PART

Emergency Aspects of Orthopedic Surgery

CHAPTER 54

Painful Syndromes of the Hand and Wrist

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This chapter addresses specific disorders that constitute the larger group of problems known as *repetitive motion disorders*. Repetitive motion disorders are a group of musculo-tendinous maladies that are generally associated with workplace or occupational etiologies. Injuries due to repetitive motions are quite common and appear to be increasing in incidence. In 1980, these injuries were noted to comprise about 18% of all reported work-related injuries. In 1990, they were reported to make up approximately 48% of all work injuries. United States Bureau of Labor Statistics data in 1994 reported over 92,500 repetitive stress injury cases involving the upper extremity. These injuries were reported to be associated with over 37,000 days of work lost. The economic impact of these injuries is enormous and represents one of the most pressing problems in occupational health and safety today.

CARPAL TUNNEL SYNDROME

Carpal tunnel syndrome (CTS) is a commonly occurring entrapment neuropathy involving the median nerve at the wrist and represents the most common of all peripheral compression neuropathies.[10]

At the level of the wrist, the median nerve exists in an anatomic tunnel or canal that is formed dorsally by the carpal bones and volarly by the fibrous, but rigid, transverse carpal ligament (Fig. 54-1). The flexor tendons and their accompanying sheaths also lie within this anatomic tunnel. The tunnel begins approximately at the level of the volar wrist crease and extends distally approximately three centimeters. Despite the fact that the carpal tunnel is open at both of its ends, the canal maintains the essential physiologic properties of a closed compartment. When intracanal interstitial pressure becomes elevated above a critical threshold, capillary blood flow rates are reduced below that which is essential for the maintenance of median nerve viability and function. Any condition that tends to decrease the internal area of the canal or increase the volume of the anatomical contents of the canal could theoretically induce carpal tunnel syndrome by resulting in impingement of the median nerve.

Numerous specific clinical conditions have been causally associated with the development of the carpal tunnel syndrome. However, it is important to remember that the idiopathic variety is probably the most common. Some authors have suggested that persons who happen to have an anatomically small canal (on a genetic basis) are more likely to develop this syndrome.[7] In addition, rheumatoid arthritis, diabetes, myxedema, and pregnancy are noted to be frequent causes of carpal tunnel syndrome. Acute fracture of the distal radius as well as fracture and dislocation of the carpus can result in carpal tunnel syndrome by either direct trauma to or compression of the median nerve secondary to bleeding. Infections involving bacterial, viral, or fungal etiologies can cause synovitis, which may result in local nerve compression with the ultimate development of CTS. Any space-occupying lesion such as tumors, as well as osteophytes resulting from malunited fractures or degenerative joint disease, can result in the development of median nerve compression with CTS symptoms.

The 1994 Bureau of Labor Statistics data indicate that about 9% of cases are associated with typing and key entry jobs, 6% with repetitive use of tools, and 12% with repetitive grasping motions. Carpal tunnel syndrome occurs more frequently in women in a ratio of greater than 2:1. Females account for over 78% of all cases. CTS is noted to be a condition of middle age and is typically seen most often in the age range of 40 to 60 with the mean age at diagnosis reported to be 50 for men and 51 for women.

CLINICAL PRESENTATION

The classically recognized clinical presentation of CTS involves paresthesias and pain at the radial aspect of the palm of the hand, frequently increased at night. The modern view of the etiology of this syndrome involves forceful, repeated hand motion associated with work. CTS is often a bilateral condition with the patient typically complaining of numbness, tingling, burning, or paresthesias in the hand along the distribution of the median nerve (the thumb, index, middle, and radial aspect of the ring fingers). These symptoms characteristically awaken the patient from sleep and may be relieved by shaking the hand and wrist. A nocturnal pain pattern is often the hallmark of CTS and as the syndrome progresses, the occurrence of symptoms may be seen to increase. The symptoms are often exacerbated by activities that maintain the wrist flexed, such as driving, holding a book, and by use of the hand such as with knitting, sewing, and hammering. Patients may also complain of weakness or clumsiness in the hand manifested by difficulty with fine motor functions such as picking up objects, holding things, and buttoning clothing.

Physical examination may reveal decreased sensation to light

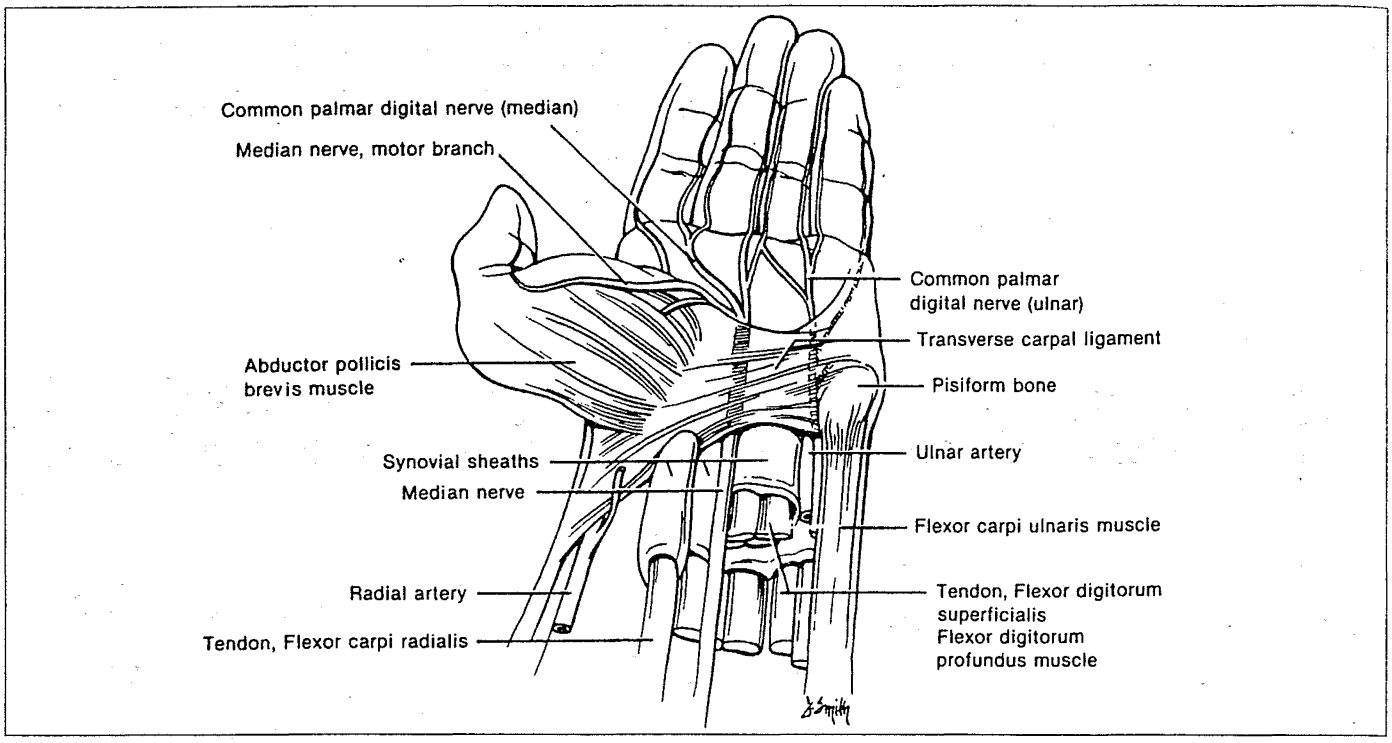

Figure 54.1. View of the carpal tunnel. The median nerve and flexor tendons, which are in the tunnel, lie below the transverse carpal ligament that forms the roof of the tunnel.

touch or decreased two-point discrimination in the median nerve distribution. Phalen's wrist-flexion test and Tinel's sign are frequently noted to be positive with carpal tunnel syndrome.[6,11] Phalen's test is considered to be positive if holding the wrist in complete, but unforced, flexion for 30 to 60 seconds reproduces or exaggerates the numbness and paresthesias in the median nerve distribution. Tinel's sign is positive if light tapping over the median nerve at the wrist causes a tingling sensation distally along the median nerve.

Phalen's test is believed to be more useful in the diagnosis of carpal tunnel syndrome.[11] In more advanced cases there may be weakness of the median-innervated thenar muscles, the abductor pollicis brevis, and the opponens. Thenar atrophy is best seen as a guttering appearance along the radial aspect of the thenar eminence that exposes the first metacarpal. In cases of carpal tunnel syndrome caused by an inflammatory synovitis (rheumatoid arthritis), there may be swelling along the volar aspect of the wrist.

Recent attempts to develop consensus criteria for the diagnosis and classification of CTS have been described. The first principle elucidated by these attempts is that no "gold standard" currently exists for the diagnosis of CTS. Rather, a combination of electrodiagnostic study results in conjunction with symptomatology will be expected to provide the most accurate diagnostic information. The use of electrodiagnostics alone is not considered sufficient to make the definitive diagnosis of CTS.

DIFFERENTIAL DIAGNOSIS

The clinical manifestations of carpal tunnel syndrome are most often characteristic and consequently there is seldom confusion with other entities. Occasionally, the clinical presentation may be unusual and consideration may need to be given to other entities that share characteristics with carpal tunnel syndrome.

Cervical root irritation, especially at the C6 and C7 levels, secondary to either a herniated cervical disc or osteoarthritis with encroachment on the neural foramina, may produce numbness in the median-innervated fingers, with pain radiating to the shoulder. However, cervical root pathology usually causes radicular symptoms that are seldom bilateral and that are worse at night.

Compression of the median nerve in the proximal forearm (pronator syndrome) may also present with numbness in the radial three and one-half digits and weak thenar muscles. However, in this case, Phalen's test would be expected to be negative and Tinel's sign would be positive in the forearm rather than at the wrist. In this case, nocturnal awakening is unlikely, and there would be pain in the volar forearm both subjectively and with palpation.

EMERGENCY DEPARTMENT EVALUATION

The diagnosis of carpal tunnel syndrome is usually made by a careful history and physical examination. Radiographs of the wrist, including a carpal tunnel view, may show structural abnormalities such as callus, osteophytes, and calcific deposits. Cervical spine films may be useful if clinical symptoms suggest the presence of a cervical radiculopathy. Thyroid function tests, fasting blood sugar, erythrocyte sedimentation rate, rheumatoid factor, antinuclear antibodies, determination of serum calcium and uric acid levels, and a white blood cell count with differential may all be useful when clinically indicated.

EMERGENCY DEPARTMENT MANAGEMENT

The initial treatment for the majority of patients involves conservative care. Wrist splints in neutral or slight extension can be worn when symptoms arise, especially at night. The patient should be told to avoid any upper extremity activities that obviously aggravate the symptoms. When underlying medical conditions are suspected (e.g., hypothyroidism, rheumatoid arthri-

tis, gout) appropriate referral to either orthopedic or plastic surgeons with special expertise in treating CTS is required. Diuretics may be useful if carpal tunnel syndrome is associated with pregnancy but should be given only in consultation with the patient's obstetrician.

EMERGENCY DEPARTMENT DISPOSITION

All patients presenting in the emergency department with symptoms of carpal tunnel syndrome should be referred to appropriate specialists for further evaluation and treatment. Outpatient electromyographic and nerve conduction studies are useful when trying to differentiate carpal tunnel syndrome from other causes of similar symptoms or when objective signs of carpal tunnel syndrome are absent but the history and subjective symptoms are compatible with carpal tunnel syndrome. These studies are not generally ordered from the emergency department but would be reserved for the consultant.

The injection of corticosteroids into the carpal canal can result in substantial symptomatic improvement, sometimes even complete relief. However, following steroid injection, the symptoms will often recur.[8] Surgical decompression of the carpal canal is the treatment of choice for those patients who fail conservative care. Indications for operation include muscle weakness, thenar atrophy, and no response to or recurrence after conservative treatment. Surgery involves what is known as carpal tunnel release (CTR), consisting of the division of the transverse carpal ligament. This surgery generally affords good results. However, recurrence of symptoms is seen in up to 20% of patients with as many as 12% requiring reoperation. More recently, endoscopic surgical release procedures have been successful.

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GUYON'S CANAL (ULNAR TUNNEL) SYNDROME

The ulnar nerve, originating from both C8 and T1 nerve roots, is formed from the medial cord of the brachial plexus. Following an anatomically tortuous course through the upper extremity, the nerve is susceptible to compressive forces at multiple locations along its course. At the elbow, the ulnar nerve travels through the cubital tunnel, which is the most common site for ulnar nerve compression. Neuropathy secondary to cubital tunnel pathology must be ruled out before a diagnosis is made of ulnar nerve compression at the wrist.

The ulnar nerve, primarily a motor and sensory nerve for the hand, does not give off important branches in the proximal upper extremity. On its entrance into the hand, the ulnar nerve provides the primary motor innervation to the intrinsic muscles therein.

The ulnar nerve innervates the abductor and opponens digiti minimi and flexor digiti minimi brevis (the hypothenar muscles); the lumbrical muscles of the fourth and fifth fingers, all of the interossei, the adductor pollicis, the medial head, and, occasionally, the lateral head of the flexor pollicis brevis are also innervated by the ulnar nerve. The ulnar nerve also supplies sensation for a significant part of the hand, which includes the palmaris brevis and the skin over the hypothenar eminence. The superficial branch of the nerve continues and divides into the common palmar digital nerves, which divide distally to form the proper digital nerves to the ulnar side of the fifth finger and the adjacent sides of the fifth and fourth fingers.

Together with the ulnar artery, the ulnar nerve passes into the wrist on the radial side of the pisiform bone via an anatomic space first described in 1861.[6] This space, the loge or canal of Guyon, is a 4-cm-long channel located between the pisiform carpal bone and the hook of the hamate.[7] The anatomy of this small space is complex.

Within the loge of Guyon, the ulnar nerve bifurcates into deep (motor) and superficial (sensory) branches. In 1969, Shea and McClain divided the canal of Guyon into three anatomic zones based on the landmarks of bifurcation.[3] Zone I represents that part of the canal that contains the ulnar nerve prior to its division into its component motor and sensory branches. Zone II is that part of the tunnel encompassing the deep motor branch distal to the bifurcation of the nerve. The sensory branch of the ulnar nerve travels through zone III of the loge of Guyon.[6] These anatomic divisions allow a more precise localization of lesions within the wrist. Lesions at the wrist, whether mass lesions, anatomic abnormalities, traumatic injuries, or ulnar artery disorders, may produce a predictable set of complaints and physical findings, depending on the level of impingement within Guyon's canal.

CLINICAL PRESENTATION

Patients exhibiting ulnar nerve compression at the level of the wrist typically present complaining of wrist pain that radiates into the ulnar two digits.[6] Patients should be asked about a history of previous falls onto an outstretched hand, which may have resulted in an occult hamate fracture. A fracture here may impinge on the nerve as it passes through zone I or zone II (or both) of Guyon's canal. Fractures of the hook of the hamate have been reported to account for 14% of all cases of ulnar nerve compression in the wrist.[3]

Patients should also be asked about repetitive trauma in both vocational and avocational settings. Repeated blows to the ulnar side of the hand and use of pneumatic tools have been implicated as potential causes of ulnar neuropathy at the wrist. Holding the wrist in forced hyperextension during such activities as bicycling may also predispose to this condition.[3] Interestingly, each of these activities has been mentioned as a cause for other painful wrist syndromes, which are discussed elsewhere in this chapter.

Physical examination of a patient complaining of a painful wrist and hand begins at the neck.[5,6] Painful or limited range of motion may suggest the presence of cervical disc disease. This should be followed by axial compression of the cervical spine, which may elicit radiculopathic pain from a compressed nerve root.

The elbow must be examined for evidence of swelling or deformity, inflammation, or masses. The ulnar nerve should be carefully palpated and percussed to assess for the presence of increased irritability. Identification of a focal site of irritation must then be compared with the opposite extremity; nearly one-fourth of normal subjects exhibited a positive Tinel's sign on percussion of the ulnar nerve.[5]

A careful assessment of the wrist and hand can help identify the level and, possibly, the exact location of an ulnar neuropathy.[6] Examination of the wrist and hands has five main components: localization of point tenderness, examination of the vascular supply, provocative testing of the nerve supply, and sensory and motor testing.[3]

Fractures of the hook of the hamate are a frequent cause of ulnar neuropathy at the loge of Guyon. They are the most common reason for symptoms from zones I and II when there is a history of trauma. Eliciting tenderness with careful palpation may indicate an occult new fracture or a poorly healed old fracture.

A determination of the patency of the ulnar artery is the second part of a focused examination of the wrist and hand. An Allen test performed on both hands will help the clinician to ascertain ulnar artery contribution to hand perfusion. The artery should also be palpated for the presence of tenderness, aneurysmal dilatation, and thrills. Arterial thrombosis or aneurysms can cause localized pressure on the ulnar nerve within Guyon's canal. Ulnar artery thrombosis has been reported as the most common cause of isolated sensory symptoms from ulnar nerve compression.[3]

Provocative testing of the ulnar nerve supply to the hand is accomplished by performing both the Tinel and Phalen tests. Percussion of the ulnar nerve at the level of Guyon's canal may elicit paresthesias to the fifth digit and the ulnar half of the fourth digit. Similarly, paresthesias in the distribution just described may result from having the patient allow his or her wrists to fall into complete volar flexion. This is considered a positive Phalen's sign.[1]

The sensory and motor examination of the hand should focus on identification, where possible, of the level of the ulnar nerve lesion. For example, impaired sensation to the ulnar dorsum of the hand (both light touch and two-point discrimination) is suggestive of an ulnar nerve lesion proximal to the wrist. Weakness to the FDP muscles of the fourth and fifth digits is similarly suggestive of more proximal compression.

Nerve compression within zone I of Guyon's canal will usually present with mixed motor and sensory findings.[3,6] Patients will exhibit weakness of the ulnar-innervated muscles in the hand, and will manifest sensory deficits to the palmar surface of the hypothenar eminence and to the ulnar two digits of the hand.

Zone II compression of the ulnar nerve results in motor branch paresis of the ulnar nerve. Wasting of the intrinsic muscles of the hand in the first dorsal interosseous space is evidence of chronic motor branch neuropathy. Further evidence of motor branch weakness can be elicited by asking the patient to extend his or her fingers. A distal ulnar neuropathy will result in a severe claw hand deformity.[6] The long flexor muscles flex the distal interphalangeal and proximal interphalangeal joints of the fourth and fifth digits. The long extensor muscles hyperextend the metacarpophalangeal joints of the same digits. The loss of intrinsic muscle function to the hand, however, results in an inability to maintain extension of the digits at the distal interphalangeal and proximal interphalangeal joints.[1]

Wartenberg's sign, an inability to abduct the fifth finger while holding the digits of the hand in extension, may be present with ulnar motor weakness. This is less likely to be present, however, with an isolated zone II injury, because the motor branch to the abductor digiti minimi exits prior to this portion of the canal. Interosseous muscle function can be evaluated by asking the patient to cross the index and middle fingers. This test is known as the Scott Earle test. Froment's sign is present when the adductor pollicis muscle, innervated by the ulnar nerve, is weak. This sign is elicited by having the patient tightly hold a piece of paper between the thumb and the radial side of the index finger. The examiner then attempts to pull the paper from the patient. Flexion at the thumb interphalangeal joint (indicating weakness of the adductor pollicis muscle) results in a positive Froment sign. Comparison with the patient's other hand should be performed.[1,5]

Injuries or compression of the ulnar nerve in zone III of Guyon's canal involves the superficial sensory branch of the nerve. Of the three zones of the canal, this is the least likely to exhibit pathology.[6]

DIFFERENTIAL DIAGNOSIS

Lesions at any location along the length of the ulnar nerve or the nerve roots from which it arises may be responsible for symptoms of wrist or hand pain. More proximal lesions, such as cervical disc disease affecting the C8 or T1 nerve roots, must be considered. (Evaluation of the cervical spine has already been described). Identification of painful and/or limited cervical motion merits further study to rule in or rule out a lesion at this level as the cause for a patient's symptoms.

Brachial plexus pathology may also be responsible for symptoms in the distribution of the ulnar nerve. Pancoast's tumor, an apical tumor of the lung, can exert pressure on or grow into the brachial plexus, resulting in symptoms distant from the primary site.[5] A careful examination would, therefore, include palpation of the supraclavicular region of the affected extremity for mass lesions. And, if clinical suspicion warrants, a posteroanterior (PA) and lateral radiograph of the chest should be ordered.

Thoracic outlet syndrome may also result in a brachial plexopathy. The diagnosis of this entity may be made by provocative testing. One such maneuver is the so-called hands-up test, in which the patient is asked to make a fist and then abduct the shoulder to 90 degrees and externally rotate the upper extremity. In the presence of thoracic outlet syndrome, this activity can trigger and reproduce the patient's symptoms.[7]

Distal median nerve function and the presence of an associated carpal tunnel syndrome must be excluded when examining a patient with complaints in the distribution of the ulnar nerve.

Whereas the most common cause for ulnar nerve compression lies at the cubital tunnel of the elbow, within the wrist itself it is the presence of a ganglion that accounts for the majority of atraumatic ulnar neuropathy.[4,6] Bednar[3] reported that, in the absence of trauma, 86% of patients presenting with a combined motor and sensory finding had a ganglion compressing the nerve. Anomalous muscle bellies and other anatomic variants, including lipomas, giant cell tumors, schwannomas, thickened ligaments, bipartite hamuli, and pisiform-hamate coalitions constitute another large class of entities responsible for ulnar nerve compression at the wrist.[3] It is unlikely, however, that a precise diagnosis of one of the aforementioned causes would be made in the emergency department.

Hamate fractures, ulnar artery thrombosis, repetitive trauma neuropathies (hypothenar hammer syndrome), and rheumatoid tenosynovitis are other potential causes of ulnar nerve compression. The presence of other joints affected by rheumatoid arthritis may help identify this disease as a potential cause of ulnar nerve compression at the wrist.[3]

EMERGENCY DEPARTMENT EVALUATION

The diagnosis of ulnar nerve compression at the wrist is usually made with a history and physical examination. Radiographs of

the wrist may prove valuable for identifying the presence of a hamate fracture or malunion. A history of trauma, recent or remote, coupled with bony point tenderness should raise the clinician's index of suspicion. Special views of the wrist or tomograms may provide additional confirmatory evidence if initial plain films do not reveal a fracture.

Complaints referable to the elbow or to the cervical spine also merit obtaining radiographs of each of these structures. Similarly a PA and lateral chest radiograph should be obtained if one is at all concerned about the possibility of an intrathoracic mass lesion causing a brachial plexus neuropathy. It is unlikely that additional laboratory studies, in the absence of supporting clinical signs or symptoms, will provide useful information in the diagnosis of this entity.

EMERGENCY DEPARTMENT MANAGEMENT

The treatment of ulnar nerve compression at the wrist may be initiated in the emergency department. As with other compressive neuropathy syndromes, in the absence of an identifiable cause, a trial period of 1 to 3 months of nonoperative intervention is recommended.[3] The application of a wrist splint, avoidance of activities that aggravate symptoms, and use of nonsteroidal antiinflammatories have been advocated.[6] Depending on the underlying cause of the neuropathy, symptoms may or may not resolve during this time. Referral for an outpatient workup by a hand surgeon is also the responsibility of the emergency department physician, and should be arranged prior to the patient's departure. For those patients who present with an identifiable cause for their symptoms, a more urgent follow-up is indicated.

DISPOSITION

Outpatient workup of ulnar nerve compression falls under the purview of the hand surgeon, who may obtain electromyographic and nerve conduction studies.[6] These studies are valuable in establishing a diagnosis. Inasmuch as the majority of causes of ulnar nerve compression neuropathy at the wrist are due to anatomic pathology, treatment for this condition is usually surgical.[2]

COMMON PITFALLS

- Perform a thorough physical examination on both upper extremities, as some abnormal findings may be physiologic and not pathologic. Comparing one side to the other is also a good technique to identify subtle pathology.
- Examine the entire course of the ulnar nerve from its origin at the nerve roots of the cervical spine to its distal end point in the digits of the hand. Ulnar neuropathy may be due to compressive forces at one or more locations along the length of the nerve.
- Consider anomalous innervations of the hand and perform a thorough evaluation of both ulnar and median nerve function to identify potential pathology.
- Remember that the most common cause of compressive ulnar neuropathy at the wrist is atraumatic anatomic pathology, which is probably most amenable to surgical repair. The most common traumatic cause of ulnar nerve compression at the wrist is fractures of the hook of the hamate.
- Examination of the painful wrist and hand involves five main components: localization of point tenderness, examination of the vascular supply, provocative testing of the nerve supply, and sensory and motor testing.

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THE HYPOTHENAR HAMMER SYNDROME

In 1934, a classic report provided the basis for the definition of the hypothenar hammer syndrome (HHS): evidence of distal ulnar artery injury following a history of trauma to the palm.[3] The distal ulnar artery lies most exposed to potential trauma at the base of the hypothenar eminence, just distal to the volar carpal ligament. At this location, the artery is no longer protected within Guyon's canal and is covered only by skin, subcutaneous fat, and (in 85% of the population) the palmaris brevis muscle.[3] Given its susceptible position, the ulnar artery is easily compressed between hard objects and the hook of the hamate.[4,5] This can occur any time that the hypothenar aspect of the base of the palm is subjected to trauma.

HHS is most commonly seen in those individuals who use the palms of their hands to push, pound, or hammer as part of their daily work activities.[4,6] Metal workers, automobile mechanics, lathe operators, and machinists are all at risk. The prevalence of HHS among carpenters has been reported to be as high as 14%.[5] This syndrome has also been found in workers who regularly use high-speed hand-held vibratory tools,[4] such as air hammers, drills, chain saws, sanders, and impact wrenches. HHS is not, however, limited specifically to this population.

Injuries to the distal ulnar artery, not unlike those caused by industrial occupational trauma, have also been seen in athletes. Baseball catchers, racquet handlers (tennis, badminton, etc.), and martial artists have all been reported as having an increased risk for developing HHS.[3] The increasing popularity of mountain biking has led to the discovery that these athletes are also prone to developing injuries of the ulnar artery. Table 54.1 lists a variety of sports that have also been implicated as potential causes HHS.

Repetitive ulnar artery injury, whether from work or sport, can result in traumatic vasospasm, thrombosis, or aneurysm formation.[4–6] Various combinations of these lesions are also possible and can complicate both diagnosis and management. Prolonged distal ulnar or digital arterial vasospasm can occlude affected blood vessels. If the source of irritation is eliminated, however, vasospasm and occlusion are reversible phenomena and a normal arterial supply may be restored.[4]

More severe trauma to the ulnar artery can damage the internal elastic lamella, leading to formation of intraarterial thrombi.[3] Embolization of traumatized intima and thrombus cause digital ischemia by occluding the end arterial supply to the fingers.[4,6] Ischemia may also occur as a result of reflex vasospasm. Thrombosis involving the adventitia may affect periarterial sympathetic fibers, resulting in vasospasm that limits surrounding collateral circulation.[6]

TABLE 54.1. Sports Reported to Cause HHS[6]

Baseball	Softball
Badminton	Karate
Handball	Weight lifting
Football	Hockey
Frisbee	Mountain biking

True palmar aneurysms may develop if the media of the arterial wall is damaged primarily.[4]

CLINICAL PRESENTATION

HHS is a treatable cause of digital ischemia that should be considered in the differential diagnosis of patients presenting with complaints of symptomatic digital ischemia.[5] As described earlier, the patient is classically a male laborer with a history of blunt trauma to his dominant hand.[4,5] Clinical findings in HHS include a male predominance, unilateral hand involvement, and acute onset of pain, followed by the more gradual development of a severe Raynaud's phenomenon (i.e., increased sensitivity of the digits to exposure to cold).[3] In a patient exhibiting Raynaud's phenomenon, coldness in the fingers of the dominant hand without a history of triphasic color changes and a lack of thumb involvement is highly suggestive of a diagnosis of HHS.[5]

The most typical occupational history of a patient with HHS includes frequent, repetitive, but comparatively minor hypothenar trauma. The daily trauma may be so much a part of the patient's regular activities that it goes unrecognized or is otherwise ignored. Careful inquiry about work and recreation is mandatory.[5]

At presentation, a patient may complain of one or more episodes of acute, lancinating pain over the hypothenar eminence that occur after striking a blow with the palm of the hand.[4] This pain typically radiates into the fourth and fifth fingers, and a dull, aching hypothenar pain may then ensue. Other patients describe an immediate pain episode that resolves completely over time. Then, days to weeks to months afterward, ischemic finger symptoms develop as a consequence of a slowly progressing thrombosis of the ulnar artery.[6]

Physical findings in HHS are largely localized to the hypothenar eminence and the digits of the affected hand. Prominent calluses over the hypothenar eminence provide a clue about recurrent trauma to this area.[5] Moreover, tenderness to palpation over the hypothenar area may indicate the presence of an ulnar artery aneurysm. Further examination may reveal the fingers or fingertips to be cold, pale, mottled, atrophic, or ulcerated.[4,5] The Allen test should be performed as part of the physical examination. A positive test suggests that either the superficial volar arch is incompletely developed or, as in the case of HHS, that the distal ulnar artery is occluded or stenotic.[4]

DIFFERENTIAL DIAGNOSIS

Many conditions are known to cause ischemia of the upper extremity. Collagen vascular diseases and various vasculitides and coagulopathies may each result in poor perfusion of the hand and fingers.[4] These disorders, however, tend to be systemic and have more generalized findings than does HHS.

Buerger disease (thromboangiitis obliterans), like HHS, affects primarily young males. Raynaud's phenomenon and hand or digital pain are historic features shared by these two conditions. The upper extremity examination of a patient with Buerger disease may be indistinguishable from someone with HHS, including an absent or diminished ulnar pulse.

Two elements may be helpful in differentiating one condition from the other. Buerger disease is found almost exclusively in cigarette smokers. Conversely, tobacco use is not a prerequisite for the development of HHS. Buerger disease is often a systemic condition involving not only the upper extremities, but also the lower ones. HHS is confined to the upper extremity only and is most often unilateral in its presentation. In the absence of clarifying historic or physical features, referral for arteriography or, potentially, arterial biopsy may be required to confirm or rule out the diagnosis of Buerger disease.

High-speed hand-held vibratory tools are known to cause both HHS and the hand–arm vibration syndrome (HAVS). Whereas both of these conditions can result in Raynaud's phenomenon of the digits of the dominant hand, the underlying pathology is different. HHS is caused by ulnar artery injury and may be differentiated from HAVS by a careful history and vascular examination at the patient's wrist.[5]

EMERGENCY DEPARTMENT EVALUATION

The emergency physician must not forget to obtain both a vocational and an avocational history from a patient presenting with complaints of hand and wrist pain. While the history will often lead the clinician to the proper diagnosis, a thoughtful and deliberate physical examination is also necessary. Performing and documenting the results of the Allen test is an important part of the physical examination. The modified Allen test is performed with the patient in a seated position. The hands, palms facing up, should be resting on the knees. The clinician then compresses the radial artery with one thumb and the ulnar artery with the other thumb, thereby externally occluding the arterial supply to the hand. The patient next rapidly clenches and opens the first several times to exsanguinate the palm. If there is pathology in the ulnar artery, the palm will remain pallid after the clinician releases his or her thumb from that vessel. Blood supply will return to the palm only after the pressure on the radial artery is removed. A pencil Doppler instrument may be used in conjunction with the Allen test to confirm the patency of the arterial blood supply to the hand.[1,5] This examination should be performed at both wrists.

EMERGENCY DEPARTMENT MANAGEMENT

Most patients with HHS will respond to proper therapy when promptly initiated. The emergency physician should therefore begin treatment after making the diagnosis of this condition. At a minimum, local wound care of necrotic or ulcerated digits can take place in the emergency department. Additional conservative measures that may be offered include counseling on the avoidance of cold environments and cessation of smoking,[5] because both will exacerbate HHS. Patients should be encouraged to wear mittens or gloves to help keep their hands and fingers warm and protected.

Aspirin and a calcium channel blocker (diltiazem or nifedipine) may be prescribed and are considered appropriate initial therapeutic agents.[3] With proper therapeutic intervention, the prognosis for HHS is good, and surgery is generally not indicated. Patients need to understand, however, that response to therapy is predicated on the elimination of the underlying trauma source. In some instances, this may require a change of occupation.[5]

DISPOSITION

Referral to a hand surgeon for angiographic studies and ongoing management is proper and necessary. Arteriography can reveal ulnar artery irregularity or aneurysms. Occlusion of the ulnar artery segment overlying the hook of the hamate is considered pathognomonic of HHS.[5] Multiple digital artery occlusions, secondary to embolization, may also be seen.

Conservative, nonsurgical modalities are favored for the treatment of HHS, except in the case of ulnar artery aneurysm. In this instance, resection of the aneurysm is performed in order to avoid or prevent further digital embolization.[3] Surgical repair is still the preferred method of treatment for this condition.[7]

COMMON PITFALLS

- Manual laborers presenting with pain to the dominant hand need a careful historic review of their daily occupational activities in order to establish a history of regular hypothenar trauma. Identification of calluses over the hypothenar eminence can provide a clue to this history.
- Recall that some athletes and even people who use canes or walkers are susceptible to ulnar artery trauma.
- Buerger disease is an important part of the differential diagnosis of HHS and must be ruled out. Angiography or arterial biopsy may be required to make the diagnosis. An important clinical clue is that Buerger disease tends to be a systemic condition and will involve both upper extremities.
- An occluded ulnar arterial supply to the hand (given the proper clinical history) is very highly suggestive of the diagnosis of HHS.
- HHS is a treatable condition, provided that the source of trauma is eliminated. The emergency department physician must introduce and then reinforce this concept once the diagnosis of HHS is made.

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DE QUERVAIN'S TENOSYNOVITIS/ DE QUERVAIN SYNDROME

De Quervain's tenosynovitis, a not infrequent cause of wrist pain, is the most common tendinitis of the extensor tendons of the hand and wrist.[2] De Quervain syndrome is a nonsuppurative tenosynovitis of the first dorsal compartment of the wrist.[6–9] The first dorsal compartment, like the other dorsal compartments of the wrist, is a "tunnel" through which the tendons of the hand's extrinsic extensor muscles pass.

The first dorsal compartment of the wrist overlies the styloid process of the radius. This compartment contains the tendons of the abductor pollicis longus (APL) and the extensor pollicis brevis (EPB).[7] The APL tendon inserts at the base of the dorsum of the thumb's metacarpal, and the EPB tendon inserts at the base of the thumb's proximal phalanx. At the level of the wrist, these tendons and their synovial sheaths lie sandwiched between the carpal bones and a fibrous band called the dorsal (extensor) retinaculum.

Typically the extrinsic tendons of the wrist glide easily through the fibroosseous canals defined by the carpal bones on one side and the extensor retinaculum on the other. The APL and EPB work in tandem to hold the thumb in abduction.[7] De Quervain syndrome represents a derangement in the normal gliding of the APL and/or the EPB tendons and, therefore, an impairment in thumb abduction.

Several hypotheses exist as to the underlying cause of de Quervain's tenosynovitis. Authors suggest that de Quervain syndrome is due to a myxoid degeneration of the tendon sheaths of the first dorsal compartment.[3]

TABLE 54.2. Vocational and Avocational Causes of de Quervain's Tenosynovitis[7]

Working at a typewriter or adding machine
Excessive writing
Washing or wringing clothes
Chopping wood
Cutting cloth with heavy scissors
Sewing, knitting, weaving
Working on a grinder or buffing machine
Fitting rubber rings over a pipe
Operating machine keyboards, sewing machines, lathes, drills, presses, grinders, and switchboards
Prolonged piano playing
Playing a reed instrument and supporting its weight on the right thumb

Chronic inflammation resulting from overuse is another postulated cause of this syndrome. According to this theory, overuse of sheathed tendons leads to a failure of inherent lubrication mechanisms.[4] Over time, friction between tendons and their synovial sheaths causes inflammatory changes in these structures. The tendon hypertrophies and the intrinsic diameter of the sheath narrows in response to inflammation. This further impedes the smooth gliding of the tendon through its synovial sheath.

A third theory explaining the pathophysiology of de Quervain's tenosynovitis identifies the condition to be the result of thickening of the extensor retinaculum overlying the first dorsal compartment.[7]

Despite various pathophysiologic theories, nearly all authors agree as to the etiology of de Quervain syndrome. Persistent repetition of specific tasks beyond the point of fatigue is commonly implicated.[7] These tasks frequently involve repeated pronation and supination of the forearm combined with forceful exertion of the thumb.

Other reported activities that can lead to a tenosynovitis of the first dorsal compartment include performing tasks that may be new or unfamiliar to the patient. Many patients begin to have symptoms within days of starting a new job or resuming an old one after a holiday.[5,7] Table 54.2 lists jobs and activities previously reported to result in de Quervain's syndrome. Minor blunt trauma and rheumatoid arthritis have also been identified as possible causes of de Quervain syndrome.

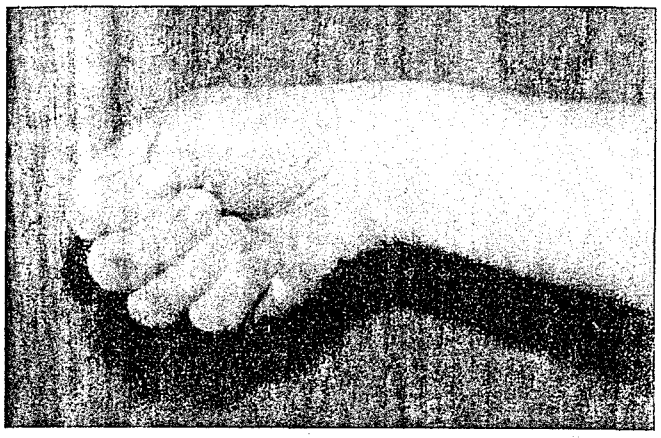
CLINICAL PRESENTATION

Pain at the radial styloid is a classic and defining symptom of de Quervain's tenosynovitis. Although the pain is more or less constant, it is exacerbated by thumb abduction, ulnar deviation of the wrist, and simple grasping. Holding the thumb at rest, in contrast, may offer only minimal relief. Previous trials of nonsteroidal antiinflammatory drugs or acetaminophen use tend to provide little, if any, symptomatic relief.

The pain, usually described by patients as severe in quality, has also been reported as a "stiffness."[7] In more advanced cases, patients may also note swelling and crepitus at the site of maximal pain. Pain can radiate proximally into the forearm or distally to the thumb. Occasionally, the pain may be so intense that sleep is disturbed. Other patients may complain of weakness or an inability to use the affected hand due to debilitating pain.

Another hallmark of de Quervain syndrome is a history of gradual onset of pain in the absence of fracture or acute trauma.[7] De Quervain syndrome typically affects women more than men (approximately 10 to 1) between the ages of 35 and 55.

Examination of the patient with de Quervain's tenosynovitis begins with a careful inspection of the forearm and wrist. Mild



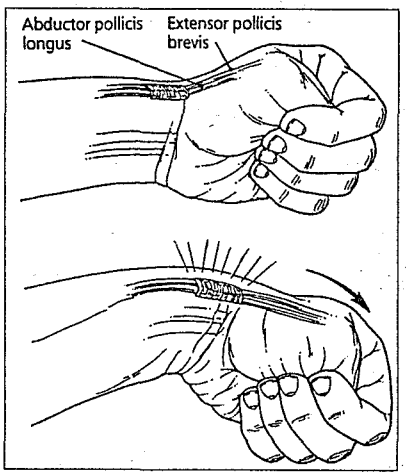


Figure 54.2. Finkelstein's test.

cases may exhibit only subtle, localized swelling at the radial styloid.[6,7] A more extensive fusiform swelling and, possibly, erythema can characterize more severe cases.

Palpation of the first dorsal compartment should be approached gently. Tenderness or crepitus at the radial styloid is highly suggestive of de Quervain syndrome.[8] A positive Finkelstein test, however, is diagnostic of this condition.[7] The Finkelstein test requires that the patient flex the thumb into the palm and then grasp it with the other fingers of the hand. The patient then actively ulnarly deviates the wrist[1] (Fig. 54.2). Severe, sharp pain (a positive test) is pathognomonic of tenosynovitis of the first dorsal compartment.[2,6] It is important to distinguish the sharp pain of de Quervain syndrome from a "pulling sensation" in the same anatomic distribution.[4] The latter is more indicative of a diagnosis of radial nerve entrapment, not tenosynovitis.

DIFFERENTIAL DIAGNOSIS

The superficial branch of the radial nerve crosses the APL and the EPB muscles in the distal forearm and then divides into terminal branches before entering the hand. The anterior terminal branch of the radial nerve, which provides sensation to the dorsum of the thumb, passes into the hand almost directly over the extensor retinaculum of the first dorsal compartment.[7] Radial nerve entrapment, therefore, should be ruled out of the differential diagnosis using the previously described technique.

Arthritis at the carpal–metacarpal (CMC) joint of the thumb is another differential diagnosis that may be excluded by the emergency department physician.[6] The pain in CMC arthritis is more accurately localized to the basal joint of the thumb, not to the first dorsal compartment. Moreover, the clinician can elicit or aggravate CMC arthritis pain by grinding the first metacarpal on the trapezium.[2] This procedure does not stretch the APL or EPB tendons and will therefore not cause pain in a patient with de Quervain syndrome. The clinician must also be alert to rule out fractures of the radial styloid, scaphoid, or trapezium.[6] A clinical history of trauma and plain film radiographs should suffice to include or exclude fractures from the diagnosis.

Intersection syndrome, another tenosynovitis of the radial extensor tendons, may be confused with de Quervain syndrome. Like de Quervain's tenosynovitis, intersection syndrome is a nonsuppurative process that develops after repeatedly performing a novel activity.[6] Patients will present complaining of radial wrist pain.

Examination of patients with intersection syndrome generally reveals swelling, pain, and crepitus in a more proximal and ulnar distribution than is seen in de Quervain syndrome.[6,9] Tenderness is localized not to the area of the radial styloid, but to the region where the radial wrist extensors of the second dorsal compartment and the tendons of the first dorsal compartment intersect.[5]

EMERGENCY DEPARTMENT EVALUATION

Diagnosis of de Quervain syndrome rests on obtaining a history of overuse of the thumb and eliciting a positive Finkelstein test. The medical literature reports no objective confirmatory electrodiagnostic tests or studies. Plain film radiographs in the absence of fracture or arthritis are usually nondiagnostic.[7]

EMERGENCY DEPARTMENT MANAGEMENT

Several methods exist for treating de Quervain's tenosynovitis. The least invasive techniques (rest, application of heat, and immobilization with a thumb spica splint) are, by themselves, ineffective for treating this condition.[6] They may be used, however, as adjunctive therapies to the mainstays of treatment: steroid injection and, when necessary, surgical release. Steroid injection into the tendon sheaths of the first dorsal compartment is an effective treatment for de Quervain syndrome that may be employed by the emergency department physician.

Between 70% and 80% of patients with de Quervain syndrome will respond to steroid injection.[5,9] One recent article cited a success rate of 84% after either one or two steroid injections into the first dorsal compartment.[8] Steroids may even be used alone without subsequent splinting or immobilization. Several studies have shown no advantage to using immobilization as a therapeutic modality after (or in the absence of) steroid injection.[7]

A corticosteroid preparation such as methylprednisolone acetate is a good choice for injection into the tendon sheath, as it tends to provide long-lasting relief. Approximately 2 cc of steroid should be drawn up and mixed in a syringe with 0.5 cc of lidocaine.[2] Using a 25- or 27-gauge needle helps minimize the pain of injection. Although techniques vary according to author, most agree that the needle should be passed through the skin at the distal end of the first dorsal compartment. One source suggests inserting the needle through the tendon sheath and tendon until it touches the radius.[6] As the needle is slowly withdrawn and as it exits the tendon, resistance will fall and the steroid–lidocaine mixture will easily be injected into the tendon sheath (Fig. 54.3). Forced injection of steroids into a tendon is not desirable.

A successful injection of the steroid–lidocaine mixture can be identified by the clinician feeling the medication track distally along the tendons. Immediate relief of pain (due to the presence of the lidocaine in the injected mixture) is also a good marker for accuracy of placement.[8] Patients need to be advised that it may take several days for the steroid to begin relieving their pain and

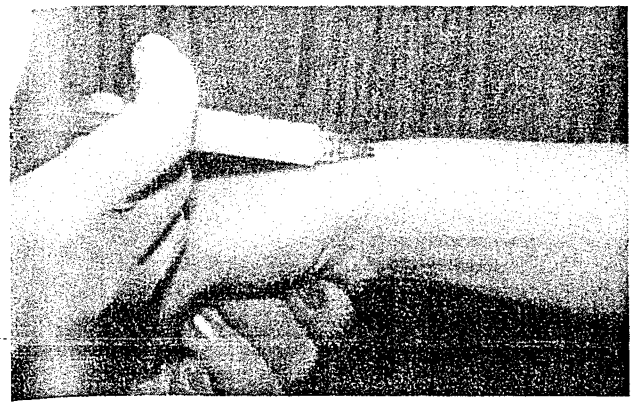


Figure 54.3. Corticosteroid injection into the tendon sheath.

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that, in the meantime, there is the possibility of a postinjection flare reaction. This painful phenomenon, sometimes seen after injection of steroids, is generally self-limited and usually resolves after a couple of days.

Failure of steroid injection to achieve a cure does occur in a small but significant percentage of those with de Quervain syndrome. These failures are believed to be due to the presence of a separate EPB compartment that is smaller and deeper than the APL compartment. If a patient has multiple sheaths within the first dorsal compartment but only one of them is injected, steroids are not likely to be effective in treating de Quervain syndrome.[6] Patients must be counseled about this possibility and advised to seek appropriate follow-up care with a hand surgeon if adequate relief is not achieved after one or two steroid injections. As a curative modality, surgery can also fail if the tendon sheath of the EPB compartment is not identified and released.

DISPOSITION

A referral to a hand surgeon is entirely appropriate when one considers that 20% to 30% of steroid injection attempts will not alleviate the pain of this condition.[6] Failure to achieve a cure after two or three injections over a 3- to 5-week period indicates the necessity for surgically releasing the stenotic tendon sheath or sheaths.[2] In general, surgically dividing the sheath(s) within the first dorsal compartment is a curative procedure.

Because multiple sheaths may lie within the compartment, the surgeon must carefully inspect this area to be certain that all sheaths have been released.

COMMON PITFALLS

- A positive Finkelstein test is virtually diagnostic for de Quervain's tenosynovitis.
- Fractures of the radial styloid and arthritis at the CMC joint of the thumb are two important differential diagnoses to be excluded. Radiographs and careful physical examination can help to rule out these possibilities.
- Radiographs in de Quervain syndrome are most likely to be normal or possibly reveal some soft-tissue swelling.
- Long-acting steroid combined with lidocaine is often curative and can be used in the emergency department to treat this condition. Patients should be warned about the possibility of postinjection flare reaction and the possibility of failure of this treatment.

CHAPTER 55
Hand Infections

Carl R. Chudnofsky

ESSENTIAL ANATOMY

The nail plate inserts into a depression on the dorsum of the finger called the proximal nail fold, which divides the proximal nail fold into superficial and deep epithelial surfaces called the dorsal roof and ventral floor, respectively. The eponychium, commonly called the cuticle, is a horny layer extension of the proximal nail fold that extends a short distance onto the dorsum of the nail. It seals the potential space between the nail plate and the proximal nail fold. Laterally, the proximal nail fold is continuous on either side with the lateral nail folds or paronychium. The soft tissue under the distal free edge of the nail is called the hyponychium (Fig. 55.1).[1]

The distal pulp space of a finger or thumb is composed of fat globules that are divided into small fascial compartments by 15 to 20 vertical fibrous septa (see Fig. 55.1). The presence of these compartments has a substantial impact on the pathophysiology and management of pulp space infections.

The flexor tendons are surrounded by a tendon sheath that is composed of an outer fibrous sheath and an inner synovial sheath. The inner synovial sheath has a visceral and parietal layer with a potential space between them. The tendon sheaths of the index, middle, and ring fingers extend from the midpalmar crease to just beyond the distal interphalangeal joints. The tendon sheath of the flexor pollicis longus tendon begins at the base of the distal phalanx of the thumb and extends to the pronator quadratus muscle in the wrist. Between the base of the thumb metacarpal and the wrist, the tendon sheath of the flexor pollicis longus tendon is called the radial bursa. The tendon sheath surrounding the flexor tendons of the little finger begins just distal to the distal interphalangeal joint and, like the tendon sheath of the flexor pollicis longus tendon, ends at the pronator quadratus muscle in the wrist. The ulnar bursa is that part of the tendon sheath between the fifth metacarpal and the pronator quadratus muscle (see Fig. 55.1). The radial and ulnar bursas communicate with each other in 50% to 80% of patients.[7,19]

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