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Review Article

Modern Treatment of Cubital Tunnel Syndrome: Evidence and Controversy



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Cubital tunnel syndrome is the second most common peripheral mononeuropathy in the upper extremity. However, the diagnosis and treatment of cubital tunnel syndrome remains controversial without a standard algorithm. Although diagnosis can often be made from the patient's history and physical examination alone, electrodiagnostic studies, ultrasound, computed tomography (CT), and magnetic resonance image (MRI) can also be useful in diagnosing the disease and selecting the most appropriate treatment option. Treatment options include conservative nonoperative techniques as well as various surgical options, including *in situ* decompression with or without transposition, medial epicondylectomy, and nerve transfer in advanced disease. The purpose of this review is to summarize the most up-to-date literature regarding cubital tunnel syndrome and propose a treatment algorithm to provide clarity about the challenges of treating this complex patient population.

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Cubital tunnel syndrome (CuTS) is the second most common peripheral mononeuropathy in the upper extremity,¹ with an estimated prevalence of 1.8% in the US population.² Despite this relatively high occurrence, modern diagnosis and treatment of CuTS remains controversial. This is in part because of the heterogeneity of patient clinical presentation and disease severity across different age groups,³ as well as a lack of standardized diagnostic tools and treatment algorithm. This has led to significant variation in practice patterns both in the United States^{4,5} and abroad.⁶

Controversy regarding the diagnosis and treatment of CuTS is abundant: more than 500 articles on CuTS have been published in the past decade. This research has led to an improvement in our understanding of the disease and has spurred the development of several diagnostic and treatment options. However, the impact of these developments on the modern treatment of CuTS remains relatively unknown. Therefore, the purpose of this review is to discuss the diagnosis and treatment of CuTS, incorporate emerging

and recent evidence, and highlight controversial topics that confound current clinical practice.

Anatomy

Ulnar nerve compression about the elbow, commonly referred to as CuTS, is caused by compression, traction, or friction of the ulnar nerve as it courses behind the elbow. The nerve provides motor innervation to the forearm and hand as well as sensory innervation to the hand. Previously described points of compression (from proximal to distal to the elbow) include the medial intermuscular septum, arcade of Struthers, medial epicondyle, Osborne's ligament, flexor carpi ulnaris (FCU) fascia, and flexor-pronator aponeurosis (Fig. 1).¹ An anomalous anconeus epitrochlearis muscle is present in as much as 20% of the population and has also been implicated in some studies as a point of compression associated with CuTS (Fig. 2).⁷ Although most patients with anconeus epitrochlearis muscle do not have CuTS,⁸ faster and more reliable symptom improvement has been reported after surgical release than in those without the anomalous muscle.⁹

In addition to static sites of compression, previous studies have also shown dynamic compression of the ulnar nerve with elbow motion.^{10,11} In 1998, Gelberman et al¹⁰ found a 30% to 41% reduction

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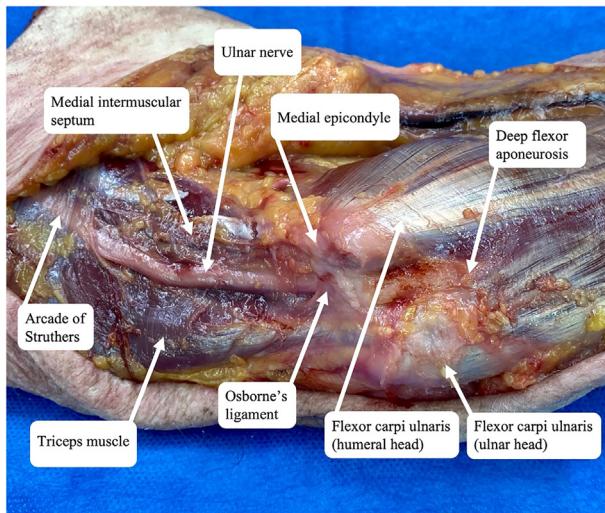


Figure 1. Points of ulnar nerve compression in CuTS.

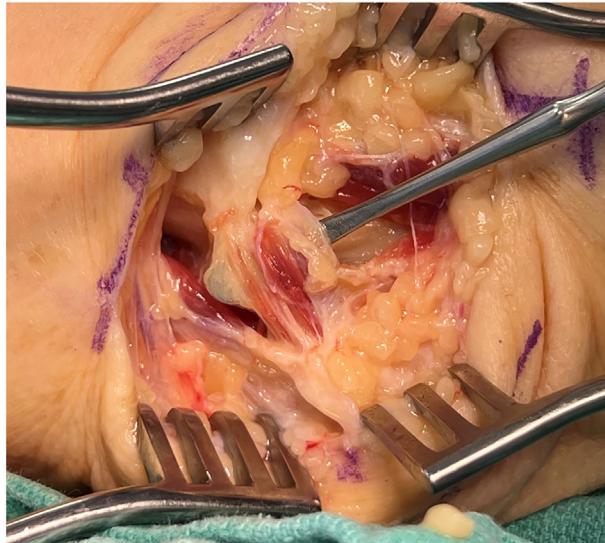


Figure 2. Intraoperative photograph of right elbow depicting an anomalous anconeus epitrochlearis (arrow) present in a patient with CuTS.

in cubital tunnel volume as the elbow was moved from full extension to 135° of flexion with a corresponding 7-fold increase in intraneuronal pressure. Furthermore, their study showed minimum intraneuronal pressures within the cubital tunnel at 30°–60° of flexion. This report established the basis of extension splinting for conservative treatment for CuTS (Fig. 3). Subsequent studies also demonstrated that an ulnar nerve excursion of 21.9 and 23.2 mm was required at the elbow and wrist for normal upper extremity motion, respectively. In addition, an ulnar nerve strain of 15% or greater can occur during elbow flexion and wrist extension and radial deviation.¹²

Ulnar nerve branching is highly variable in number and level.¹³ At the level of the medial epicondyle, 1–2 posterior articular branches are typically present, followed by 2–3 distal motor branches (Fig. 4).¹⁴ The most proximal motor branch typically arises 1.6 cm distal to the epicondyle and innervates the FCU alone, whereas a second motor branch typically arises 3.6 cm on average to the medial epicondyle and innervates the FCU, flexor digitorum profundus, or both.¹³ Intrafascicular dissection of these nerve



Figure 3. Elbow extension splinting.

branches has been shown to facilitate increased nerve excursion anteriorly during transposition.¹³ At the cubital tunnel, 3 main intrafascicular groups are present, which are divided into distinct patterns as they travel down the forearm to innervate the hand. The location of these groups within the nerve is often referred to as the “intraneuronal topography,” which may suggest as to why certain signs/symptoms prelude others. In the proximal forearm, the following intrafascicular groups can be identified: the dorsal cutaneous (ulnar), motor (central), and sensory (radial) nerves (Fig. 5).¹⁵ This topography is crucial when performing a supercharged nerve transfer during advanced surgery for CuTS.¹⁵

As the ulnar nerve enters the hand, it passes through another possible point of compression: Guyon’s canal. The boundaries of Guyon’s canal include the volar carpal ligament volar and the transverse carpal ligament dorsal. The pisiform, pisohamate ligament, and abductor digiti minimi form the border medially and the hook of the hamate laterally.¹⁶ The dorsal branch of the ulnar cutaneous nerve arises about 5 cm proximally to Guyon’s canal to provide sensation to the medial dorsal hand. This anatomy can be important in distinguishing between distal compression at Guyon’s canal and proximal compression in the case of CuTS.

Three main arteries contribute to the blood supply of the ulnar nerve at the elbow: the superior ulnar collateral artery, the inferior ulnar collateral artery, and the posterior ulnar recurrent artery.¹⁷ When possible, these branches should be preserved during decompression and/or transposition (Fig. 6). Previous studies have reported that the ulnar nerve can be transposed up to 2.5 cm anteriorly without significantly disrupting its vascular supply.¹⁷ Disruption of vascular supply has been shown to cause segmental nerve ischemia,¹⁸ which in animal models have led to decreased nerve conduction.¹⁹

Diagnosis

Patients with CuTS often delay seeking treatment and are more likely to present with advanced disease.²⁰ Specific risk factors associated with advanced disease at presentation include older patients, patients with higher body mass index, diabetes mellitus, and economic distress.²¹ White females in their fourth and fifth decade of life comprise the most common patient demographic associated with carpal tunnel syndrome.² The most common signs and symptoms of CuTS are intermittent numbness and tingling in the ulnar ring and small fingers. Symptoms can be exacerbated by activities requiring elbow flexion, such as talking on the phone.²² Additional signs/symptoms may include hand weakness, atrophy, diminished dexterity, and pain of the medial elbow, forearm, or wrist.^{1,23} Consideration of differential

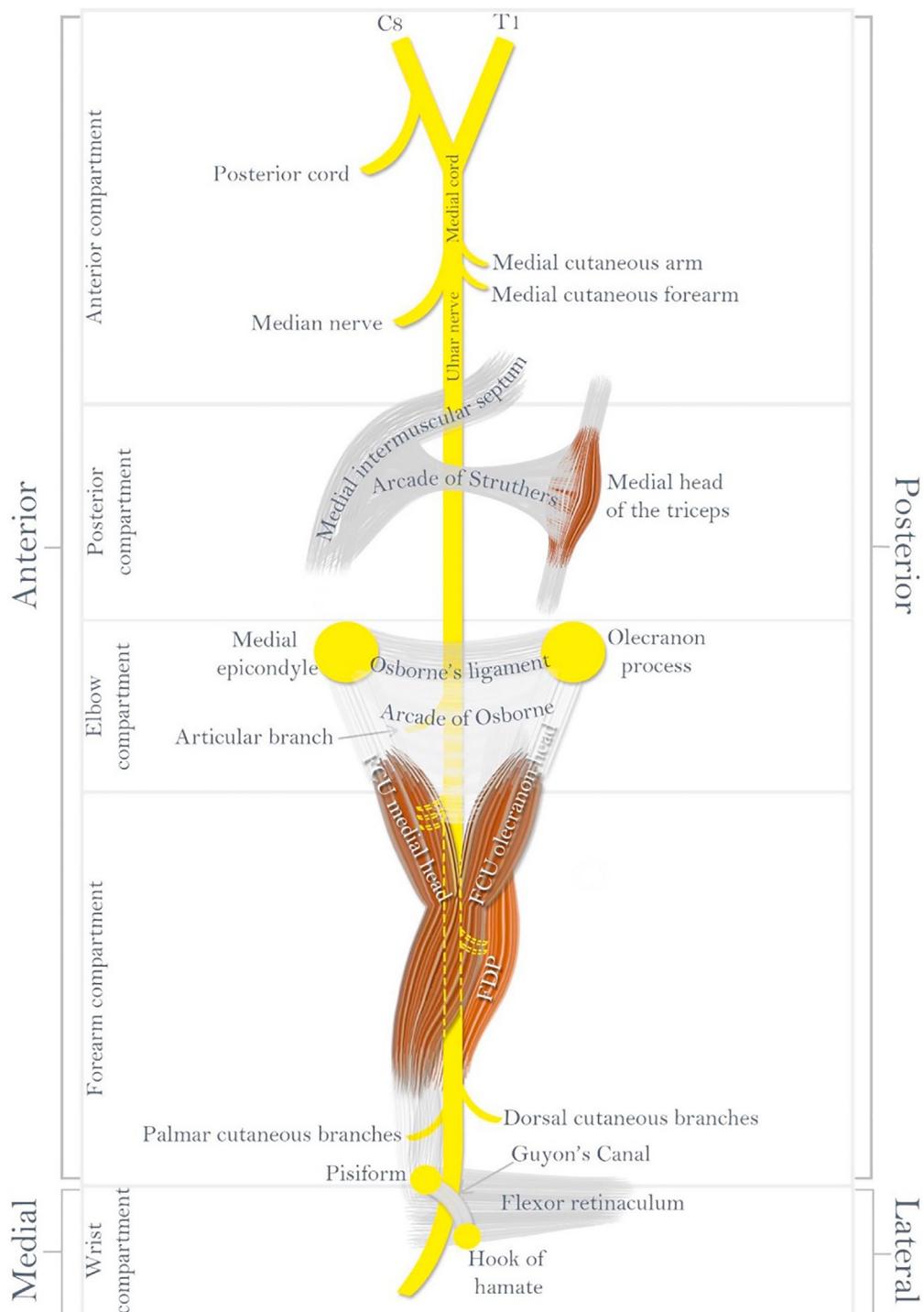


Figure 4. Ulnar nerve branching patterns at the elbow and wrist. Used with permission from Andrews et al.²²

diagnoses such as cervical radiculopathy, Pancoast tumor, thoracic outlet syndrome, medial epicondylitis, elbow arthritis, FCU tendinitis, ulnar tunnel syndrome, and hypothenar hammer syndrome must be assessed during the history and physical examination (Table 1).

Common physical examination findings associated with CuTS include impaired sensation in ulnar nerve distribution, muscular atrophy of first dorsal interosseous muscle, as well as Wartenberg, Froment, and Jeanne signs (Fig. 7).¹ Clawing of the fourth and fifth fingers is a result of weakened intrinsics (third and fourth

lumbricals) being overpowered by the flexor digitorum profundus tendons. This is considered a late finding owing to the axonal loss of the intrinsics of the hand.²⁴ Provocative maneuvers that may be positive in patients with CuTS include Tinel sign test with percussion at the retrocondylar groove, as well as elbow flexion and flexion compression tests (Fig. 8).¹ Novak et al²⁵ previously reported higher sensitivity of elbow flexion compression test (91%) compared with Tinel sign test (70%) and elbow flexion test (32%) at 30 seconds. More recently, the glenohumeral internal rotation test has been shown to demonstrate

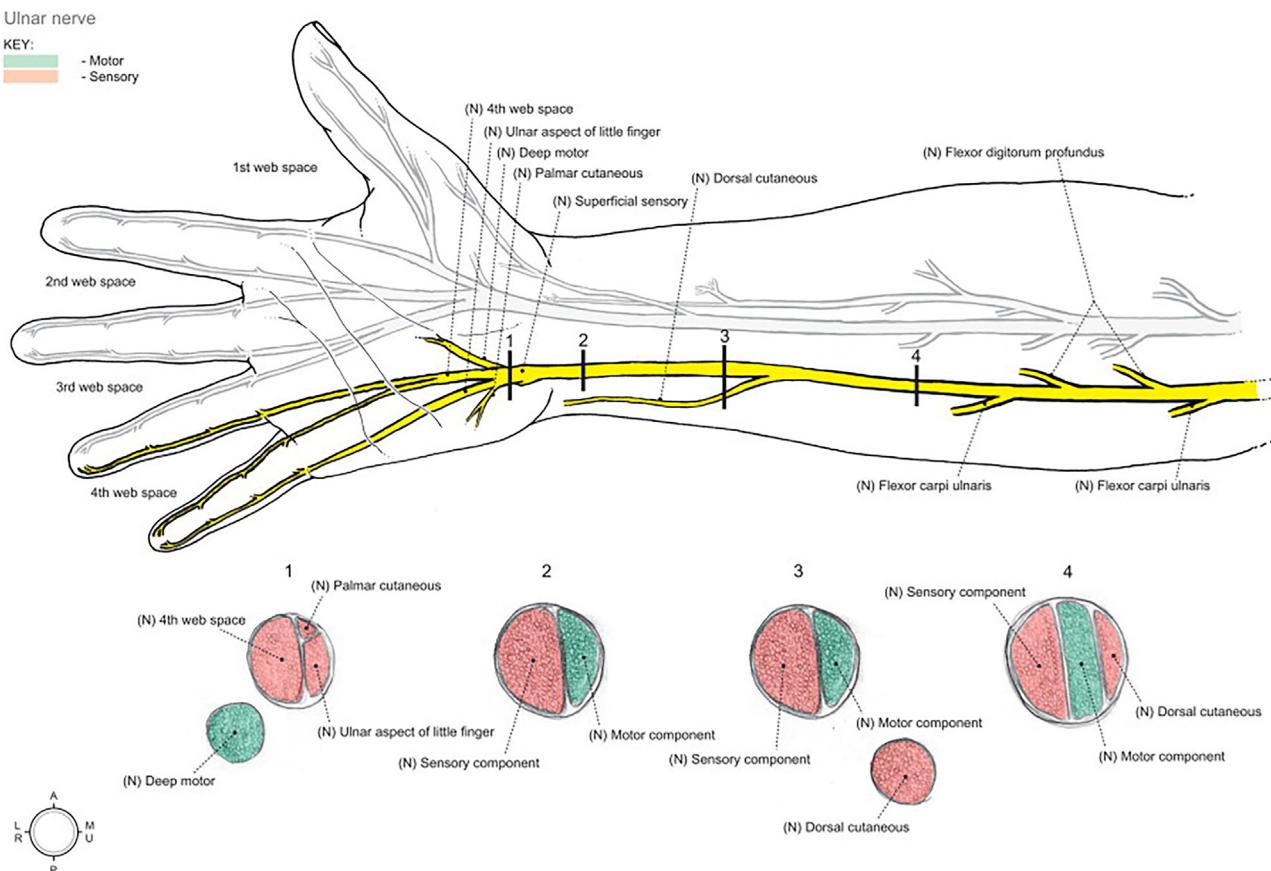


Figure 5. Topography of the ulnar nerve in the forearm and hand from Moore et al.¹²⁶ Used with permission from Elsevier.

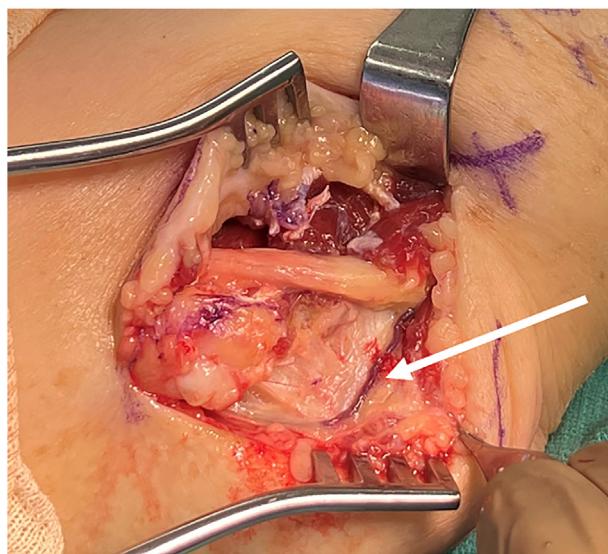


Figure 6. Intraoperative photograph of the right elbow depicting preservation of blood supply during anterior transposition of the nerve for treatment of CuTS. The arrow indicates the intact posterior recurrent ulnar artery.

positive findings in less than 5 seconds and is 87% sensitive and 98% specific.^{26,27} The scratch collapse test can also be performed and has been shown to have a sensitivity of 69% (Fig. 8).²⁸ However, recently, the sensitivity and interrater reliability of the scratch collapse test have been questioned.²⁹

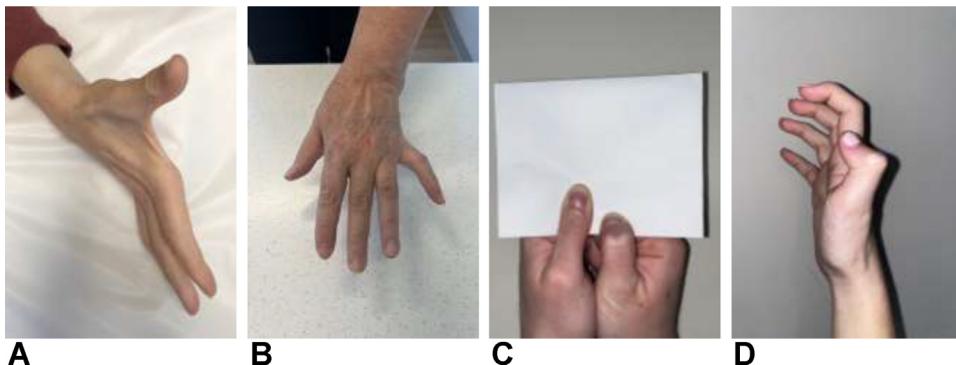
Nerve instability should be assessed on clinical examination with palpation of the ulnar nerve in the retrocondylar groove as the elbow is moved from extension to full flexion.³⁰ "Subluxation" is thought to occur when the nerve moves anteriorly out of the groove and perches on the medial epicondyle and "dislocate" when it fully translocates anterior to the medial epicondyle. However, definitions of degrees of ulnar nerve instability are not uniformly agreed upon.³⁰ In addition, hypermobility of the ulnar nerve has been found to be present and asymptomatic in one-third of the general population (Fig. 9).³¹ More recently, preoperative dynamic ultrasound (US) has been shown to more accurately predict the degree of ulnar nerve instability following *in situ* decompression compared with physical examination (88% vs 12%).³²

Electrodiagnostic studies (EDX) can aid in the diagnosis of CuTS as the disease progresses from dynamic ischemia to demyelination and finally axonal loss.²⁶ In the early stages of the disease, EDX can present as normal as the dynamic ischemia from intermittent nerve compression has not yet reduced perfusion enough to decrease nerve conduction velocity in the fastest-conducting nerve fibers.³³ Once demyelination has occurred, nerve conduction will slow to the point of ulnar nerve compression. According to the American Association of Neuromuscular and Electrodiagnostic Medicine, the greatest strength of evidence for the diagnosis of CuTS is a conduction velocity of <50 m/s across the elbow or when conduction velocity from the *above-elbow* to *below-elbow* segment is slowed to >10 m/s compared with the *below-elbow* to *wrist* segment.³⁴ However, the diagnostic accuracy of EDX in CuTS is highly variable and secondary to biological (ie, low elbow temperature) and technical factors.³⁵ Shubert et al³⁶ previously reported that the

Table 1

Differential Diagnosis of CuTS

Differential Diagnosis for CuTS	Special Considerations
Cervical radiculopathy	<ul style="list-style-type: none"> Dermatomal pattern of pain and sensory disturbance Positive Spurling test Pain and limitations with neck motion
Pancoast tumor	<ul style="list-style-type: none"> Rare lung tumor in upper lobe identified on X-ray Horner's syndrome History of smoking
Thoracic outlet syndrome	<ul style="list-style-type: none"> Adson, Roos, and Wright tests Symptoms with overhead activity Tinel sign over the coracoid process Change in pulse with overhead activity Pain without numbness, tingling, and weakness
Medial epicondylitis	<ul style="list-style-type: none"> Pain localized to flexor pronator origin Pain worse with resisted flexion and pronation Pain at rest with decreased range of motion Mechanical symptoms (loose bodies)
Elbow arthritis	<ul style="list-style-type: none"> Evidence of joint space narrowing and osteophyte formation on X-ray Pain over FCU throughout elbow and forearm can be aggravated with repetitive and resisted wrist flexion and ulnar deviation
FCU tendinitis	<ul style="list-style-type: none"> No numbness and tingling on dorsal hand (dorsal cutaneous nerve is spared)
Ulnar tunnel syndrome	<ul style="list-style-type: none"> Positive Tinel sign over Guyon's canal X-ray with carpal tunnel view can be used to rule out hook of hamate fracture in traumatic setting
Hypothenar hammer syndrome	<ul style="list-style-type: none"> Pain on ulnar side of wrist History of repetitive activity History of pulsatile mass on ulnar side CT angiogram with evidence of thrombosis, irregularity, or aneurysm.

**Figure 7.** Physical examination findings and signs in patients with CuTS. **A** Ulnar claw hand with atrophy of the FDI. **B** Wartenberg sign. **C** Froment sign. **D** Jeanne sign.

application of American Association of Neuromuscular and Electrodiagnostic Medicine criteria increased the sensitivity of EDX in CuTS from 33.3% to 87.5%. However, this variable sensitivity is the reason why EDX is not considered the gold standard for diagnosing CuTS, especially in early disease. In their study consisting of 118 patients with clinically diagnosed CuTS, Shubert et al³⁶ demonstrated that cubital tunnel release provided considerable relief in 94% of patients, even though EDX reports showed that only 11% of patients had clear CuTS, 23% had ulnar neuropathy, and 66% had negative findings.

Axonal loss occurs as a result of severe and prolonged nerve ischemia and manifests itself as EDX changes with a decrease in amplitude, which can reflect the overall decrease in the number of functioning nerve fibers.³³ Electromyography shows abnormal activity during the insertional phase (indicating muscle denervation), fibrillations during the resting phase, and the presence of motor unit action potentials during the recruitment phase (indicating attempted reinnervation by either collateral sprouting or axonal reinnervation; Fig. 10).³³ Friedrich et al³⁷ previously showed that stringent EDX findings have prognostic value following surgical treatment. They found that recovery (86%) was strongly associated with a combination of conduction block across the elbow to the first

dorsal interosseous (FDI) and normal distal compound muscle action potential (CMAP) amplitude from the abductor digiti minimi.³⁷ Conversely, reduced FDI amplitude has been associated with pre-operative weakness in grip and key pinch strength, indicating more severe disease with less predictable recovery potential.³⁸

Table 2 provides a summary of the presentation, physical examination, ultrasound, and EDX parameters used in the diagnosis of CuTS. Additional imaging modalities, such as US, have been incorporated recently for diagnosing CuTS. High-resolution US enables a relatively fast, convenient, well-tolerated, and accurate evaluation of the ulnar nerve (Fig. 11).^{26,39,40} Volpe et al⁴¹ have previously shown that the maximum cross-sectional area (CSA), defined as the largest CSA of the ulnar nerve and recorded 4 cm proximal and distal to the medial epicondyle, was 14.6 mm² in patients with EDX-confirmed ulnar neuropathy at the elbow versus 7.1 mm² in control patients. In addition, a positive relationship was observed between the disease severity and nerve size of CuTS. In their study, patients with axonal loss demonstrated a mean CSA of 18.3 mm², whereas patients with less severe symptoms, such as demyelination, demonstrated a CSA of 11.1 mm.^{2,41} The distance between the medial epicondyle and ulnar nerve at full elbow extension has also been found to be diagnostic for CuTS, with a distance of 0.53 cm having good sensitivity

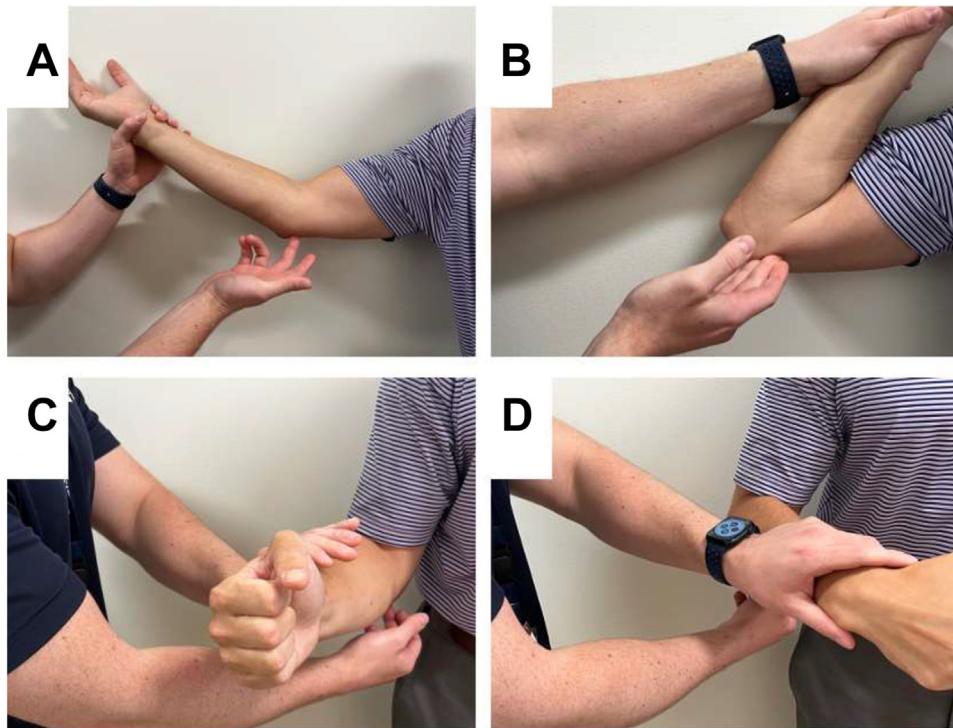


Figure 8. Provocative physical examination maneuvers that are useful in the diagnosis of CuTS. **A** Tinel sign test. **B** Flexion compression test. **C** Scratch collapse test before collapse. **D** Scratch collapse test after collapse.

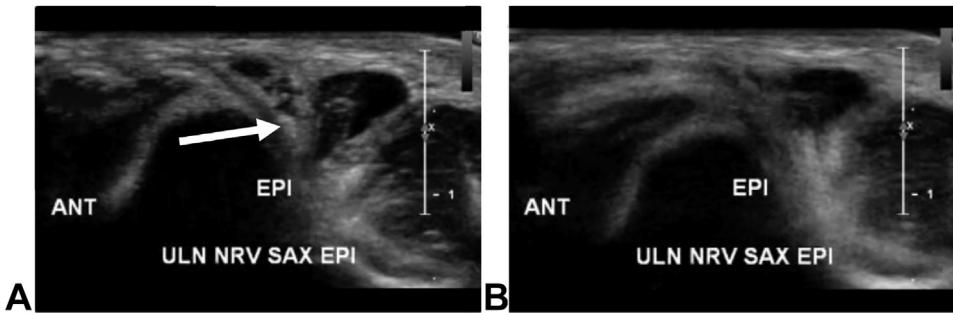


Figure 9. High-resolution ultrasound depicting dynamic instability of a hypermobile ulnar nerve (arrow). **A** The elbow in extension with the ulnar nerve reduced. **B** The elbow in hyperflexion with the ulnar nerve subluxated and no longer visible.

Spectrum of Disease	Mild (Dynamic ischemia)	Moderate (Demyelination)	Severe (Axonal loss)
Conduction Velocity	Normal	Slowed	Slowed
CMAP amplitude	Normal	Normal	Decreased
Electromyography	Normal	Normal	Abnormal

Figure 10. EDX findings in CuTS.

(71%) and specificity (90.7%).⁴² US has also been used to diagnose patients with CuTS with negative EDX⁴³ as well as in patients with recurrent disease.^{44,45} However, compared with EDX, US has been shown to have lower sensitivity for detecting CuTS in the primary setting (71% vs 89%).⁴⁶

In addition to US, CT and MRI have also been used in the diagnosis of CuTS. Computed tomography scans of the cubital tunnel have shown decreased CSA compared with normal controls as well

as differences in bony morphology that may increase the odds of developing CuTS.¹⁴ Magnetic resonance image studies have been shown to have higher diagnostic sensitivities than EDX in some studies,⁴⁷ especially in patients with nonlocalizing neurophysiological response. The most frequent MRI findings include a combination of high signal intensity and nerve enlargement (63%), followed by nerve compression (27%) and isolated high signal intensity (23%) or nerve enlargement (2%; Fig. 12).⁴⁷ In addition, an increase in T2 signal intensity and nerve caliber enlargement have been shown to positively correlate with disease severity and progression over time.⁴⁸

Treatment

Nonoperative treatment

Patients with intermittent symptoms and clinical examination consistent with CuTS without evidence of axon loss, weakness, or

Table 2Presentation, Physical Examination, Ultrasound,⁴¹ and Electrodiagnostic Parameters³⁶ for the Diagnosis of CuTS

Spectrum of Disease	Mild (Dynamic ischemia)	Moderate (Demyelination)	Severe (Axonal loss)
Presentation	Intermittent paresthesias in ulnar nerve distribution with elbow flexion	Sensory symptoms more constant, some motor weakness	Hand weakness, clawing
Physical examination	Positive findings on provocative testing (elbow flexion test, Tinel sign, scratch collapse test)	Decreased 2-point discrimination, weakness on strength testing (grip and apposition pinch)	Atrophy of intrinsic hand muscles with profound sensory disturbances
US CSA of ulnar nerve	11.1 ± 3.4 mm ²	15.8 ± 3.8 mm ²	18.3 ± 5.1 mm ²
Electromyography	Normal	Normal	Abnormal
Conduction velocity	Normal	Slowed	Slowed

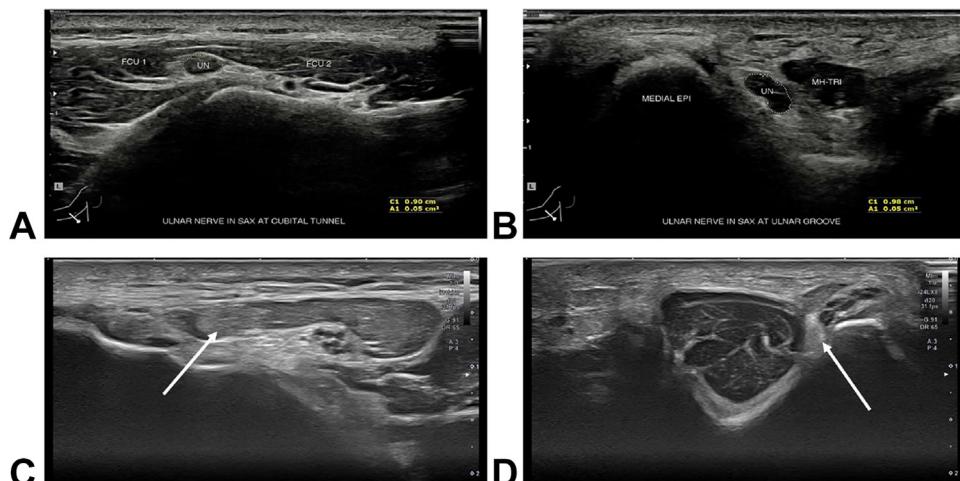


Figure 11. High-resolution ultrasound for diagnosis in CuTS. **A** The ulnar nerve (UN) as it courses between the 2 bellies of the FCU. **B** The UN as it travels around the elbow in the ulnar groove between the medial epicondyle (MEDIAL EPI) of the humerus and the medial head of the triceps (MH-TRI). **C** The UN (arrows) in a patient which CuTS at the FCU. **D** The UN (arrows) in a patient with CuTS at the FCU and perched on the medial epicondyle. The nerve's morphological appearance is abnormally hypoechoic and has lost its normal fascicular echo pattern.

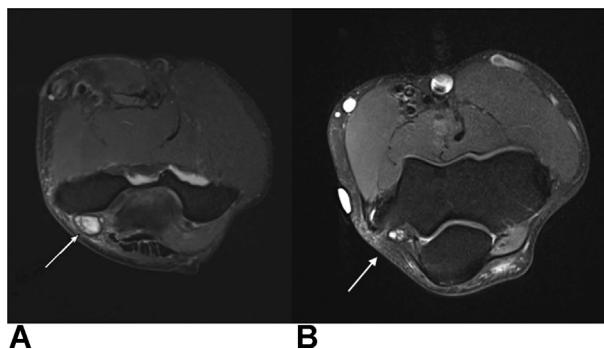


Figure 12. Axial MRI using T2-weighted imaging of the right elbow. **A** An enlarged and high signal intensity ulnar nerve in a patient with CuTS. **B** Comparison MRI in a patient without CuTS depicting the internal topography of the ulnar nerve fascicles.

atrophy are candidates for nonoperative treatment. Previous studies have reported 50% to 88% success using nonoperative approaches^{2,49,50} and found a positive correlation between symptomatic relief and improved EDX findings.⁴⁹ Successful nonoperative treatment is lower in pediatric and adolescent patients but should be attempted prior to surgical intervention.⁵¹ Nonoperative approaches include orthoses to prevent elbow flexion, elbow pads, avoidance of triceps exercises, postural/behavior modification, nonsteroidal anti-inflammatory drugs, night splints, physical therapy, US, pulsed signal therapy, ergonomics education, and corticosteroid injections.^{22,52} In a randomized trial, Svernlöv et al⁵³ found 90% clinical improvement in patients with

early disease regardless of whether they received education separately, wore nighttime orthosis, or performed nerve glides.

Surgical treatment

Although the surgical treatment of CuTS is increasing,¹ there remains a lack of consensus or standardized algorithm for treatment. Modern treatment strategies fall into one of 2 categories: *in situ* decompression or transposition, with multiple technical options and nuances inclusive to both. Proponents of simple decompression cite equivalent clinical⁵⁴ and electrodiagnostic outcomes⁵⁵ with lower morbidity,⁵⁶ whereas proponents of transposition cite improved clinical outcomes in some studies⁵⁷ with less revision for postoperative nerve instability⁵⁸ and decreased reoperation rate in the long term (12% vs 25%).⁵⁹ Furthermore, biomechanical evidence of decreased ulnar nerve pressure⁶⁰ and strain,⁶¹ especially in elbow flexion, has historically favored transposition relative to *in situ* release. This dilemma is currently being researched as part of a clinical trial funded by the National Institutes of Health (clinicaltrials.gov identifier NCT04254185).

In a recent large systematic review, outcomes from 30 studies were evaluated, in which 8 different surgical techniques were utilized on more than 2800 elbows. The study found an improvement rate of 87% with surgery, 3% risk of complication, and 2% recurrence rate.⁶² Comparative analysis of surgical treatment techniques revealed *in situ* decompression to be the most effective procedure with the least amount complications and lowest risk of reoperation and recurrence.⁶² It should be noted that transpositions were not



Figure 13. Intraoperative photograph of in situ decompression of the ulnar nerve in a patient with CuTS. The arrow indicates preservation of the medial antebrachial cutaneous nerve.

performed randomly in these studies, creating an important selection bias.

In situ decompression

In situ decompression (Fig. 13) was first described by Buzzard⁶³ in 1922 and later by Osborne⁶⁴ in 1957. This technique is relatively faster than transposition,⁵ more cost-effective,⁶⁵ and can be performed under local anesthesia.⁶⁶ The procedure involves releasing the points of compression from the ulnar nerve without mobilizing it anterior to the medial epicondyle and performing a soft tissue stabilization procedure.

In a recent survey of American Society for Surgery of the Hand members, in situ decompression was the treatment of choice for the majority of members in patients without ulnar nerve subluxation.⁴ This mirrors the trend observed in other studies that have found an overall rise in in situ release but a decrease in transposition over time,^{5,67} both in the United States and abroad.⁶⁸ Explanations for the rise of in situ release are likely multifactorial. However, improved understanding of disease etiology, increased ability to detect CuTS earlier in its course, faster surgical time, decreased morbidity, equivalent reimbursement, and surgeon preference all likely play a role.⁶⁹

Both open and endoscopic approaches have been described and have demonstrated equivalent efficacy for postoperative clinical improvement and similar complication rates.^{70,71} Patient satisfaction has been reported to be higher (79%) for endoscopic versus open (60%) release.⁷² In addition, a smaller incision of visualization through a

smaller (2 cm vs 4 cm) has been reported.⁷³ Despite lower compensation and higher procedural cost than open in situ release, the frequency of endoscopic cubital tunnel release appears to be increasing.⁷⁴

Revision surgery following in situ decompression has been reported to be as high as 19%.⁷⁵ Reasons for persistent or recurrent symptoms are often secondary to incomplete release, persistent tension on the nerve during elbow flexion, and nerve instability.⁷⁶ Patients with prior elbow fracture or dislocation (OR 7.1) as well as patients requesting surgery for mild clinically graded disease (OR 3.2), have a higher rate of revision surgery. Young patients with a sharp-angled ulnar nerve groove have previously been found to be at a higher risk of anterior dislocation of the ulnar nerve after simple decompression.^{77,78} Interestingly, some studies found that age, sex, body mass index, tobacco use, and diabetes status are not associated with a greater likelihood of revision surgery following in situ release.⁷⁵

Transposition

Transposition has historically been the most widely used method of treatment with the final position of the nerve located subcutaneous,⁷⁹ intramuscular,^{80,81} or submuscular (Fig. 14).⁸² This technique appears the most logical as it treats both the compression and traction components of the disease.⁸³ Subcutaneous transposition was first described by Curtis⁷⁹ in 1898 and allows for immediate mobilization.⁶⁹ However, concerns with neuritis and recurrent subluxation posterior to the medial epicondyle later led Learmonth⁸⁴ in 1942 to modify the transposition technique to include excising the medial intermuscular septum and placing the ulnar nerve next to the median nerve beneath the flexor-pronator mass. Although this modification provided nerve stability, the requisite postoperative immobilization to protect the flexor-pronator mass repair to the medial epicondyle was associated with significant stiffness, fibrosis, and decreased nerve gliding.⁶⁹ Several modifications of the technique have since been developed to help facilitate earlier range of motion, such as musculofascial lengthening of the flexor-pronator mass.⁸⁵ For subcutaneous transposition, the use of a fasciодermal sling⁸⁶ has been described to prevent posterior subluxation, as well as adipose flaps to decrease perineural scarring and neuritis (Fig. 15).^{87,88}

Randomized controlled trials and meta-analyses have yet to elucidate a superior treatment outcome between subcutaneous and submuscular transposition⁸⁹ as well as transposition versus in situ release.⁵⁶ It remains unclear which preoperative factors predict positive outcomes for anterior transposition.⁹⁰ Although some studies support transposition for unstable ulnar nerves,⁹¹ others have found that older age, weak preoperative grip strength, and abnormal 2-point discrimination are associated with unfavorable outcomes at 2 years after surgery regardless of treatment.⁹¹ Currently, history or examination consistent with ulnar nerve instability, recurrent disease, and muscle atrophy are the most common indicators for transposition.³⁰ Submuscular transposition is most commonly performed in thin patients but has greater wound complications and peri-incisional numbness compared with subcutaneous transposition.¹ Subcutaneous transposition is the preferred technique for throwing athletes and has demonstrated good clinical results.⁹²

Other areas in which transposition of the ulnar nerve can be beneficial include traumatic elbow reconstruction and total elbow arthroplasty. McKee et al⁹³ previously reported neurolysis and transposition during reconstruction of failed elbow fixations that resulted in functional improvements, return of strength, and patient satisfaction among patients with varying levels of ulnar nerve injury. However, in the primary care setting, a recent multicenter randomized controlled trial failed to show any significant difference between prophylactic in situ decompression and transposition for

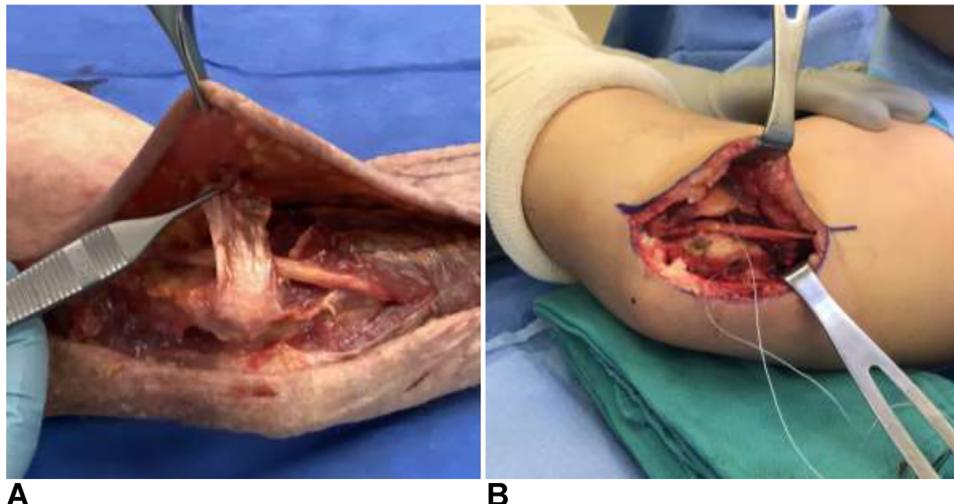


Figure 14. Ulnar nerve transposition surgery. **A** Anterior subcutaneous transposition. **B** Submuscular ulnar nerve transposition.

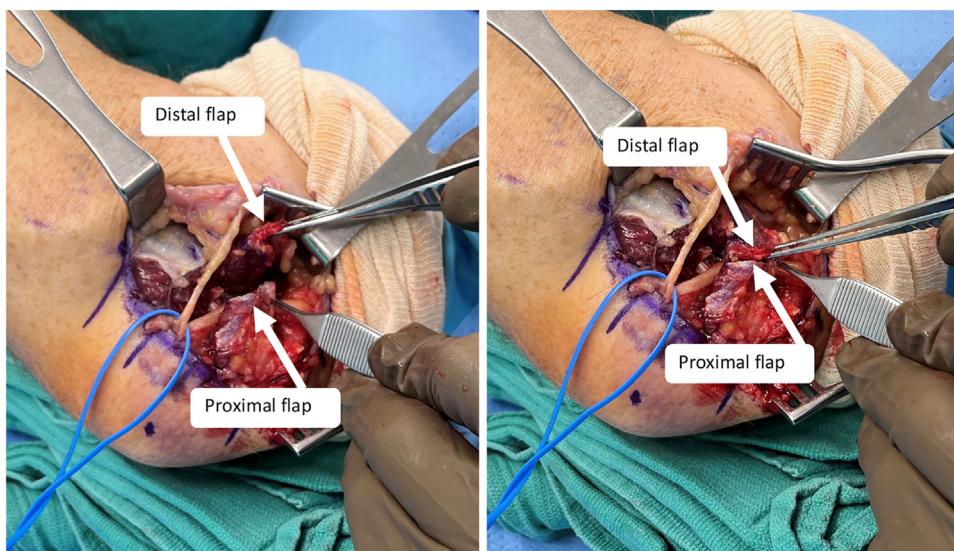


Figure 15. Submuscular transposition of the ulnar nerve demonstrating both the musculofascial lengthening of the flexor-pronator mass and the distal and proximal flaps of the fasciodermal sling.

distal humerus fractures treated with bicondylar fixation.⁹⁴ For primary total elbow arthroplasty, ulnar nerve transposition is recommended only if preoperative elbow flexion is markedly limited or abnormal tracking or increased nerve tension is identified intraoperatively after insertion of the prosthesis.⁹⁵ Otherwise, an *in situ* release is sufficient to minimize ulnar nerve complications after total elbow arthroplasty, which occurs approximately 3% of the time and can have devastating consequences.⁹⁵

Regardless of the final position of the nerve, the success of any ulnar nerve transposition procedure requires creation of a completely untethered path for the ulnar nerve without kinking or tension.⁹⁶ In addition to proximal excision of the medial intermuscular septum, structures distal to the medial epicondyle must also be considered. These structures include but are not limited to the medial antebrachial cutaneous nerve (whose branches cross the ulnar nerve approximately 2.0, 3.7, and 7.7 cm distal to the medial epicondyle⁹⁶), Osborne's fascia, FCU nerve branches of the ulnar nerve, crossing vascular branches from the ulnar artery, distal intermuscular septum between FCU/FDS, flexor-pronator origin, and investing fascia of the FDS over the ulnar nerve.⁹⁶

Medial epicondylectomy

Medial epicondylectomy was first described by King and Morgan⁹⁷ in 1959 and involves resection of the medial epicondyle of the humerus to decrease both compression and tension on the ulnar nerve without creating secondary nerve instability. Although this procedure was effective in improving ulnar nerve symptoms, medial elbow pain and iatrogenic elbow instability limited its widespread adoption.⁷⁶ Following O'Driscoll's⁹⁸ discovery that only 20% of the width of medial epicondyle could be removed without risking injury to the origin of the medial collateral ligament (MCL), a modified MCL-sparing technique was subsequently developed utilizing an oblique osteotomy halfway between the coronal and sagittal planes.⁹⁹ Outcomes of this technique have been favorable, with 93% of patients reporting good or excellent results with no subsequent elbow instability.⁹⁹ Furthermore, a prospective randomized study reported superior outcomes and patient satisfaction following medial epicondylectomy compared with transposition.¹⁰⁰ Current indications for medial epicondylectomy include patients with hypermobile nerves, pre-existing vascular

Table 3

Clinical Guidelines to Optimize Surgical Treatment for CuTS

Preoperative	Intraoperative	Postoperative
In patients that show clinical symptoms of CuTS but a negative EDX, we recommend US of the ulnar nerve given the high false-negative rate of EDX. This will provide the best treatment results when CuTS is diagnosed early.	Infiltration with 1% lidocaine with epinephrine prior to incision around cubital tunnel can help with hemostasis and avoid the need for a tourniquet. Identification of the ulnar nerve in between the 2 heads of FCU is fast and reliable, especially in obese patients.	For posttransition patients, a short arm volar-based wrist splint is better tolerated than a long arm posterior-based splint and effectively protects the flexor-pronator mass.
Assess for proximal (cervical) and distal (Guyon's canal) sites of compression when evaluating CuTS (double crush syndrome). For concomitant cervical radiculopathy and CuTS of ulnar nerve compression, treat CuTS first. For concomitant CuTS and Guyon's canal compression, treat both at index procedure.	Maintain vascularity to ulnar nerve by minimizing dissection posterior to the nerve in an <i>in situ</i> release.	We encourage the use of a neoprene sleeve postoperatively to aid with swelling and incisional discomfort.
Nerve transfers are best used in patients with viable motor endplates and should not be used when atrophy and sever clawing are present. Success is dependent on appropriate indications.	Retrograde dissection of the ulnar nerve proximal to the medial epicondyle to the arcade of Struthers is safe as there are no nerve branches between these anatomic sites	In the revision setting, we recommend anterior submuscular transposition.
	During anterior submuscular transposition, a blunt hemostat passed beneath flexor-pronator mass can help protect the underlying ulnar collateral ligament during flap dissection.	

disease where transposition could potentially worsen ischemia, and thin patients.^{1,76} A 13% revision rate has been reported in the literature with younger age, associated workers' compensation claims, mild disease, and preoperative narcotic use being identified as risk factors for reoperation.¹⁰¹

Revision cubital tunnel surgery

Evaluation and treatment of recurrent and/or persistent symptoms after cubital tunnel release is challenging.¹⁰² Recurrent CuTS has been attributed to inaccurate preoperative diagnosis, incomplete nerve decompression, iatrogenic injury, postsurgical perineural adhesions, irreversible nerve pathology, or conditions associated with secondary nerve compression.¹⁰³ In addition to evaluating for medial antebrachial cutaneous nerve neuroma and more proximal sites of nerve compression in the neck and thoracic outlet, psychiatric comorbidities and psychological coping skills must also be assessed.¹⁰²

It is estimated that as many as 25% of patients treated for CuTS will experience recurrence.¹⁰⁴ Younger age at presentation (<50 years)¹⁰¹ as well as greater static 2-point discrimination and history of diabetes have been associated with a greater number of revision surgeries.¹⁰⁵ In a large systematic review of patients undergoing revision cubital tunnel surgery, transposition surgery was the most common procedure for primary surgery (51%), perineural scarring was the most common intraoperative finding at revision surgery (79%), and the medial intermuscular septum was the most frequent entrapment site (33%).¹⁰⁶ In their study, Novak and Mackinnon¹⁰⁷ examined a series of 100 patients who underwent reoperation following cubital tunnel surgery. The authors found that the most common operative findings included a medial antebrachial cutaneous nerve neuroma ($n = 73$) and a distal kink of the ulnar nerve ($n = 57$) caused by fascial flaps or tendinous bands.¹⁰⁷ Outcomes following revision surgery are inferior to those following primary surgery, with only 75% to 80% of revision patients reporting symptomatic improvement, and worse outcomes reported on all measured standardized questionnaires.^{108,109} In addition, patients over the age of 50 years, electromyographic evidence of denervation, and previous submuscular transposition have been associated with poor outcomes after revision surgery.¹⁰⁹

External neurolysis,¹¹⁰ anterior submuscular¹¹¹ and subcutaneous¹¹² transposition, and medial epicondylectomy have been

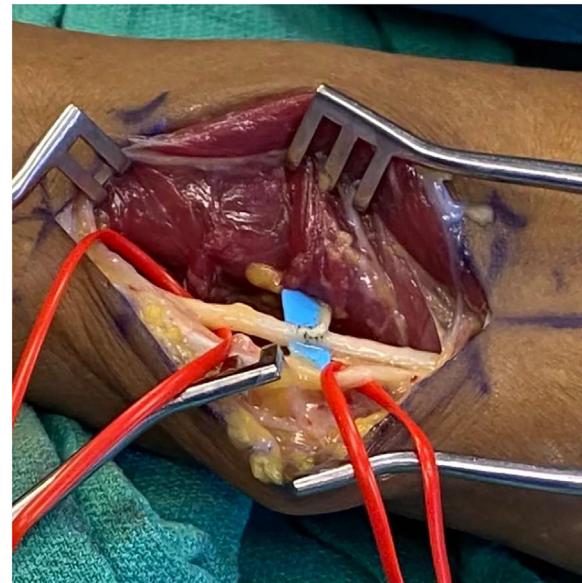


Figure 16. Intraoperative photograph of anterior interosseous nerve (AIN)-to-ulnar motor nerve transfer for CuTS. Anterior interosseous nerve (arrow) as it is transferred to the ulnar nerve.

described as revision treatment options. The most common revision surgery is submuscular transposition of the ulnar nerve (75%).¹⁰⁶ Table 3 provides a comprehensive approach to the optimization of the treatment of CuTS. More recently, vein,¹¹³ collagen nerve wraps,¹⁰⁴ and porcine extracellular matrix wraps¹¹⁴ have been used as adjuvants to help prevent perineural scarring with promising results. However, randomized prospective studies and long-term results are currently lacking to support the use of such expensive options.

Nerve transfers

For severe primary or recurrent CuTS, nerve transfers have been described as a technique to "supercharge" the ulnar nerve, and thus facilitate faster and more complete recovery with minimal morbidity (Fig. 16).^{15,115–117} Patient selection is guided by preoperative EDX: the best candidate demonstrates

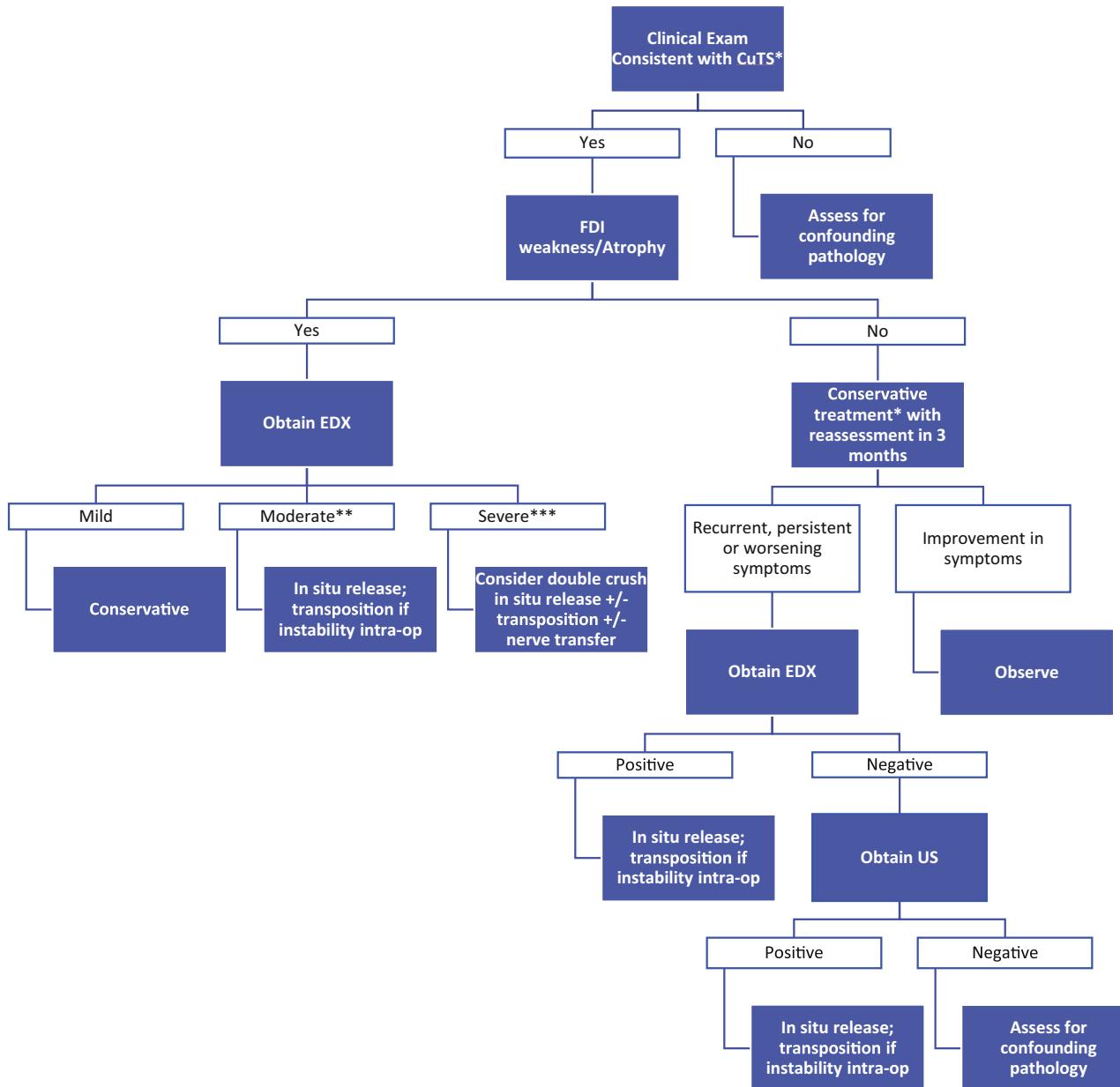


Figure 17. Algorithm for CuTS diagnosis and treatment. *Clinical examination consistent with CuTS is defined as having paresthesias in the distribution of the ulnar nerve, symptoms caused by elbow flexion, a positive Tinel sign at the medial elbow, and/or widened 2-point discrimination in ulnar nerve distribution. **Moderate findings of EDX include decreased conduction velocity. ***Severe findings of EDX include decreased CMAP with/without abnormal electromyography findings.

decreased CMAP amplitudes that reflect a lower number of axons crossing the compression site, and the presence of fibrillations/positive sharp waves indicating that at least some motor endplates remain available and receptive to reinnervation.¹¹⁸ The terminal branch of the anterior interosseous nerve (AIN) innervates the pronator quadratus and provides an expendable donor in the forearm with approximately 900 axons.¹¹⁹ Recently, Doherty et al¹¹⁵ found that more than three quarters of patients with advanced CuTS had partial or complete resolution of clawing or muscle wasting following end-to-side AIN to ulnar nerve motor transfer. Improvement in function within the first 2 to 3 months is thought to be related to remyelination, 4 to 5 months for axonal regeneration, and approximately 6 to 7 months after supercharge nerve transfer.¹¹⁸ The mean time for observing nascent units was 8.5

months in some studies; however, continued recovery has been observed at 12 and 24 months at the level of endplate and as a result of neuroplasticity.¹²⁰ Additional procedures that may augment recovery include concomitant Guyon's release, side-to-side profundus tenodesis, and cross-palm nerve grafts from the median to ulnar sensory nerve.^{118,121}

Another adjunct treatment for severe CuTS is postsurgical electrical stimulation (PES). Recently, Power et al¹²² reported on PES enhanced muscle reinnervation and functional recovery following surgery for severe CuTS. In their randomized, double-blind, placebo-controlled study, patients underwent cubital tunnel release followed by either a sham stimulation or a 1-hour treatment of 20 Hz PES. At the 3-year follow-up, the motor unit number estimation increased and key pinch strength was 3-fold higher in the cohort that underwent

PES.¹²² Although the ideal PES timing and intensity has yet to be determined, PES is an emerging technology likely to be used more often in the future.

Conclusions

The reason for the current lack of standardized algorithm for diagnosing and treating CuTS is multifactorial. However, controversies regarding the diagnostic tools, treatment, and outcomes at research level all play a role. Diagnostic challenges include heterogeneous patient population that often present symptoms at a later stage of the disease when treatment outcomes are less reliable. In early disease, when CuTS is most reliably treated, EDX are less dependable³⁶ and history and physical examination can be equivocal. Therefore, it is not surprising that additional imaging modalities are being studied to assist in accurately identifying CuTS prior to demyelination, axon loss, or before further irrecoverable changes occur. This would decrease time to surgery in patients who fail conservative treatment and increase their likelihood of favorable results with a less invasive surgical approach.

Treatment controversies stem from a lack of prospective, randomized, controlled trials of patients with a similar diagnosis being treated with different techniques using standard objective and subjective outcomes measures. In addition, the presence of pre-operative nerve instability in many studies, including systematic reviews, has been shown to influence treatment options and present an important selection bias.¹²³ It is also important to develop new techniques and consistently use reliable and reproducible outcome measures that are validated in the CuTS patient population.¹²⁴ A previous systematic review has identified 45 unique outcomes and 31 postoperative outcome measures using 101 published studies on the surgical treatment of CuTS.¹²⁵ This heterogeneity limits the interpretation of current studies and represents a barrier we must overcome in future studies moving forward. The included treatment algorithm acknowledges the limitation of the evidence supporting each technique with the aim to provide clarity about the challenges for treating this complex patient population (Fig. 17).

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