Neuroanatomy (2004) Volume 3 / Pages 18–24

Review Article

Published online 24 May, 2004 © neuroanatomy.org

Entrapment neuropathies of the upper extremity

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Received 12 April 2004; accepted 20 May 2004

Abstract

Painful tingling, numbness, weakness of the hands or upper extremities may be the result of the entrapment of a peripheral nerve. Although these problems are common, they sometimes lead to diagnostic and management difficulties. A thorough knowledge of the anatomy of the common entrapment sites, the pathophysiology of the nerve injury, a detailed history and physical examination together with electrodiagnostic studies usually lead physicians to the right management. Most common upper extremity entrapment neuropathies are encountered in this review.

Key words: [entrapment neuropathy] [electromyography]

Introduction

Entrapment or compressive neuropathies are important and widespread debilitating clinical problems, especially in patients with predisposing occupations or with certain medical disorders. They are caused by mechanical dynamic compression of a short segment of a single nerve at a specific site, frequently as it passes through a fibro-osseous tunnel, or an opening in fibrous or muscular tissue. By far the most common is median nerve entrapment in the wrist leading to so-called carpal tunnel syndrome (CTS). CTS is one of the most common orthopedic conditions, with an estimated incidence of nearly 1% annually in the USA, which makes almost 2.8 million new cases per year [1] and the surgery performed for CTS accounts for the most common operation performed on the hand [2, 3]. Its overall incidence varies between 0.125% and 5.8% of the population, depending on the criteria used for population surveying [4–9]. In the upper limbs, ulnar nerve and posterior interosseous branch of the radial nerve can also be entrapped leading to cubital tunnel syndrome (CuTS) and radial tunnel syndrome (RTS) respectively. Other entrapment neuropathies have been recognized in the upper extremity, involving the superficial sensory radial nerve, the anterior interosseous nerve, the median nerve in the elbow region (e.g. pronator syndrome), the ulnar nerve at the base of the palm (Guyon's canal), the palmar cutaneous branch of the median nerve, and various components of the brachial plexus (neurogenic thoracic outlet syndrome). Anatomical variations, a hypertrophied subscapularis muscle may lead to entrapment of the suprascapular nerve [10, 11].

Detailed anatomical studies have been performed to clarify the basic mechanical aspects of these syndromes. Yet, there is still discussion regarding the best methods for evaluating patients both initially and in follow-up, and the most appropriate treatment. New radiographic and electromyographic methods may allow rapid and accurate assessment of the location and severity of nerve compression. This review focuses on most common upper extremity entrapment neuropathies which are the CTS, CuTS, RTS and neurogenic thoracic outlet syndrome. Anatomical considerations, patient evaluation, indications for conservative treatment and surgical intervention, outcomes, and complications are discussed.

Carpal Tunnel Syndrome

Anatomy and Pathophysiology

The carpal bones and intercarpal ligaments at its medial, lateral and posterior borders form the carpal tunnel. The anterior border is formed by the transverse carpal ligament and flexor retinaculum [12, 13]. The flexor retinaculum (FR) as a whole can be divided into three parts from proximal to distal. The antebrachial fascia forms the proximal part of the FR. A superficial

fascial layer is inseparable from the thickened deep investing antebrachial fascia, which is anterior to the median nerve and continuous with the transverse carpal ligament distally. The two layers separate to enclose the flexor carpi radialis tendon radially and the contents of Guyon's canal and flexor carpi ulnaris tendon ulnarly. Thus, the deep investing antebrachial fascia at this level is volar to the contents of the carpal tunnel and dorsal to Guyon's canal [14]. The transverse carpal ligament proper represents the middle third of the FR and forms the palmar 'roof' of the carpal tunnel. It inserts into the scaphoid tuberosity and ridge of the trapezium radially and the hamulus and pisiform ulnarly where it is narrowest between the hamate hook and trapezial ridge. The distal third is the aponeurosis between the thenar and hypothenar muscles, from which these muscles originate. The thickness of the FR over the carpal tunnel is 10 times that of the antebrachial fascia [14].

A cross-section at the wrist reveals a tunnel tightly packed with the median nerve and the nine extrinsic flexor tendons of the thumb and fingers (flexor pollicis longus tendon, four flexor digitorum superficialis tendons and four flexor digitorum profundus tendons) and synovium. The median nerve normally divides into six branches at the distal end of the FR. The six branches include the recurrent motor branch encompassing: a proper digital nerve to radial side of the thumb; a short common digital nerve to the first web space that quickly divides into a proper digital nerve to the ulnar side of the thumb and a proper digital nerve to the radial side of the index finger; and two common digital nerves to the second and third web spaces.

Lanz defined four categories of variations found in this nerve in the carpal tunnel: 1) variations in the course of the thenar branch; 2) accessory branches at the distal carpal tunnel; 3) high division of the distal median nerve; and 4) accessory branches proximal to the carpal tunnel. Furthermore, the motor branch may arise in the forearm, or may be separated by the persistent median artery or an aberrant muscle only to join distal to the transverse carpal ligament [15].

Studies of carpal tunnel dimensions using computed tomography or magnetic resonance imaging have yielded some consistent and some inconsistent anatomical findings [16–18]. In one of them, post-operative tunnel volume was found to be increased by 24.2% at 6 weeks and this increase in volume was also found at 8 months follow-up. Tunnel contents were displaced anteriorly after decompression at both 6 weeks and 8 months, but the carpal arch width was not changed [17]. In the other three studies, however, carpal arch width increased by between 7% and 11% after surgery [16, 18].

It has been hypothesized that median nerve compression most likely occurs in wrist flexion at the proximal edge of the transverse carpal ligament where it joins the deep investing fascia of the forearm, the anatomic explanation for Phalen's sign. Alternately, the median nerve may be compressed where the carpal tunnel is narrowest at the level of the hook of the hamate by either synovial hypertrophy or a space-occupying lesion [14]. Using dynamic imaging techniques, several investigators have shown changes in position of carpal contents [17–18], and changes in pressure within the canal [21–24]. Imaging studies of median nerve during wrist flexion have demonstrated that patients with carpal tunnel syndrome are more likely than normal patients to have limited median nerve motion in the carpal canal. The nerve in normal patients moved radially and posteriorly to a position interposed between the flexor tendons during wrist flexion. The nerve in patients with carpal tunnel syndrome was more likely to remain in position at the FR. The limited motion of the nerve in these cases may predispose the nerve to compression during wrist flexion leading to carpal tunnel symptoms [25]. It was reported by Szabo and Chidgey that tunnel pressure after repeated flexion/extension movements took significantly longer to recover in patients with CTS than in normal subjects [24]. Decreased median nerve motion during flexion was observed in patients with non-specific forearm pain with median nerve compression in another study [26]. The sliding of median nerve in response to not only wrist, but also elbow, shoulder and neck movements was analyzed in a study by Dilley et al. [27] and the median nerve was found to be unloaded when the shoulder was adducted or elbow flexed. It was reported in the same study that even when the nerve was loaded in the shoulder abducted, elbow straight and wrist in 60º extension, the blood flow to or conduction in the median nerve was unaffected. It was concluded that the median nerve was well designed to cope with changes in bed length caused by limb movements [27].

The three leading theories of causation of CTS are: 1) repeated compression leading to ischemia, edema formation in the subendoneurial space and the synovium and eventually fibrosis [28], 2) tethering of the nerve due to scar tissue leading to reduced nerve gliding and ischemia [29–31], and 3) localized mechanical pressure from structures such as the FR causing local nerve damage [32]. These theories may overlap, e.g., an increase in extraneurial pressure may push the nerve against a stiff tissue and lead to a localized injury due to mechanical pressure.

Anatomic variations

Various congenital variations intrinsic to the carpal tunnel, anomalous lumbrical origins, presence of a flexor digitorum superficialis muscle belly [33, 34], bifid median nerves, either at the forearm or wrist level [35–38] have been reported. It should be kept in mind that anomalous muscles are occasionally found in this region and can lead to CTS [39, 40]. A patient having both carpal and ulnar tunnel symptoms was reported to have a muscle originating at the tendon of the palmaris longus and ulnar antebrachial fascia that split and extended through Guyon's canal to join portions of the abductor digiti minimi and the FR [40]. Transfer of sensory information between ulnar and median nerves is described [41]. A communicating branch conveying sensory information from the little finger passing from median to ulnar just proximal to the wrist was reported by Saeed and Davies [42]. It was not until this anomalous

communication was decompressed separately that the symptoms were relieved although open release of the carpal tunnel was performed 7 years before.

Patient Evaluation

Clinical assessment includes Phalen's test (appearance or worsening of paresthesia with maximal passive wrist flexion for one minute) and Tinel's sign (paresthesia in the median territory elicited by gentle tapping over the carpal tunnel). Tinel's sign has a sensitivity of 60% and a specificity of 67%; the corresponding values for Phalen's test are 75% and 47% [43,44]. When conducted in the proper setting, these tests can provide useful information. In a clinical setting, an assessment of strength, sensory loss, and pain is sufficient to monitor the progress of the syndrome.

Electrodiagnostic testing should be carried out in most cases. Sensory fibers are used to measure the nerve conduction velocity from the finger or palm to the wrist and motor conduction velocity from the wrist to the thenar muscles. The palmar serial sensory study – sequential measurements at short distances over the course of the nerve in the palm – improves the sensitivity of sensory conduction testing [45]. Electromyography of thenar muscles innervated by the median nerve is also usually done.

Evaluation of selected patients with imaging in addition to nerve conduction studies may be important, as a proportion of symptomatic patients fail to show a decreased median nerve conduction velocity [19,46]. Mesgarzadeh et al. noted four general findings by MRI in CTS regardless of the etiology, including swelling of the median nerve (best evaluated at the pisiform level), flattening of the nerve at the hamate level, palmar bowing of the FR, and increased T2 signal in the median nerve [47]. MRI was also shown to be able to discern unexpected causes of typical CTS prior to surgery, such as a large adductor pollicis muscle, a persistent median artery, an excessive amount of fat tissue within the tunnel, a ganglion cyst, and synovial hypertrophy related to rheumatoid arthritis.

Other methods of imaging the region of the carpal tunnel, like ultrasound and thermography have been evaluated [48–50]. The high spatial resolution and exquisite flow detection methods of ultrasound allows analysis of many superficial soft tissue structures. Abnormalities such as tenosynovitis, synovial hypertrophy, ganglia, giant cell tumor of the tendon sheath, lipomas, aberrant musculovascular anatomy and bursae, metabolic edema and infiltrative processes (e.g. amyloidosis) have all been identified as causes of carpal and tarsal tunnel syndromes [51–53]. Thermography shows clear abnormalities in CTS, but is not reliable for the diagnosis of bilateral cases and has limited value in the differential diagnosis [50].

The standard means of diagnosis is accepted to be the electrodiagnostic testing together with clinical evaluation [54]. Most electromyographers consider the following results abnormal (with control for the patient's age and limb temperature): an absolute sensory latency of more than 3.7 msec, a difference of 0.4 msec or more between values obtained for the median nerve and those obtained for the radial or ulnar nerve, a motor conduction latency of more than 4.0 msec, and an incremental change of 0.4 msec in the palmar serial sensory study with the use of measurements made at standard distances [55].

Evaluation of F-wave responses [56,57], or combined indices of median versus ulnar nerve conduction data [58,59], could be used for better diagnosing the possibility of the so called 'double crush' phenomenon [60], and in demonstrating higher sensitivity and specificity in routine CTS diagnosis [58,59].

Treatment

Conservative treatment includes the avoidance of the use of wrist, using a wrist splint in a neutral position for day and night time, anti-inflammatory medication. Patients with minimal or intermittent symptoms usually get benefit from this kind of nonsurgical management. According to the Practice Parameter of the American Academy of Neurology, local steroid injections are considered a treatment for mild CTS [61]. Several investigators have reported that local steroid injection into the carpal tunnel is an effective treatment of CTS [62–65]. Improvements of nerve conduction parameters beginning as early as 1 month and lasting for at least 6 months by local steroid injections were reported by Hagebeuk and de Weerd [66]. Subjective measures were also improved in this study, although these measures were not correlated with the improvements in nerve conduction parameters.

Surgical intervention is recommended for patients with CTS if they have failed conservative management, or if they have intolerable pain, constant numbness, or any weakness limiting their activities. The procedure is usually done on an outpatient basis and has a good record of success. The surgical technique is either standard open release or endoscopic release. The complication and success rates are similar, but patients return to work sooner and with less pain and debilitation after endoscopic procedures [67].

Complications of carpal tunnel release can be generally reported as incomplete release, neuropraxia or injury to the median or ulnar nerve, inadvertent entrance into Guyon's canal, injury to the digital nerves, the ulnar artery and the superficial palmar arch [68,69].

Ulnar Neuropathy

Ulnar neuropathy at elbow is the second most common entrapment neuropathy after CTS. The ulnar nerve can become compressed primarily in two areas: the wrist (at Guyon's canal) and the elbow. As the nerve passes through the ulnar groove at the elbow, it is prone to several types of compressive injuries.

Anatomy and pathophysiology

The cubital tunnel is the aponeurosis between the two heads of the flexor carpi ulnaris muscle and it contains the ulnar nerve. In some people, the aponeurosis is drawn taut over the nerve, particularly with elbow flexion which was described by O'Driscoll et al. as Type IA [70]. They reported in a cadaver study that the cubital

tunnel retinaculum (which forms the roof of the tunnel) is likely a remnant of the anconeus epitrochlear muscle. The anatomy was classified into four types, ranging from no band at all (Type 0) to having the muscle itself remain (Type II). The nerve is most compressed where it passes through the olecranon notch behind the medial epicondyle [71] if the elbow is repeatedly rested on a flat surface, as well as other points on its passage across the elbow joint, including the epicondyle and epicondylar groove, the flexor muscle mass including its deep aponeurosis and the arcade of Struthers. Most consider the entire area as part of the cubital tunnel or, if not, refer to ulnar nerve entrapment at the elbow overall as CuTS if the appropriate symptom complex has occurred.

Patient evaluation

The clinical picture is typically characterized by nocturnal paresthesias involving 4^{th} and 5^{th} fingers, pain at elbow radiating toward the hand, and sensory symptoms related with prolonged flexion of the elbow. If weakness occurs, it may affect many functions of the hand, including finger abduction, thumb abduction, pinching of the thumb and forefinger, and eventually, power grip. Musicians and other people who require fine control of the digits may note a decline in performance with minimal ulnar compression [72]. The symptom complex may vary in intensity and even disappear for months or years at a time, or acutely with extension of the arm. This syndrome sometimes presents many years after an initial injury to the elbow, often referred to as 'tardy ulnar palsy'. The nerve is often palpably enlarged in the ulnar groove and may be tender. A history of trauma (a fracture or dislocation), arthritis or repetitive minor trauma is helpful. Tophaceous gout [73], calcium pyrophosphate dihydrate crystal deposition [74] and extraneural hemangioma [75] may also lead to acute compression.

Electrodiagnostic studies show a delay of at least 10 msec, or if significant atrophy is present, 15 msec in the affected arm relative to the unaffected arm. A positive Tinel sign is helpful, but it is less precise. One of the earliest findings is the 'Wartenberg's sign' where the little finger is abducted due to weakness of the third palmar interoseeous muscle. Froment's sign is an indicator of motor involvement in ulnar neuropathy, the thumb-forefinger pinch is distorted because of the weakness of the adductor pollicis, the ulnar portion of the flexor polllicis brevis, and the first dorsal interosseous. Montagna in 1994 reported the 'motor' tinel sign in a patient with entrapment of the ulnar nerve at the elbow [76]. Percussion or manipulation of the ulnar nerve at the ulnar groove provoked an electric shock sensation in the hand ('sensory' Tinel sign) associated with a visible motor jerk of the ulnar innervated muscles, which corresponded to myoclonic bursts on electromyographic recordings from the abductor digiti minimi and first dorsal interosseous muscles. It was suggested that 'motor' Tinel sign could be of use in the diagnostic evaluation of entrapment neuropathies.

Differential Diagnosis

Several other entities leading to numbness of the little finger and possible motor weakness should be considered. The ulnar nerve can be compressed at the wrist rather than the elbow by repeated trauma to the palm (often occupational) or by a ganglion or a tumor. Distinction is accomplished by nerve conduction studies. Clinical findings also help to localize the lesion. With entrapment at the wrist, there is no loss of dorsal sensation because the dorsal cutaneous branch of the ulnar nerve leaves the main trunk 5–8 cm proximal to Guyon's canal. Lower cervical radiculopathy, neurogenic thoracic outlet syndrome, amyotrophic lateral sclerosis, syringomyelia and other cord lesions are also to be considered for differential diagnosis.

Treatment

Both conservative and operative modalities exist to treat the CuTS. A good diagnosis that includes an explanation of the anatomical condition of the nerve during postures and movements represents the first therapy [77]. Some cases improve spontaneously without surgical treatment. A part of the improvements could be due to the changing of arm postures, ie, avoidance of frequent or prolonged flexion of the elbow. The patient may try to wear a loose, bivalved cast at night to prevent elbow flexion, but this is usually poorly tolerated. Steroid injections and oral anti-inflammatory agents are not useful. Any external trauma should be eliminated.

In patients with disability, particularly weakness, or with mild to moderate neuropathy but have failed conservative management, a surgical procedure is recommended. Complete decompression, followed by restoration of the nerve to a safe and unkinked location is the primary goal of surgery. The results of the surgery are more related to the degree of preoperative motor or sensory loss than to the type of the surgical procedure. The results are satisfactory in general. Local tenderness and painful paresthesias might persist in a few patients.

Posterior Interosseous Nerve Entrapment (Radial Tunnel Syndrome)

Anatomy

Fractures of the humerus, wrist lacerations and intravascular line placements commonly cause injury to the radial nerve and decompression and repair is needed in most of the cases [78–79]. Idiopathic entrapment of the nerve, however, is quite rare. The posterior interosseous nerve (PIN) branches from the radial nerve immediately distal to the elbow joint, after turning, it enters the supinator muscle through the 'arcade of Frohse', which is a fibrous ring region. Before the distal radial nerve branches into the PIN and the radial sensory nerve, a branch to the extensor carpi radialis leaves it. As the PIN continues distally, it innervates the supinator and extensors of the wrist and fingers. Branches to the extensor carpi radialis brevis may arise just proximal to the site of compression of the nerve. In this case, the extensor carpi radialis longus is intact, and the patient with a PIN compression might not have a wrist

drop, although in extension there is a radial drift. Some branches to the supinator often arise proximal to the arcade of Frohse and supination remains intact.

Patient evaluation

The patient with entrapment of the PIN may have a painless palsy, or have pain that is often difficult to distinguish from lateral epicondylitis. The painful condition is called 'resistant tennis elbow' or 'radial tunnel syndrome'. Either painful or painless, the condition may develop after strenuous use of the forearm. While in lateral epicondylitis the pain is directly over the lateral epicondyle, in case of PIN compression, tenderness is over the nerve in extensor muscle group approximately 3 cm distal to the elbow. A provocative test to differentiate lateral epicondylitis from PIN compression is resisted supination and extension of the middle finger [78].

The probable compression sites of PIN in the forearm region are adhesions between the brachialis and brachioradialis muscles, the edge of the extensor carpi radialis brevis [80], fibrous bands associated with the supinator muscle, and a set of vascular branches sometimes termed 'the leash of Henry' [78, 81]. The exit from the distal supinator is a rare entrapment site.

Treatment

Rest, behavior modification, anti-inflammatory medications and sometimes steroid injections are recommended initially, but the patients eventually require an operation [71, 81].

Thoracic Outlet Syndrome

Pathophysiology

Tingling in the hands with shoulder abduction or elevation is a common finding in patients with neurogenic thoracic outlet syndrome, while measurable neurogenic deficit is very rare. Neurogenic thoracic outlet syndrome is caused by abnormal bands crossing the brachial plexus, often inserting on a rudimentary cervical rib [82]. The symptoms often occur after a whiplash injury, cervical muscle spasm may have a role in the pathogenesis.

Patient Evaluation

Classic features are weakness of all the intrinsic muscles of the hand and sensory loss over the ulnar side of the hand and forearm [82]. Hyperabduction test, which is a change in pulse with abduction of the arm is not a reliable indicator because it can be found in some normal persons, too. Electrodiagnostic testing usually reveals no abnormality.

Treatment

Therapeutic exercises increasing the range of motion of the neck and shoulders, strengthening the rhomboid and trapezius muscles and inducing a more erect posture help patients with reversible paresthesias. For rare patients with worsening of neurologic functions, exploration of the brachial plexus may be recommended although it has some risk [83].

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