those fascicles versus a recently described anatomical variant in which the dorsal ulnar cutaneous nerves travel with the superficial radial nerve.³⁰⁻³¹ Depending on the location and severity of the entrapment, weakness may be seen in all of the ulnar innervated muscles as no muscles are innervated

above the elbow. The distal muscles of the hand are more likely to be affected, and proximal muscles may remain normal despite severe distal weakness and atrophy. This has been attributed to fascicular involvement of the ulnar nerve at the elbow as well as length dependent axonal susceptibility.³² This phenomenon may cause an ulnar neuropathy at the elbow to partially mimic a distal ulnar neuropathy.

There are many clinical signs described to aid in the examination of the suspected ulnar neuropathy. Froment's sign is positive when flexor pollicis longus is substituted for weakness of adductor pollicis, flexor pollicis brevis, and first dorsal interosseous muscles. Wartenberg's sign is described as one of the earliest motor signs of ulnar neuropathy and is present when there is an abducted posture of the fifth digit due to weakness of the third palmar interosseous muscle. The ulnar claw hand, sometimes referred to as Duchenne sign, is the clawing posture of the fourth and fifth digits with no clawing of digits two and three due to preserved function of the lumbrical muscles for these digits (related to median innervation). The ulnar claw hand is often contrasted with the hand of benediction seen in a severe proximal median nerve injury. In the hand of benediction, the thumb, index, and middle fingers fail to flex when the patient tries to make a fist. In comparison, the ring and small finger fail to fully extend in the patient with an ulnar claw hand. These signs, while useful, cannot replace the detailed examination of individual muscles of the upper limb in the patient with a suspected peripheral nerve injury or entrapment.

Diagnosis: The differential of UNE may be quite broad. Lesions of the brachial plexus, specifically the lower trunk and medial cord as well as a C8 radiculopathy, can produce symptoms with significant overlap with that of UNE. Motor predominant processes including motor neuron disease as well as multifocal motor neuropathy may focally involve the ulnar nerve. Cervical myelopathy as well as a cervical cord lesion may produce hand predominant symptoms occasionally mimicking symptoms of UNE. Leprosy has a strong predilection to involve the ulnar nerve at the elbow.

The diagnosis of UNE can usually be made clinically, but the presentation may be variable making localization difficult. EMG and NCS are usually necessary to help confirm the diagnosis, determine the severity, and to exclude other disorders. Similar to the median nerve, the ulnar nerve may be imaged with relative ease using ultrasound which may provide supplemental information to electrodiagnostic studies. Other imaging modalities may be helpful to investigate for contributing structural abnormalities, particularly in individuals with apparent osseous malalignment or a history of traumatic injury.

Treatment: The natural history of idiopathic ulnar neuropathy at the elbow is largely unknown. The limited available studies suggest that the majority of patients will improve with conservative measures alone.³³⁻³⁴ Treatment strategies are broadly grouped into either conservative aimed at reducing mechanical pressure on the nerve versus surgical intervention. Bartels severity classification is helpful in determining whether to pursue conservative versus surgical management (see table 2).^{27, 35-36} Grade 1/mild includes sensory symptoms, normal sensory examination, and no muscle atrophy or weakness. Grade 2/moderate includes sensory symptoms, detectable sensory loss, and grade 4-4+ muscle weakness with or without atrophy. Grade 3/severe includes sensory loss and grade 4 minus or worse muscle weakness associated with atrophy.³⁵ Conservative measures should usually be recommended for patients with mild or moderate UNE (grade 1 or 2) unless there is evidence for a structural abnormality or prolapsing nerve. Conservative measures include modification of daily activities that may predispose to increased pressure on the ulnar nerve, wearing external padding to reduce pressure at the elbow, and sleeping with straight elbows with or without the assistance of splinting. Surgical management is limited to patients with significant motor weakness. If significant weakness (even if severe) is associated with electrodiagnostic findings of prominent motor conduction block, greater than 50%, the prognosis may be more benign and consideration for surgical intervention delayed.³⁷ The surgical approaches to ulnar neuropathy at the elbow include cubital tunnel decompression, transposition, and medial epicondylectomy.

Bartel's Criteria of Ulnar Neuropathy at the Elbow and Management Considerations			
Severity Classification	Sensory	Motor	Management
Mild (Grade 1)	Sensory symptoms	Normal	Usually Conservative
Moderate (Grade 2)	Detectable sensory loss	Grade 4 to 4+	Conservative unless persistent or worsening symptoms
Severe (Grade 3)	Detectable sensory loss	Grade 4- or less	Consider should be given to surgical intervention

Table 2: Bartel's Severity Classification of Ulnar Neuropathy at the elbow³⁵

Ulnar Neuropathy at the Wrist

Ulnar neuropathy at the wrist (UNW) is the most common site of ulnar neuropathy distal to the elbow. UNW may involve the distal ulnar branches in isolation or combination determining the sensory and/or motor presenting features. Involvement of only the deep palmar motor branch will result in interosseous muscle weakness and potentially clawing with no sensory loss. Other branches distal to the Guyon's canal include the superficial sensory, hypothenar motor, and digital sensory branches. Types of ulnar nerve lesions at the wrist are characterized and identified by which branches are involved.³⁸ Type I involves compression just proximal to Guyon's canal affecting the superficial sensory, hypothenar motor, and deep motor branches. Type II involves compression within the canal with involvement of only the superficial sensory branch. Type III involves compression distal to the superficial sensory branch with compression of the hypothenar and the deep motor branch. Type IV involves compression distal to the superficial sensory and hypothenar motor branches and involves only the deep motor branch. Type V involves compression of the deep motor branch just proximal to the branches to the adductor pollicis and first dorsal interosseous muscles.

There are no available studies comparing surgical versus conservative treatment of ulnar neuropathy at the wrist. In ulnar neuropathy at the wrist, commonly the patient will describe an external compression that preceded the onset of symptoms. If a history of mild or repetitive trauma is elicited during the history, initial treatment measures should include avoidance of such activities. If no preceding trauma or provoking activity is elicited, structural lesions such as a ganglion cyst or osseous abnormalities should be excluded.

Other Focal Neuropathies of the Upper Limb:

Radial

The radial nerve may be injured at multiple sites producing a number of clinical presentations and syndromes. These are uncommonly related to chronic compression. Thus this presentation will not cover the radial nerve in detail. The most common presentation of radial neuropathy is related to acute compression at the spiral groove, the so-called Saturday night's palsy. Patients typically awaken with symptoms of numbness, tingling, and motor weakness in the distribution of the radial nerve with relative sparing of the triceps muscle. In most cases this is a self limited process and is not, strictly speaking, an entrapment neuropathy. Other radial neuropathy syndromes may include chialgia paresthetica or handcuff palsy, posterior interosseous syndrome, and radial tunnel syndrome. Chialgia paresthetica is related to compression of the superficial radial nerve causing sensory symptoms of the dorsal lateral hand and thumb, index, and middle fingers.

Posterior interosseous neuropathy is a rare example of a focal radial neuropathy sometimes attributed to compression as the nerve passes through the supinator muscle or compression at by the extensor carpi radialis brevis muscle tendon, or the arcade of Frohse. The clinical presentation of posterior interosseous nerve syndrome includes dull pain at the elbow associated with weakness of finger and thumb extension and relatively spared wrist extension strength. MR imaging and ultrasound may be helpful in the evaluation to help demonstrate posterior interosseous nerve compression and associated swelling.³⁹⁻⁴⁰ Similar to the anterior interosseous branch of the median nerve, neuralgic amyotrophy has been implicated in posterior interosseous neuropathy.

A highly controversial disorder attributed to radial nerve entrapment is radial tunnel syndrome. Symptoms include pain in the lateral forearm over the brachioradialis muscle. Exacerbating maneuvers may include forearm supination and finger extension. Some believe that this disorder may represent early posterior interosseous nerve syndrome.⁴¹ Sites of compression attributed to this disorder include the tendinous edge of the extensor carpi radialis brevis muscle, the supinator muscle, and the arcade Frohse (tendinous origin of the supinator muscle).⁴⁰

Suprascapular

The suprascapular nerve arises from the C5 and C6 roots and the upper trunk of the brachial plexus. It supplies motor input to the supraspinatus and infraspinatus muscles and has limited to no associated cutaneous sensory territory. There is some evidence that the suprascapular nerve supplies the majority of the sensation to the shoulder joint.⁴² Suprascapular neuropathies have been described in association with chronic compression at the suprascapular and the spinoglenoid notch. Tightening of the spinoglenoid ligament has been shown to occur in the shoulder is positioned for overhead throwing, and several reports detail an increased prevalence of suprascapular neuropathy in athletes involved in overhead sports such as volleyball.⁴³⁻⁴⁴ The typical presentation includes an aching pain that is localized in the posterior aspect of the shoulder pain is associated with complaints of shoulder weakness. Electrodiagnostic testing is a crucial component of the evaluation. Nerve conduction studies to the supraspinatus and infraspinatus with side-to-side comparison may be helpful to demonstrate axonal loss as well as features of demyelination. In true chronic compression findings of demyelination would be expected. Findings of pure axonal loss may suggest an alternate etiology. The needle electrode examination will help to demonstrate evidence of active or chronic axonal loss. In addition to confirming the abnormalities expected in suprascapular neuropathy, electrodiagnostic studies are helpful in excluding other mimicking conditions such as radiculopathy, plexopathy, or neuralgic amyotrophy. Neuralgic amyotrophy is not uncommonly associated with suprascapular nerve involvement, and is the most important mimicking condition.⁴⁵⁻⁴⁷ Other musculoskeletal disorders, such as a rotator cuff tears, may limit the use of the shoulder leading to atrophy of the rotator cuff muscles and occasionally mimic this condition as well.

Musculocutaneous

Nerve fibers from the C5, C6, and C7 roots form the upper and middle trunks and later the lateral cord prior to becoming the musculocutaneous nerve. The musculocutaneous nerve supplies motor input to the coracobrachialis, biceps brachii, and brachialis muscles. The lateral antebrachial cutaneous nerve is a pure sensory nerve continuation of the musculocutaneous nerve supplying the lateral ventral forearm. Focal neuropathies involving the musculocutaneous nerve are very uncommon. Focal neuropathies related to compression by nearby muscles, such as coracobrachialis, have been suggested. Similar to the other proximal nerves of the shoulder, the musculocutaneous nerve is commonly involved in neuralgic amyotrophy which likely explains the majority of isolated musculocutaneous neuropathies.

Axillary

Nerve fibers from the C5 and C6 ventral rami form the upper trunk and later pass through the posterior cord forming the axillary nerve. The axillary nerve supplies motor input to the deltoid and teres minor muscles. The upper lateral cutaneous nerve of the arm branches from the axillary nerve to supply a patch of skin over the deltoid muscle. Similarly to the musculocutaneous and suprascapular nerve branches of the brachial plexus, the axillary nerve is commonly involved in neuralgic amyotrophy.⁴⁵ Quadrilateral space syndrome involves the axillary nerve being compressed within the quadrilateral space, but the syndrome has similar, considerable overlap with that of neuralgic amyotrophy.

Specific Lower Limb Entrapment neuropathy Syndromes

Lateral Femoral Cutaneous Nerve (Lateral cutaneous nerve of the thigh)

Anatomy: The lateral femoral cutaneous (LFC) nerve typically arises from the dorsal divisions of the ventral primary rami of the L2-L3 spinal nerves. The nerve emerges along the lateral border of the psoas muscle, travels along the lateral aspect of the pelvis, and passes under the inguinal ligament approximately 1 cm medial to the anterior superior iliac spine to enter the thigh.⁴⁸

Meralgia Paresthetica

LFC neuropathy is responsible for the syndrome of meralgia paresthetica (MP). The constellation and distribution of symptoms may vary between patients, but pain, numbness, and paresthesia are typically present in the distribution of the anterior lateral thigh. Occasionally patients will describe a very limited distribution of sensory symptoms in only part of the LFC distribution. The LFC nerve is a purely sensory nerve. Thus no motor signs or symptoms are present, but occasionally a patient will present with difficulty walking related to pain. Patients may describe varying provoking positions, and worsening of symptoms may be seen with hip flexion or extension.

Varied mechanisms of injury have been described including surgical incision, injection, direct blow, malignant invasion, as well as acute and chronic compression. Predisposing or causative factors related to compression may include abdominal protuberance (pregnancy, obesity, or ascites), external compression from tight clothing, or prolonged positioning (lithotomy positioning, bicycling, or prolonged hip extension). The most common site of chronic compression occurs as the LFC nerve passes under the inguinal ligament.

Clinical Features: Early in the course of MP or in patients with mild symptoms, examination may be normal. With increasing severity sensory loss is present and may involve all or only part of the classically described distribution of the LFC nerve. Otherwise the neurological examination is normal. Reflexes, most importantly the patellar reflex, remain intact. Motor weakness is characteristically absent, and if present other mimicking conditions such as upper lumbar radiculopathy, lumbar plexopathy, or femoral neuropathy are more likely.

Diagnosis: The diagnosis of MP can usually be made clinically. Diagnostic investigations are usually only necessary in atypical cases. In this instance, imaging studies and EMG and NCS are helpful to exclude mimicking conditions. Nerve conduction studies to confirm LFC neuropathy can be unreliably present in healthy individuals, and are increasingly difficult in the obese patient. Diagnostic injections at the common site of entrapment can be helpful to confirm the diagnosis if clear relief of symptoms is provided. Imaging modalities may include CT, MR, and ultrasound.

Treatment: The symptoms of MP are typically transient, and even without specific treatment the majority of patients will have complete resolution within weeks to months. Thus conservative treatment should be encouraged. Conservative treatments should include removing sources of extrinsic compression such as compressive clothing or provoking postures. NSAIDs or neuropathic pain medications may be helpful for symptomatic management. In cases that do not respond with time and conservative measures infiltration with local anesthetic and steroids may provide temporary relief and can be repeated at intervals. In patients with significant persistent symptoms, surgical intervention (decompression or sectioning) may be considered and has been reported to be effective in about 80% of cases.⁴⁹

Tibial Nerve

Anatomy: The tibial nerve, composed of L4, L5, S1, and S2 nerve roots, courses through the lumbosacral plexus and through the thigh within the sciatic nerve, along with the peroneal component. The sciatic nerve divides into the peroneal and tibial nerves proximal to the popliteal fossa. In the thigh, the tibial component of the sciatic nerve supplies all of the hamstrings muscles and ½ of adductor magnus. The tibial nerve travels within the posterior compartment of the leg innervating all of the posterior compartment muscles and distally passes posterior to the medial malleolus through the tarsal tunnel dividing into the medial and lateral plantar nerves.

Tarsal Tunnel Syndrome

Tarsal tunnel is an uncommon and much debated syndrome associated with compression of the tibial nerve at the level of the tarsal tunnel. The tarsal tunnel is a fibro-osseous space, formed by the flexor retinaculum posterior and distal to the medial malleolus. The contents of the tarsal tunnel include the tibial nerve, posterior tibial artery, posterior tibial vein, flexor digitorum longus tendon, posterior tibialis tendon, and flexor hallucis longus tendon. Contributing factors of tarsal tunnel syndrome include previous trauma, biomechanical issues such as joint hypermobility, space occupying lesions, or idiopathic.

Clinical Features: Symptoms of tarsal tunnel syndrome may include ankle pain and associated numbness, tingling, burning, and pain of the plantar aspect of the foot. The presentation of tarsal tunnel syndrome may be variable as the entire tibial nerve may be involved versus selective involvement of either the lateral or plantar branches.

Diagnosis: Mimicking conditions may include other neuropathic processes such as peripheral neuropathy or radiculopathy particularly S1 radiculopathy. Common musculoskeletal mimicking conditions include posterior tibial tendon dysfunction and plantar fasciitis. Tarsal tunnel syndrome is typically a clinical diagnosis supported by electrodiagnostic findings. Nerve conduction studies may help to localize the lesion and to exclude mimicking conditions. Imaging studies are helpful to exclude structural or space occupying lesions. If typical foot pain and paresthesia, a Tinel's sign at the tarsal tunnel, and classic electrodiagnostic findings are present, the diagnosis can be conferred with reasonable certainty.⁵⁰

Treatment: Individuals without a contributing structural or space occupying lesion may respond adequately to conservative management which may include NSAIDs, neuropathic pain medications, activity modification, physical therapy, and biomechanical modification with shoes, inserts, or orthoses. Surgery is reserved for individuals with a definite diagnosis who have been resistant to conservative strategies.

Peroneal (Fibular) Nerve

Anatomy: The peroneal nerve is composed of the L4, L5, and S1 nerve roots and travels through the lumbosacral plexus into the sciatic nerve along with the tibial nerve component. The peroneal and tibial components of the sciatic nerve are divided by a connective tissue sheath, and proximal to the popliteal fossa the sciatic nerve separates into the peroneal and tibial nerves. Only one muscle proximal to the knee, the short head of the biceps, is innervated by the peroneal component of the sciatic nerve. After dividing from the sciatic nerve the peroneal nerve gives off the lateral cutaneous nerve of the calf supplying sensation of the lateral upper leg. Traveling around the fibular head it divides into the superficial and deep peroneal nerves. The superficial branch carries motor axons innervating the ankle evertors and sensory axons supplying the lateral aspects of the lower leg and foot. The deep peroneal nerve travels through the anterior compartment supplying sensory input from the first dorsal web space and motor input to the ankle dorsiflexors and toe extensors.

Common Peroneal neuropathy at the Fibular Head

The common peroneal nerve (CPN) is susceptible to compression as it travels laterally around the fibular head. The mechanism of injury is typically related to external compression of the nerve against the fibular in contrast to compression occurring between anatomic structures. True entrapment neuropathy is described at the fibular tunnel, but this occurs uncommonly. Other possible etiologies of common peroneal neuropathy may include mass lesions, tumors, stretch injuries, trauma and vascular. A complete lesion of the CPN produces weakness in toe extension, foot eversion, and ankle dorsiflexion associated with sensory loss on the anterior lateral surface of the lower leg and dorsum of the foot. With a lesion at the fibular head there is sparing of the proximal anterior lateral leg related to the preserved lateral cutaneous nerve of the calf which branches in the lateral popliteal fossa.

The most common mimicking condition of peroneal neuropathy at the fibular head is L5 radiculopathy, but other mimicking conditions may include sciatic neuropathy, motor neuron disease, and lumbosacral plexopathy. Preservation of ankle inversion is a feature of peroneal neuropathy that helps to distinguish from L5 radiculopathy.

Peroneal Neuropathy at the Ankle (Deep peroneal neuropathy/anterior tarsal tunnel syndrome)

Deep peroneal nerve entrapment is sometimes referred to as anterior tarsal tunnel syndrome. There is disagreement regarding the use of this term due to the lack of a clearly defined fibro-osseous tunnel. DPN compression may be acute as in a traumatic injury or chronic external pressure related to ill fitting footwear, etc.

Other Compressive Neuropathies of the Lower Limb:

Nerve entrapments associated with groin neuralgia

Inguinal herniorrhaphy surgery and pelvic surgery are occasionally associated with entrapment of the ilioinguinal, genitofemoral, and iliohypogastric nerves. There are no nerve conduction study techniques available to assess these nerves. Diagnosis is usually established by demonstrating sensory disturbance in the distribution of the individual nerve and relief of symptoms with infiltration with local anesthetic. Conservative treatments may include oral anti-inflammatory or neuropathic pain medications, avoidance of activities that provoke pain, nerve blocks, and application of TENS unit. Good results have been reported with surgical intervention for select cases with persistent pain.

Obturator neuropathy

The obturator nerve is occasionally entrapped as it exits the obturator foramen entering the proximal thigh. Obturator neuropathy has been associated with traumatic, postsurgical, and idiopathic causes. Typically patients present with exercise induced aching pain in the medial thigh or groin and occasional weakness of obturator innervated muscles. Electrodiagnostic studies may reveal signs of denervation on the needle electrode examination, but there are no reliable conduction studies to confirm focal demyelination. Resolution of pain symptoms with infiltration of local anesthetic helps to confirm the diagnosis. Surgical intervention has been suggested for release of entrapment of the nerve as it enters the thigh.⁵¹

Sciatic neuropathy

Piriformis syndrome is a much debated syndrome associated with sciatica nerve entrapment due to compression by the piriformis muscle. Under diagnosis and over diagnosis have both been debated in this syndrome. Mimicking conditions include lumbosacral radiculopathy and musculoskeletal disorders of the lumbosacral spine or sacroiliac joint. Treatment strategies include conservative measures of physical therapy, oral anti-inflammatory and neuropathic pain medications, and avoidance of provoking activities. Cases resistant to conservative measures have been treated with surgical decompression, corticosteroid injections, and botulinum toxin injections.

Interdigital neuropathy (Morton neuroma)

Interdigital neuropathy is a common entrapment neuropathy of the lower limbs, often referred to as Morton neuroma. The underlying pathophysiology involves compression of the interdigital nerve beneath the intermetatarsal ligament at the level of the metatarsal heads. The most commonly involved interdigital nerve is that between the third and fourth digits which is formed by communicating branches of the lateral plantar and medial plantar nerves. Predisposing factors include ill fitting shoes with narrow toe boxes, high heeled shoes, flexible feet associated with hyperpronation, and repetitive activities such as running.⁵² Typical symptoms include numbness, tingling, burning, and pain. The symptoms are usually exacerbated with activity and relief with rest and massage of the forefoot area. Mimicking conditions may include musculoskeletal etiologies such as metatarsalgia or tenosynovitis as well as other distal neuropathic syndromes such as peripheral neuropathy, radiculopathy (S1), plantar neuropathy, or tarsal tunnel syndrome. The evaluation of possible interdigital neuropathy is predominantly to exclude mimicking conditions. There are a few nerve conduction studies that can assess the interdigital nerves, but there is limited data regarding reliability.⁵³ Diagnostic interdigital nerve blocks are usually more helpful to confirm the diagnosis. Treatment strategies include activity modification, improved footwear with or without supportive insoles, physical therapy to maximize gastrocnemius-soleus flexibility, and symptomatic medications such as nonsteroidal anti-inflammatory drugs or neuropathic medications. Treatment with steroid injection, alcohol ablation of the neuroma, or surgical resection should be reserved for refractory cases.

Conclusions

Peripheral nerve entrapment is a common neurologic problem, and the associated pain, sensory disturbance, and weakness may lead to significant disability. Appropriate management of peripheral nerve entrapment requires a firm diagnosis and a clear understanding of contributing factors. The diagnosis can often be established on clinical grounds, but an electrodiagnostic evaluation should be pursued in most cases to confirm the diagnosis and exclude other mimicking focal or generalized disorders. Occasionally imaging modalities may be helpful, and ultrasound imaging is increasing in availability and application. Most entrapment syndromes carry an excellent prognosis even with conservative management. Surgical management is necessary in some patients but should be reserved for severe cases or in cases with persistent symptoms despite conservative measures.

References:

1. Simpson JA. Electrical signs in the diagnosis of carpal tunnel syndrome and related syndromes. *J. Neurol. Neurosurg. Psychiatry.* 1956;19(275):275-280.
2. Kozin SH. The anatomy of the recurrent branch of the median nerve. *Journal of Hand Surgery American.* 1998;23(5):852-858.
3. Atroshi I, Gummesson C, Johnsson R, Ornstein E, Ranstam J, Rosen I. Prevalence of carpal tunnel syndrome in a general population. *Jama.* 1999;282(2):153-158.

4. Keir PJ, Rempel DM. Pathomechanics of peripheral nerve loading. Evidence in carpal tunnel syndrome. *Journal of Hand Therapy*. 2005;18(2):259-269.
5. Mackinnon SE, Dellon AL, Hudson AR, Hunter DA. Chronic nerve compression--an experimental model in the rat. *Annals of Plastic Surgery*. 1984;13(2):112-120.
6. Mackinnon SE, Dellon AL, Hudson AR, Hunter DA. Chronic human nerve compression--a histological assessment. *Neuropathology & Applied Neurobiology*. 1986;12(6):547-565.
7. Fuchs PC, Nathan PA, Myers LD. Synovial histology in carpal tunnel syndrome. *Journal of Hand Surgery American*. 1991;16(4):753-758.
8. Schnetzler KA. Acute Carpal Tunnel Syndrome. *Journal of the American Academy of Orthopaedic Surgeons*. 2008;16(5):276-282.
9. Hess H, Baumann F. On the familial occurrence of bilateral carpal tunnel syndrome. *Zeitschrift fur Orthopadie und Ihre Grenzgebiete*. 1969;106(3):565-569.
10. Elstner M, Bettecken T, Wasner M, et al. Familial carpal tunnel syndrome: further evidence for a genetic contribution. *Clinical Genetics February*. 2006;69(2):179-182.
11. Centers for Disease C. Occupational disease surveillance: carpal tunnel syndrome. *MMWR Morbidity & Mortality Weekly Report*. 1989;38(28):485-489.
12. Atcheson SG, Ward JR, Lowe W. Concurrent medical disease in work-related carpal tunnel syndrome. *Archives of Internal Medicine*. 1506;158(14):1506-1512.
13. Palmer KT, Harris EC, Coggon D. Carpal tunnel syndrome and its relation to occupation: a systematic literature review. *Occupational Medicine*. 2007;57(1):57-66.
14. Loong S. The carpal tunnel syndrome: A clinical and electrophysiological study in 250 patients. *Clinical and Experimental Neurology* 1975;14:51-65.
15. Borg K, Lindblom U. Diagnostic value of quantitative sensory testing (QST) in carpal tunnel syndrome. *Acta Neurologica Scandinavica*. 1988;78(6):537-541.
16. Katz JN MLaASea. The carpal tunnel syndrome: diagnostic utility of the history and physical examination findings. *Annals of Internal Medicine* 1990;112:321-327.
17. Jablecki CK, Andary MT, Floeter MK, et al. Practice parameter: Electrodiagnostic studies in carpal tunnel syndrome. Report of the American Association of Electrodiagnostic Medicine, American Academy of Neurology, and the American Academy of Physical Medicine and Rehabilitation. *Neurology*. 1589;58(11):1589-1592.
18. Padua L, Padua R, Aprile I, Pasqualetti P, Tonali P, Italian CTSSGcts. Multiperspective follow-up of untreated carpal tunnel syndrome: a multicenter study. *Neurology*. 1459;56(11):1459-1466.
19. Gerritsen AA, de Vet HC, Scholten RJ, Bertelsmann FW, de Krom MC, Bouter LM. Splinting vs surgery in the treatment of carpal tunnel syndrome: a randomized controlled trial. *Jama*. 1245;288(10):1245-1251.
20. Katz JN, Keller RB, Simmons BP, et al. Maine Carpal Tunnel Study: outcomes of operative and nonoperative therapy for carpal tunnel syndrome in a community-based cohort.[Erratum appears in *J Hand Surg [Am]* 1999 Jan;24(1):201]. *Journal of Hand Surgery American*. 1998;23(4):697-710.
21. Korthals-de Bos IB, Gerritsen AA, van Tulder MW, et al. Surgery is more cost-effective than splinting for carpal tunnel syndrome in the Netherlands: results of an economic evaluation alongside a randomized controlled trial. *BMC Musculoskeletal Disorders*. 2006;7(86).
22. Ablove RH, Ablove TS. Prevalence of carpal tunnel syndrome in pregnant women. *Wmj*. 2009;108(4):194-196.
23. Jenkins PJ, Sohaib SA, Akker S, et al. The pathology of median neuropathy in acromegaly. *Annals of Internal Medicine*. 2000;133(3):197-201.
24. Purnell DC, Daly DD, Lipscomb PR. Carpal-tunnel syndrome associated with myxedema. *Archives of Internal Medicine*. 1961;108:751-756.
25. Morgenlander JC, Lynch JR, Sanders DB. Surgical treatment of carpal tunnel syndrome in patients with peripheral neuropathy. *Neurology*. 1159;49(4):1159-1163.
26. Taggart TF, Allen TR. Surgical treatment of a tomaculous neuropathy. *Journal of the Royal College of Surgeons of Edinburgh*. 2001;46(4):240-241.
27. Stewart JE. *Focal Peripheral Neuropathies*. Fourth ed. West Vancouver: JBJ Publishing; 2010.
28. Kara M, Emlakcioglu E, Kaymak B, Ozcakar L. Brachial neuritis mimicking severe anterior interosseous syndrome. *Acta Reumatologica Portuguesa*. 2010;35(1):114-115.
29. Love JR GaJG. Diagnosis and Treatment of Tardy Paralysis of the Ulnar Nerve. *J. Bone Jt Surg*. 1947;29:1087.
30. Leis AA, Wells KJ. Radial nerve cutaneous innervation to the ulnar dorsum of the hand. *Clinical Neurophysiology*. 2008;119(3):662-666.
31. Leis AA, Stetkarova I, Wells KJ. Martin-Gruber anastomosis with anomalous superficial radial innervation to ulnar dorsum of hand: a pitfall when common variants coexist. *Muscle & Nerve*. 2010;41(3):313-317.

32. Stewart JD. The variable clinical manifestations of ulnar neuropathies at the elbow. *Journal of Neurology, Neurosurgery & Psychiatry*. 1987;50(3):252-258.
33. Eisen A, Danon J. The mild cubital tunnel syndrome. Its natural history and indications for surgical intervention. *Neurology*. 1974;24(7):608-613.
34. Padua L, Caliandro P, La Torre G, Padua R, Giannini F. Treatment for ulnar neuropathy at the elbow [Protocol]. *Cochrane Database of Systematic Reviews*. 2009;1:1.
35. Bartels RH, Menovsky T, Van Overbeeke JJ, Verhagen WI. Surgical management of ulnar nerve compression at the elbow: an analysis of the literature. *Journal of Neurosurgery*. 1998;89(5):722-727.
36. McGowan A. The results of transposition of the ulnar nerve for traumatic ulnar neuritis. *J Bone Joint Surg* 1950;32:293-301.
37. Dunselman HH, Visser LH. The clinical, electrophysiological and prognostic heterogeneity of ulnar neuropathy at the elbow. *Journal of Neurology, Neurosurgery & Psychiatry*. 1364;79(12):1364-1367.
38. Kothari MJ. Ulnar neuropathy at the wrist. *Neurologic Clinics*. 1999;17(3):463-476.
39. Toros T, Karabay N, Ozaksar K, Sugun TS, Kayalar M, Bal E. Evaluation of peripheral nerves of the upper limb with ultrasonography: a comparison of ultrasonographic examination and the intra-operative findings. *Journal of Bone & Joint Surgery British*. 2009;91(6):762-765.
40. Ferdinand BD, Rosenberg ZS, Schweitzer ME, et al. MR imaging features of radial tunnel syndrome: initial experience. *Radiology*. 2006;240(1):161-168.
41. Ritts GD, Wood MB, Linscheid RL. Radial tunnel syndrome. A ten-year surgical experience. *Clinical Orthopaedics & Related Research*. 1987;219:201-205.
42. Brown DE, James DC, Roy S. Pain relief by suprascapular nerve block in gleno-humeral arthritis. *Scandinavian Journal of Rheumatology*. 1988;17(5):411-415.
43. Plancher KD, Luke TA, Peterson RK, Yacoubian SV. Posterior shoulder pain: a dynamic study of the spinoglenoid ligament and treatment with arthroscopic release of the scapular tunnel. *Arthroscopy*. 2007;23(9):991-998.
44. Lajtai G, Pfirrmann CW, Aitzetmuller G, Pirkl C, Gerber C, Jost B. The shoulders of professional beach volleyball players: high prevalence of infraspinatus muscle atrophy. *American Journal of Sports Medicine*. 1375;37(7):1375-1383.
45. van Alfen N, van Engelen BG, van Alfen N, van Engelen BGM. The clinical spectrum of neuralgic amyotrophy in 246 cases. *Brain*. Feb 2006;129(Pt 2):438-450.
46. Chisholm K, Scala S, Srinivasan J. Delay in diagnosis of neuralgic amyotrophy in patients initially evaluated by non-neurologists. *European Journal of Neurology*. Mar 2008;15(3):e18.
47. Sahin E, Senocak O, Bacakoglu AK, et al. [Neuralgic amyotrophy as the primary cause of shoulder pain in a patient with rotator cuff tear]. *Acta Orthop Traumatol Turc*. Mar-Apr 2009;43(2):190-192.
48. de Ridder VA, de Lange S, Popta JV. Anatomical variations of the lateral femoral cutaneous nerve and the consequences for surgery. *Journal of Orthopaedic Trauma*. 1999;13(3):207-211.
49. Siu TL, Chandran KN. Neurolysis for meralgia paresthetica: an operative series of 45 cases. *Surgical Neurology*. 2005;63(1):19-23.
50. Lau JT, Daniels TR. Tarsal tunnel syndrome: a review of the literature. *Foot & Ankle International*. 1999;20(3):201-209.
51. Bradshaw CFACSP, McCrory. Obturator Nerve Entrapment. *Clinical Journal of Sport Medicine* July. 1997;7(3):217-219.
52. Peck E, Finnoff JT, Smith J. Neuropathies in runners. *Clinics in Sports Medicine*. 2010;29(3):437-457.
53. Uludag B, Tataroglu C, Bademkiran F, Uludag IF, Ertekin C. Sensory nerve conduction in branches of common interdigital nerves: a new technique for normal controls and patients with morton's neuroma. *Journal of Clinical Neurophysiology*. 2010;27(3):219-223.