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REVIEW

Evidence-Based Management of Ulnar Neuropathy at the Elbow

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Abstract

Purpose of Review Ulnar neuropathy at the elbow (UNE) is the second most common entrapment neuropathy and can result in severe loss of function of the hand. This review will cover up to date anatomy, pathophysiology, diagnostic assessment, and treatment of UNE.

Recent Findings In cases of suspected UNE with normal electrodiagnostic assessment (EDx), neuromuscular ultrasound (NMU) and MRI have been shown to increase diagnostic sensitivity. A variety of treatment options exist including activity modification, bracing, physical therapy, ESWT, injections, hydrodissection, surgical decompression, and surgical functional restoration. A Delphi survey recommended standardized outcome measures to be utilized for ongoing research due to heterogenous existing literature.

Summary UNE diagnosis can be reliably achieved in patients with compatible signs and symptoms in conjunction with the use of diagnostics tests including EDx, NMU, and MRI. A large variety of non-surgical and surgical treatment options exist, however, there is lacking consensus regarding optimal treatment choice.

Keywords Ulnar nerve · Ulnar neuropathy · Cubital tunnel syndrome · Nerve compression syndromes

Introduction

Ulnar neuropathy at the elbow (UNE) is the second most common entrapment neuropathy in the upper extremity, second only to carpal tunnel syndrome [1]. UNE is also known as cubital tunnel syndrome, however, given a multitude of possible entrapment sites at the elbow, is better described as ulnar neuropathy at the elbow. The ulnar nerve is essential to the function of the upper extremity, as it provides both motor and sensory innervation to both the hand and forearm. Clinical symptoms include pain at the elbow, muscle weakness in the hand, and paresthesias in the ring and small fingers. Risk factors for UNE include general medical conditions such as diabetes mellitus and rheumatologic diseases. Certain occupations that require long periods of elbow flexion, such as working on a telephone, lead to increased risk for UNE [2]. Similarly, athletes who repetitively flex their elbow, such as baseball players, have demonstrated an increased risk for developing UNE [3].

Detailing the most updated evidence-based management of UNE is crucial for researchers, healthcare providers, and patients. UNE can cause significant impairment in hand function, which can negatively affect activities of daily living and work. If not appropriately treated, functional impairments can lead to disability and can progress to permanent nerve damage [4]. Outlining current evidence-based management is critical, as treatment modalities vary, and management should be tailored to each patient's particular presentation and preferences to preserve ulnar nerve function. Understanding evidence-based diagnosis and treatment of UNE is important to empower healthcare providers to collaborate with their patients and develop the most efficacious treatment plans.

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Anatomy and Pathophysiology

The ulnar nerve arises from the medial cord of the brachial plexus, originating from nerve roots C8-T1. The ulnar nerve provides both motor and sensory functions. In the forearm, the ulnar nerve innervates the medial half of the flexor digitorum profundus and the flexor carpi ulnaris. In the hand, the ulnar nerve innervates the interosseus muscles, third and fourth lumbricals, adductor pollicis, palmaris brevis, hypothenar muscles, flexor pollicis brevis and the skin on the medial hand as seen in Fig. 1. Clinical motor deficits include difficulty lifting, loss of dexterity, and decreased grip strength. Sensory symptoms include referred pain in the forearm, paresthesias, and sensory loss of the dorsal and palmar aspects of the medial side of the hand, little finger, and ulnar side of the ring finger.

UNE occurs at the elbow prior to any of the motor or sensory branches of the ulnar nerve. The anatomy and pathophysiology of this region is important to understand for appropriate diagnosis and treatment. Within the elbow, there are multiple locations where the ulnar nerve may be entrapped. The sites of entrapment proximal to the elbow include the medial intermuscular septum and the Arcade of

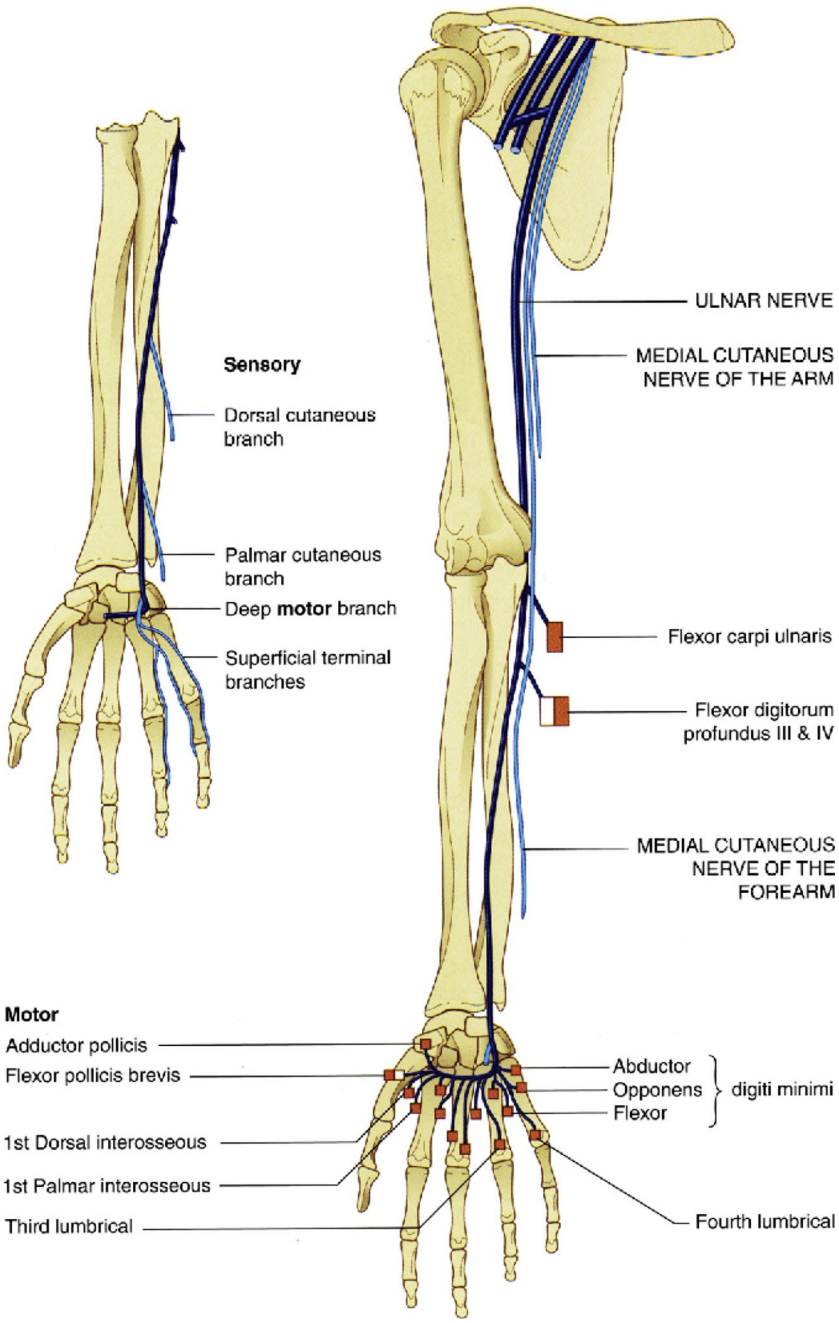

Fig. 1 Depiction of the ulnar nerve's course and ordinal innervation. Reproduced from: Evaluation and Treatment of Upper Extremity Nerve Entrapment Syndromes. Primary Care: Clinics in Office Practice December 2013. Illustration used with the permission of Elseiver Inc. All rights reserved

Struthers, which lie 6 to 10 cm proximal to the medial epicondyle [5]. At the elbow, the common entrapment sites are at the retroepicondylar groove and under the humeroulnar aponeurotic arcade (HUA) also known as Osborne's ligament. The HUA originates in the medial epicondyle region and contains three layers of tissue overlying the ulnar nerve. During elbow flexion, the cross-sectional area drops by 30 to 41%, which may restrict mobility and compress the ulnar nerve [6, 7]. Other sources of compression include the epitrochleoanconeus muscles, and the two heads of the flexor carpi ulnaris [6], the true "cubital tunnel". Space occupying lesions such as ganglion, heterotopic ossification, elbow osteoarthritis, and olecranon bursitis can also lead to UNE. The ulnar nerve may be compressed due to reduction in tunnel volume, compression during movement, or tension forces leading the nerve to ischemia, inflammation, and fibrosis. When damage occurs to the blood-nerve barrier, protein-rich fluid extravasates leading to edema and eventual neural fibrosis. Nerve conduction is slowed by fluid impairing function of Schwan cells at the nodes of Ranvier. Sustained compression may even lead to death of Schwann cells, and loss of the myelin sheath makes the axons within nerves more vulnerable to compression, damage, and axon death [8]. A hypermobile ulnar nerve can subluxe or dislocate around the medial epicondyle. Ulnar nerve subluxation/dislocation is frequently present in asymptomatic individuals. There is conflicting literature regarding subluxation and/or dislocation as it relates to UNE [9]. There are proposed models of ulnar nerve hypermobility causing a tractional/frictional neuritis. It is important to consider ulnar nerve subluxation/dislocation in cases of symptomatic UNE as it may alter treatment, especially if surgery is being considered.

Physical Examination

Physical examination in patients with UNE should include relevant tests of motor and sensory function in addition to relevant special tests. Inspection of the hand may demonstrate marked atrophy of the hypothenar eminence, hand intrinsics, and in severe cases the "ulnar claw". Weakness of ulnar innervated musculature in the forearm and hand should be documented. Measures such as key pinch and grip strength can be useful to measure longitudinally for objective outcome assessment. Sensory loss in ulnar distribution and 2-point discrimination measurements should also be assessed. Careful assessment should be undertaken with palpation or dynamic ultrasound to evaluate for ulnar nerve subluxation or dislocation over the medial epicondyle. Special tests including Tinnel's, Froment sign, Wartenberg sign, Jeanne sign, and Duchenne sign can be helpful, although there is no documented sensitivity and specificity. The shoulder internal rotation elbow flexion test has been proposed as specific and quick provocative test for diagnosing UNE, with a sensitivity of 87% and specificity of 98% [10]. In this test, 90° abduction, maximum internal rotation, and 10° flexion of the shoulder is combined with maximum elbow flexion, forearm supination and wrist extension as depicted in Fig. 2. If symptoms of UNE develop within 5 s, the test is considered positive. The scratch test is another special test with excellent documented sensitivity of 69% and specificity of 99% [11]. In this test, the patients resist bilateral shoulder external rotation with their elbows flexed at 90° to determine baseline external rotation strength. Then the area of suspected nerve compression (medial elbow) is lightly scratched. Resisted shoulder external rotation is immediately repeated, and the test is considered positive if there is momentary loss of shoulder external rotation resistance as depicted in Fig. 2. The scratch test has been shown to have significantly higher sensitivity and specificity compared to the Tinel's test and the elbow flexion/nerve compression test for UNE [11].

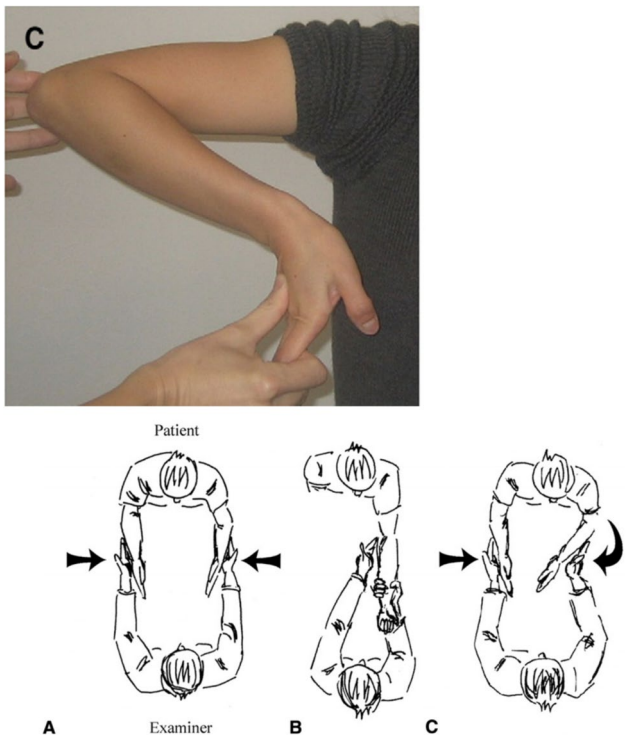

Fig. 2 Example positioning for the shoulder internal rotation elbow flexion test and the scratch test. Images reproduced with permission from Ochi et al. [11] and Cheng et al. [11]

UNE Severity Classification

Severity grading of UNE has typically been performed with the McGowan Classification. In this scale, UNE is classified between grade I and III. Patients who have symptoms of UNE, without objective weakness or atrophy, are classified as grade I. Grade II classification consists of mild objective sensory loss, muscle weakness, and incomplete

muscle atrophy. In grade III, patients experience functionally disabling sensory disturbances, motor weakness, and muscle atrophy [12]. Under the McGowan Classification with Goldberg modification, patients in grade II are subdivided into IIA and IIB. Grade IIA classification consists of patients with no atrophy of intrinsic muscles, while grade IIB consists of patients with some atrophy of intrinsic muscles [13].

UNE Diagnostic Testing

The diagnosis of UNE is classically based on a typical clinical presentation with supportive electrodiagnostic studies (EDx). Edx are useful for confirming the diagnosis, localizing the lesion, and ruling out conditions that can mimic UNE such as ulnar neuropathy at the wrist, polyneuropathy, cervical radiculopathy, and brachial plexopathy. Additionally, Edx can be used to classify the severity of injury per the Seddon and Sunderland criteria and prognosticate recovery by delineating demyelinating from axonal ulnar nerve injuries [14–16]. Edx can also be useful in UNE by guiding the type of treatment utilized based on the severity of nerve injury at presentation per the Padua classification [17]. Edx are not without pitfalls, though. Edx are time-consuming and uncomfortable for patients. In UNE, the literature suggests that Edx has a sensitivity of 37–87% and surgical treatment trials have demonstrated a false negative rate of more than 10% [18, 19]. Furthermore, in purely axonal lesions, localization of the ulnar neuropathy to the elbow as the entrapment site is not possible [16]. In this setting, neuromuscular ultrasound (NMU) has gained popularity for the diagnosis of UNE [20]. Measuring maximal cross-sectional area (CSA) of the ulnar nerve (as depicted in Fig. 3) within a few centimetres of the elbow is the most commonly used parameter for diagnosis, although no clear diagnostic criteria have been established [20]. A large retrospective study of patients with clinical UNE demonstrated similar sensitivities of Edx and NMU and a combined sensitivity of 98% with a maximal CSA of 10 mm^2 [21•]. NMU adds the added utility of localizing the area of entrapment more precisely, identifying possible anatomic abnormalities responsible, identifying nerve edema, and assessing for ulnar nerve subluxation. Magnetic resonance (MR) imaging techniques and MR neurography techniques have also improved [22]. Despite lower reported sensitivity than NMU, MR techniques can also identify nerve compression, edema, muscle denervation, and soft tissue structures with great anatomic detail [22]. MR imaging is especially useful at characterising deeper soft tissue structures and lesions suspicious for malignancy [22]. Clinicians need to understand the relative strengths and weaknesses of each diagnostic test when assessing and treating patients with suspected UNE.

UNE Treatment

Activity Modification

Education and activity modification forms the backbone of physical medicine and rehabilitation treatment plans. In

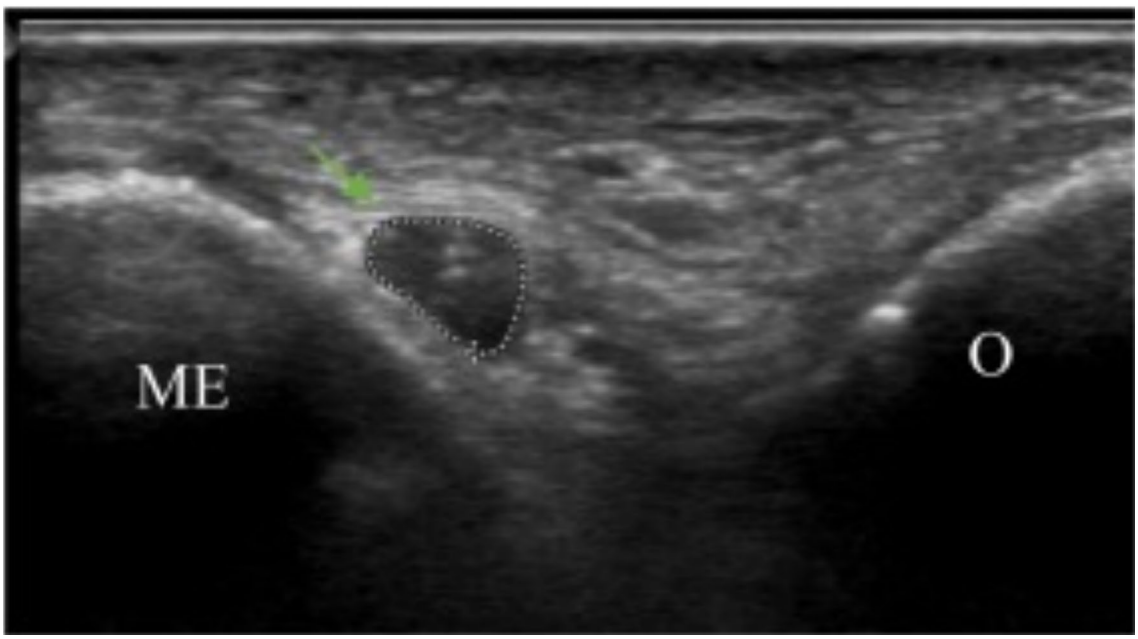

Fig. 3 Ultrasound images of ulnar nerve at the level of the medial epicondyle with cross-sectional area calculation. ME = medial epicondyle, O = olecranon. [23]

the setting of UNE, certain positions are particularly provocative in exacerbating transient ulnar nerve compression. Avoidance of prolonged elbow flexion and supination, minimization of resting of medial elbow on hard surfaces, and ergonomic workstation setup are all steps that can improve symptoms. Multiple studies have demonstrated that patient education about the pathophysiology of UNE, corresponding provocative positions and appropriate activity modification can alleviate symptoms in 35–82% of cases. The symptom alleviation was persistent months to years after the intervention was employed [24–27]. One RCT was performed comparing nighttime splinting, nerve gliding, and a control group of education and activity modification in patients with mild to moderate severity UNE [28]. At 6 months, 90% of all patients had symptom improvement in all three groups. Strength, electrophysiology, and functional outcomes improved similarly in all three groups in most patients as well. Education and activity modification is an easy, safe, and no-cost intervention that should be employed for all cases of UNE and may provide adequate symptom relief in certain cases.

Bracing

Bracing/splinting the elbow is another commonly used non-operative treatment modality for UNE [29]. Cadaveric studies have been undertaken to determine the relationship of elbow flexion, ulnar nerve traction, intraneural interstitial pressure, and ulnar nerve compression below Osborne's ligament within the cubital tunnel [30]. The result of the body of work in this area indicate the optimum position for elbow immobilization to reduce pressure on the ulnar nerve at the elbow is at approximately 45 degrees of elbow flexion [30]. Several investigational trials have assessed the role of elbow bracing/splinting in the treatment of UNE. A systematic review and meta-analysis showed that elbow splinting can improve symptoms of UNE in 89% of patients (95% CI 69–99%) with I^2 of 92% [31]. The type of splint and splinting protocol was heterogenous in the included studies. All splints limited elbow flexion but did not necessarily prevent elbow extension. The splinting protocols varied from isolated nighttime splinting to wearing them as much as possible. The only RCT assessing nighttime splinting utilized comparator groups of neurodynamic mobilization and a control group of education and activity modification. This RCT demonstrated positive outcomes in all groups, but did not show a significant difference between any of them [28]. The optimal type of splint and splinting protocol remains to be determined and requires additional research. The cause of patient symptoms and aggravating factors may help guide clinicians regarding splint type and timing. Given the pathophysiology of UNE and cadaveric studies, the authors suggest that rigid nighttime elbow splinting at 45 degrees of elbow flexion is a reasonable first-line intervention to employ, although there is only low quality literature to support its use, similarly to the treatment of carpal tunnel syndrome [32].

Physical Therapy

Other non-surgical treatment interventions frequently utilized include physical therapy. There is minimal literature investigating these types of interventions in UNE. One case-series investigated the role of neurodynamic mobilization and demonstrated positive results in all cases [33]. This finding could not be reproduced in an RCT comparing neurodynamic mobilization to an education and activity modification control with no significant differences between groups [28]. Another small (n = 32) case–control study investigated the role of ultrasound therapy compared to low-level laser therapy for the treatment of UNE [34]. Both interventions were successful at improving clinical and electrophysiologic parameters in the short term. UNE can also present in addition to other musculoskeletal conditions of the elbow such as medial epicondylopathy, ulnar collateral ligament insufficiency, and valgus extension overload [35–37]. In this setting, physical therapy interventions targeting the main biomechanical issue are effective at treating the concomitant ulnar neuritis [35–37].

Pharmacology

Symptomatic relief may be achieved with nonsteroidal anti-inflammatory drugs (NSAIDs), oral steroids, and neuropathic agents (gabapentinnoids, tricyclic antidepressants, SNRIs) but there is no evidence to suggest long term benefit or disease modifying activity. These medications should only be used for short duration on a patient-specific basis.

Extracorporeal Shockwave Therapy (ESWT)

ESWT is an emerging therapy for a multitude of musculoskeletal and neurologic diseases [38–40]. ESWT has been shown to induce neuro-recovery in rodent models and is proposed to help regenerate damaged peripheral nerves through mechanisms including proliferation of Schwann cells, activation of macrophages, and increased vascularization [39, 41, 42]. A recent single-blinded RCT (n = 50) investigated radial ESWT vs sham ESWT for the treatment of UNE [43]. They demonstrated that three sessions of radial ESWT performed one week apart improved clinical, functional, and some electrophysiologic parameters at 2 weeks, 1 month, and 3 months after treatment was completed. A small pilot study demonstrated similar findings of pain and functional outcome scores with radial ESWT [44]. ESWT is a promising emerging therapy for

the treatment of UNE. Further research regarding the optimal dosing and timing is important to better elucidate this treatment modality further.

Injections and Hydrodissection

Injection treatments are the most invasive of the non-operative treatment modalities. Injection safety, precision, effectiveness has increased with the use of ultrasound-guidance [45]. Despite significant literature investigating the role of injections in carpal tunnel syndrome, there is minimal literature investigating them in UNE [31, 46]. A 2023 systematic review and meta-analysis of the available literature pooled the results of 6 studies and found that 54% of patients (95% CI 41–67%) improved after an average of 4.3 months after receiving a perineural steroid and lidocaine injection. Of the included 6 studies, only 1 was a placebo-controlled RCT (n = 55) and this trial did not find a significant difference between corticosteroid (1 mL methylprednisolone and 1 mL 1% lidocaine) and normal saline injections (1 mL NaCl) for UNE [47].

More recent studies have investigated the role of dextrose and hydrodissection in UNE, utilizing the successful literature in carpal tunnel syndrome treatment as a guide [48, 49]. Chen et al. performed a double-blind RCT (n = 33) comparing perineural dextrose (5 mL 5% dextrose) and corticosteroid injections (3 mL 10mg/mL triamcinolone and 2 mL NaCl) for UNE [50]. Both groups had clinically and statistically significant improvements in clinical, functional, electrophysiologic, and ultrasound parameters for 3–6 months. The better clinical results after both dextrose and corticosteroid injection in this trial may be attributed to higher volume causing greater hydrodissection, type of injectate, or injection technique. Another high quality study investigating the role of dextrose injections in UNE was performed by Mansiz-Kaplan et al. [51]. They performed a double-blind RCT (n = 40) comparing perineural dextrose (1 mL 5% dextrose at 5 sites = 5 mL) and normal saline (1 mL NaCl at 5 sites = 5 mL) for the treatment of UNE. The injections were performed 4 cm proximal to the medial epicondyle, 2 cm proximal to the medial epicondyle, at the level of the medial epicondyle, 2 cm distal to the medial epicondyle, and 4 cm distal to the medial epicondyle. The same procedure was repeated 2 weeks later. The dextrose group had statistically and clinically significant improvements in pain, function, ulnar nerve CSA, and electrophysiologic parameters that continued to improve at each time point until final measurement at 12 weeks compared to placebo. These results indicate that 5% dextrose may have anti-inflammatory and/or neuro-regenerative effects on peripheral nerves independent of hydrodissection.

Finally, platelet-rich plasma (PRP) is another injectable that has recently gained popularity for a multitude of injection indications [52]. PRP is proposed to have a potential neuronal repair mechanisms through its release of various growth factors such as platelet-derived growth factor, VEGF, transforming growth factor-b1, and insulin-like growth factor. Naggar et al. performed a RCT (n = 60) comparing leukocyte poor PRP (3 mL) and triamcinolone (1 mL 40 mg/mL and 1 mL 1% lidocaine). Both groups showed improvements in clinical, functional, nerve CSA and electrophysiologic parameters. The corticosteroid group had more rapid improvements at 1 month, however, similar improvements in both groups were demonstrated at 3 months. No long-term outcomes were reported.

Taken together, there are several injection treatments that have shown positive outcomes for the treatment of UNE. Although initially equivocal, more recent corticosteroid injection techniques with an element of hydrodissection appear to have at least short-term symptom and functional relief. Other injection treatments including 5% dextrose and PRP show promise, however, additional research is needed. Specifically, long-term outcomes need to be investigated to identify if these procedures are temporizing measures or disease-modifying interventions with long-term promise. Furthermore, these promising non-surgical interventions should be studied in more severe cases of UNE since all studies were conducted in grade 1–2 UNE per the McGowan classification.

Surgical Treatments

Ulnar Nerve Decompression

Surgical management of idiopathic UNE is considered the most effective long term option with a 75–90% success rate [53, 54]. Indications for surgery include failed conservative management, high-grade disease severity, and patient preference. A variety of surgical techniques are described in the literature; however, there seems to be no one superior method.

Simple decompression (SD) is the original and most performed surgical technique where the structures superficial to the ulnar nerve in the cubital tunnel are released and the nerve is left in its original position. According to a Cochrane systematic review and meta-analysis performed in 2016, compared to other surgical techniques, SD had equivalent clinical and functional outcomes, similar revision rates (~ 2%), and the smallest rate of complications (~ 3%) [8, 55].

Endoscopic decompressions approach treatment of UNE similarly, but with a minimally invasive surgical wound with the goal of reduced surgical morbidity and reduced risk of medial antebrachial cutaneous nerve (MABCN) injury [8]. The added risk of endoscopic decompression is incomplete ulnar nerve decompression. A comparative study between open and endoscopic simple decompression demonstrated less pain and greater patient satisfaction with the endoscopic approach and no differences in objective outcome measures [56].

Anterior transposition techniques involve relocation of the ulnar nerve anterior to the medial epicondyle with the goal of

reduced nerve tension and prevention of nerve subluxation/dislocation during elbow flexion. Transposition techniques are indicated for significant ulnar nerve subluxation and dislocation. There is a higher incidence of complications with anterior transposition procedures including damage to blood supply, MABCN injury, and symptom recurrence through new compression sites [8]. Different transposition locations include subcutaneous, intra-muscular and submuscular sites. The available literature does not demonstrate evidence to support one technique over the others [57].

An alternative technique to transposition involves simple decompression with medial epicondylectomy. The benefit of this technique is controlling ulnar nerve subluxation/dislocation and preserving blood supply without introducing new entrapment sites that can occur with transposition. Medial epicondylectomy presents its own set of other complications including pain at the osteotomy site, elbow flexion contracture, and elbow instability [8]. There is no clear surgical consensus on how to balance adequate bone excision for ulnar nerve release with preservation of the medial collateral ligament and flexor pronator group insertion [58].

Taken together, a variety of surgical techniques are available for the treatment of UNE. A patient-specific and surgeon-specific approach is necessary to employ for optimal outcomes.

Functional Hand Surgical Restoration Techniques

In chronic and severe cases of UNE (McGowan grade 3), motor recovery of the hand intrinsic musculature can be limited [59]. Adequate ulnar nerve decompression can allow motor recovery through a multitude of mechanisms including reversal of conduction block, distal axonal collateral sprouting, muscle hypertrophy, and axonal regeneration [60]. Unfortunately, due to the long distance of nerve injury at the elbow to the motor endplates in the hand, axonal regeneration cannot always occur before irreversible degeneration and fibrosis of the motor endplates. Alternative techniques have been developed to improve functional outcomes in this subgroup of patients with different surgical techniques. Tendon and soft tissue procedures including claw hand reconstructions, metacarpophalangeal capsulodesis, flexor digitorum superficial (FDS) lasso, FDS transfer, wrist extensor to intrinsic transfer, and flexor digitorum profundus side-to-side transfers are describes options with good success, but associated morbidity with loss of function from sacrificed structures [61]. Nerve transfers are an alternative option that have gained popularity in the last two decades with less associated functional morbidity and restoration of original muscle biomechanics compared to soft-tissue procedures [62]. Several nerve transfer options exist such as end-to-side anterior interosseous to ulnar motor nerve, end-to-end motor branch of opponens pollicis to deep palmar ulnar motor, and cross-palm sensory median to ulnar grafts [59, 63]. It is important for clinicians to understand and appreciate the role of functional augmentation procedures for the treatment of UNE.

Conclusions

UNE is a common neuropathy that can cause significant morbidity and mortality if not treated early and appropriately. Diagnosis can be reliably achieved in patients with compatible signs and symptoms and the use of diagnostics tests including Edx, NMU, and MRI. A large variety of non-surgical and surgical treatment options exist, however, there is lacking consensus regarding optimal treatment choice. Lack of consensus is partly due to lacking literature and partly due to inconsistent outcome measures utilized [64••]. A recent Delphi survey was conducted to develop consistent outcome measures to be utilized moving forwards [64••].

If caught early, UNE can usually be effectively treated with a variety of non-surgical treatment options. Education and activity modification is one of the most important interventions in cases of idiopathic UNE. When signs and symptoms are more severe (McGowan grade 2 or 3), the evidence suggests surgical treatment without initial trial of non-surgical management. Interestingly, there is minimal available evidence to support the concept that non-surgical management is less effective in higher grade idiopathic UNE [55]. All available non-surgical treatment studies excluded patients with higher grade UNE. Intuitively, higher grade UNE may require more urgent and definitive treatment, however, promising new non-surgical treatments and nerve hydrodissection in conjunction with education and activity modifications may prove to be effective in this patient category as well. Moving forward, treatment plans can and should be tailored by patient preference to optimize symptom relief and ulnar nerve function.

Author Contributions All authors whose names appear on the submission made substantial contributions to the conception or design of the work, drafted the work or revised it critically for important intellectual content, approved the version to be published, and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Data Availability No datasets were generated or analysed during the current study.

Declaration

Ethical We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

Conflicts of Interest The authors declare that they have no conflict of interest.

Competing Interests The authors declare no competing interests.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- • Of importance
- •• Of major importance

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64.•• Wilson TJ, Davis GA, Dengler NF, Guedes F, Hébert-Blouin M-N, Jack MM, et al. Core outcomes in nerve surgery: development of a core outcome set for ulnar neuropathy at the elbow. J Neurosurg. 2023;1(aop):1–9. (**This expert consensus paper outlines core outcome measures that should be utilized as the standard for treatment outcome assessment in ulnar neuropathy at the elbow.**)