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Ulnar tunnel syndrome.

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KEY POINTS
A zone I compression elicits motor and sensory signs and symptoms, a zone II compression results in isolated motor deficits, and a zone III compression causes purely sensory deficits.
In select cases, conservative treatment such as activity modification may be helpful, but often, surgical exploration of the ulnar tunnel with subsequent ulnar nerve decompression is indicated.
The anatomy of the ulnar tunnel is complex, but numerous anatomic studies have described the tunnel in significant detail. Because organic lesions are often implicated in the cause, surgical exploration and decompression of the ulnar tunnel represent a common treatment modality.

INTRODUCTION

Ulnar tunnel syndrome (UTS) is broadly defined as a compressive neuropathy of the ulnar nerve at the level of the wrist. The term "ulnar tunnel syndrome" was coined by DuPont and colleagues[1] in 1965 to describe the condition of 4 patients with acquired ulnar neuritis. The ulnar tunnel proper, also known as "Guyon's canal," is one potential but not exclusive site of ulnar nerve compression at the wrist. The eponym comes from Guyon's description in 1861 of a space at the base of the hypothenar eminence at which the ulnar nerve bifurcates and that is vulnerable to compression from surrounding structures.[2,3] Numerous factors may precipitate the onset of UTS, including space-occupying lesions, vascular lesions, and repetitive trauma. Patient presentation depends on the anatomic zone of ulnar nerve compression and therefore may be purely motor, purely sensory, or a combination of both. In select cases, conservative treatment such as activity modification may be helpful, but often, surgical exploration of the ulnar tunnel with subsequent ulnar nerve decompression is indicated.

ANATOMY

As with all nerve-related disease, a thorough understanding of anatomy and potential sites of compression is critical. The ulnar nerve emerges from the medial cord (C8–T1) of the brachial plexus and passes through the axilla into the anterior compartment of the arm, before piercing the intermuscular septum and traveling in the posterior compartment medially. It then courses superficially and passes posterior to the medial epicondyle, into the anatomic cubital tunnel. The nerve then continues deep along the flexor digitorum profundus in the forearm. Before its

entrance to the ulnar tunnel, approximately 3.4 cm proximal to the ulnar styloid, the ulnar nerve gives off the dorsal cutaneous branch, which innervates the ulnar and dorsal side of the hand.[4] The main nerve resurfaces at the level of the wrist where it passes through the ulnar tunnel, which is a fibro-osseous structure. The anatomy of the tunnel is complex, and variations in the nomenclature and structures surrounding the tunnel have previously been a source of confusion and misinterpretation.[5,6] For example, the terms pisohamate tunnel,[5,6] pisohamate hiatus,[6–8] and pisohamate arcade[6] have been used variably and interchangeably to describe the ulnar tunnel in part or whole. The entrance of the tunnel is triangular and begins at the proximal edge of the palmar carpal ligament. It extends distally to the fibrous arch of the hypothenar muscles at the level of the hook of the hamate and is approximately 40 to 45 mm in length. The boundaries of the tunnel vary along its length.[3,6] Generally, the roof of the canal consists of the palmar carpal ligament, palmaris brevis, and hypothenar connective tissue. The floor of the canal consists of the transverse carpal ligament, pisohamate ligament, pisometacarpal ligament, and the tendons of the flexor digitorum profundus and opponens digiti minimi. The medial wall of the canal is formed by the pisiform, the abductor digiti minimi, and the tendon of the flexor carpi ulnaris. The lateral wall is formed by the hook of the hamate, the transverse carpal ligament, and the flexor tendons.[3,9,10]

Within the boundaries of the canal lie the ulnar nerve, ulnar artery, accompanying veins, and connective fatty tissue.[6] The ulnar nerve lies slightly deep and ulnar to the ulnar artery. During its course in Guyon's canal, the ulnar nerve bifurcates into a superficial and a deep branch approximately 6 mm distal to the distal pole of the pisiform.[6] The superficial branch innervates the palmaris brevis and provides the sensory fibers over the hypothenar eminence and small and ring fingers. The motor branch of the nerve exits deep in the canal and courses around the ulnar edge of the hamulus and then runs radially between the abductor digiti minimi and flexor digit minimi and dorsal to the flexor tendons of the small finger (Fig. 1).[6,11]

Compression of the ulnar nerve at the wrist is not limited to the confines of the ulnar tunnel. Shea and McClain and, later, Gross and Gelberman studied the relationship between the symptoms of ulnar neuropathy and the anatomic location of nerve compression about the wrist.[3,11] They classified compressive ulnar neuropathy at the wrist into 3 types (Fig. 2). Type I syndrome,[11] or a zone I[3] compression, occurs as a result of nerve compression proximal to or within Guyon's canal, before any nerve bifurcation, and manifests as motor weakness of all the ulnar innervated intrinsic muscles and sensory deficits over the hypothenar eminence and the small and ring digits. Type II syndrome, or a zone II compression, manifests exclusively as motor weakness of the hand. The sensory branch is spared and therefore sensation along the ulnar nerve distribution remains intact. Compression of the deep ulnar branch may occur as it exits Guyon's canal at the level of the hamulus. Type III syndrome, or a zone III compression, occurs secondary to compression of the superficial sensory branch of the ulnar nerve and manifests as isolated sensory loss.

COMMON PATHWAY OF COMPRESSIVE PERIPHERAL NEUROPATHIES

The details of nerve degeneration and regeneration as a result of compression loading have been studied extensively over the past few decades and have yielded a tremendous amount of information regarding the pathophysiology of nerve compression.[12–19] In general, these studies have demonstrated that nerve injury correlates to both the degree and duration of compression, with both mechanical and ischemic factors contributing to neurologic dysfunction.[20] Situations such as trauma or sustained compression will induce the accumulation of edema into the endoneurial space of the nerve trunk.[21] Because of the diffusion barrier created by the perineurium and the lack of lymphatic vessels in the endoneurial space, the fluid may not easily escape. The result is an increase in endoneurial fluid pressure and encroachment of the normal endoneurial microcirculation. A study by Lundborg and colleagues showed that after 2 to 8 hours of experimental compression induced ischemia (80 mm Hg) in nerves of animals, the endoneurial fluid pressure may increase rapidly and persist for 24 hours or longer.[22] Another example of metabolic conduction block is the sensory loss and motor paralysis that can occur after deflating the tourniquet around the upper arm. This type of metabolic block, caused by local arrest of intraneural microcirculation, is immediately reversible when the compression is removed. With extended compression, however, edema within the fascicles can result in increased endoneurial

pressure, which could compromise endoneurial capillary flow for hours or days, potentially permanently affecting function of the nerve.[23]

CAUSES

No reports have specifically addressed the incidence and prevalence of UTS in the widespread population. It is generally accepted that the incidence of UTS is much less than that of either carpal tunnel syndrome (CTS) or cubital tunnel syndrome. Numerous factors may cause UTS, and in fact, a large proportion of the literature on UTS is dedicated to case reports that describe the various causes of the disease.[24–38] Shea and colleagues[11] reported that the mass effect of ganglion cysts and then occupational neuritis were the 2 leading causes of UTS. During the past several decades, however, reports on isolated ulnar neuropathy secondary to occupational activities have been scarce.[39] Other causes include benign lesions, hook of hamate fractures, ulnar artery pathologic conditions or aberrancy, deviant hypothenar muscles, and crystal deposition disease. Chronic, repetitive trauma or compression over the hypothenar eminence has also been implicated as a cause of UTS[40] and is not uncommon in long-distance cyclists.[41,42] Idiopathic disease has also been reported.[37] Several studies have reported a strong association between the presentation of CTS and UTS,[1,43–46] whereas others have disputed this relationship.[47,48] The carpal and ulnar tunnels lie adjacent to each other. Although the transverse carpal ligament constitutes the roof of the carpal tunnel, it also constitutes the floor of the ulnar tunnel. Pressure changes within the carpal tunnel are transmitted to the ulnar tunnel, and vice versa. A relevant clinical correlate demonstrates this fact: to completely decompress the motor branch of the ulnar nerve, the transverse carpal ligament needs to be sectioned distally beyond the hook of the hamate, as the motor branch runs on the floor of the carpal tunnel. Silver and colleagues[46] reported a series of 59 hands with CTS and found concurrent ulnar sensory deficiencies in 34% of cases. After carpal tunnel release only, they found that 94% of their patients had improvements in ulnar nerve sensation according to the Semmes-Weinstein test. Ablove and colleagues[45] measured the pressure changes in the carpal tunnel and the ulnar tunnel before and after endoscopic and open carpal tunnel release. Following the release, they found that pressure dropped significantly in both anatomic tunnels. They also suggested that carpal tunnel release alone could be used to successfully treat patients with concurrent disease. The association between CTS and UTS has been debated, but the trends in evidence seem to favor a true association between the two syndromes.

In sum, with regard to the cause of UTS, it seems that most cases are secondary to impingement of an organic lesion on a segment of the ulnar nerve. This contrasts with ulnar neuropathy at the elbow (cubital tunnel syndrome) or CTS, in which the most common causes are believed to be idiopathic.

HISTORY AND PHYSICAL EXAMINATION

A complete patient history includes information on cervical and any other peripheral joint pain (particularly elbow pain or trauma), the duration and progress of symptoms, aggravating and relieving scenarios, and the common occupational or leisure activities. Patients with a history of club or racquet playing have been reported to suffer hamate fractures during instrument use,[49,50] whereas long-distance cyclers may acquire ulnar nerve compression as a result of prolonged grasping of the bicycle's handlebars and direct pressure on the wrist.[41,42] Manual labor that requires constant hammering or repetitive traumatic activities may damage the ulnar artery and perhaps the ulnar nerve as well, a condition often referred to as "hypothenar hammer syndrome."[40]

Ideally, the physical examination should rule out more proximal sites of nerve entrapment, and if possible, the examiner should be able to localize the zone of compression within the wrist. As a result, a thorough examination of the cervical spine and elbow is recommended. CTS should also be ruled out because of the likelihood of concurrent presentation. Careful observation for any gross masses over the dorsal or volar wrist is an important initial step. Hypothenar or interossei wasting, clawing, or the inability to cross fingers may be observed in cases with motor branch involvement (Fig. 3).[1,28,38,51] Palpation may help in identifying the type and location of a lesion. Point tenderness over the hook of the hamate would place a hamulus fracture high in the differential diagnosis, whereas the hardness and consistency of any overt lesion may provide clues about the nature of the lesion. Vascular examination of the wrist is useful because UTS associated with

ulnar artery pathology has been reported frequently.[26–28,34,37] Doppler examination for bruits or thrills over the ulnar artery may indicate pseudoaneurysmal dilation, and the Allen test may be useful to determine patency of the ulnar artery. Provocative tests such as the Tinel or Phalen test are often performed as part of the examination, but their usefulness has not been fully established. In 31 patients with type 1 disease (compression of both motor and sensory components of the ulnar nerve), Grundberg reported that 92% of his patients had a positive Phalen test, whereas the Tinel sign elicited paresthesias in only 44% of patients.[52] Objective tests that examine sensation, such as the Semmes-Weinstein monofilament test and 2-point static discrimination, can provide useful information about the location and magnitude of the disease and can provide baseline information. Similarly, comparing side-to-side grip strength and pinch strength can provide useful objective data to determine the initial status and gauge the progress of the intrinsic hand muscles.

DIAGNOSTIC TESTS

Imaging studies are useful for confirming a suspected diagnosis or when the cause is not entirely clear. If a hamulus fracture is suspected, a carpal view or hamate hook view radiograph or a computed tomography scan can provide useful information. Magnetic resonance imaging is frequently used and is a suitable tool for localizing and diagnosing soft tissue masses, aberrant muscle, and vascular lesions, and it provides useful information for preoperative planning.[28,33,53,54] The use of ultrasound in UTS has not been fully investigated, but Harvie and colleagues[55] screened 58 asymptomatic volunteers to determine the prevalence and morphology of anomalous muscles within the ulnar and found that 47% of volunteers had anomalous variants of abductor digiti mini. It was not clear, however, how anomalous muscles were defined. Ultrasound has the advantages of being a noninvasive, safe, cost- effective tool that can aid in diagnosis,[35] and in select cases it may aid in ganglion aspiration.[56] Arteriography is appropriate when an ulnar artery pathologic condition is suspected.

Electrodiagnostic studies such as electromyography and nerve conduction studies are often used as confirmatory studies after positive motor tests and positive sensory findings to establish a diagnosis of nerve compression. Although not an adequate substitute for a thorough and detailed physical examination, quality electrodiagnostic studies can complement the clinical findings by localizing the lesion and predicting the likelihood of neural regeneration and recovery. Concerns include the highly operator-dependent nature of the tests and the confounding effect of systemic disease-related and age-related changes to expected values. Nerve conduction testing cannot objectively evaluate pain and paresthesias because these sensations are transmitted by unmyelinated fibers that are left untested in the setting of a study, which assesses large myelinated nerve tracts.

CONSERVATIVE TREATMENT

Nonoperative treatment is usually initiated in mild cases or when a specific structural abnormality has not been identified. Little evidence exists regarding the effectiveness of conservative treatment. Protective splinting and anti-inflammatory medications may be appropriate in mild cases and in the event of concurrent CTS and UTS.[1] Activity modification has been shown to be an effective intervention when the cause of UTS is related to repetitive compression or trauma.[35,39,41] Ganglion aspiration alone has been performed to successfully treat UTS.[56] Little or no evidence exists regarding the role of steroid injections into Guyon's canal for idiopathic cases.

SURGERY

If an organic compressive lesion is causing signs and symptoms or if conservative treatment has failed, the patient should be offered surgery. Surgical exploration, removal of any space-occupying lesion, and decompression of the ulnar tunnel form the standard surgical treatment. Under either general, regional, or local anesthesia and tourniquet control, the patient's hand is placed in the supine position. It is our preference to perform this procedure under either general or regional anesthesia to avoid obscuring landmarks with local anesthetic administration; however, it certainly acceptable to use local anesthesia. An appropriate length incision is made longitudinally parallel to the thenar crease between the hamulus and pisiform and extended proximally approximately 3 to 4 cm beyond the distal wrist crease (Fig. 4). The palmaris brevis is incised proximally. Farther distally along the incision, some patients may exhibit an ulnar palmar cutaneous branch of large caliber. It is preferable to preserve this branch to limit postoperative paresthesias. Moving deep to the incision proximally, the thick antebrachial fascia over the

proximal wrist is released to decompress the ulnar nerve in zone I. Once the roof of Guyon's canal is released, the contents of the canal, particularly the ulnar artery and ulnar nerve before bifurcation, should be visible and accessible. The neurovascular bundle is gently moved medially and the hook of the hamate is identified. The fibrous, archlike origin of the hypothenar muscles should become evident coming off the hook of hamate (Figs. 5 and 6). Beneath the arch lies the deep motor branch of the ulnar nerve. The tendinous hypothenar muscle origin is carefully released close to the hook to further decompress the distal end the tunnel. The superficial sensory branch of the ulnar nerve courses superficial to the fibrous arch or the hypothenar muscles. Throughout the procedure, the ulnar artery should be carefully inspected to rule out any overt vascular pathologic condition. After completion of the decompression, the tourniquet is deflated and adequate hemostasis is ensured. The wound is closed and a bulky dressing or plaster splint is applied. Sutures are frequently removed at 10 to 14 days postoperatively.

SUMMARY

The approach to the diagnosis and treatment of UTS has changed little during the past several decades. UTS is less common than either CTS or cubital tunnel syndrome, and in a high proportion of cases, the cause can be localized to a compressive space-occupying lesion or repetitive trauma. The anatomy of the ulnar tunnel is complex, but numerous anatomic studies have described the tunnel in significant detail. Because organic lesions are often implicated in the cause, surgical exploration and decompression of the ulnar tunnel is a common treatment modality. Reported surgical results have yielded good results, although at this time, case series and comparative studies are uncommon, perhaps as a result of both the infrequency and heterogeneity of the causes.

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Fig. 1. Superficial structures of the palm and wrist. The superficial and deep branches of the ulnar nerve are indicated. ADM, abductor digiti minimi; FDM, flexor digiti minimi. (From Standring S. Gray's anatomy: the anatomical basis of clinical practice. 40th edition. New York: Churchill Livingstone; 2008. p. 891; with permission.)
Fig. 2. Surgical anatomy in the Guyon canal and the 3 zones of injury as described by Gross and Gelberman. FCU, flexor carpi ulnaris; H, hamate; M, motor branch; P, pisiform; PHL, pisohamate ligament; S, sensory branch; UA, ulnar artery; UN, ulnar nerve. (From Kokkalis ZT, Efstathopoulos DG, Papanastassiou ID, et al. Ulnar nerve injuries in guyon canal: a report of 32 cases. Microsurgery 2012;32(4):296–302, with permission; and data from Gross MS, Gelberman RH. The anatomy of the distal ulnar tunnel. Clin Orthop Relat Res 1985;196:238–47.)
Fig. 3. First dorsal interosseous muscle wasting in the left hand. (Courtesy of Dr D.G. Efstathopoulos, KAT Accident Hospital, and Dr Z.T. Kokkalis, Attikon University Hospital, Athens, Greece.)
Fig.4. Outline of a standard approach for decompression of the ulnar tunnel. The pisiform is labelled A, while the hook of the hamate is labelled B. (Courtesy of A.M. Ilyas.)
Fig. 5. Demonstration of the fibrous archlike origin of the hypothenar muscles. The hook of the hamate is ink-marked. The deep motor branch of the ulnar nerve courses deep to the fibrous arch. (From Washington University School of Medicine in St Louis. Peripheral nerve surgery: a resource for surgeons. Available at: nervesurgery.wustl. edu.)
Fig. 6. Demonstration of the leading edge of the hypothenar muscles. (From Washington University School of Medicine in St Louis. Peripheral nerve surgery: a resource for surgeons. Available at: nervesurgery.wustl.edu.)