CUBITAL TUNNEL SYNDROME

Oleh:

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Introduction

Prior to 1957, ulnar neuropathy was felt to be a "stretch neuritis" caused by cubitus valgus. In fact, the predominant cause of ulnar neuropathy was secondary to elbow injuries. Osborne proposed the concept of compression of the ulnar nerve in 1957, with Feindel and Stratford defining the "cubital tunnel" the following year. Since the advent of more successful orthopedic management of complex elbow injuries, the more frequent cause of ulnar nerve entrapment has become idiopathic or related to a "susceptible" patient. Nonetheless, it appears that ulnar nerve entrapment is increasing in prevalence (although no data are available), with pain often being a predominant morbidity. In 1898, Curtis performed the first published case of management for ulnar nerve neuropathy at the elbow, which consisted of a subcutaneous anterior transposition. *(1,2)*

It is hoped that the reader may develop an increased awareness of cubital tunnel syndrome and its place in the differential diagnosis of the painful upper extremity. An overview of the diagnostic and treatment options are presented, along with complications that may occur. *(1,2)*

Kata kunci:

Surgical Anatomy of The Ulnar Nerve

The ulnar nerve is formed from the medial cord of the brachial plexus, which originates from the C8 and T1 cervical roots along the lateral wall of the axilla. As it passes through the proximal arm, it maintains a posteromedial relationship to the brachial artery, initially lying between the coracobrachialis and triceps muscles. In the middle one third of the arm, the ulnar nerve accompanies the superior ulnar collateral artery posteriorly through the intermuscular septum to lie on the anterior aspect of the medial head of the triceps muscle. The nerve travels on the
posterior surface of the intermuscular septum medial to the humerus, to reach the elbow. It traverses the elbow region bounded medially and anteriorly (superiorly) by the medial humeral epicondyle, laterally by the olecranon and by a connective tissue roof spanning the two bony prominences-the "epicondylar groove". The nerve then enters the "cubital tunnel" by passing deep to the arcuate ligament (Osborne's ligament), which connects the ulnar and humeral heads of the flexor carpi ulnaris (FCU) muscle. The nerve then passes between the two heads of the FCU and passes deep to the deep flexor pronator aponeurosis. It then travels through the forearm between the FCU and flexor digitorum profundus (FDP), giving off motor branches to the FDP of the small and ring fingers. The nerve enters the wrist through Guyon's canal; a fibrous-osseous canal, extending 4 cm from the palmar carpal ligament to the fibrous edge of the hypothenar muscles. This is also a common site of ulnar nerve entrapment. (3)

The ulnar nerve is primarily a motor and sensory nerve for the hand. As such, it does not give off any branches in the arm (except small sensory articular branches to the elbow). It supplies the FCU muscle and the ring and small FDP muscles in the elbow/proximal forearm region. Proximal to the wrist, it gives off the dorsal sensory branch to the ulnar aspect of the hand. In the hand, it is the primary innervation to the intrinsic muscles (except for some of the thenar muscles; superficial head of the flexor pollicis brevis, opponens pollicis, and abductor pollicis brevis), the index and long finger lumbricals, and the sensation to the ulnar 1.5 digits.

The ulnar is also well known to have anomalous connections (Martin-Gruber anastomosis) to the median nerve, primarily occurring in the forearm, which alter the motor contribution of the two
nerves (primarily the intrinsic motor supply). This is important when assessing for distal weakness caused by proximal median or ulnar nerve lesions. \(^{(3,4)}\)

O'Driscoll (1991) believes that the roof of the cubital tunnel, or Osborne ligament, is a remnant of the anconeus epitrochlearis muscle. He also identified a retinaculum at the proximal edge of the arcuate ligament in all but 4 of 25 cadaveric specimens. He classified this retinaculum as 1 of 4 types, as follows:

1. An absent retinaculum
2. A thin retinaculum that becomes tight with full flexion without compressing the nerve
3. A thick retinaculum that compresses the nerve between 90° and full flexion
4. An accessory anconeus epitrochlearis muscle

Upon entering the cubital tunnel, the ulnar nerve gives off an articular branch to the elbow. It then passes between the humeral and ulnar heads of the FCU, the next potential site of compression. The nerve then descends into the forearm between the FCU and the FDP muscles. \(^{(4,5)}\)

About 5 cm distal to the medial epicondyle, the ulnar nerve pierces the flexor pronator aponeurosis, the fibrous common origin of the flexor and pronator muscles. The flexor-pronator aponeurosis is another point of possible compression, with compression of the ulnar nerve beneath the muscle belly of the FCU.

The ligament of Spinner is an additional aponeurosis between the flexor digitorum superficialis (FDS) of the ring finger and the humeral head of the FCU. This septum is independent of the other aponeuroses and attaches directly to the medial epicondyle and medial surface of the coronoid process of the ulna. This structure was found in 4 of 20 specimens in one study, and it is important to recognize and to release with anterior transposition of the ulnar nerve to prevent kinking. \(^{(6)}\)

In the forearm, the ulnar nerve extends motor branches to the FCU and the FDP of the ring and small fingers. The ulnar nerve may extend as many as 4 branches to the FCU, ranging from 4 cm above to 10 cm below the medial epicondyle. Proximal dissection of the first motor branch to the FCU from the
ulnar nerve may be performed up to 6.7 cm proximal to the medial epicondyle, facilitating anterior transposition of the nerve.

An aberrant muscle, the anconeus epitrochlearis, has been found in 3-28% of cadaver elbows and in as many as 9% of patients undergoing surgery for cubital tunnel syndrome. This muscle arises from the medial humeral condyle and inserts on the olecranon, crossing superficially to the ulnar nerve, where it may cause compression.

The arcade of Struthers must be differentiated from the ligament of Struthers, which is found in 1% of the population and extends from a supracondylar bony or cartilaginous spur to the medial epicondyle. This supracondylar spur can be found on the anteromedial aspect of the humerus, 5 cm proximal to the medial epicondyle, and it can often be seen on radiographs. The ligament of Struthers may occasionally cause neurovascular compression. This compression generally involves the median nerve or the brachial artery; however, the ulnar nerve can also be compressed by this structure.

Posterior branches of the medial antebrachial cutaneous nerves cross the ulnar nerve anywhere from 6 cm proximal to 4 cm distal to the medial epicondyle. These branches are often cut when making the skin incision for a cubital tunnel release, creating an area of dysesthesia or resulting in potential neuroma formation.

Extrinsic blood supply to the ulnar nerve is segmental and involves 3 vessels. These include the superior ulnar collateral artery, the inferior ulnar collateral artery, and the posterior ulnar recurrent artery. Typically, the inferior ulnar collateral artery (and often the posterior ulnar recurrent artery) is sacrificed with anterior transposition. At the level of the medial epicondyle, the inferior ulnar collateral artery is the sole blood supply to the ulnar nerve. In an anatomic study, no identifiable anastomosis was found between the superior ulnar collateral artery and the posterior ulnar recurrent arteries in 20 of 22 arms. Instead, communication between the 2 arteries occurred through proximal and distal extensions of the inferior ulnar collateral artery.
Intrinsically, the blood supply is composed of an interconnecting network of vessels that run along the fascicular branches and along each fascicle of the ulnar nerve itself. The surface microcirculation of the ulnar nerve has been shown to have an anastomotic stepladder arrangement. The inferior ulnar collateral artery is consistently found 5 mm deep to the leading edge of the medial intermuscular septum on the surface of the triceps.

Finally, acute ulnar neuropathy may have a sex predilection. This perioperative condition is found 3-8 times more frequently in men than in women. Contreras et al (1998) revealed that the medial aspect of the elbow has 2-19 times more fat content in women than in men. In men, the coronoid tubercle is approximately 1.5 times larger. He suggests that the coronoid process may be a potential site for ulnar nerve compression in men, and the increased subcutaneous fat around the ulnar nerve in women may provide a protective advantage against acute ulnar neuropathy.

The most common potential sites of compression of the ulnar nerve at the elbow are the medial intermuscular septum, the arcade of Struthers, the retrocondylar groove, the cubital tunnel, and the deep flexor-pronator aponeurosis. The 2 most common sites of compression are the retrocondylar groove and the true cubital tunnel, where the ulnar nerve passes between the 2 heads of the FCU.

**Frequency**

The elbow is the most common site of compression of the ulnar nerve. Cubital tunnel syndrome is the second most common compressive neuropathy (after carpal tunnel syndrome). Cubital tunnel syndrome affects men 3-8 times as often as women.

**Etiology**

Cubital tunnel syndrome may be caused by constricting fascial bands, subluxation of the ulnar nerve over the medial epicondyle, cubitus valgus, bony spurs, hypertrophied synovium, tumors, ganglia, or direct compression. Occupational activities may aggravate cubital tunnel syndrome secondary to repetitive elbow flexion and extension. Certain occupations are associated with the development of cubital tunnel syndrome;
however, a definite relationship with occupational activities is not well defined. (1,2)

**Pathophysiology**

Cubital tunnel syndrome is the common term used for ulnar compressive neuropathies at the elbow (from the mid arm to the mid forearm). Ulnar entrapment neuropathy develops because of the predisposing anatomy of the elbow region and the biomechanics of the ulnar nerve at the elbow; it is based on compressive, traction, and frictional forces, with the possible association of a nerve at risk. Systemic diseases such as diabetes, chronic alcoholism, renal failure, and malnutrition may predispose the patient to compressive neuropathy (i.e., a nerve at risk). Ultimately, the cumulative effect on the nerve is to cause a region of ischemia and inflammation resulting in ulnar nerve dysfunction or cubital tunnel syndrome. (3,4)

Compression of the ulnar nerve at the elbow may be idiopathic, but often there is a component of extrinsic compression aiding in the entrapment. Moreover, the compression of the nerve may be dynamic or static. The dynamic anatomy and biomechanics of the cubital tunnel dramatically affect the ulnar nerve, resulting in relative regional ischemia to the nerve. Dynamic impingement tends to occur early in the clinical course of the disease (i.e., position-dependent, intermittent). Although initially reversible, fixed structural changes occur over time, leading to static compression. Static compression also can develop from structural abnormalities of osseous architecture or space occupying lesions (Table 1).

With flexion of the elbow, the aponeurosis covering the cubital tunnel stretches, changing the cross-sectional geometry of the cubital tunnel from smooth and round to flattened and triangular. This both decreases the volume of the tunnel by 55%, and significantly increases the intraneural pressure, therefore putting the nerve at risk of ischemia. Intraneural pressure can be increased up to 600% with shoulder abduction, elbow flexion, and wrist extension. Moreover, contraction of the FCU muscle may increase the pressure
on the ulnar nerve. Normally, the ulnar nerve at the cubital tunnel is known to elongate 4.7 mm during elbow flexion. Should the nerve be tethered by perineural fibrosis (e.g., postoperative, post trauma), it can no longer elongate and may experience up to doubled intraneural pressures. (7)

The ulnar nerve has five common sites of compression in the elbow region (Fig. 1). The most common sites are at the level of the elbow. As the nerve passes by the medial epicondyle (or epicondylar groove) and into the cubital tunnel (passing deep to the aponeurosis linking the two heads of the FCU), it is at its greatest risk of compression. These are the primary sites of idiopathic disease. The other three tend to be sites of secondary or iatrogenic compression (often because of inadequate release). The most proximal site is the arcade of Struthers, a fascial band 8 cm proximal to the medial epicondyle, extending from the medial head of the triceps to the medial intermuscular septum. (Note: This is different from the ligament of Struthers, which extends from a supracondylar process to the medial humeral epicondyde and is involved primarily with proximal median nerve compression. (7,8)

Table 1. Causes Of Cubital Tunnel Syndrome

<table>
<thead>
<tr>
<th>Category</th>
<th>Causes</th>
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<tbody>
<tr>
<td>Idiopathic</td>
<td>Acute compression or direct injury</td>
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<tr>
<td></td>
<td>Entrapment in distal humeral fracture or elbow dislocation</td>
</tr>
<tr>
<td></td>
<td>Heterotopic ossification-post traumatic, secondary to burns, secondary to head injury</td>
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<tr>
<td></td>
<td>Aberrant or abnormal musculature</td>
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<tr>
<td></td>
<td>Anconeus epitrochlearis reverse flexor carpi ulnaris, triceps brachii (snapping)</td>
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<tr>
<td></td>
<td>Cumulative trauma disorders-keyboard operator, baseball pitcher</td>
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<tr>
<td></td>
<td>Arthritides</td>
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<tr>
<td></td>
<td>Osteoarthritis-secondary to osteophytes, loose bodies, synovial cysts</td>
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<tr>
<td></td>
<td>Inflammatory arthritis-synovitis</td>
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<tr>
<td></td>
<td>Synovial chondromatosis</td>
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<tr>
<td></td>
<td>Vascular bands--often branches of ulnar artery</td>
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<tr>
<td></td>
<td>Iatrogenic-postanesthetic palsy</td>
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<tr>
<td></td>
<td>Space-occupying lesions of the cubital tunnel and epicondyle region--e.g., lipoma</td>
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Although Struthers described the deep fascia of the arm, Spinner coined the term arcade when he noted that it appeared as a distinct entity in revision ulnar nerve decompression (following
anterior transposition). This structure is often indistinguishable from the deep fascia at the primary surgery, however. The strong, taut intermuscular septum, normally not a structure to entrap the nerve, runs anterior to the nerve, inserting throughout the length of the medial epicondyle. Following anterior transposition, the intermuscular septum acts secondarily as a point of local entrapment if its distal 3 cm is not excised. Finally, the last site is at the deep flexorpronator aponeurosis. Although uncommon as a primary cause, it is often a secondary compression site following transposition.

There is increasing evidence that cumulative or repetitive trauma work disorders are a cause of cubital tunnel syndrome. Keyboard operators, for instance, appear to be at increased risk. It is postulated that poor seating is a significant ergonomic factor; the keyboard placed too high and too close to the operator causes shoulder flexion, elbow flexion, wrist extension, and, therefore, traction on the ulnar nerve.

Figure 1. Five potential levels (boldface type) of compression of the ulnar nerve in the region of the elbow. (From Amadic, PC: Anatomical basis for a technique of ulnar nerve transposition. Surg Radiol Anat 8:158, 1986; by permission of the Mayo Foundation.)

When considering the painful upper extremity, reflex sympathetic dystrophy (RSD) often comes to mind. Although often reported as a posttraumatic or postoperative condition, it can occur without insult. In a series of 35 patients with RSD, 30 were noted to have peripheral nerve entrapment (single or multiple). Grundberg and Reagan also noted that, in their study of 93 cases of RSD, patients did not respond to standard treatment. Further investigation of those demonstrated carpal tunnel syndrome in and cubital tunnel syndrome in 5. Surgical decompression in those 27 patients led to complete resolution of their symptoms. RSD therefore may be a
pertinent painful manifestation of upper-limb peripheral nerve compression syndromes such as cubital tunnel syndrome.\(^{(3,6)}\)

**Clinical Presentation**

Patients with cubital tunnel syndrome tend to present with a pain, often aching or lancinating, primarily in the region of the elbow. It may radiate proximally or distally. The most frequent complaint, however, is of paresthesias in the ring and small fingers, initially intermittent and position-related, often waking them at night. Objective sensory loss usually occurs later, with progression of the disease (Table 2). A clue to the location of proximal ulnar nerve entrapment is the presence of dorsal sensory loss. There is usually no sensory loss along the medial forearm because it is supplied by the medial antebrachial cutaneous nerve, a branch of the medial cord of the brachial plexus. Most importantly, examining for Tinel's sign helps localize the entrapment.\(^{(8)}\)

Figure 2. Percussion test (Tinel sign): Tapping over the ulnar nerve at the elbow causes a reproduction of symptoms.

Patients often note they have "weak or clumsy hands," often dropping objects or being unable to open jars. Muscle weakness or atrophy often occurs and, in many patients, in the absence of any objective sensory change. The fascicles to the intrinsic ulnarly supplied hand muscles are more susceptible because of their superficial topographic location within the ulnar nerve at the cubital tunnel (FCU and FDP often are spared). Muscle weakness may be demonstrated by clawing of the hand, Wartenberg's sign or Froment's sign although these indications are not helpful in localizing the lesion. 23,41 Because of the common anomalous connections between the median and ulnar nerves (17% incidence
of Martin-Gruber anastomosis) that primarily affect the intrinsic innervation, however, weakness from significant cubital tunnel syndrome may be absent.

Histologically, severe demyelination of the nerve may occur in ulnar neuropathy. Demyelination may be located in the bulbous swelling just proximal to the entry of the nerve into the cubital tunnel.

McGowan (1950) established the following classification system:

1. Grade I - Mild lesions with paresthesias in the ulnar nerve distribution and a feeling of clumsiness in the affected hand; no wasting or weakness of the intrinsic muscles
2. Grade II - Intermediate lesions with weak interossei and muscle wasting
3. Grade III - Severe lesions with paralysis of the interossei and a marked weakness of the hand

Diagnosis

The elbow flexion test, a provocative test, is analogous to Phalen's test for carpal tunnel syndrome. Patients are positioned with their arms at their side, and elbows flexed approximately 120 degrees. The patient maintains that position for 3 minutes in an attempt to reproduce their symptoms (by compression or tension on the nerve). Rayan found this test positive in 10% of the control group and therefore questioned its usefulness. Novak and Mackinnon recently demonstrated that elbow flexion combined with pressure over the ulnar nerve for 30 seconds was the most sensitive and specific provocative test for cubital tunnel syndrome. (6,7)

Routine radiographs are often desirable to establish the presence of abnormal skeletal anatomy, malalignment, or compressive osseous structures. On specialized plain radiographs of the elbow, approximately 20% to 29% of patients with cubital tunnel syndrome had abnormalities, compared with 6% of controls.

Imaging Studies

Radiography

- Obtain a cubital tunnel projection radiograph with a history of trauma or
arthritis to exclude medial trochlear lip osteophytes.
• If a supracondylar process on the medial aspect of the humerus is suspected, obtain an elbow radiograph 5 cm proximal to the medial epicondyle.
• Obtain a chest radiograph if the patient has a history of smoking and symptoms in the ulnar nerve distribution to exclude a Pancoast tumor in the apical lung.

Magnetic resonance imaging (MRI)
• MRI is both sensitive and specific in the diagnosis of ulnar nerve entrapment at the elbow. It may be useful if the patient has previously undergone an anterior transposition of the ulnar nerve. On MRI, increased signal intensity is better than enlargement of the nerve for detecting ulnar nerve entrapment. A disadvantage of MRI in diagnosing cubital tunnel syndrome is its expense.
• Britz et al (1996) examined the use of MRI in diagnosing cubital tunnel syndrome using a short tau inversion recovery sequence. They studied 31 elbows with documented ulnar nerve entrapment and found increased signal intensity in the ulnar nerve in 97% of their cases and enlargement of the ulnar nerve in 75%.

High-resolution ultrasonography
• High-resolution ultrasonography has been used to evaluate the morphologic changes in the ulnar nerve at the cubital tunnel in ulnar nerve neuropathy.
• Using high–resolution ultrasonography, Chiou et al (1998) found that the mean value of the area of the ulnar nerve at the level of the medial epicondyle in symptomatic patients was significantly larger than that of the control group and that of the unaffected, contralateral side. Their P value was less than 0.001. Their conclusions were that if the area of the ulnar nerve was greater than 0.075 cm², at the level of the medial epicondyle, the patient probably had cubital tunnel syndrome.
Electromyography

- An electromyograph (EMG) is not essential when the diagnosis of cubital tunnel syndrome is obvious on clinical examination, as a false test result can be misleading; however, it is important to perform an EMG when the diagnosis of cubital tunnel syndrome is unclear or to determine the efficacy of conservative treatment.

- EMG findings are considered positive for cubital tunnel syndrome when the motor conduction velocity across the elbow is less than 50 m/s or when the difference between the motor conduction velocity across the elbow and that below the elbow is greater than 10 m/s. During the test, it is important to stimulate the nerve over 2-cm intervals to precisely localize the area of entrapment. Compression of the ulnar nerve is probably at the level of the retrocondylar groove when the point of maximum conduction delay and drop in amplitude of the compound muscle action potential is 2 cm distal to the medial epicondyle. Unfortunately, false-positive results are obtained in 15% of cases.\(^{(5,7)}\)

<table>
<thead>
<tr>
<th>Table 2. Staging of Ulnar Nerve Compression</th>
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<tr>
<td><strong>Mild</strong></td>
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<tr>
<td>Sensory, Intermittent paresthesias; vibratory perception increased</td>
</tr>
<tr>
<td>Motor, Subjective weakness, clumsiness, or loss of coordination</td>
</tr>
<tr>
<td>Tests, Elbow flexion test or Tinel's sign may be positive</td>
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</tbody>
</table>

| **Moderate**                               |
| Sensory, Intermittent paresthesias; vibratory perception normal or decreased |
| Motor, Measurable weakness in pinch or grip strength |
| Tests, Elbow flexion test or Tinel's sign is positive; finger crossing may be abnormal |

| **Severe**                                 |
| Sensory, Persistent paresthesias; vibratory perception decreased; abnormal two-point discrimination (static >6 mm, moving >4 mm) |
| Motor : Measurable weakness in pinch and grip plus muscle atrophy |
| Tests : Positive elbow flexion test or positive Tinel's sign may be present; finger crossing usually abnormal |
Electrophysiologic Studies

Electrophysiologic investigations are very helpful in the diagnosis of cubital tunnel syndrome, particularly in a difficult diagnostic problem, such as the patient with multiple complaints or diffuse upper-extremity pain. In some circumstances, however, the studies may prove to be normal in a clinically diagnostic case of cubital tunnel syndrome (often mild and intermittent). One review noted nerve conduction study abnormalities ranged from 23% to 93% in classic cases of cubital tunnel syndrome. Moreover, the severity of the clinical manifestations does not always correlate with the objective electrodiagnostic changes. Determination of exact location of entrapment may be difficult and, more importantly, the accuracy of these studies is user-dependent. The most commonly described abnormality is a decrease of 10 meters/second in conduction velocity over the region of the elbow. Raynor et al. recently found that surface-recorded sensory and mixed nerve studies are more sensitive than motor studies in the diagnosis of cubital tunnel syndrome, particularly in cases with subtle clinical involvement. (7,8)

Many do not believe that nerve conduction studies are necessary if the diagnosis of cubital tunnel syndrome is obvious. In fact, Mackinnon and Dellon stated: "It is clear that there is no reason to deny surgery to a patient with a normal preoperative electrical study, or to require all patients to have such a study if the history and physical examination of the patient are themselves consistent with ulnar nerve compression at the elbow." Ideally, however, most upper-extremity surgeons believe that an electrodiagnostic abnormality should be present to establish a diagnosis of cubital tunnel syndrome. Moreover, some have stated that a normal electrophysiologic study is a contraindication to surgery.

Electrodiagnostic studies postoperatively do not have a strong correlation with the recovery the patient experiences. There is a high rate of velocity and latency recovery following surgery, but the nerve conduction recovery does not always correlate with the functional recovery, particularly the distal motor recovery.
Differential Diagnosis of Cubital Tunnel Syndrome

The diagnosis of cubital tunnel syndrome is based on the clinical symptoms and signs, along with the objective electrophysiologic findings. Reaching the correct diagnosis in a timely fashion is difficult but plays an important role in outcomes. Chan noted that, if treated within 1 year of onset of symptoms, 88% of patients with cubital tunnel had a good result, compared with only 67% showing improvement in those treated longer than 1 year after onset. A delay in diagnosis therefore puts the patient at increased risk of chronic neuritis and pain caused by the prolonged and increasing injury.\(^{(2,4)}\)

Awareness of the differential diagnosis is important to be aware of to arrive at the correct diagnosis in a timely manner. Compression of the ulnar nerve at the cubital tunnel is most often confused with other sites of ulnar nerve (or its fibers) compression, proximal or distal to the elbow.

1. Cervical disc disease-in particular, that of the C8 root (C7-T1 level) often causes paresthesias in the small and ring fingers, sometimes with associated weakness of the intrinsic muscles of the hand. It often has associated painful, limited cervical motion, however.

2. Thoracic outlet syndrome is a compression of the medial structures of the brachial plexus, primarily the medial cord, as they exit the thoracic outlet. It often mimics cubital tunnel syndrome (pain, paresthesias, and weakness). The clinical diagnosis can be made or at least suspected with provocative testing ("hands up" test 90 degree shoulder abduction, external rotation while making a fist, often replicates the symptoms). Electrodiagnostic studies are often not reliable.

3. Compression of the ulnar nerve at Guyon's canal, at the level of the wrist, is often very similar in presentation. The ulnar nerve may have sensory, motor, or combined dysfunction, depending on the level of compression in the canal.
The discriminating clinical findings are a lack of sensory changes on the dorsum of the hand and more distal pain (variable).

Uncommon sites of compression may occur proximally, such as by a Pancoast tumor. So called double crush syndromes may also occur, whereby the nerve is compressed or irritated at two sites, with the effect being additive. Examples include thoracic outlet syndrome plus ulnar nerve compression at the elbow and compression of the cubital tunnel along with Guyon's canal. Indeed, we have observed a number of cases in which clinical or electrodiagnostic examination suggested pathology at both the cubital tunnel and Guyon's canal. Our practice has been to decompress both sites (with good results) when we were unable to identify a single predominant site preoperatively.\(^{(1,5)}\)

Errors or delay in the diagnosis of cubital tunnel syndrome may have a profound impact on the outcome. With continued neural dysfunction caused by any unusual repetitive stress, load, or anatomical abnormality in the region, chronic compression may lead to irreversible neural inflammation and fibrosis. The longer the injury to the nerve continues without treatment, the higher the likelihood the nerve will not recover completely. In fact, Gabel and Amadio noted the time from onset of symptoms to index treatment in their group of failed decompressions averaged 35 months (range 8-180 months). A delay in diagnosis therefore puts the patient at increased risk of chronic neuritis and pain because of the accumulating injury. Systemic- Diabetes, renal disease, multiple myeloma, amyloidosis, chronic alcoholism, malnutrition, leprosy, others

**Treatment of Cubital Tunnel Syndrome**

Progress in the management of ulnar nerve syndromes has been affected by: (1) delayed diagnosis and (2) treatment that is often based on personal bias rather than scientific results. The principles of treatment of ulnar nerve compression and chronic arm pain are:

1. Identification of the presenting signs and symptoms
2. Reaching the correct diagnosis and ruling out other common differential diagnoses
3. Treating the patient appropriately to optimize recovery of neural function and lower the risk of postoperative complications (neuroma, neuritis, inadequate release)

Nonoperative Treatment

As discussed, idiopathic ulnar neuropathy often initially manifests as intermittent or mild symptoms. At the early, mild stage, the process may be reversible if treated prior to the onset of chronic neural changes. The goals of conservative treatment are to reduce the inflammatory state of the perineural tissues, to enhance vascular perfusion of the nerve, and, indirectly, to restore normal axonal transport. The results are quite encouraging for those with mild symptoms. Dimond and Lister reported on 73 patients with cubital tunnel syndrome, treated with long arm splints. Dellon et al noted that, with more progressive clinical and electrophysiologic findings, it is more likely that the patient will require surgery. Specifically, 89% of those with only mild, intermittent disease were successfully treated nonoperatively, whereas only 38% of those with moderate disease (persistent paresthesias, muscle weakness, abnormal two-point discrimination \(< 10 \text{ mm})\) were successfully managed conservatively. Moreover, if the neuropathy was posttraumatic in nature, there was an increased chance of failure with conservative treatment.\(^{(1)}\)

To date, no guidelines have been widely agreed upon as to whom should be treated conservatively. Urbaniak believes patients with the following traits should undergo a trial of conservative treatment:

1. Early symptoms, intermittent episodes
2. Mild paresthesias without significant pain
3. Minimal physical findings (slight numbness), with normal motor examination

It is well recognized that those with severe findings of weakness, decreased two-point discrimination, and electromyographic evidence of denervation potentials should undergo operative exploration without a trial conservative treatment.
The principles of conservative treatment are: (1) avoiding aggravating factors (i.e., pressure on the elbow, repetitive flexion/extension), (2) avoiding full flexion (particularly at night), and (3) optimizing the environment (nutrition, decrease inflammation). Effective measures include the avoidance of pressure with elbow pads, splints (particularly at night) to prevent elbow flexion, avoidance of repetitive movements, and ergonomic workplace modification. The use of nonsteroidal anti-inflammatory drugs, local corticosteroids, and vitamin B6 may be beneficial. Patients should be followed at 1 to 3 month intervals until improvement is noted and maintained. If no improvement occurs or if the symptoms progress, repeat electrodiagnostic studies are warranted, particularly in noncompliant patients. (3,5)

Operative Treatment

If the symptoms are moderate or severe, or if the patient has failed nonoperative treatment, surgical decompression is the next treatment option. Most authors agree that the patient should have an electrodiagnostic abnormality to be considered an operative candidate.

Local procedures to decompress the ulnar nerve aim to restore a favorable environment for the nerve but maintain its normal anatomic position, whereas transposition procedures reposition the ulnar nerve into an uninolved environment. Understanding the effects of surgery on the nerve is critical to deciding which procedure to undertake. Ogata et al have shown that the segmental blood supply of the ulnar nerve is significantly reduced for 72 hours following transposition, whereas neither of the local procedures alter its vascularity. Conversely, if there is a disease process in the region of the cubital tunnel, a transposition procedure removes the nerve from the pathology. Urbaniak and Gabel make the following recommendations about primary procedures:

1. If decompressed in situ or by medial epicondylectomy, there should be no evidence of abnormalities within the cubital tunnel (i.e., no structural changes, inflammatory arthritis, or prior fractures)
2. If the nerve is transposed, the surgeon should take care to ensure no new foci of compression are created. (1)

Local Procedures

Local decompression of the ulnar nerve can be achieved in situ by either simple decompression or by medial epicondylectomy. In situ decompression involves the release of the deep fascia overlying the nerve in the epicondylar groove and of the FCU aponeurosis. In one review, 90% of those with mild symptoms had relief with any surgical procedure, but local decompression was rarely successful for moderate disease. Bednar recommends that this procedure be limited to those with compression isolated to the FCU aponeurosis. Ferlic recommends decompression alone only when (1) symptoms are mild or intermittent, (2) there is no subluxation or instability of the ulnar nerve (anterior to the epicondyle), (3) there is absence of pain, (4) osseous architecture of the elbow is normal, and (5) the findings at surgery are consistent with compression in the cubital tunnel. In situ decompression is therefore recommended for those with a short history, normal architecture of the cubital tunnel and osseous structures, and in whom the ulnar nerve is compressed by the FCU arcade (Osborne's ligament). (4,5)

The most common complication of in situ decompression is a failure to relieve symptoms. Anterior subluxation is a problem that tends to become a complication only if it is not detected at surgery (and treated with anterior transposition).

Medial epicondylectomy has been advocated by many for treatment of all stages (mild to severe) of ulnar nerve compression at the elbow. Dellon's review,13 however, noted good results in 90% of those with mild disease but in only 50% of those with moderate disease. Moreover, the procedure had the highest rate of recurrence of all procedures reviewed. Heithoff et all found that extensive or complete epicondylectomy was associated with improved results but controversy surrounds the possibility of medial collateral ligament (MCL) instability by excessive resection of the epicondyle (and MCL origin). With only a single report of instability following epicondylectomy, this concern is
reasonable but remains theoretical. O'Driscoll et al all reported the precise anatomy of the origin of the MCL and described an oblique osteotomy of the epicondyle to preserve its origin (Fig. 2A, B). We believe this technique provides excellent decompression of the nerve, at the same time preserving stability. Other possible complications include failure to fully decompress the nerve and inadequate resection, with persistent symptoms. (8)

We believe that local decompression with or without epicondylectomy should include neurolysis proximal (to include the arcade of Struthers) and distal into the FCU (aponeurosis and flexor pronator aponeurosis). We have had good success employing an extensive local decompression combined with medial epicondylectomy. Care must be taken to preserve the origin of the MCL with an oblique osteotomy of the epicondyle, to repair the flexor-pronator origin, and to cover the exposed bone with fascia and muscle to prevent nerve adherence to bone.

Figure 3. A, Medial part of the distal humerus removed by orthogonal osteotomies. On right 2 mm thick coronal slice through the widest part of the AMCL origin and the tip of the epicondyle. (Mean values are shown.) B, As the AMCL origin is anterior and inferior on the medial epicondyle, an osteotomy in a plane between the sagittal and coronal planes would permit removal of more bone while minimizing violation of the AMCL origin. (From O'Driscoll, et al: Origin of the medial ulnar collateral ligament. J Hand Surg. 17(A): 165-166, 1992; with permission.)

Transposition Procedures

Anterior transposition procedures move the ulnar nerve anterior to the axis of elbow motion and thereby theoretically decrease the traction and compressive forces upon the nerve. There are three types of transposition-subcutaneous, submuscular, and intramuscular. Subcutaneous transposition moves the nerve into the subcutaneous plane and holds it there with a fascial sling.
Submuscular transposition moves the nerve deep to the flexor pronator mass. Intramuscular transposition places the nerve in a channel within the flexor pronator mass without damaging the muscle origin.\(^5\)

Gabel and Amadio have observed that "anterior transposition can remove the nerve from the environment of the cubital tunnel and medial epicondyle, but the surgeon may overlook or even create other levels of mechanical impingement." Does the decreased blood flow caused by anterior transposition predispose the nerve to fibrosis? The results of anterior subcutaneous transposition were 90% good for mild disease, 70% good for moderate disease, and 50% good for severe disease. The results of submuscular and intramuscular transfer were equally good for mild and moderate disease, but the intramuscular transpositions had the worst results of any treatment of severe disease. The risk of scar formation and perineural fibrosis appears to be equal with both intramuscular and submuscular transposition, according to an in vivo study by Dellon et al.\(^7\)

**Internal Neurolysis**

The question has arisen as to whether decompression should include internal neurolysis. The recommendation for internal neurolysis is based on anecdotal results, with no studies demonstrating its efficacy in ulnar neuropathy. Moreover, internal neurolysis may incite prolonged pain along the ulnar nerve.

External neurolysis tends to cause minimal trauma to the nerve whereas internal neurolysis has been shown to be detrimental in both clinical and experimental studies. It disrupted the segmental as well as the longitudinal blood supply over the neurolysed segment. Gabel and Amadic, reported that two of six patients who underwent internal neurolysis at the index procedure experienced permanent loss of motor/sensory function upon repeat decompression. Very extensive external neurolysis may also be harmful and it should be limited to the extent needed to decompress the nerve.\(^{1,3,4}\)
Complications of Treatment of Cubital Tunnel Syndrome

Complications of Nonoperative Management

1. Poor outcome of treatment of moderate or severe compression
2. Poor outcome in the noncompliant patient
3. Failure to recognize progression of symptoms

Complications of Operative Treatment

Complications of operative management are primarily:

1. Injury to the medial antebrachial cutaneous nerve
2. Failure to completely decompress the nerve
3. Formation of new points of constriction

Injury to the medial antebrachial cutaneous nerve (MACN) is the most common complication of ulnar nerve surgery. In fact, in one study, 7 of 14 failed decompressions had painful neuromata of the MACN. Understanding the anatomy of the MACN is the key to preventing this painful complication. Masear et al46 noted at least one branch of the MACN (from the posterior division) crossing the region of the classic cubital tunnel incision in 40% of cadavers. Most have a branch at the level of the medial humeral epicondyle. These branches, one to three in number, can lie from 6 cm proximal to 6 cm distal to the humeral epicondyle. If transected, the manifestations of injury to the MACN are hypesthesia, painful scar "neuroma pain," or hyperalgesia. Hypesthesia is usually of little concern. The painful "neuromatous" scar and hyperalgesic nerve are often extremely symptomatic complications. The pain, localized to the elbow and the scar, is often aggravated by movement or touching the region. It may overshadow a successful restoration of ulnar nerve function. The diagnosis is usually by MACN block and treatment is initially nonoperative, with a 6-month course of desensitization. Surgery is reserved for those who fail therapy and have a well-localized, tender scar. (1,2,4)

Surgical treatment is varied. If injury to the MACN is noted at the time of initial surgery, then the nerve may be either primarily repaired 46 or resected
and buried in muscle. Re-exploration for a neuroma usually involves resecting the neuroma and burying the nerve end deep into muscle, away from the scar tissue. Awareness, recognition, and prevention are the keys to successful treatment.

**Failed Treatment of Cubital Tunnel Syndrome**

"The patient's best opportunity for full, permanent relief of symptoms is at the first operation. Undoubtedly, no matter how diligent the surgeon, there will always be patients who fail their initial management procedure. Although Dellon noted that approximately 20% of all patients treated had fair or poor results (prior to 1987), very little is reported on the failure and management of failed ulnar nerve decompression.

Typically, treatment failures are attributable to "Pain." Rogers et al all noted that the primary complaint leading to repeat surgery in all of their patients was unremitting pain. Gabel and Amadio, found that of pain and sensory and motor dysfunction, on average, pain was the most severe finding in their 30 patients failing initial treatment.\(^{(3,7)}\)

Unfortunately, very little is reported in the literature on why these patients have painful outcomes following their initial surgery. Gabel and Amadio\(^{,25}\) reported in their series that, in the surgeon's opinion, on average, 2.2 levels were found to be compressed (from the arcade of Struthers to the flexor pronator aponeurosis) at revision neurolysis, whereas, on average, only one level was involved at the initial surgery. The failure to adequately release the nerve or the creation of new levels of entrapment by transposing over a short distance at the initial decompression certainly contribute to poor and typically painful outcomes. Although 20 of 30 patients for revision neurolysis did not experience improvement following their index procedure, 10 patients initially had complete resolution of symptoms, only to relapse after approximately 5 months. This latter group, hypothetically, may have developed fibrosis around and, possibly, within the nerve. At repeat decompression, they noted tremendous fibrosis at the level of the epicondyle (both medially and anteriorly) in 80% of their patients.\(^{(5)}\)
Our experience is similar with respect to re-explorations of ulnar nerves. The primary surgeon may have never looked for a second site of involvement (i.e., double crush syndrome with involvement usually at Guyon’s canal) or did not release extensively enough, especially into the FCU. We have seen secondary compression by the arcade of Struthers in a few instances. Although failures may have sensory or motor deficits, perhaps worse than preoperatively, it is usually pain that brings the patient back for further treatment. \(^{(3,4,8)}\)

When we find a clear site(s) of entrapment at the primary operation, the outcome is usually very good. Pain and sensory recovery are consistent and even motor deficits recover a significant percentage. If, at the primary surgery, however, the nerve is not found to be particularly compressed, but rather is inflamed (indicative of intrinsic neuritis or neuropathy), the results are often poor or, at least, recovery is slow to occur.

The indications for repeat ulnar nerve decompression at the elbow have not been well established. Broudy and colleagues believed significant pain or progressive motor or sensory loss after surgery, unresponsive to conservative measures, warranted repeat decompression. One must be diligent, however, to ensure another cause is not the source of complaints. In particular, the double crush syndrome, proximal or distal ulnar nerve entrapment, and MACN injury may have similar complaints. Repeat electromyography is warranted in all cases. \(^{(3)}\)

The best method of treating a failed ulnar nerve decompression remains a matter of discussion. Rogers et al all used submuscular transposition in all cases, whereas Kleinmann advocates intramuscular transposition as his procedure of choice. Dellon noted that most repeat decompressions are done using submuscular transposition to correct failures of other procedures. In one of the few reports on the treatment of failed ulnar nerve decompression, Urbaniak and Gabel recommend that the key elements of revision surgery involve (1) re-exploration and decompression of each level of the ulnar nerve from the midarm to the midforearm (to locate
missed and second degree or iatrogenic entrapment). And (2) at reoperation, placing the nerve in a healthy environment, preferably void of scar tissue (i.e., if initially decompressed in situ, then transpose anteriorly to healthy tissue. If subcutaneously transposed, then transpose intra- or submuscularly). (1)

Repeat decompression of the ulnar nerve achieves its objective in many cases. Broudy et al all reported all of their patients had a diminution of pain and all were improved clinically. Rogers and associates 65 noted that repeat procedures were highly successful for relief of pain (all 14) and paresthesias, but recovery of motor function and return of sensibility were variable and unpredictable. Gabel and Amadio state that revision ulnar nerve decompression gave the greatest benefit in relief of pain, moreso than sensory and motor improvement. In fact 25 of 30 were improved, 3 were unchanged, and only 2 were worse following repeat surgery. But motor and sensory recovery were not improved as reliably. They identified the following factors associated with a poor outcome:

1. Previous submuscular transposition
2. Age greater than 50 years at index procedure
3. Denervation on electromyography

Amadio and Gabel suggest that medial epicondylectomy is ineffective as a revision procedure (because of fibrosis), but we have had success in a few cases. We agree, however, that the presence of significant inflammation or fibrosis of the nerve or its bed mandates transposition to a healthy bed. In situations of multiple reoperations or post trauma, the local tissue bed may be too compromised even for sub- or intramuscular transposition. In these cases, there are few other good alternatives. We, as others (Masear VR, personal communication, 1987) have had some success in a small number of cases using autogenous saphenous vein wrapping. Allograft and xenograft vein wrapping are also reported in small series or anecdotally but they may incite increased fibrosis (Masear VR, personal communication). Few local flaps are available to cover a fibrotic or ischemic nerve bed. In some cases, an island fasciocutaneous forearm flap may be
rotated on the ulnar artery pedicle. In the case of extensive or proximal damage post trauma, we have had success rotating or freely transferring a latissimus dorsi flap to provide healthy soft-tissue coverage. Recent reports note improvement of chronic neuritic pain with free or pedicle tissue transfers to circumferentially wrap the fibrotic or ischemic nerve. (2)

Authors’ Preferred Surgical Approach. When planning a decompression of the ulnar nerve at the elbow, the method of treatment is decided upon with consideration given to the patient, the degree of compression, the architecture of the elbow, the soft-tissue bed, and, often most importantly, the findings at the time of surgery. With this in mind, our preferred surgical approach is:

1. External neurolysis alone for very localized compression and a stable nerve
2. External neurolysis with medial epicondylectomy for more extensive compression, subluxation of the nerve, or excessive nerve tension when the elbow is flexed
3. Anterior transposition for an environment compromised by fibrosis of the tissue bed, joint pathology, architectural abnormality of the elbow region, or extensive entrapment or fibrosis of the nerve. This is usually reserved for revision or posttraumatic procedures. (1)

Summary

Cubital tunnel syndrome is the secondmost-common compressive neuropathy. With the increasing prevalence of entrapment neuropathies, the presentation of ulnar nerve compression with a painful upper extremity appears to be more common. Although our knowledge and understanding of this disease are increasing, the principles of management remain constant. We are obliged to reach a timely and appropriate diagnosis to minimize the extent of neurologic injury and institute an appropriate treatment regimen to preserve and restore normal neural function. Although there are many ways to reach these goals, the avoidance of complications is paramount to achieve a reliable and pain-free outcome. Preventing injury to the medial
antebrachial cutaneous nerve, complete release of all sites of compression, and avoidance of creating new compressive sites are the keys to this end.

References