Nerve Entrapment Syndromes in the Wrist

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Abstract

The patient with compression neuropathies of the median and ulnar nerves at the wrist commonly presents with pain, paresthesias, and weakness in the hand and digits. Diagnosis of these conditions is becoming more widespread with the increased attention given to "cumulative trauma disorders" during the past decade. Successful management requires a thorough understanding of the pathophysiology of compression neuropathy and how it relates to the various diagnostic tests available today. The authors review the epidemiology, etiology, and evaluation of compression neuropathy and discuss common clinical presentations, treatment recommendations, and controversies surrounding carpal and ulnar tunnel syndromes.

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Compression neuropathy at the wrist is not a single disease, but rather a constellation of symptoms resulting from compression of either the median or the ulnar nerve caused by a disparity between the size of the corresponding tunnel and its contents. History and physical examination will localize the site of compression and direct further diagnostic studies. Once the cause is determined, appropriate therapy can, in most cases, yield a successful outcome.

Epidemiology

Upper-extremity compression neuropathy remains one of the most frequently encountered disorders seen by orthopaedists and hand surgeons. Classically, these syndromes have presented as either posttraumatic conditions or the gradual onset of paresthesias and pain in a patient, typically female, in late middle age. In the past decade, these two forms have been surpassed by another presentation—symptoms developed in the younger industrial worker in relation to repetitive motions.

This syndrome is one of a group of nonspecific conditions termed "cumulative trauma disorders." Workers' compensation litigation and labor-management hostilities, as well as psychological and economic factors, are often an important part of the picture. Workstation and task-related modifications should be undertaken first, as these may obviate the need for surgical treatment. Objective evidence of a specific nerve disorder should be demonstrated before surgical intervention is recommended. It also should be emphasized that a large number of patients who obtain relief of symptoms after surgical decompression ultimately will require job retraining.[1,2]

Industry continues to seek a screening tool for identifying patients at risk for upper-extremity compression neuropathies. Preemployment screening is controversial and can lead to discriminatory practices. The only clearly documented intrinsic risk factors appear to be female sex, pregnancy, diabetes, and rheumatoid arthritis. Occupational factors include task repetition, force, mechanical stresses, posture, vibration, and temperature. However, the relative importance of these factors and the mechanisms by which they produce neuropathy are poorly understood.

The growing importance of work-related factors has required a compensatory change in the physician's approach to managing these conditions. The surgeon must treat the patient, rather than focus on the injured extremity. This may best be accomplished with a team approach, with contributions by a physical therapist, an occupational therapist, a psychologist, a kinesiologist, and, most important, the patient. A successful outcome is more likely if the patient becomes an active participant in his or her own rehabilitation. Progress toward correction of obesity, alcohol abuse, or tobacco abuse is good evidence of the patient's commitment. If specific objective evidence of a compression neuropathy is lacking, it is best to institute a trial of nonoperative management and to let other members of the team assume the primary role in treatment.

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Etiology

Between the cervical spine and the wrist there are a number of specific sites where nerve compression is common, giving rise to various well-known nerve compression syndromes. The most common site for compression is at the wrist in the region of the carpal and ulnar tunnels. Here both median and ulnar nerves may be entrapped in their anatomic compartments (Fig. 1).

Some of the factors associated with the development of carpal tunnel and ulnar tunnel syndromes are listed in Tables 1 and 2, respectively. A careful history and physical examination can usually identify the specific causative factor. In most cases, the appropriate surgical procedure to decompress the involved nerve has been established. However, the concept that a static anatomic structure is the sole cause of a nerve compression syndrome is too simple; other factors enter into the clinical picture. For example, in idiopathic carpal tunnel syndrome, the point of compression is the flexor retinaculum. The pathologic changes, however, are related to fibrous hypertrophy of the flexor tendon synovium, probably secondary to repeated mechanical stresses that induce local necrosis with edema and collagen fragmentation.

The principle that chronic inflammation is the underlying cause of idiopathic carpal tunnel syndrome has also been challenged. Only 4% to 10% of biopsy specimens of tenosynovium from over 800 wrists that underwent carpal tunnel release revealed the presence of inflammatory cells, while edema and vascular sclerosis were consistently observed (98% of cases).[3,4] Recently, two investigative groups examined tenosynovium specimens from patients with idiopathic carpal tunnel syndrome and found amyloid deposition in an overwhelming majority.[5,6]

Systemic Conditions

Diabetes, alcoholism, hypothyroidism, and exposure to chemical toxins may cause systemic depression of peripheral nerve function, which lowers the threshold for manifestation of a compression neuropathy. Aging may have a similar systemic effect. The importance of systemic conditions may be reflected in the high prevalence of bilateral occurrence and multiple-nerve involvement, even if only one extremity is used in the activity that provokes symptoms.

Children with mucopolysaccharidosis or mucolipidosis, a rare group of disorders, frequently have carpal tunnel syndrome and benefit from early carpal tunnel release. Systemic conditions that alter interstitial fluid equilibrium (e.g., pregnancy, myxedema, long-term hemodialysis, and rheumatoid arthritis), extreme wrist positions, and proliferation of flexor tendon tenosynovium also may cause nerve compression.

Ischemia and Mechanical Compression

Experimental and clinical studies and intraoperative observations suggest ischemic causation for many compression neuropathies.[7] Reduced epineural blood flow is the earliest manifestation of low-grade peripheral nerve compression and can occur experimentally at compression pressures as low as 20 to 30 mm Hg. Axonal transport becomes impaired at 30 mm Hg, with a subsequent increase in endoneural fluid pressure. Neurophysiologic changes and symptoms of paresthesias have been induced in human volunteers with 30 to 40 mm Hg of compression on the median nerve. Experimental compression at 50 mm Hg for 2 hours caused epineural edema and axonal transport block in animal studies. Pressures greater than 60 mm Hg cause total intraneural ischemia with a complete sensory block followed by complete motor block.

In chronic cases of nerve compression, recovery following decompression may be very slow, or progression of the condition may halt without improvement of symptoms. In these cases, the initial vascular causation is superseded by other processes, particularly fibrosis of the nerve, that diminish potential for recovery.

Recognition of these physiologic changes in peripheral nerves secondary to progressive ischemia has led to the classification of nerve compression lesions into early, intermediate, and late stages. Early, low-grade compression responds most favorably to conservative management, such as splinting and modification of activities and limb position. Intermediate-stage nerve

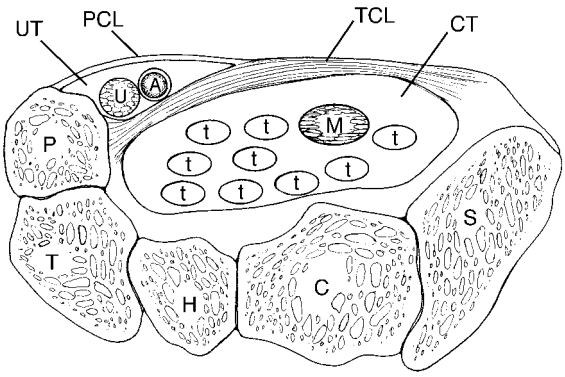

Fig. 1 Cross-section of the wrist demonstrating the relationship of the carpal tunnel (CT) and the ulnar tunnel (UT). A = ulnar artery, C = capitate, H = hamate, M = median nerve, P = pisiform, PCL = palmar carpal ligament, S = scaphoid, t = flexor tendon, T = triquetrum, TCL = transverse carpal ligament, U = ulnar nerve.

Table 1
Factors in the Pathogenesis of Carpal Tunnel Syndrome*

Anatomy
Decreased size of carpal tunnel
Abnormalities of the carpal bones
Thickened transverse carpal ligament
Acromegaly
Increased contents of canal
Neuroma
Lipoma
Myeloma
Abnormal muscle bellies
Persistent median artery (thrombosed or patent)
Hypertrophic synovium
Distal radial fracture callus
Posttraumatic osteophytes
Hematoma (hemophilia, anticoagulation therapy)
Physiology
Neuropathic conditions
Diabetes
Alcoholism
Proximal lesion of median nerve (double-crush syndrome)
Inflammatory conditions
Tenosynovitis
Rheumatoid arthritis
Infection
Gout
Physiology (continued)
Alterations of fluid balance
Pregnancy
Eclampsia
Myxedema
Long-term hemodialysis
Horizontal position and musle relaxation (sleep)
Raynaud's disease
Obesity
Congenital
Mucopolysaccharidosis
Mucolipidosis
Position and use of the wrist
Repetitive flexion / extension (manual labor)
Repetitive forceful squeezing and release of a tool
Repetitive forceful torsion of a tool
Finger motion with the wrist extended
Typing
Playing many musical instruments
Vibration exposure
Weight-bearing with the wrist extended
Paraplegia
Long-distance bicycling
Immobilization with the wrist flexed and ulnar deviation
Casting after Colles fracture
Awkward sleep position

* Adapted with permission from Szabo RM, Madison M: Carpal tunnel syndrome. *Orthop Clin North Am* 1992;23:106.

compression is caused by persistent interference with intraneural microcirculation and is characterized by symptoms of constant paresthesias and numbness. This is best treated by decompression of the nerve. In late-stage cases, long-standing endoneural edema induces fibroblast invasion and endoneural fibrosis. Patients in this stage have permanent sensory loss and muscle atrophy; decompression alone may not eliminate all symptoms. These patients were once thought to benefit from internal neurolysis, but several recent studies have shown that neurolysis offers no additive benefits.[8,9]

Traction Injuries

Nerves of the upper extremity have considerable mobility throughout their length. Focal compression may tether the nerve, restricting its mobility, and thereby cause traction in response to joint motion. Traction alone can cause conduction block. It is likely, though not yet demonstrated, that many upper-extremity compression neuropathies are due, at least in part, to traction on the nerve.

Double-Crush Syndrome

Normal function of the axon depends on the synthesis of various enzymes, polypeptides, polysaccharides, free amino acids, neurosecretory granules, mitochondria, and tubulin subunits by the proximal nerve cell body. Fast and slow axoplasmic transport mechanisms regulate the distal flow of these substances along the axon and the proximal return of degradation products. Any disruption of the synthesis or transport of these materials will increase susceptibility of the axons to compression. A compression lesion at one point on a peripheral nerve will lower the threshold for occurrence of compression neuropathy at another locus, distal or proximal, on the same nerve, possibly by restricting axonal transport kinetics.[10] In such cases, both areas of entrapment may need to be decompressed. For instance, when a proximal cervical lesion is present, less compression of the median nerve at the carpal tunnel level is necessary to produce symptoms. Coexistent cervical root compression is one of the reasons for persistent symptoms following carpal tunnel release.

Table 2
Factors in the Pathogenesis of Ulnar Tunnel Syndrome

Anatomy
Ganglia
Soft-tissue masses
Abnormal muscle bellies
Hook of hamate fracture
Distal radial fracture
Thickening of proximal fibrous hypothenar arch
Hypertrophic synovium
Iatrogenic (after opponensplasty)
Physiology
Inflammatory conditions
Tenosynovitis
Rheumatoid arthritis
Edema secondary to burns
Gout
Coexistent carpal tunnel syndrome
Vascular conditions
Ulnar artery thrombosis
Ulnar artery pseudoaneurysm
Neuropathic conditions
Diabetes
Alcoholism
Proximal lesion of ulnar nerve (double-crush syndrome)
Occupation-related
Vibration exposure
Repetitive blunt trauma
Direct pressure on ulnar nerve with wrist extended
Typing
Cycling

Appearance of Symptoms

In most cases, nerve compression is gradual in onset and symptoms are chronic. In dynamic or exertional compression, symptoms appear in response to a specific provocative activity and resolve when the activity is stopped. The more classic presentation of entrapment is gradual, with less obvious relationships to activity. The patients' symptoms often are worse at night. It is important to distinguish these two presentations by obtaining a careful history.

Rarely, nerve compression at the wrist develops rapidly secondary to trauma. An acute presentation, which is analogous to a compartment syndrome, should be considered a surgical emergency requiring prompt decompression. For instance, acute carpal tunnel syndrome may be seen following a distal radial fracture or bleeding from a malfunctioning radial arterial line. Acute compression presents with significant swelling over the carpal tunnel and progressive deterioration in median nerve function. This should be differentiated from contusion of the median nerve. In the latter, swelling over the carpal tunnel is usually less tense, and the patient will report paresthesias in the median nerve distribution that occurred at the time of injury and have not changed over time. If there is any doubt, the physician should measure carpal tunnel pressures. Median nerve decompression is indicated when the pressure exceeds 40 mm Hg. An anatomic or metabolic double-crush syndrome should also be considered in the differential diagnosis.

Carpal Tunnel Syndrome

Compression of the median nerve at the wrist is the most common compression neuropathy of the upper extremity. The clinical presentation consists of pain and paresthesias on the palmar-radial aspect of the hand, often worse at night and/or exacerbated by extreme wrist positions (e.g., those used in driving and prolonged typing) or repetitive forceful use of the hand. The frequent complaint of dropping items is often related to alterations in sensibility, although it can be secondary to thenar weakness in patients with a chronic and advanced stage of compression.

Diagnosis

A variety of diagnostic tests are available for characterizing carpal tunnel syndrome (Table 3). In most cases, radiographic information is of limited value. Plain radiographs in two orthogonal planes should be obtained to rule out posttraumatic deformity and soft-tissue calcifications or Kienböck's disease. A carpal tunnel view rarely adds any useful information.

In general, there is a trade-off between tests that have only modest accuracy but are easily performed (e.g., Phalen's test) and tests that are highly specific but difficult, expensive, or invasive (e.g., electrodiagnostic tests and direct measurement of carpal tunnel pressures). The use of liquid crystal thermography and ultrasonography has received some attention, but the sensitivity of these techniques is quite low, and they are not useful in the diagnosis of either carpal or ulnar tunnel syndrome. Although magnetic resonance (MR) imaging and computed tomography (CT) are helpful in visualizing certain anatomic factors responsible for compression, they are not useful for specifically diagnosing entrapment neuropathy at the wrist unless one suspects a mass lesion. Symptomatic nerve compression does not correlate with alterations in MR signals or anatomic details seen on CT.

Sensibility testing is an important part of the workup of a patient with a nerve compression lesion. A clear understanding of the nature of what each test is measuring has eliminated much of the controversy surrounding the supposed superiority of the various tests. Different fiber populations and receptor systems are evaluated by four available sensory tests. Touch fibers (group A-beta) can be divided into slowly and quickly adapting fiber systems. A quickly adapting fiber responds to an on-off event, and a slowly adapting fiber continues to fire through-

Table 3
Diagnostic Tests for Carpal Tunnel Syndrome*

Test	How Performed	Condition Measured	Positive Result	Interpretation of Positive Result†
Phalen's test	Patient places elbows on table, forearms vertical, wrists flexed	Paresthesias in response to position	Numbness or tingling on radial-side digits within 60 sec	Probable CTS (sensitivity, 0.75; specificity, 0.47)
Percussion test (Tinel's)	Examiner lightly taps along median nerve at the wrist, proximal to distal	Site of nerve lesion	Tingling response in fingers at site of compression	Probable CTS if response is at the wrist (sensitivity, 0.60; specificity, 0.67)
Carpal tunnel compression test	Direct compression of median nerve by examiner	Paresthesias in response to pressure	Paresthesias within 30 sec	Probable CTS (sensitivity, 0.87; specificity, 0.90)
Hand diagram	Patient marks sites of pain or altered sensation on outline diagram of the hand	Patient's perception of site of nerve deficit	Signs on palmar side of radial digits without signs in palm	Probable CTS (sensitivity, 0.96; specificity, 0.73); negative predictive value of a negative test = 0.91
Hand-volume stress test	Measure hand volume by water displacement; repeat after 7-min stress test and 10-min rest	Hand volume	Hand volume increased by 10 ml or more	Probable dynamic CTS
Direct measurement of carpal tunnel pressure	Wick or infusion catheter is placed in carpal tunnel; pressure is measured	Hydrostatic pressure while resting and in response to position or stress	Resting pressure of 25 mm Hg or more (this value is variable and may not be valid in and of itself)	Hydrostatic compression at wrist is probable cause of CTS
Static two-point discrimination	Determine minimum separation of two points perceived as distinct when lightly touched on palmar surface of digit	Innervation density of slowly adapting fibers	Failure to discriminate points more than 6 mm apart	Advanced nerve dysfunction
Moving two-point discrimination	As above, but with points moving	Innervation density of quickly adapting fibers	Failure to discriminate points more than 5 mm apart	Advanced nerve dysfunction
Vibrometry	Vibrometer head is placed on palmar side of digit; amplitude at 120 Hz increased to threshold of perception; compare median and ulnar nerves in both hands	Threshold of quickly adapting fibers	Asymmetry with contralateral hand or between radial and ulnar digits	Probable CTS (sensitivity, 0.87)
Semmes-Weinstein monofilament test	Monofilaments of increasing diameter touched to palmar side of digit until patient can tell which digit is touched	Threshold of slowly adapting fibers	Value greater than 2.83 in radial digits	Median nerve impairment (sensitivity, 0.83)
Distal sensory latency and conduction velocity	Orthodromic stimulus and recording across wrist	Latency and conduction velocity of sensory fibers	Latency greater than 3.5 msec or asymmetry greater than 0.5 msec compared with contralateral hand	Probable CTS
Distal motor latency and conduction velocity	Orthodromic stimulus and recording across wrist	Latency and conduction velocity of motor fibers of median nerve	Latency greater than 4.5 msec or asymmetry greater than 1.0 msec	Probable CTS
Electromyography	Needle electrodes placed in muscle	Denervation of thenar muscles	Fibrillation potentials, sharp waves, increased insertional activity	Very advanced motor median nerve compression

* Adapted with permission from Szabo RM, Madison M: Carpal tunnel syndrome. *Orthop Clin North Am* 1992;23:105.
†CTS = carpal tunnel syndrome.

out the duration of the stimulus. Slowly adapting fibers are evaluated by static two-point discrimination and Semmes-Weinstein monofilament tests. Vibration and moving two-point discrimination tests assess the quickly adapting fibers. Each fiber system, in turn, is associated with a specific sensory receptor. Each clinical test of sensibility is related to one of these receptor groups and is classified as either a threshold or an innervation density test.

A threshold test measures a single nerve fiber innervating a receptor or group of receptors and is more sensitive in evaluating nerve com-

pression. Semmes-Weinstein monofilament and vibration tests are threshold tests and are more likely to detect a gradual, progressive change in nerve function. An innervation density test measures multiple overlapping peripheral receptive fields and the density of innervation in the region being tested. Static and moving two-point discrimination are innervation density tests, which require overlapping of different sensory units and complex cortical integration. Innervation density tests are reliable when assessing functional nerve regeneration after nerve repair but are not sensitive to the gradual decrease in nerve function seen in nerve compression.[11,12] Two-point discrimination may remain intact even if only a few fibers are conducting normally to their correct cortical end points; it will be abnormal only in advanced cases of nerve compression. At present, Semmes-Weinstein monofilament testing is simpler and less expensive than vibration testing, but just as reliable and sensitive.

Provocative testing is crucial to the diagnosis of dynamic nerve compression. Most physicians are familiar with nerve percussion and wrist flexion tests (Table 3). A modification of Phalen's test, adding some measure of objectivity, has been described by Koris et al.[13] Sensory testing with Semmes-Weinstein monofilaments can be performed before flexion and after the wrist has been maintained in flexion for 60 seconds in order to detect early sensibility changes.[14]

More specialized forms of provocative testing are crucial to the diagnosis of dynamic nerve compression. Many patients with these disorders are asymptomatic at rest and manifest symptoms only after a period of a specific activity. For this reason, diagnostic tests performed in an office setting may produce false-negative results. Braun et al[14] have shown that carpal tunnel syndrome can be provoked and that associated physiologic changes, such as the volume of water displaced by the hand, can be objectively measured. If the history suggests a dynamic condition, the patient should be tested after a provocative activity during or after which symptoms are experienced, such as typing, shoveling, or playing the violin.

Electrodiagnostic testing remains the benchmark examination; however, several caveats are in order. It is highly operator-dependent; different operators and equipment, different electrodes and their placement, and varying testing environments may influence results. Systemic conditions (including age-dependent alterations in nerve conduction) may confound the comparisons. Electrodiagnostic measurements have been reported as normal in 8% to 20% of patients with clinically or surgically proved nerve entrapment.[15,16] Nerve-conduction velocities and latencies can be compared with published population norms, with those in the contralateral nerve or in other nerves in the same extremity, or with those obtained in previous tests on the same patient. Studies of a particular nerve repeated on several occasions can document progression or resolution of a neuropathy. Inching (nerve-conduction studies done over small segments of the median nerve at the wrist) and antidromic/orthodromic palmar techniques are useful in localizing a lesion. The true value of nerve-conduction studies is that they often provide the only objective evidence of the neuropathic condition.

It is important not to concentrate too early on compression at the wrist, but to consider the carpal tunnel syndrome in view of the patient's overall health. If the condition is bilateral, metabolic abnormalities or other systemic causes should be sought. Similarly, it is important to look for evidence of proximal nerve compression, such as cervical radiculopathy, thoracic outlet syndrome, and pronator syndrome. Patients with poliomyelitis or paraplegia, whose upper extremities become weight-bearing in extremes of wrist extension through the use of wheelchairs and other ambulatory aids, are predisposed to carpal tunnel syndrome. This group of patients also is more refractory to surgical intervention.[17]

Conservative Treatment

Conservative therapy includes splinting the wrist in neutral position, oral anti-inflammatory drugs to reduce synovitis, diuretics to reduce edema, and medical management of underlying systemic diseases. The great interest in pyridoxine (vitamin B_6) for treatment of carpal tunnel syndrome has faded, as it does not appear to modify the natural history of this disease. Corticosteroid injections will offer transient relief to 80% of patients; however, only 22% will be symptom-free 12 months later.

Those likely to benefit the most from a combination of steroid injection and splinting have had symptoms for less than 1 year, accompanied by mild and intermittent paresthesias. Their physical examinations reveal normal two-point discrimination and no weakness or thenar atrophy. Neurophysiologic studies show no denervation potentials on electromyography and only 1- to 2-msec prolongation of distal motor and sensory latencies.[18] Forty percent of this group will remain symptom-free for longer than 12 months.

Workstation evaluation and redesign, ergonomic tool modification, simple hand and wrist exercises during breaks, and patient education will often alleviate the symptoms associated with work-related carpal tunnel syndrome.

Surgical Treatment

Failure of nonoperative treatment is an indication for surgical release

of the transverse carpal ligament. The choice between open and endoscopic release remains an area of controversy. We believe that the reliability and good visualization possible with an open procedure make it still the preferred technique, especially for the surgeon who does not do a large volume of these surgeries.[19-21]

Reconstruction of the transverse carpal ligament has been proposed as a better method than carpal tunnel release alone in the young laboring individual.[22] The operation requires considerably more dissection, with release of Guyon's canal and mobilization of the ulnar nerve and artery. Until prospective randomized studies confirm any benefits, this procedure should be reserved for situations in which repair of the ligament is necessary. Repair of the ligament is indicated to prevent bow-stringing when it is necessary to immobilize the wrist in some flexion after releasing the carpal tunnel (e.g., if a flexor tendon was repaired).

Previously, internal neurolysis was a commonly used adjunctive procedure in operative treatment of carpal tunnel syndrome. Several clinical studies have failed to demonstrate any benefit from neurolysis, and it is no longer recommended.[8,9]

Patients with carpal tunnel symptoms occasionally may have paresthesias in the little finger. Some surgeons have recommended simultaneous release of Guyon's canal. This is no longer recommended. Recent MR imaging evidence shows that the dimensions of Guyon's canal enlarge with carpal tunnel release alone.[23] Clinically, this finding has been substantiated because patients' ulnar nerve symptoms, if truly coming from compression of Guyon's canal, get better after carpal tunnel release alone.

Ulnar Tunnel Syndrome

Ulnar tunnel syndrome, due to pathologic compression of the ulnar nerve at the wrist, occurs where the nerve passes through the confines of the canal of Guyon (Fig. 1). The patient may present with numbness along the little finger and the ulnar half of the ring finger and / or weakness of grip, particularly in activities in which torque is applied to a tool. Rarely, a patient may first appear with wasting of the intrinsic musculature in the hand. Pain is usually a less significant aspect of the presentation than it is in carpal tunnel syndrome.

Diagnosis

Ganglia and other soft-tissue masses are responsible for 32% to 48% of cases of ulnar tunnel syndrome. Another 16% of cases are due to muscle anomalies.[24] Computed tomography or MR imaging may be useful in visualizing these abnormalities. Fractures of the distal radius and ulna and the hook of the hamate may cause compression of the ulnar nerve in the ulnar tunnel. Plain radiographs, including carpal tunnel and oblique views of the wrist, are frequently diagnostic, although hamate fractures are best identified on CT scans. Other causes of ulnar tunnel syndrome include thrombosis or pseudoaneurysms of the ulnar artery, edema secondary to burns, and inflammatory arthritis.

Ulnar tunnel syndrome may present with pure motor, pure sensory, or mixed symptoms, depending on the precise location of entrapment. The distal ulnar tunnel is divided into three zones to allow more accurate localization of the site of ulnar nerve compression (Fig. 2).[25] Zone 1 is the area proximal to the bifurcation of the nerve. It begins at the edge of the palmar carpal ligament and is about 3 cm in length. Compression in zone 1 causes combined motor and sensory deficits and is most likely due to ganglia or fractures of the hook of the hamate. Zone 2 surrounds the deep motor branch. Compression in this region will produce pure motor deficits. Ganglia and fractures of the hook of the hamate are the most likely causes. Zone 3 surrounds the superficial branch of the ulnar nerve. Compression in this region produces sensory deficits without motor abnormalities. Synovial inflammation has been reported to cause compression in zone 3. More frequently, however, compression in zone 3 is due to thrombosis or an aneurysm of the ulnar artery. The Allen test and Doppler studies are useful in making this diagnosis.

Differential diagnosis includes cubital tunnel syndrome, thoracic outlet syndrome, and cervical root compression.

The elbow is the most common site of ulnar nerve entrapment. The site of the compression should be delineated by careful physical examination before concluding that the ulnar tunnel is causative. Sensory involvement on the ulnar dor-

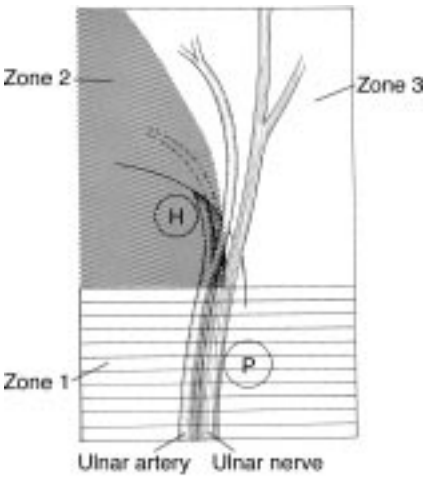

Fig. 2 Schematic drawing of the distal ulnar tunnel showing the location of the three zones. H = hook of hamate; P = pisiform.

sal aspect of the hand suggests compression proximal to the wrist, as the dorsal cutaneous branch of the ulnar nerve originates in the forearm. Weakness of the deep flexors to the ring and little fingers, as well as weakness of the flexor carpi ulnaris, also signals proximal ulnar nerve entrapment. A chest radiograph to rule out a Pancoast tumor should be obtained whenever a history of smoking, ulnar nerve symptoms, or shoulder pain is given by the patient.

Treatment

Initial conservative care for ulnar tunnel syndrome is similar to that for carpal tunnel syndrome. In the absence of an identifiable lesion, alterations of repetitive activities, splint immobilization of the wrist in neutral, and nonsteroidal anti-inflammatory agents may alleviate symptoms. Operative intervention is recommended for patients who are refractory to conservative care or who have documented anatomic lesions. Regardless of the suspected site of compression in Guyon's canal, the ulnar nerve should be visualized and released in its entirety within the ulnar tunnel.

Summary

Compression neuropathy at the wrist is one of the most frequently encountered disorders in the upper extremity. A thorough history and physical examination will localize the site of compression and aid in determination of a cause. Appropriate laboratory, imaging, and sensibility studies will guide the physician in diagnosis and staging of nerve compression. While electrodiagnostic testing remains the benchmark examination, provocative sensibility testing is very sensitive in many early cases of neuropathy.

Patients with cumulative trauma are best treated with a team approach including evaluation of the work environment; symptoms can often be alleviated with nonoperative intervention. Conservative therapy for nerve compression at the wrist includes a combination of splinting, activity modification, and treatment of underlying systemic disease. Evolving concepts of the pathophysiology of compression neuropathy at the wrist may challenge the traditional roles that oral anti-inflammatory agents and corticosteroid injections have played in treating these disorders.

Failure of conservative therapy and the presence of documented surgical lesions are indications for operative intervention. While endoscopic carpal tunnel release has gained popularity, the versatility, lower complication rate, and more satisfactory long-term follow-up of the open procedure indicate that this remains the preferred technique for surgical release of compression neuropathy at the wrist.

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