Cubital Tunnel Syndrome

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Cubital tunnel syndrome is the second most common compression neuropathy in the upper extremity. Patients complain of numbness in the ring and small fingers, as well as hand weakness. Advanced disease is complicated by irreversible muscle atrophy and hand contractures. Ulnar nerve decompression can help to alleviate symptoms and prevent more advanced stages of dysfunction. Many surgical treatments exist for the treatment of cubital tunnel syndrome. *In situ* decompression, transposition of the ulnar nerve into the subcutaneous, intramuscular, or submuscular plane, or medial epicondylectomy have all been shown to be affective in the treatment of this disease process. Comparative studies have shown some short–term advantages to one or another technique, but overall results between the treatments have essentially been equivocal. The choice of surgical treatment is based on multiple factors, and a single surgical approach cannot be applied to all clinical situations. Through careful consideration of the potential sites of nerve compression and the etiologies for these local irritations, the appropriate surgical technique can be selected and a good outcome anticipated in most patients. *(J Hand Surg 2010;35A:153–163. © 2010 Published by Elsevier Inc. on behalf of the American Society for Surgery of the Hand.)*

Key words Cubital tunnel syndrome, nerve compression, nerve transposition, ulnar nerve.

ENTRAPMENT OF THE ulnar nerve is the second most common compression neuropathy in the upper extremity after carpal tunnel syndrome.[1–4] Although the ulnar nerve may be compressed at multiple points along its course, the most common location is at the elbow. A thorough knowledge of the anatomy of the ulnar nerve can assist with diagnosis and guide treatment. Patients often present with paresthesias in the ulnar nerve distribution and weakness or atrophy of the intrinsic musculature of the hand. Pain is not the predominant feature early in the course of ulnar nerve compression. Various surgical techniques for decompression of the ulnar nerve have been described in the literature, and a definitive gold standard does not exist. A thorough understanding of the pathology of cubital tunnel syndrome will help guide treatment and lead to successful outcomes.

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ANATOMY

The ulnar nerve is composed of branches from the C8 and T1 nerve roots. These 2 roots combine to form the lower cord of the brachial plexus and transition into the medial cord. The ulnar nerve is the terminal branch of the medial cord. The course of the ulnar nerve continues between the medial head of the triceps brachii and the brachialis muscles (Fig. 1). The nerve is posteromedial to the brachial artery and just posterior to the intermuscular septum. The arcade of Struthers is a band of fascia that connects the medial head of the triceps with the intermuscular septum of the arm. This fascial band crosses the ulnar nerve approximately 8 cm proximal to the medial epicondyle. The ulnar nerve then becomes more superficial and enters the ulnar sulcus approximately 3.5 cm proximal to the medial epicondyle. The nerve courses posterior to the medial epicondyle and medial to the olecranon. The nerve then enters the cubital tunnel. The roof of the cubital tunnel is defined by the arcuate ligament of Osbourne, or Osbourne's ligament. Osbourne's ligament is a thickened transverse band between the humeral and ulnar head of the flexor

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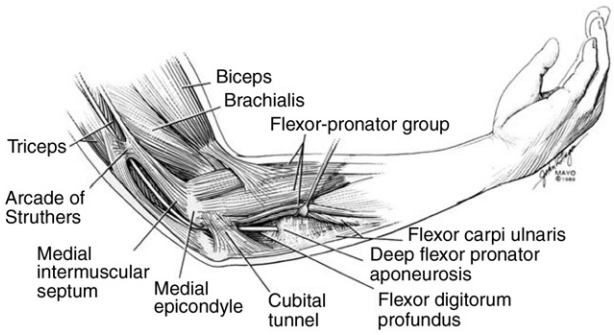


FIGURE 1: The course of the ulnar nerve across the elbow. Note the 5 common sites of compression of the ulnar nerve: the arcade of Struthers, the medial intermuscular septum, the medial epicondyle, the cubital tunnel, and the deep flexor pronator aponeurosis. From Elhassan B, Steinman SP. Entrapment neuropathy of the ulnar nerve. J Am Acad Orthop Surg 2007;15:672–681. Copyrighted and used with permission of Mayo Foundation for Medical Education and Research, all rights reserved.

carpi ulnaris (FCU). The floor of the cubital tunnel consists of the medial collateral ligament of the elbow, the elbow joint capsule, and the olecranon. After passing through the cubital tunnel, the ulnar nerve courses deep into the forearm between the ulnar and humeral heads of the FCU.

Potential ulnar nerve entrapment can occur at 5 sites around the elbow: the arcade of Struthers, the medial intermuscular septum, the medial epicondyle, the cubital tunnel, and the deep flexor pronator aponeurosis. The most common site of entrapment is the cubital tunnel.[5] Recent anatomic studies have shown variability in the level of previously unidentified fibrous bands. These finds suggest that the recurrence of symptoms after decompression could be due to inadequate release of these structures. It is suggested that the proximal and distal ends of the cubital tunnel be explored carefully to prevent incomplete release.[6]

The anatomic relationship of the posterior branch of the medial antebrachial cutaneous nerve and its proximity to the cubital tunnel is also an important anatomic consideration in ulnar nerve surgery. The proximity of the medial antebrachial cutaneous nerve to the medial epicondyle makes it particularly prone to injury during ulnar nerve decompression. One of the most common causes of pain after ulnar nerve surgery is secondary to injury of the posterior branch of the medial antebrachial cutaneous nerve.[7] In a recent anatomic study, medial antebrachial cutaneous nerve branches were found to cross the surgical incision of cubital tunnel release at, or proximal to, the medial epicondyle 61% of the time. These branches crossed the incision at an average distance of 1.8 cm proximal to the medial epicondyle in 97 randomly selected patients having cubital tunnel decompression. Medial antebrachial cutaneous nerves were noted to cross distal to the medial epicondyle 100% of the time at an average distal distance of 3.1 cm from the epicondyle. Knowledge of these structures should be taken into account when approaching the ulnar nerve to help prevent iatrogenic injury to the cutaneous nerves.[8]

DIAGNOSIS

A thorough history should be obtained from all patients with suspected upper-extremity nerve entrapment. The patient should be asked about comorbidities such as diabetes, thyroid disease, hemophilia, and general peripheral neuropathies. One should inquire about onset of symptoms, grip or pinch weakness, subjective findings of numbness, and whether the numbness is dorsal or volar. It should be noted if there are any positional or temporal patterns appreciated. The symptoms should be questioned with regard to aggravating activities and positions that can alleviate the symptoms.

Maybe the most important historical piece of information gathered is whether or not the symptoms are constant. With intermittent symptoms, there are times or positions when there is normal nerve function. The symptoms develop as a result of transient, local nerve ischemia. Interventions to prevent this transient ischemia (bracing, surgery, etc.) are likely to restore normal function. Once patients complain of a constant low level of numbness, with intermittent exacerbations of their symptoms related to position or activity, the results of intervention are less predictable.

Numbness and paresthesias are the predominant presenting features early in the disease; pain is less common.[9] Paresthesia and numbness of the ulnar digits is common, but patients will frequently have difficulty localizing their symptoms. In patients with complaints of medial elbow pain, careful history and examination is required to rule out other etiologies. Pain is typically localized to the cubital tunnel region, but symptoms can also be localized to the medial epicondyle and into the forearm.

A complaint of hand and grip problems secondary to intrinsic muscle weakness and atrophy is often seen at presentation. Patients with less pronounced muscle dysfunction may complain of a vague feeling of clumsiness. Specific questions focusing on precision hand pinch activities, which are controlled by the intrinsics, should be asked. Difficulty buttoning buttons, opening bottles, difficulty with typing, and generalized fatigue

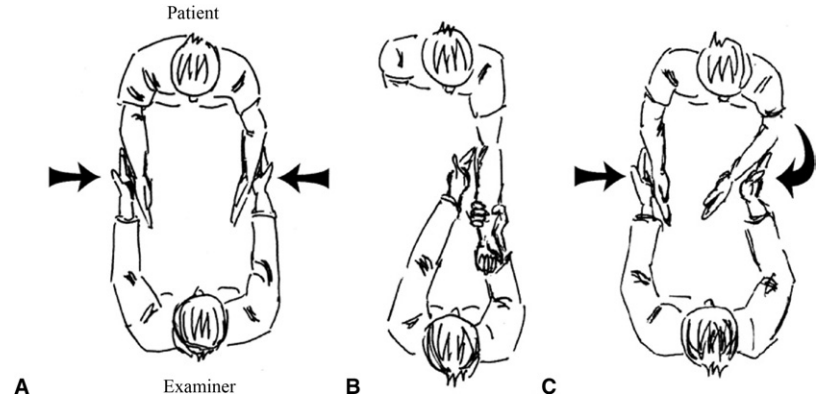


FIGURE 2: The scratch collapse test. The patient faces the examiner with arms adducted, elbows flexed, and hands outstretched with the wrists at neutral. **A** The patient resists bilateral shoulder adduction and internal rotation as the examiner applies these forces to the forearm. **B** The examiner "scratches" or swipes the fingertips over the course of the compressed ulnar nerve. **C** The force is reapplied to the forearm. A positive result occurs when the patient has a temporary loss of external rotation resistance tone (as seen in the diagram). From Cheng CJ, Mackinnon-Patterson B, Beck JL, Mackinnon SE. Scratch collapse test for evaluation of carpal and cubital tunnel syndromes. J Hand Surg 2008;33A:1518–1524. Copyright 2008, with permission from Elsevier.

are often described by the patient. Patients with cubital tunnel are 4 times more likely to present with atrophy than patients with carpal tunnel.[10] The extent of ulnar nerve dysfunction has been divided into 3 categories by McGowan[11] and modified by Dellon.[12] Mild nerve dysfunction implies intermittent paresthesias and subjective weakness. Moderate dysfunction presents with intermittent paresthesias and measurable weakness. Severe dysfunction is characterized by persistent paresthesias and measurable weakness.

Provocative tests for cubital tunnel have been described in the literature. Two of the most frequently used tests are a Tinel test along the course of the ulnar nerve and the elbow flexion test. In addition, a pressure provocation test (where direct pressure is applied to the cubital tunnel for 60 seconds) and a combined elbow flexion-pressure test can be performed. A positive Tinel test is only 70% sensitive, whereas the elbow flexion test is 75% sensitive after 60 seconds. However, after 60 seconds, the pressure test is 89% sensitive, and the combined elbow flexion-pressure test is 98% sensitive. These examination findings can be used in combination to best diagnose cubital tunnel syndrome.[13]

Recently, the "scratch collapse" test has been described (Fig. 2). To perform the scratch collapse test, the examiner scratches the patient's skin lightly over the area of nerve compression while the patient performs resisted bilateral shoulder external rotation. A brief loss of muscle resistance will be elicited if the patient has allodynia due to the compression neuropathy. Sensitivity for the scratch collapse was 69% compared with 54% and 46% for Tinel test and elbow flexion-compression test, respectively. Tinel test, however, had the highest negative predictive value (98%) of all tests for cubital tunnel.[14]

A thorough elbow exam is needed to look for other sources of pain and to rule out other etiologies for the patient's symptoms. In the athlete, signs of elbow instability such as chronic valgus stress can lead to cubital tunnel syndrome symptoms. Signs of old trauma, such as a childhood supracondylar fracture, can lead to a tardy ulnar nerve palsy. The ulnar nerve should be inspected through a full range of elbow motion to make certain the nerve does not subluxate over the medial epicondyle. Medial elbow pain can be seen after elbow fractures that are treated without ulnar nerve transposition (olecranon fractures, distal humerus fractures, medial epicondylar fractures). Medial elbow swelling after these injuries and surgeries can lead to ulnar nerve compression with elbow flexion that prevents progression of postoperative rehabilitation. In these cases, ulnar nerve transposition may be required to facilitate rehabilitation.

After long-standing ulnar nerve palsy, intrinsic weakness develops. Paralysis of both lumbrical and interosseous muscles will result in hypertension of the proximal phalanx with flexion of the middle and distal phalanges: the intrinsic minus or claw hand also known as Duchenne's sign. Clawing limited to the ring and small fingers is the most frequent presentation. The index and long fingers are often spared by median nerve innervated lumbrical muscle. Masse's sign is described as a flattening of the dorsal transverse metacarpal arch, and the hand appears flattened. This finding is due to hypothenar muscle paralysis, which eliminates the normal flexion and supination of the fifth metacarpal. At-

rophy of the interosseous muscles is often most evident in the dorsal thumb web space, and finger abduction and adduction is lost. Interosseous weakness leads to Wartenberg's sign, which presents as ulnar deviation of the small finger and weakness of adduction of the small finger. Often, patients with Wartenberg's sign will complain of the small finger interfering when trying to place their hand in a pants pocket. As ulnar nerve palsy progresses, thumb adduction and metacarpophalangeal joint flexion is weakened. Key pinch strength may be diminished by as much as 80%. To compensate, patients often pinch by flexion of the distal phalanx of the thumb against an index finger that lapses into supination and is buttressed by the adjacent middle finger. This compensation is known as Froment's sign. Metacarpophalangeal instability develops causing a hyperextension deformity of this joint. This thumb posture deformity is known as Jeanne's sign.[15,16]

Radiographs should be obtained in all patients to evaluate for elbow arthritis, which may lead to osteophytic impingement on the cubital tunnel. Additionally, radiographs may show signs of instability, deformity from old trauma, or the presence of a supracondylar process (which can cause median nerve compression).

Electrodiagnostic testing is typically performed in patients with symptoms of ulnar nerve compression. Ulnar nerve motor conduction velocity across the elbow is considered positive for cubital tunnel syndrome if it is measured to be less then 50 m/s. Electrodiagnostic studies are useful to confirm the clinical diagnosis and can help to localize the site of compression. In addition to identifying other sites of compression, other diseases processes such as upper motor neuron disease or other peripheral neuropathies can be diagnosed. Recently, it has been suggested that electrodiagnostic testing is unnecessary to predict the surgical outcomes.[17] Despite this, the authors typically recommend electrodiagnostic testing for cubital tunnel syndrome. In patients with mild or moderate compression, electrodiagnostic testing can substantiate or refute the diagnosis. In patients with advanced findings, the testing can be used for prognosis and help predict the expectations for nerve and muscle recovery.

Diagnosis of cubital tunnel syndrome is made from a combination of clinical data and electrodiagnostic testing. However, in patients with clinical evidence of cubital tunnel syndrome, electromyography and nerve conduction velocities may have a false-negative rate in excess of 10%. False-negative electrodiagnostic tests may occur as few functional axons are required for a study to be interpreted as normal.[17] Therefore, reliable localization may necessitate use of several diagnostic approaches, including the recording of sensory nerve action potentials or mixed nerve potentials and determination of motor conduction velocity change across the elbow.[18] These advanced techniques of electrodiagnostics are particularly helpful in the patient without classic findings on history and physical examination or when other diagnoses are being considered.

High-resolution ultrasound is a relatively new technique in the evaluation of nerve entrapment syndromes.[19] Studies have shown that enlargement of the ulnar nerve is seen in cubital tunnel syndrome, and use of ultrasound in the diagnosis of ulnar neuropathy has been investigated.[20] A recent clinical study evaluating the diagnostic value of elbow ultrasound compared 14 patients with cubital tunnel syndrome based on symptoms, clinical examination, and nerve conductions velocities, with 60 normal elbows. The cross-sectional area of the ulnar nerve at the cubital tunnel was statistically significantly smaller in the control group, suggesting that ultrasound may provide a valuable adjunct to electrodiagnostic evaluation. However, more standardization of ultrasound techniques are required to determine the gold standard for image-based diagnosis of cubital tunnel.[21] It has been suggested that ultrasound's ability to visualize nerves may prove to be useful in cases of peripheral nerve trauma, tumors, or revision surgery.[22] The ability to see the course of the nerve, and to determine if its path has been negatively affected by these processes, may be of some clinical benefit.

NONSURGICAL TREATMENT

Mild cubital tunnel syndrome can often be treated without surgery. There is a tendency toward spontaneous recovery among patients with mild and/or intermittent symptoms if provocative causes can be avoided. Patients with constant symptoms or muscle atrophy usually require surgical treatment. The most commonly described methods of conservative treatment are activity modification, splints to obstruct maximum and repetitive flexion, and physical therapy (nerve mobilization techniques).[4] Svernlov et al. looked at conservative treatment of patients with mild or moderate cubital tunnel. One group was instructed to wear a splint at night for 3 months. The splint limited flexion to 45°. The second group was instructed in nerve gliding exercises as described by Bryon.[23] The third group received education regarding cubital tunnel syndrome and activity modification. In this study, 89.5% of patients improved at follow-up. This result suggests the majority of patients with mild or moderate cubital tunnel will benefit from conservative treatment. There were no

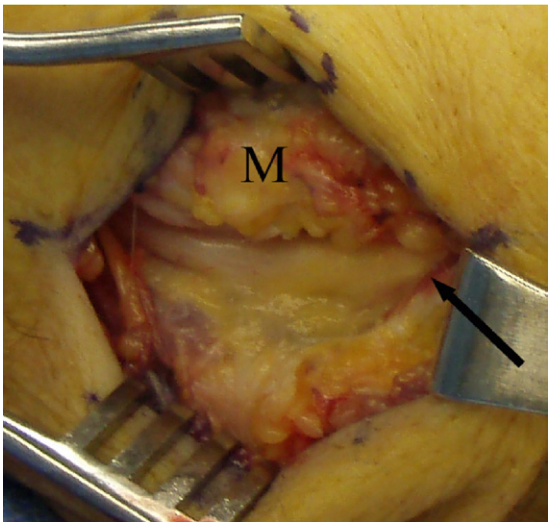


FIGURE 3: *In situ* release of the ulnar nerve. Note that the FCU has been released distally (black arrow). The ulnar nerve remains posterior to the medial epicondyle (M).

statistical differences between groups, suggesting that patient education may be sufficient treatment.[24]

SURGICAL TREATMENT

Surgical treatment of cubital tunnel syndrome is indicated with motor weakness or when conservative measures have failed.[24] There are multiple techniques currently recommended for treatment of cubital tunnel syndrome, and there is ongoing controversy as to which is the optimal surgical treatment of this nerve entrapment.[16] The most common surgical treatments include *in situ* decompression, subcutaneous transposition, intramuscular transposition, submuscular transposition, and medial epicondylectomy. More recently, endoscopic techniques of simple decompression have been described.

In situ decompression

In situ decompression has been proposed by various authors as a treatment for cubital tunnel syndrome.[25–29] For simple decompression, a 6- to 10-cm incision is made along the course of the ulnar nerve between the medial epicondyle and the olecranon. Care should be taken to avoid the branches of the medial antebrachial cutaneous nerve. Osbourne's ligament is released as is the FCU superficial and deep fascia (Fig. 3). The nerve is retained in its bed and not circumferentially dissected of the surrounding connective tissue.[16] Simple decompression has been shown to be successful in treating cubital tunnel syndrome. Prospective randomized studies have shown results of simple decompression to be equal to those of anterior transposition.[30–33] *In situ* decompression also appears to have a low failure rate. A recent study of 56 patients (69 extremities) who had *in situ* decompression of the ulnar nerve showed that 5 limbs (7%) had persistent symptoms postoperatively. These recurrent symptoms were relieved after anterior submuscular transposition. The data suggest that *in situ* decompression is a reliable treatment with a low failure rate, and anterior transposition can be used to treat those patients with recurrent symptoms.[34]

Subcutaneous anterior transposition

Subcutaneous anterior transposition of the ulnar nerve is another common surgical treatment for cubital tunnel syndrome. With elbow flexion the ulnar nerve is placed under tension with a concomitant decrease in the carpal tunnel volume. Both factors lead to a decrease in neural blood flow. The goal of ulnar nerve transposition is to move the nerve anterior to the elbow axis of flexion, decreasing the tension on the nerve. Concomitantly, removing the nerve from the tunnel eliminates the pressure produced from the decreased cubital tunnel volume. There are concerns, however, that dissecting the nerve from surrounding connective tissue compromises the blood supply to the nerve.[35] Care is taken to ensure no new sites of compression are created proximal or distal after anterior nerve transposition. A longer incision is required to perform anterior transposition compared with that for *in situ* decompression. The proximal nerve is identified as in simple *in situ* decompression. The intramuscular septum is removed so that it does not become a proximal site of compression after transposition (Fig. 4). The large venous plexus near the intermuscular septum must be coagulated prior to sectioning the septum to avoid a hematoma. The nerve is then released through the cubital tunnel and traced distally through the two heads of the FCU. A vessel loop is placed around the nerve to provide a gentle traction while the nerve is circumferentially dissected free from the surrounding connective tissue. The nerve is lifted from its bed and transposed anterior to the medial epicondyle. Care is taken to preserve the motor branches to the FCU and the flexor digitorum profundus. The ulnar nerve should be examined along its course for any remaining points of compression or severe angulations. A small sling can be created to prevent the nerve from returning to its anatomic position (Fig. 5). This can be done either with a sling of subcutaneous tissue that is sutured to the fascia over the medial epicondyle or with a sling of muscle fascia that is sutured to the subcutaneous tissue. In either case, no

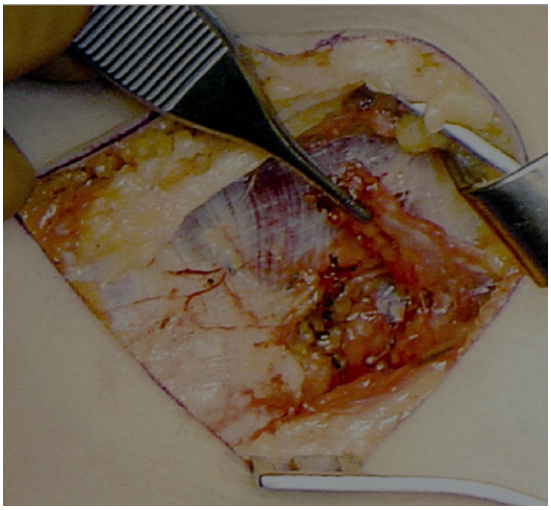


FIGURE 4: In cases where the ulnar nerve is to be transposed, the medial intermuscular septum should be resected (in forceps). This prevents tension on the nerve as it crosses from the posterior compartment of the arm to the anterior. Large vessels running through the septum should be coagulated or ligated prior to resecting the intermuscular septum. From Henry M. Modified intramuscular transposition of the ulnar nerve. J Hand Surg 2006;31A:1535–1542. Copyright 2006, with permission from Elsevier.

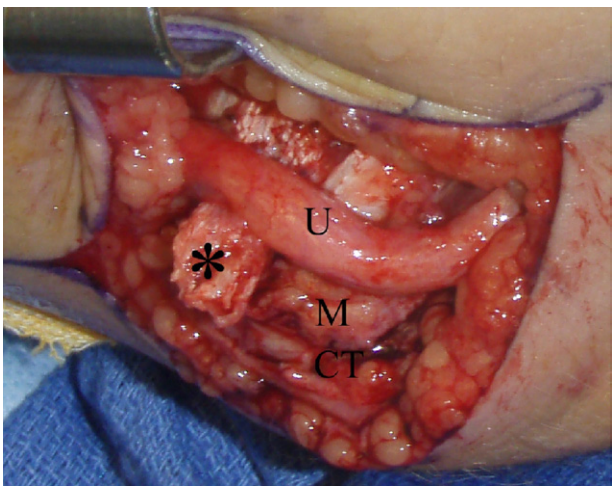


FIGURE 5: A patient had marked ulnar nerve symptoms that limited elbow flexion after fixation of a lateral condyle and capitellar fracture. The patient had anterior transposition of the ulnar nerve. The ulnar nerve (U) is removed from the cubital tunnel (CT) and transposed anterior to the medial epicondyle (M). A fascial sling (*) is sewn to the subcutaneous tissue to prevent the nerve from subluxating back into the cubital tunnel.

fascia is sewn over the nerve directly, avoiding any iatrogenic compression on the nerve from this sling.[16]

Naghan et al. reported on 66 patients diagnosed with clinically and electromyographically proven cubital tunnel syndrome. The patients were prospectively randomized into nerve decompression without transposition and subcutaneous anterior transposition of the nerve. Follow-up examinations at 3 and 9 months after surgery were for pain, motor and sensory deficits, and nerve conduction velocities. There were no statistical differences between outcomes of the 2 groups at either follow-up interval.[33]

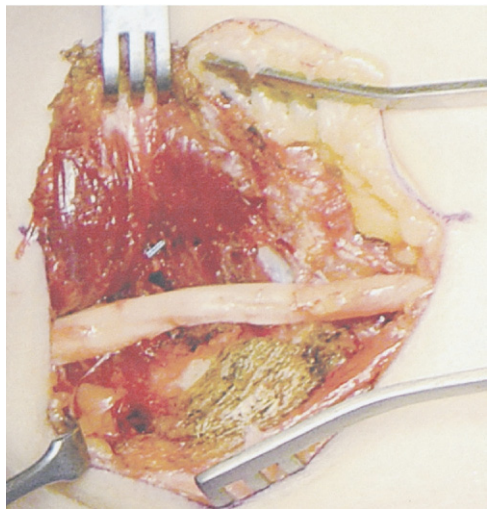


FIGURE 6: In an intramuscular transposition, the ulnar nerve is placed in a straight line. It is enveloped in muscle after the transposition. From Henry M. Modified intramuscular transposition of the ulnar nerve. J Hand Surg 2006;31A: 1535–1542. Copyright 2006, with permission from Elsevier.

Intramuscular transposition

Intramuscular transposition is another technique employed in combination with anterior transposition of the nerve. Proponents of the technique believe that this places the nerve in a straighter line across the elbow joint (Fig. 6). Opponents of the technique argue that it can cause scarring of the nerve, which serves as the bed for the transposed nerve. The procedure is similar to the subcutaneous transposition, however a groove is created in the flexor-pronator muscle mass to serve as a tract into which the nerve is transposed (Fig. 7).[16,36]

Submuscular transposition

After anterior transposition, some surgeons prefer to place the nerve complete beneath the flexor-pronator mass. The submuscular transposition requires the largest incision and most extensive dissection. The branches of the medial antebrachial cutaneous nerve are identified and protected. The ulnar nerve is identified and decompressed as in subcutaneous transposition. The flexor-pronator muscle mass is incised 1 to 2 cm

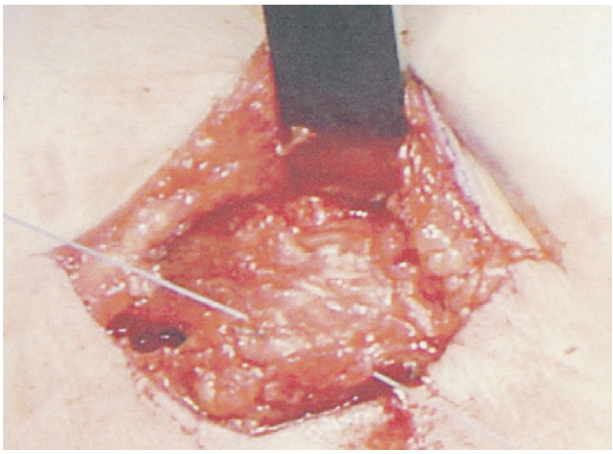


FIGURE 7: The superficial muscle is repaired to the medial epicondyle. Care needs to be taken that there is no compression of the nerve when the muscle is repaired. From Henry M. Modified intramuscular transposition of the ulnar nerve. J Hand Surg 2006;31A:1535–1542. Copyright 2008, with permission from Elsevier.

distal to the medial epicondyle in a step-cut fashion to allow for fractional lengthening of the muscle. Identification and protection of the ulnar collateral ligament and the median nerve is required. The ulnar nerve is transposed anteriorly and is placed adjacent and parallel to the median nerve. The reflected flexor-pronator muscle is repaired over the transposed ulnar nerve.[16] Submuscular anterior transposition has been compared with simple ulnar nerve decompression in a prospective randomized study. The patients were evaluated on subjective symptoms only, and there were no statistical differences between the 2 groups at follow-up.[37] A retrospective comparison of the clinical outcomes of submuscular and subcutaneous transposition has also been performed. There was no statistical difference between the subjective symptoms of the 2 groups postoperatively. Both groups had significant improvements in motor and sensory function after surgery, but there was no difference between the groups.[38] Two recent meta-analyses of the literature compared the clinical outcomes of simple decompression and anterior transposition (submuscular, intramuscular, and subcutaneous). Both found no statistical differences in reported outcomes between simple decompression of the ulnar nerve and anterior transposition of any type in patients with cubital tunnel syndrome.[39,40]

Medial epicondylectomy

Medial epicondylectomy was described by King for the treatment of ulnar nerve palsy.[41] Since that time, the surgery has been refined to decrease complications secondary to overzealous resection of the medial epicondyle, which can lead to instability.[42,43] In medial epicondylectomy, the nerve is decompressed as described for simple *in situ* decompression. The medial epicondyle is exposed subperiosteally, leaving the flexor/pronator origin in continuity with the periosteal sleeve. An osteotome is used to score the leading edge of the epicondyle. A plane is chosen between the sagittal and coronal planes of the humerus to avoid detachment of the anterior band of the ulnar collateral ligament. Care is taken not to enter the elbow joint. The flexor/pronator origin is reattached to the periosteal sleeve with absorbable sutures.[16] Treatment of cubital tunnel syndrome with partial medial epicondylectomy has been shown to relieve symptoms without elbow instability; however, 45% of patients had medial elbow pain at 6 month follow-up.[44] A study comparing minimal medial epicondylectomy with anterior subcutaneous transposition showed no statistical differences. In this retrospective study, however, patients were treated with either minimal medial epicondylectomy alone or in combination with anterior subcutaneous transposition. Obviously, the results from this study are affected by the fact that the transposition group also received a partial medial epicondylectomy.[45] Prospective, randomized trials comparing medial epicondylectomy to other surgical treatment options are needed to better evaluate medial epicondylectomy as a primary treatment for cubital tunnel.

Endoscopic decompression

Endoscopic decompression of the ulnar nerve at the elbow was first described in 1995 by Tsai et al.[46] Multiple variations of endoscopic techniques have been described since then. All techniques used a small 15-mm to 35-mm incision located over the ulnar nerve at the condylar groove. The variations rely on different techniques of retraction of subcutaneous tissues for visualization of the nerve. Use of tunneling forceps to elevate the subcutaneous tissues has been described. A space is made between the fascial covering of the nerve in the cubital tunnel and the subcutaneous adipose tissue. An illuminated septum and endoscope is placed in this space, and blunt-tipped dissecting scissors are used to release any proximal and distal fascial constrictions over the ulnar nerve. This surgery was performed in 76 nerves in 75 patients with idiopathic cubital tunnel syndrome. Sensory loss improved in 96% of patients, and grip strength measurements showed a significant improvement. Nerve conduction studies also showed an improvement after nerve decompression. Four patients developed superficial hematomas, which resolved over

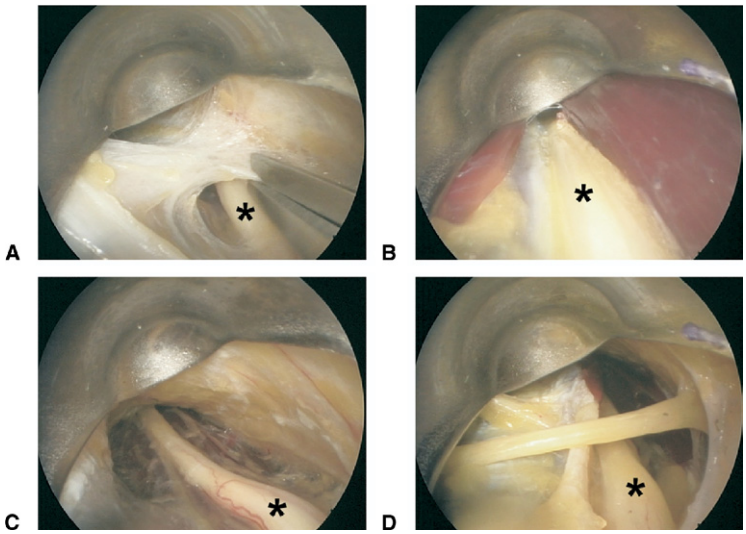


FIGURE 8: Endoscopic ulnar nerve release. **A** Releasing the ulnar nerve (*) by cutting the forearm fascia. **B** Ulnar nerve (*) between the heads of the FCU muscle. **C** Ulnar nerve (*) released proximal to the elbow. **D** Crossing branch of the medial antebrachial cutaneous nerve. From Ahčan U, Zorman P. Endoscopic decompression of the ulnar nerve at the elbow. J Hand Surg 2007;32A:1171–1176. Copyright 2007, with permission from Elsevier.

time. Nine patients developed decreased feeling in the medial antebrachial cutaneous nerves, which resolved by 3 months in 8 patients.[47] In another described technique, a custom guiding-dissecting tool is placed between the fascial and subcutaneous layers. A 4-mm standard 18-cm-long endoscope is inserted into the guiding tool. Long blunt-tipped scissors are then used to divide the flexor-pronator aponeurosis and release the cubital tunnel both distally and proximally to the level of the arcade of Struthers. This technique was used on 36 patients. Excellent outcomes were obtained in 21 (58%) patients and good outcomes in 12 (33%) patients. All patients demonstrated some degree of improvement, and 64% had normalization of electrodiagnostic studies. One patient suffered from a postoperative hematoma, which resolved. No patients reported hypoesthesia of the medial antebrachial cutaneous branches (Fig. 8).[48] A recent comparison between endoscopic techniques and *in situ* decompression demonstrated statistically significant less pain and greater satisfaction with the endoscopic technique.[49] Objective outcomes were not statistically different. More studies investigating the outcomes of endoscopic decompression are needed.

TREATMENT ALGORITHM

Choosing a surgical treatment has been as much a matter of preference as it has been based on evidence-based medicine. Most comparative studies demonstrate equivalent results, and no statistical difference in outcomes has been proved with any particular technique.[27,30–33,37–40,43,45,49] The authors have adopted an approach that the simplest surgical option that will address the pathology should be pursued. In most cases, simple decompression of the cubital tunnel is adequate. Whereas in the future the simplest technique may be an endoscopic release, the authors do not believe that a smaller incision equates with "simplest." Therefore, each treating surgeon must decide which technique is most reproducible in their hands, and for most surgeons the endoscopic technique is unfamiliar.

Although cubital tunnel symptoms can frequently be treated effectively with *in situ* decompression, certain situations will likely recommend a different surgical treatment. Nerve subluxation is an uncommon but not a rare entity. In cases where the nerve is hypermobile at the elbow, simple decompression will not effectively treat the underlying source of nerve irritation, which is its translation across the medial epicondyle. In these cases, some form of transposition is recommended. Another group of patients in whom a transposition is more likely to address the source of pathology is those patients with posttraumatic elbow stiffness whose flexion is limited by ulnar nerve symptoms. In these patients, decompression alone may alleviate symptoms. However, the associated scarring of surrounding elbow ligaments, capsule, and muscular tissues from the initial trauma may limit ulnar nerve mobility, and circumferential dissection of the nerve is likely to decrease the chance of recurrent symptoms in this group. In contrast,

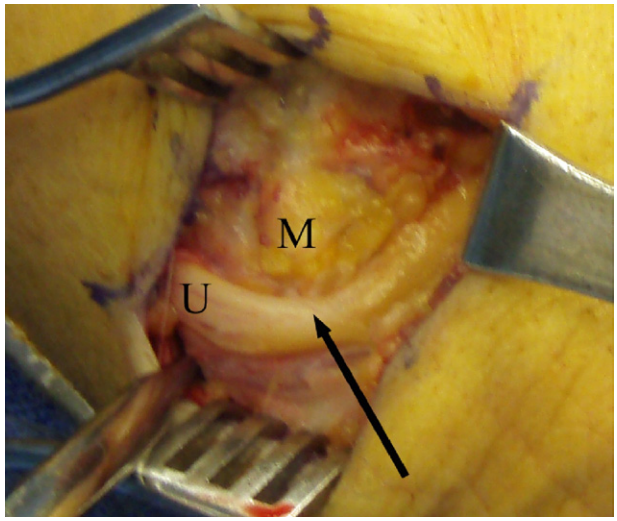


FIGURE 9: After *in situ* decompression of the ulnar nerve (U), the elbow is placed through a range of motion. The nerve begins to subluxate and is seen to ride up (arrow) higher than the medial epicondyle (M).

more extensive dissection may lead to nerve subluxation. Additionally, the lengthening of the nerve that occurs with elbow flexion may be greater if tissues around the elbow are scarred or swollen and the nerve must travel a greater distance. In this subgroup of patients, the authors prefer to transpose the ulnar nerve, thereby performing a more complete neurolysis and allowing nerve relaxation with elbow flexion. Similarly, in overhead-throwing athletes with valgus instability and in patients with a tardy ulnar nerve palsy, the authors believe that transposition is the preferred technique. In these cases, the stretch on the ulnar nerve with progressive valgus deformity will only be addressed with an anterior transposition.

After an *in situ* decompression, the nerve is assessed for signs of persistent compression or nerve subluxation (Fig. 9). The elbow is placed through a full range of motion, and if the nerve is found to subluxate, then transposition is necessary. In most patients, a subcutaneous transposition is likely adequate. In thin patients, a deeper transposition (intramuscular or submuscular) should be considered.

COMPLICATIONS

The posterior branch of the medial antebrachial cutaneous nerve is encountered in all surgical approaches to the ulnar nerve. Injury to the nerve can cause painful neuroma, hyperesthesia, hyperalgesia in the forearm, and a painful scar.[8] Subluxation may occur with simple decompression alone. Intraoperative assessment of the nerve's stability is necessary to avoid this complication. If it is appreciated postoperatively, transposition is required to eliminate the symptoms of a painful subluxating nerve. Recurrent symptoms after cubital tunnel surgery are usually the result of incomplete decompression or perineural scarring.[50] The treatment of recurrent disease requires complete assessment of each potential site of compression. This includes the arcade of Struthers, the medial intramuscular septum, the medial epicondyle, the cubital tunnel, Osbourne's ligament, and the aponeurosis of the flexor-pronator mass. Incomplete release of these structures can lead to recurrent or persistent symptoms. After transposition, attention should be focused on these areas as well as on the proximal and distal sites of transposition, where the nerve crosses from posterior to anterior and back again.[51] Surgical options for failed cubital tunnel syndrome include anterior submuscular transposition, anterior intramuscular transposition, and anterior subcutaneous transposition.[52,53] Medial epicondylectomy is another option; however, anterior transposition remains the preferred technique.[54] An adjunctive procedure for cubital tunnel revision is the addition of some form of soft tissue coverage. These procedures are designed to provide a more hospitable bed for the nerve after transposition to limit perineural scarring. Options include vein-wrapping, triceps muscle flap, and pedicle fat flap.[55,56]

Cubital tunnel syndrome is a common nerve compression with a variety of treatment options. Multiple surgical options exist that have been shown to alleviate symptoms. Selection of a surgical approach is based on the etiology of nerve compression, anatomic variations, and the surgeon's experience. Good results can be obtained with careful protection of the medial antebrachial cutaneous nerve and careful complete decompression of the nerve around the elbow, with or without transposition.

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