Entrapment Neuropathy of the Ulnar Nerve

Bassem Elhassan, MD

Scott P. Steinmann, MD

Abstract

Ulnar nerve entrapment is the second most common nerve entrapment syndrome of the upper extremity. Although it may occur at any location along the length of the nerve, it is most common in the cubital tunnel. Ulnar nerve entrapment produces numbness in the ring and little fingers and weakness of the intrinsic muscles in the hand. Patient presentation and symptoms vary according to the site of entrapment. Treatment options are often determined by the site of pathology. Many patients benefit from nonsurgical treatment (eg, physical therapy, bracing, injection). When these methods fail or when sensory or motor impairment progresses, surgical release of the nerve at the site of entrapment should be considered. Surgical release may be done alone or with nerve transposition at the elbow. Most patients report symptomatic relief following surgery.

Dr. Elhassan is Hand Consultant and Assistant Professor of Orthopedic Surgery, Department of Orthopedics, Mayo Clinic, Rochester, MN. Dr. Steinmann is Hand Consultant and Associate Professor, Department of Orthopedics, Mayo Clinic.

In the past 20 years, there has been an increase in the incidence of compression neuropathy involving the upper extremity. In particular, the number of reported work-related cases has increased. Several articles have been published recently on the basic science, diagnosis, and treatment of compression neuropathies of the upper extremity.[1-4]

Ulnar nerve entrapment is the second most common nerve entrapment syndrome of the upper extremity; carpal tunnel syndrome (median neuropathy) is the most common. Symptoms associated with ulnar neuropathy may arise from impingement of one of the ulnar nerve roots (C8, T1) in the cervical spine; impingement in the brachial plexus (lower trunk and branches), manifested as thoracic outlet syndrome; or entrapment around the elbow, forearm, or wrist.

Despite widespread awareness of and the abundance of literature regarding ulnar nerve compression syndromes, diagnosis remains difficult. Often, the patient will not recognize the presence of nerve compression until the symptoms are severe, such that the patient presents with sensory and motor functional deficits. Late presentation decreases the likelihood of full recovery and good clinical outcome.

Peripheral Nerve Anatomy

Microanatomy

Peripheral nerve anatomy has been studied in detail.[5-7] Peripheral nerves consist of a connective tissue framework, with an inner and outer epineurium covering the nerve trunk; a perineurium surrounding the fascicles; and an endoneurium, which is intimately associated with each myelinated nerve fiber or group of unmyelinated nerve fibers. These

structures are supplied by a longitudinal network of epineurial, perineurial, and endoneurial vessels.

A single nerve cell is composed of a cell body, an axon, and terminal branches. A nerve fiber may be composed of multiple unmyelinated axons enveloped within a Schwann cell or may be a single myelinated axon wrapped by a Schwann cell. The areas between myelinated segments, called nodes of Ranvier, are devoid of myelin. These nodes transmit nerve signals from the cell body (central nerve body) proximally to the nerve endings distally. Schwann cells are interposed between the axon and the endoneurium. These cells provide the axon with necessary nutrients. The cell body cytoplasm extends down the axon, forming the axoplasm, which contains microtubules and neurofilaments, elements necessary for axonal transport. Loss of axonal transport and function of the nodes of Ranvier at any location along the peripheral nerve produces similar loss of nerve ending function and denervation at the myoneural junction.

Macroanatomy

Compression neuropathies of the ulnar nerve occur at anatomic sites that are susceptible to intrinsic and extrinsic compressive factors.[8] Intrinsic sources of nerve compression include anatomic structures, pathologic lesions (eg, perineuroma), and congenital anomalies (eg, hamartoma of the ulnar nerve). Extrinsic factors include fibrous tendon or muscle bands, hypertrophied muscles, and vascular lesions, any of which may directly compress the ulnar nerve at the elbow or wrist.

Acute compression may be caused by intraneural bleeding from blood dyscrasia; blunt trauma; fracture or dislocation; open injury (eg, knife or gunshot wound); or iatrogenic crush injury caused by surgical instruments, positioning, or prolonged tourniquet use. Chronic compression may be caused by hy-

pertrophic callus resulting from olecranon or distal humerus fracture or fracture malunion. Cubitus valgus is a cause of ulnar neuropathy resulting from distal humeral physeal fracture. Other etiologies of ulnar neuropathy include perineural scarring, ganglion cysts, tumors, and elevation of intracompartmental pressures.

Proliferation of the joint synovium and tenosynovium in rheumatoid disease has led to cubital tunnel syndrome and other forms of neural entrapment (eg, radial nerve compression). Tophaceous gout may cause entrapment neuropathy as the result of uric acid crystal deposits in the tenosynovium or in the nerve itself. The patient with hemophilia is susceptible to compression from perineural, intramuscular, and intraneural hematoma.

Some patients have a congenital predisposition to nerve compression because of the presence of an aberrant muscle (anconeus epitrochlearis muscle) in the cubital tunnel, a cervical rib that produces thoracic outlet syndrome, or malformation (eg, supracondylar process proximal to the elbow). Repetitive injury caused by vibrating hand tools as well as highly repetitive and forceful motion of the upper extremity have been implicated in ulnar nerve compression disorders at the wrist.

Pathophysiology of Nerve Compression

Classification

Peripheral nerve injuries are most easily grouped into three types: neurapraxia, axonotmesis, and neurotmesis. The latter has been divided into five degrees.[6,7] In first-degree injury (neurapraxia), local myelin damage blocks axonal conduction, but all nerve components remain intact. Second-degree injury (axonotmesis) is characterized by loss of axonal continuity, with wallerian degeneration of axons and myelin distally. First- and second-degree le-

sions recover spontaneously. In third-degree nerve injury, the perineurium is intact but the Schwann cells, axons, and endoneurium are disrupted. Regeneration may be incomplete, and fibrosis develops within the nerve. In fourth-degree injury, the epineurium remains intact. In fifth-degree injury, the nerve is transected. A combination of all of the above degrees of nerve injury recently has been categorized as sixth-degree injury. Compression neuropathy typically is a first- or second-degree injury, although there are instances of third-degree involvement.

Acute Effects

Acute nerve dysfunction, which occurs within hours of injury, often is related to compression secondary to ischemic changes. Basic research has shown that nerve dysfunction can be induced with extraneural pressure of 4.0 kPa less than the diastolic pressure.[5,7,9] Rydevik et al[10] demonstrated that 6.7 kPa extraneural compression applied for 2 minutes altered the shape of myelin sheaths. Higher pressures resulted in severe splitting and distortion of the myelin. Cyclic loading of nerves has been shown to decrease nerve function, which is similar to the effects of constant loading at the mean pressure.

Short-term Nerve Compression

Sustained low intraneural pressure may cause intraneural edema. Increased epineurial and endoneurial vessel permeability have been found after just 2 hours of compression. This edema has been shown to persist for longer than 24 hours, even after removal of the compressive insult.[2] Fracture-dislocation of the wrist and elbow may produce acute ulnar neuropathy secondary to sustained intraneural pressure from displaced bone fragments.

Figure 1
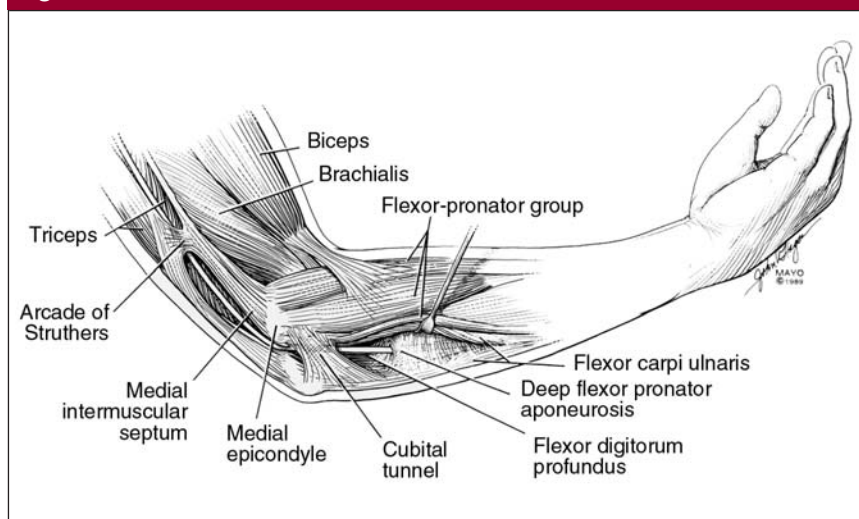
The five sites of potential ulnar nerve entrapment around the elbow: arcade of Struthers, medial intermuscular septum, medial epicondyle, cubital tunnel, and deep flexor pronator aponeurosis. (Copyright Mayo Foundation.)

Long-term Effects

Extraneural compression for longer durations (≤28 days) has been studied.[10] As with the short-term studies, subperineurial edema may persist even after the removal of the extraneural compression. Inflammatory and fibrin deposits occur within hours, followed by proliferation of endoneurial fibroblasts and capillary endothelial cells. Fibrous tissue from endoneurial fibroblasts proliferates within several days, followed by invasion of mast cells and macrophages into the endoneurial space. Axonal degeneration is noted in nerves subjected to compression for 4 weeks.

Histopathology

Histologic examination of nerves at the site of compression injury reveals proliferation of the endoneurial and perineurial microvasculature, edema in the epineurial space, and fibrotic changes.[5,7,10] Initial changes occur in the nerve-blood barrier, followed by edema and epineurial fibrosis. Thinning of the myelin sheath then occurs at the periphery of the nerve. Axonal degeneration follows prolonged compression. The rate and severity of these changes differ throughout the nerve, possibly because of variations in the amount of connective tissue. These pathologic changes appear to be dose-dependent, based on the duration and force of compression.

Anatomy

The ulnar nerve arises from the C8 and T1 nerve roots of the medial cord of the brachial plexus.[3,11] Five branches arise at the level of the cord; from proximal to distal, they are the medial pectoral nerve, medial brachial nerve, medial antebrachial cutaneous nerve, medial contribution to the median nerve, and ulnar nerve. Impingement of the C8, T1 root, caused by cervical spondylosis with narrowing of the intervertebral foramina, may lead to symptoms that mimic ulnar nerve compression.

Within the neck, compression or injury of the brachial plexus (ie, medial cord and distal branches) may occur secondary to a Pancoast tumor in the lung, metastatic disease (eg, breast cancer, lymphoma), or a cervical rib entrapping any part of the brachial plexus before it divides into the branches for the upper extremities. These passages begin at the level of the thoracic inlet, which is composed of the first rib laterally, the spine dorsally, and the manubrium sterni anteriorly. The passages move over the first rib to enter a potential space bordered anteromedially by the anterior scalene muscle, posterolaterally by the middle scalene, and at the base by the first rib. The medial cord and the ulnar nerve then enter the thoracic outlet at the level of the apex of the axilla, which is bordered by the first rib medially, the upper border of the scapula and subscapularis muscle dorsally, and the clavicle and subclavius muscle anterolaterally.

At the level of the axilla, the ulnar nerve is located between the axillary artery and vein, posterior to the pectoralis minor. The nerve continues in a sheath medial to the brachial artery at the level of the upper arm, then separates from the vascular bundle at the level of the insertion of the coracobrachialis muscle and travels posteriorly with the ulnar collateral artery. The ulnar nerve next pierces the medial intermusclar septum, emerging from under the arcade of Struthers, and lying on the medial head of the triceps (Figure 1). The arcade of Struthers, which is present in approximately 70% of the population, is a thickening of the deep investing fascia of the distal arm. It extends from the medial head of the triceps to the intermuscular septum.[12]

At the level of the elbow, the ulnar nerve continues distally toward the posterior aspect of the condylar groove, passing between the medial epicondyle and olecranon to enter the cubital tunnel. The floor of this tunnel is formed by the medial collateral ligament of the elbow, the joint capsule, and the olecranon. The roof is formed by the arcuate (Osborne's) ligament. This ligament

blends distally with the antebrachial fascia superficial to the aponeurosis and connects the ulnar and humeral heads of the FCU. The ulnar nerve continues further in the forearm between the FCU and the flexor digitorium profundus (FDP) muscles.

The ulnar nerve begins branching at the elbow, with a sensory articular branch to the elbow joint, after which it provides two to four branches to the FCU. In addition, it gives either a single branch or multiple branches to the medial one half of the FDP. The intraneural topography of the ulnar nerve at the cubital tunnel region shows that the location of the nerve fibers to the FCU and FDP is more central, whereas the motor fibers to the hand muscles and sensory fibers to the hand and fingers are located more superficial, which places the latter at a higher risk for compression and ischemia and thus, earlier clinical manifestation of entrapment neuropathy.

Distal to the axilla within the arm, forearm, and wrist, the ulnar nerve can be compressed by any of the following structures: arcade of Struthers, medial intermuscular septum, medial epicondyle, Osborne's ligament, fascia of the FCU, and aponeurosis of the proximal edge of the flexor digitorum superficialis. The presence of an anomalous muscle, the anconeus epitrochlearis, at the level of the cubital tunnel, also may lead to ulnar nerve compression at the elbow.

At the level of the middle to distal one third of the forearm, the ulnar nerve gives two cutaneous branches. The dorsal cutaneous branch, which arises 6 to 10 cm proximal to the wrist flexion crease, supplies the dorsomedial aspect of the hand and fingers. The palmar cutaneous branch of the ulnar nerve originates near the middle of the forearm and accompanies the ulnar artery into the hand. It gives branches to the ulnar artery, perforates the flexor retinaculum, and ends in the anteromedial skin of the palm, communicating with the palmar branch of the median nerve. The proximal dorsal cutaneous sensory branch of the ulnar nerve helps to separate more proximal (cubital tunnel) from distal (Guyon's canal) compression neuropathy of the ulnar nerve.

At the level of the wrist, the ulnar nerve enters Guyon's canal.[13] This canal is approximately 4 cm long, and it is divided into three zones: 1, located proximal to the bifurcation of the nerve; 2, surrounding mostly the deep motor branch; and 3, surrounding mostly the superficial sensory branch.[14] The roof of the canal is formed by the volar carpal ligament, the floor by the pisohamate ligament and the transverse carpal ligament, the radial wall by the abductor digiti minimi and the hook of hamate, and the ulnar wall by the pisiform.

In the hand, the ulnar nerve divides into the superficial sensory branch and the deep motor branch. The superficial sensory branch supplies the palmar surfaces of the little finger and the ulnar half of the ring finger. The deep motor branch supplies all of the small muscles of the hand except the radial two lumbricals and the median innervated thenar muscles, which include all of the thenar muscles, with the exception of the deep head of the flexor pollicis brevis.

Causative factors leading to ulnar tunnel syndrome include ganglion of the triquetrohamate joint, ulnar artery thrombosis, palmaris brevis hypertrophy, and nonunited fracture of the hook of hamate.

Diagnosis

History

A detailed history should be obtained for all patients who present with symptoms suggestive of upper extremity nerve entrapment. The patient usually presents with ill-defined upper extremity pain, which may be localized to the medial as-pect of the elbow.[15,16] Paresthesia and numbness of the small finger and the ulnar half of the ring finger are common, as is hand weakness secondary to intrinsic muscle weakness, which leads to reduced grip and pinch strength, difficulty opening bottles, and fatigue.

The patient should be asked about the onset of symptoms, subjective feelings of numbness (dorsal hand versus volar), grip or pinch weakness, loss of spreading of the fingers or thumb, exacerbating and alleviating factors, comorbidities (eg, diabetes, hemophilia, general peripheral neuropathy), and occupation. Ulnar hammer syndrome is a likely diagnosis in the patient who works with vibrating tools. The positions of the shoulder, elbow, and wrist that provoke the symptoms are recorded. These positions include elbow flexion (compression of the cubital tunnel), overhead elevation (thoracic outlet syndrome), and wrist flexion (entrapment in Guyon's canal).

Physical Examination

In the patient with ulnar nerve symptoms, the examination must be performed from the proximal origin of the nerve to its most distal aspect.[15] A detailed examination of the cervical spine, neck, and axilla is performed to rule out thoracic outlet syndrome and cervical nerve root compression. The Adson maneuver and the Allen test for thoracic outlet syndrome, and the Spurling test for cervical nerve root compression, may aid in differentiating between these differential diagnoses. Within the axilla, palpation, for a mass or Tinel sign should be performed. The ulnar nerve should be traced by palpation, and the Tinel sign should be sought along the medial aspect of the arm, from the axilla to the cubital tunnel.

Next, a detailed examination of the elbow should be performed, paying attention for any mass or deformity. The carrying angle of the el-

bow must be determined. It is also important to check for ulnar nerve subluxation with elbow flexion and extension, and to differentiate ulnar neuropathy from the snapping triceps syndrome.[17]

When examining the wrist and hand, the surgeon should examine for swelling or a pulsatile mass, as well as for point tenderness over the hook of hamate or pisiform. In addition, a vascular examination should be performed, including the Allen test at the wrist; detection of thrills or bruits in the axilla, arm, or wrist; and, if necessary, a Doppler examination.

Sensory Examination

Sensory touch fibers (group A-β) are divided into slowly and quickly adapting fibers.[16] A slowly adapting fiber continues its pulse response throughout the duration of the impulse, while a quickly adapting fiber signals an on-off event. Useful testing tools include a two-point discrimination-measuring device, the Semmes-Weinstein monofilament test, a 256 cps tuning fork, and a vibrometer. The static two-point discrimination and Semmes-Weinstein monofilament tests evaluate the slowly adapting fibers, whereas the moving two-point discrimination and vibration tests evaluate the quickly adapting fibers. As part of the sensory assessment, the examiner should note changes in the threshold of stimulus intensity and innervation density. Threshold tests, which include the Semmes-Weinstein and the vibration test, measure a single nerve fiber innervating a receptor or a group of receptors. These tests are particularly useful for detecting gradual, progressive changes in nerve function.

Mild nerve compression results in transient hypersensitive responses to vibratory perception. Moderate to severe compression results in decreased cutaneous vibratory perception. Innervation density tests, which include static and moving two-point discrimination, measure multiple overlapping peripheral receptive fields and the density of innervation in the region being tested. These tests are reliable for assessing functional nerve regeneration after nerve repair, but they are not sensitive to the gradual decrease in nerve function seen in nerve compression. Only when nerve compression results in wallerian degeneration does the diminution in the density of innervation lead to increased two-point discrimination (<6 mm, normal; >10 mm, poor).

Several easily performed tests are useful in diagnosing ulnar neuropathy, including Tinel's nerve percussion test, direct compression of the nerve, elbow flexion, and use of a hand diagram on which the patient marks on an outline of the location of numbness, tingling, or pain over the dorsal and palmar aspects of the hand. The Semmes-Weinstein monofilament, vibrometry, and direct compression tests are the most sensitive, and the hand diagram and Tinel sign are the most specific, for diagnosing ulnar nerve compression at Guyon's canal. However, with cubital tunnel syndrome, it has been shown that both the Tinel sign and the Phalen test, which consists of elbow flexion, forearm supination, and wrist extension for 1 minute, are falsely positive in 24% of cases.[14] Thus, none of the aforementioned tests is highly sensitive or specific for cubital tunnel syndrome.

Motor Examination

Motor function is graded on a scale of 0 to 5. Grip and pinch strength in the hand are measured using a dynamometer and a pinch gauge. Motor dysfunction usually is not associated with mild compression. Moderate compression can cause muscle weakness, and severe compression may lead to muscular atrophy.[1,7,15]

The motor examination should be performed by resistive testing of the FDP to the ring and little fingers, as well as the small muscles in the hand, with the exception of the median innervated muscles. However, the examiner should be aware of anomalous innervation patterns, such as the Martin-Gruber communication, which exists in 7.5% of the population.[1,3,15] In this anomaly, the anterior interosseous nerve, a branch of the median nerve, communicates with the ulnar nerve in the proximal forearm. Thus, the FDP innervation may vary from all ulnar and all median, to completely dual, with the thenar muscles possibly innervated by the ulnar nerve.

Another nerve anomaly is the Riche-Cannieu anastomosis, which consists of communication between the motor branch of the ulnar nerve and the recurrent branch of the median nerve. The median nerve may innervate all of the lumbrical muscles, and the dorso-ulnar surface of the hand may be innervated by a superficial branch of the radial nerve.

Intrinsic muscle testing is done on the first dorsal interosseous, abductor digiti quinti, and adductor muscles. Weakness of the adductor pollicis can be tested by detecting either a positive Jeanne's sign, indicating loss of key pinch of the thumb, or Froment sign (thumb flexion), indicating weakness involving the 1st dorsal interosseous, 2nd palmar interosseous, or adductor pollicis muscles. In the Froment test, during key pinch (straight thumb interphalangeal joint), the thumb flexes at the interphalangeal joint as a result of the overpull of the flexor pollicis longus caused by paralysis or weakness of the adductor pollicis. The patient attempts to lateral pinch on a piece of paper or cloth against resistance. To compensate for the adductor paralysis and to allow for the cloth to be held and not pulled away from the thumb, the FPL overly contracts, producing the Froment sign.

The crossed fingers test is done to evaluate the function of the 1st palmar and 2nd dorsal interossei. The surgeon also should check for the

Duchenne sign (clawing of the ring and little finger), as well as the Wartenberg sign (spontaneous abduction of the little finger when the patient attempts to extend the fingers). The Wartenberg sign is caused by unopposed ulnar insertion of the extensor digiti quinti. The examiner may notice more severe claw deformity in the little finger than in the ring finger. This disparity is caused by inherent increased laxity in the little finger metacarpophalangeal joint volar plate. In approximately one half of patients, the median nerve has cross-innervation to the lumbrical of the ring finger, thus preventing claw deformity of the ring finger. Clawing should not occur in either the long or the index finger.

Testing the innervation of the dorsal sensory branch of the ulnar nerve aids in distinguishing proximal (cubital tunnel) from distal (Guyon's canal) lesions. An abnormal sensory examination over the ulnar dorsopalmar aspect of the hand indicates a more proximal lesion.

Diagnostic Tests

Radiographs

Radiographs of the involved extremity should be obtained to rule out any bony deformity or anomaly as a cause of nerve entrapment at the elbow or the wrist.[15] Occasionally, classic peripheral neuropathy is caused by anatomic variations that may be visible on plain radiographs (eg, median nerve compression caused by a supracondylar process).

Cervical spine radiographs, including outlet views and transaxillary views, are required to rule out cervical radiculopathy and cervical rib involvement. A chest radiograph is recommended to rule out Pancoast tumor in the patient with a history of smoking, shoulder pain, and ulnar nerve–related symptoms. Anteroposterior, lateral, and epicondylar tunnel views of the elbow are recommended to rule out arthritis, posttraumatic changes, and ab-

normal carrying angle (eg, cubitus valgus). Recently, ultrasound of the cubital tunnel was used to determine the size of the ulnar nerve and compare it with a normal value.[12] The authors demonstrated a correlation between decreased diameter of the nerve and progressive ulnar neuropathy at the elbow.

At the level of the hand, carpal tunnel and oblique views should be obtained for the patient with ulnar tunnel (ie, Guyon's canal) syndrome. Additional studies, including a computed tomography scan of the hand, should be considered to rule out fracture of the hook of hamate. Soft-tissue anomalies may be evident on magnetic resonance imaging, including space-occupying lesions causing nerve compression, such as ganglion, soft-tissue tumors, or aneurysm.[18] Thrombosis of the ulnar artery leading to ulnar nerve symptoms in the hand can be confirmed by either a duplex scan ultrasound or an angiogram.

Electrodiagnostic Studies

Electromyography (EMG) records electrical activity within a muscle during voluntary contraction.[19] The relaxed muscle is neutral. Weak voluntary contraction produces a few motor action potentials. Maximal effort produces a signal with multiple action potentials firing simultaneously. EMG is helpful in diagnosing advanced compression neuropathy with muscular denervation, which results in spontaneous action potential muscle fibrillations and decreased interference patterns with maximal voluntary effort.

In motor nerve conduction studies, amplitude, velocity, and latency of the muscular action potential are measured. Conduction velocity and latency are related to nerve myelination; electrical amplitude is related to the integrity of the axon. Compression neuropathy results in increased distal latency and decreased conduction velocity. Absence of motor response suggests severe neurop-

athy. One of the earliest signs of compression neuropathy is an increase in the threshold of excitability to stimulation of the involved nerve.

The patient with cubital tunnel syndrome may show focal slowing of conduction in the ulnar nerve segment crossing the elbow. The lower limit of normal with the elbow flexed to 135° should be >49 m/sec or within 11 m/sec of the forearm segment. Conduction study with the inching technique can aid in localizing a specific area of compression at the elbow (ie, supracondylar process).

Laboratory Studies

Laboratory studies should be used judiciously to eliminate differential diagnoses and to monitor comorbid conditions in the patient with nerve compression syndrome. Thyroid abnormalities, diabetes, and autoimmune disorders should be assessed and treated before any surgical intervention for peripheral neuropathy.

Treatment

Nonsurgical

Nonsurgical management of ulnar neuropathy is indicated for mild to moderate ulnar nerve entrapment.[20] Night splinting with the elbow in 45° of flexion and the forearm in neutral rotation, padding of the nerve, and daily use of a cushion splint or protective splinting of the wrist are recommended, in addition to nonsteroidal anti-inflammatory drugs and, occasionally, steroid injection. Occupational therapy emphasizing ergonomic upper extremity positioning at work and at rest also may aid in recovery. The patient with intermittent symptoms, no atrophy, and mild electrodiagnostic findings may respond well to nonsurgical management.

Surgical

Surgical intervention is indicated when nonsurgical methods have

failed.[8,11,18,19,21-24] Because there are multiple areas of potential ulnar nerve compression, the surgical options are many and should be tailored to the particular patient. The principle of surgery is to decompress a peripheral nerve by removing the pressure applied to the nerve at rest and when the joint across which the nerve travels is taken through a range of motion. All sites of compression must be eliminated, and the nerve must be tension-free and allowed to glide. Many surgical techniques have been described to decompress the ulnar nerve at the elbow, including in situ decompression, medial epicondylectomy, and anterior transposition of the subcutaneous, intramuscular, or submuscular positioning of the nerve. More recently, endoscopic in situ decompression has been described.[25]

Decompression

With in situ decompression, all sites of compressions are relieved, keeping the nerve in its bed. Osborne[26] reported on the technique of in situ release. An incision is placed midway between the medial epicondyle and the olecranon. The ulnar nerve is identified proximally and then exposed. Using scissors, the cubital tunnel retinaculum (ie, Osborne's ligament) is divided proximal to distal. The fascia between the two heads of the FCU then are incised. The motor branch to the FCU is identified and preserved. Further release is performed distally through the flexor-pronator aponeurosis; when indicated, the nerve is decompressed proximally through the medial intermuscular septum.

Wilson and Krout[27] reported on 16 consecutive patients (17 elbows) treated with simple decompression. They reported eight excellent, five good, and four fair outcomes, as well as one revision. The authors concluded that the ulnar nerve could be restored to normal by simple decompression.

Miller and Hummel[28] reported on 12 patients with progressively worsening paresthesia, moderate to severe weakness, and EMG changes who were managed with simple decompression. Six of 7 patients had improvement of pain, 9 of 12 had improvement in paresthesia, and 11 of 12 had improvement on EMG. One ulnar nerve required transposition. The authors concluded that the best outcome of simple decompression is obtained in the patient with mild weakness, recent onset of symptoms, and mild abnormality of sensory action potentials.

The advantages of simple ulnar nerve decompression include simplicity of incision, use of local anesthesia, and lack of postoperative immobilization. Disadvantages include potentially inadequate decompression, ulnar nerve subluxation, and failure to address all potential pathology in the patient with advanced motor and sensory loss and abnormal bony anatomy (ie, cubitus valgus).

Medial Epicondylectomy

In 1959, King and Morgan[29] reported their technique, in which a 12- to 15-cm incision is made over the medial epicondyle. The medial antebrachial cutaneous nerve is identified and protected. The ulnar nerve is identified and released in the same way as for simple decompression, from a proximal to distal direction. The medial epicondyle is exposed subperiosteally, and osteotomy of the medial epicondyle is performed, taking care to avoid the joint and the origin of the ulnar collateral ligament. The osteotomy surface is finely rasped, and the flexor-pronator origin is then reattached to the periosteum with the elbow in extension.

Craven and Green[30] studied 30 patients who reported symptoms of cubital tunnel syndrome lasting for an average of 10.8 months. These patients were treated with medial epicondylectomy. Twenty-eight of 30 patients had complete pain relief;

improvement in conduction velocity from an average of 33 m/sec to 54 m/sec; and no recorded flexor-pronator weakness. The authors recommended this technique for cubital tunnel syndrome in the patient with abnormal nerve conduction velocity across the elbow.

Heithoff et al[31] reviewed 43 patients with chronic ulnar neuropathy who were treated with medial epicondylectomy. At an average follow-up of 2.3 years, 8 patients had excellent, 23 had good, 9 had fair, and 3 had poor results. The poor results were related to failure of the neuropathy to improve over time. No problems were reported related to osteotomy site tenderness, elbow instability, flexor-pronator weakness, or increase in ulnar nerve vulnerability. The authors concluded that medial epicondylectomy is a satisfactory means of decompression.

In a primate study, Ogata et al[32] demonstrated that anterior transposition is associated with significant decrease in blood flow for several days. No decrease in blood flow was reported with simple release or epicondylectomy.

Advantages of medial epicondylectomy include the relative simplicity of the procedure and, because medial epicondylectomy requires less dissection than ulnar nerve transposition, a lower risk of ulnar nerve injury. Disadvantages include bone tenderness, risk of elbow instability, wrist flexor-pronator muscle weakness, and heterotopic bone formation.

Transposition

Anterior ulnar nerve transposition relieves the biomechanical mechanism of cyclic traction and compression on the ulnar nerve by placing the nerve anterior to the axis of elbow motion. In subcutaneous transposition, the nerve is placed superficial to the flexor-pronator origin, and a fascial sling is created to prevent nerve migration.

Eaton et al[22] reported on 14 patients (16 elbows) who underwent subcutaneous transposition of the ulnar nerve. Their technique consisted of complete release of the ulnar nerve from proximal to distal, followed by creation of a noncompressing fasciodermal sling that was placed posterior to the transposed nerve at the level of the medial epicondyle. At an average 18-month follow-up, 14 patients had improvement of their symptoms, 5 of 6 patients with weakness regained normal strength, and 3 of 4 patients with sensory loss regained two-point sensation. The authors concluded that subcutaneous transposition, a simple alternative to submuscular transposition, offers good results.

Richmond and Southmayd[33] reported on 18 subcutaneous transpositions in which the epineurium was sutured to the fascia (16 patients). At an average follow-up of 23 months, results were excellent in 15 extremities (83%), satisfactory in 1 (6%), and unsatisfactory in 2 (11%). The patients with unsatisfactory results had double lesions. The authors concluded that subcutaneous transposition using an epineural sling is an effective minimally invasive option for managing ulnar neuropathy.

Subcutaneous ulnar nerve transposition is technically easier to perform than either submuscular or intramuscular transposition. However, its disadvantages include inadequate decompression of all areas of compression and, especially in the thin patient, the vulnerability of the nerve to repeated trauma because of its position in the subcutaneous tissue.

Intramuscular transposition places the nerve within the muscle mass by creating a tunnel in the flexor-pronator origin.[15] The proximal border of the pronator teres fascia is incised in line with the medial intermuscular septum from mid humerus to the elbow. The ulnar nerve is transposed anteriorly. A 5-mm trough is created in the muscle, after which the flexor-pronator fascia is repaired over the nerve with the forearm pronated and flexed to 90°.

Kleinman and Bishop[34] reported on 45 extremities that underwent intramuscular transposition. At an average 28-month follow-up, 87% of the extremities had excellent or good results, 4% had fair results, and 9% had poor results based on sensory recovery and ulnar motor strength testing. The factors that negatively affected outcome included preoperative advanced EMG abnormalities and cases involving workers' compensation. No problems with intraneural scarring were reported. The authors concluded that anterior intramuscular transposition is a reliable procedure with good results.

In the Learmonth submuscular transposition technique, the flexor-pronator mass is completely detached, and the nerve is transposed anteriorly under the muscle mass. As described by Leffert,[35] the flexor-pronator mass is detached and reflected posteriorly from the medial epicondyle. The ulnar nerve is carefully dissected from proximal to distal, the medial intermuscular septum is released and resected, and restrictive fascia or abnormal muscles are divided. The ulnar nerve is transposed anteriorly adjacent and parallel to the median nerve. Then the flexor-pronator group is reattached with the elbow flexed and the forearm pronated.

Amadio[36] reported anatomic findings of the entrapped ulnar nerve in 58 patients who underwent submuscular transposition. The author reported multiple potential sites of ulnar constriction, particularly in revision cases, and concluded that submuscular transposition more predictably released all sites of compression. Dellon[21] performed a literature review of 50 articles on ulnar nerve entrapment that were published from 1898 through 1988. He reported that 50% of patients with minimal compression could obtain excellent results with nonsurgical treatment, and nearly 100% could obtain excellent results with any of five surgical techniques. For moderate to severe compression, anterior submuscular transposition offered the best results with the lowest recurrence rate, whereas anterior intramuscular transposition gave the fewest excellent results and the most clinical presentations of recurrence of ulnar nerve symptoms.

The advantages of anterior submuscular transposition include release of all potential compression sites and protection of the nerve in a less vulnerable position beneath the flexor-pronator origin. The disadvantages include longer postoperative immobilization, possible weakness of the flexor-pronator mass, and a more technically demanding procedure.

In a prospective randomized study, Nabhan et al[37] compared simple decompression with anterior subcutaneous transposition in 66 patients with cubital tunnel syndrome. Thirty-two patients underwent simple decompression, and 34 underwent anterior subcutaneous transposition. No significant difference in pain, motor and sensory deficits, or nerve conduction velocity studies was found between the two groups at 3- to 9-month follow-up. The authors recommended simple decompression of the ulnar nerve.

Biggs and Curtis[38] reported on 23 in situ neurolysis and 21 submuscular transpositions. The results were equally effective, but three deep wound infections developed in the transposition group. The authors concluded that in situ release is equally effective and offers fewer complications.

Bartels et al[39] reported on 75 simple decompressions and 77 anterior subcutaneous transpositions in a prospective randomized trial. The authors found no difference in clinical outcome between the two groups. The complication rate was 9.6% in the simple decompression group and 31.1% in the transposi-

tion group. The authors concluded that simple decompression was easier to perform because of reduced soft-tissue dissection and the absence of muscle detachment. Additionally, simple decompression was associated with fewer complications, even in the presence of subluxation.

Postoperative Complications of Cubital Tunnel Release

Persistent symptoms after ulnar nerve transposition are usually the result of incomplete decompression or perineural scarring.[40] The posterior branch of the medial antebrachial cutaneous nerve often crosses the surgical field, particularly with anterior or submuscular transposition. Injury to this branch can lead to hyperesthesia, hyperalgesia in the forearm, and occasionally, a painful surgical scar. Subluxation of the ulnar nerve may occur with simple decompression alone. Injury to the medial collateral ligament of the elbow may occur as a complication of a submuscular transposition procedure. Recurrent entrapment has been reported following intramuscular and submuscular transposition. To prevent nerve transfer–related adhesions, Dellon[21] recommended early mobilization after ulnar nerve surgery. Dellon believes that immobilizing the elbow for more than 2 weeks to permit reattachment of either the muscle origin or the sling for the anterior subcutaneous transposition after submuscular transposition might result in adherence of the ulnar nerve in its new position.

Revision surgery may provide some benefit, but the results are unpredictable. In a review of 50 studies, 75% of patients reported some residual symptoms, although many symptoms were improved at initial surgery, particularly with submuscular transposition.[21] The good results with submuscular transposition led to it becoming the recommended surgical technique for revision cases of failed ulnar nerve decompression.

Management of Ulnar Tunnel Syndrome

Treatment of the ulnar nerve in Guyon's canal (ie, ulnar tunnel syndrome) follows the same principles as management of cubital tunnel syndrome. With ulnar tunnel syndrome, the possible etiologies include local masses (ie, ganglia), fracture of the hook of hamate, or distal radius fracture with marked dorsal displacement.[14,41] Surgical treatment is indicated in the patient with both motor and sensory deficit, especially in the presence of a specific anatomic lesion. On surgical exploration, Guyon's canal is opened, and the distal arcade at the hypothenar muscle origin is released. Release should include all four compartments, starting proximal to the wrist flexion crease and extending to the mid palmar line. Loss of motor function only should be managed with release of the pisohamate ligament and exploration of a deeply located ganglion cyst.

Summary

Ulnar nerve entrapment syndrome is a common compression neuropathy of the upper extremity. Cubital tunnel syndrome is the most common form of this entrapment. Surgical release of the cubital tunnel alone or with anterior transfer of the ulnar nerve is recommended. For the elderly patient, subcutaneous transposition is recommended in the presence of both motor and sensory deficit. For the patient with persistent symptoms, no objective motor loss, and mild sensory loss, release of the cubital tunnel alone may be sufficient. A subluxating ulnar nerve should be managed with anterior transposition. Submuscular transposition is recommended for the throwing athlete, as well as for the patient with more than one area of compression and recurrence of cubital tunnel syndrome despite surgical release.

Release of Guyon's canal is recommended when there is clear evidence of a compression neuropathy caused by a ganglion cyst, tumor, hamate fracture, or thrombosis of the ulnar artery (ie, ulnar hammer syndrome). Simultaneous release of the carpal tunnel and Guyon's canal can be performed in the properly selected patient.

References

Evidence-based Medicine: There are two level I studies (references 38 and 39) and several case-control series (level III/IV) (references 1, 2, 4, 6, 7, 11-16, 19-21, 36, and 41). The remainder are primarily expert opinion references (level V).

Citation numbers printed in **bold type** indicate references published within the past 5 years.

1. Eversmann WW Jr: Entrapment and compressive neuropathies, in Green DP (ed): *Operative Hand Surgery*, ed 3. New York, NY: Churchill Livingstone, 1993, p 1341-1389.
2. Dahlin LB, Rydevik B: Pathophysiology of nerve compression, in Gelberman RH (ed): *Operative Nerve Repair and Reconstruction*. Philadelphia, PA: JB Lippincott, 1991, pp 847-866.
3. Norkus SA, Meyers MC: Ulnar neuropathy of the elbow. *Sports Med* 1994;17:189-199.
4. Osterman AL: The double crush syndrome. *Orthop Clin North Am* 1988; 19:147-155.
5. Rempel D, Dahlin L, Lundborg G: Pathophysiology of nerve compression syndromes: Response of peripheral nerves to loading. *J Bone Joint Surg Am* 1999;81:1600-1610.
6. Seddon H: *Surgical Disorders of the Peripheral Nerves*, ed 2. Edinburgh, Scotland: Churchill Livingstone, 1975.
7. Sunderland S: *Nerve and Nerve Injuries*, ed 2. Edinburgh, Scotland: Churchill Livingstone, 1978.
8. Amadio PC, Beckenbaugh RD: Entrapment of the ulnar nerve by the deep flexor-pronator aponeurosis. *J Hand Surg [Am]* 1986;11:83-87.
9. Lundborg G: Structure and function of

the intraneural microvessels as related to trauma, edema formation, and nerve function. *J Bone Joint Surg Am* 1975;57:938-948.

10. Rydevik B, Lundborg G, Bagge U: Effects of graded compression on intraneural blood flow: An in vivo study on rabbit tibial nerve. *J Hand Surg [Am]* 1981;6:3-12.

11. Rayan GM: Proximal ulnar nerve compression: Cubital tunnel syndrome. *Hand Clin* 1992;8:325-336.

12. Wiesler ER, Chloros GD, Cartwright MS, Shin HW, Walker FO: Ultrasound in the diagnosis of ulnar neuropathy at the cubital tunnel. *J Hand Surg [Am]* 2006;31:1088-1093.

13. Moneim MS: Ulnar nerve compression at the wrist: Ulnar tunnel syndrome. *Hand Clin* 1992;8:337-344.

14. Gelberman RH: Ulnar tunnel syndrome, in Gelberman RH (ed): *Operative Nerve Repair and Reconstruction.* Philadelphia, PA: JB Lippincott, 1991, pp 1131-1143.

15. Taleisnik J, Szabo RM: Compression neuropathies of the upper extremity, in Chapman MW, Madison M (eds): *Operative Orthopaedics*, ed 2. Philadelphia, PA: JB Lippincott, 1993, vol 2, pp 1419-1465.

16. Dellon AL: Patient evaluation and management considerations in nerve compression. *Hand Clin* 1992;8:229-239.

17. Spinner RJ, O'Driscoll SW, Jupiter JB, Goldner RD: Unrecognized dislocation of the medial portion of the triceps: Another cause of failed ulnar nerve transposition. *J Neurosurg* 2000;92:52-57.

18. Ruocco MJ, Walsh JJ, Jackson JP: MR imaging of ulnar nerve entrapment secondary to an anomalous wrist muscle. *Skeletal Radiol* 1998;27:218-221.

19. Bradshaw DY, Shefner JM: Ulnar neuropathy at the elbow. *Neurol Clin* 1999;17:447-461.

20. Spinner M: Management of nerve compression lesions of the upper extremity, in Omer G, Spinner M (eds): *Management of Peripheral Nerve Problems.* Philadelphia, PA: WB Saunders, 1980, pp 569-605.

21. Dellon AL: Review of treatment results for ulnar nerve entrapment at the elbow. *J Hand Surg [Am]* 1989; 14:688-700.

22. Eaton RG, Crowe JF, Parkes JC III: Anterior transposition of the ulnar nerve using a non-compressing fasciodermal sling. *J Bone Joint Surg Am* 1980; 62:820-825.

23. Geutjens G, Langstaff RJ, Smith NJ, Jefferson D, Howell CJ, Barton NJ: Medial epicondylectomy or ulnar-nerve transposition for ulnar neuropathy at the elbow? *J Bone Joint Surg Br* 1996;78:777-779.

24. Froimson AI, Anouchi YS, Seitz WH Jr, Winsberg DD: Ulnar nerve decompression with medial epicondylectomy for neuropathy at the elbow. *Clin Orthop Relat Res* 1991;265:200-206.

25. Porcellini G, Paladini P, Campi F, Merolla G: Arthroscopic neurolysis of the ulnar nerve at the elbow. *Chir Organi Mov* 2005;90:191-200.

26. Osborne G: Compression neuritis of the ulnar nerve at the elbow. *Hand* 1970;2:10-13.

27. Wilson DH, Krout R: Surgery of ulnar neuropathy at the elbow: 16 cases treated by decompression without transposition. Technical note. *J Neurosurg* 1973;38:780-785.

28. Miller RG, Hummel EE: The cubital tunnel syndrome: Treatment with simple decompression. *Ann Neurol* 1980;7:567-569.

29. King T, Morgan FP: Late results of removing the medial humeral epicondyle for traumatic ulnar neuritis. *J Bone Joint Surg Br* 1959;41:51-55.

30. Craven PR Jr, Green DP: Cubital tunnel syndrome: Treatment by medial epicondylectomy. *J Bone Joint Surg Am* 1980;62:986-989.

31. Heithoff SJ, Millender LH, Nalebuff EA, Petruska AJ Jr: Medial epicondylectomy for the treatment of ulnar nerve compression at the elbow. *J Hand Surg [Am]* 1990;15:22-29.

32. Ogata K, Shimon S, Owen J, Manske PR: Effects of compression and devascularisation on ulnar nerve function: A quantitative study of regional blood flow and nerve conduction in monkeys. *J Hand Surg [Br]* 1991;16:104-108.

33. Richmond JC, Southmayd WW: Superficial anterior transposition of the ulnar nerve at the elbow for ulnar neuritis. *Clin Orthop Relat Res* 1982; 164:42-44.

34. Kleinman WB, Bishop AT: Anterior intramuscular transposition of the ulnar nerve. *J Hand Surg [Am]* 1989;14: 972-979.

35. Leffert RD: Anterior submuscular transposition of the ulnar nerves by the Learmonth technique. *J Hand Surg [Am]* 1982;7:147-155.

36. Amadio PC: Anatomical basis for a technique of ulnar nerve transposition. *Surg Radiol Anat* 1986;8:155-161.

37. Nabhan A, Ahlhelm F, Kelm J, Reith W, Schwerdtfeger K, Steudel WI: Simple decompression or subcutaneous anterior transposition of the ulnar nerve for cubital tunnel syndrome. *J Hand Surg [Br]* 2005;30:521-524.

38. Biggs M, Curtis JA: Randomized, prospective study comparing ulnar neurolysis in situ with submuscular transposition. *Neurosurgery* 2006;58: 296-304.

39. Bartels RH, Verhagen WI, van der Wilt GJ, Meulstee J, van Rossum LG, Grotenhuis JA: Prospective randomized controlled study comparing simple decompression versus anterior subcutaneous transposition for idiopathic neuropathy of the ulnar nerve at the elbow: Part I. *Neurosurgery* 2005;56: 522-530.

40. Rogers MR, Bergfield TG, Aulicino PL: The failed ulnar nerve transposition: Etiology and treatment. *Clin Orthop Relat Res* 1991;269:193-200.

41. Bednar MS: Ulnar tunnel syndrome. *Hand Clin* 1996;12:657-664.