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Ulnar neuropathy: evaluation and management

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Abstract Ulnar neuropathy is commonly encountered, both acutely after elbow trauma and in the setting of chronic compression neuropathy. Careful clinical evaluation and discerning evaluation of electrodiagnostic studies are helpful in determining the prognosis of recovery with nonoperative and operative management. Appreciation of the subtleties in clinical presentation and thoughtful consideration of the timing and type of surgical intervention are critical to optimizing outcomes after treatment of ulnar neuropathy. The potential need for decompression at both the cubital tunnel and Guyon's canal must be appreciated. Supplementation of decompression with supercharged end-to-side nerve transfer can expedite motor recovery of the ulnar intrinsic muscles in the appropriately selected patient. The emergence of nerve transfer techniques has also changed the management of acute ulnar nerve injuries.

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Introduction

Regardless of discipline or subspecialty, all surgeons treating complex elbow conditions should be aware of the principles in evaluation and management of acute and chronic ulnar neuropathy. Treatment of chronic compression neuropathy of the ulnar nerve remains a complex and challenging issue. Appraisal of the literature via systematic review and meta-analyses indicates that the best treatment for ulnar neuropathy remains unclear [1–3]. Appreciation of the subtleties in clinical presentation and thoughtful consideration of the timing and type of surgical intervention are critical to optimizing outcomes after treatment of ulnar neuropathy. Similarly, management of acute ulnar nerve injuries has recently changed with the introduction of nerve transfer techniques.

Clinical presentation

Patients with compressive neuropathy of the ulnar nerve typically describe numbness and tingling of the ulnar-sided digits of the hand, classically in the small finger and ulnar aspect of the ring finger. Among the general population, symptoms usually begin intermittently and are often worse at night, particularly if the elbow is flexed while sleeping. As the disease progresses, paresthesias may occur more frequently and during the daytime. Position of the elbow likely contributes to the symptom characteristics, as cadaveric studies have demonstrated that the ulnar nerve at the elbow is predisposed to both compression and traction during elbow flexion [4, 5].

Prominent bands of the Osborne ligament at the leading edge of the flexor carpi ulnaris muscle will aggravate ulnar nerve symptoms with gripping activities, such as driving. For throwing athletes, laborers, and others with high physical demands on their elbows, symptoms may be precipitated by intense or prolonged periods of increased activity. These individuals may also present with concurrent bony and soft tissue disease at the elbow, such as medial epicondylitis, lateral epicondylitis, ligamentous instability, and stress fractures. Cyclists and others who place prolonged pressure on the hypothenar eminence are at increased risk for compression of the ulnar nerve at Guyon's canal. For these reasons, detailed inquiry into specific provocative activities and positions, as well as the timing and chronicity of symptoms, is necessary to inform clinical decision-making. Clumsiness and loss of dexterity imply intrinsic muscle involvement. Cases of motor weakness without any sensory deficit should receive special attention, with concurrent cervical radiculopathy and/or myelopathy receiving consideration. Rarely, an upper motor neuron disease or a compressive mass may be present. These varied, but significant, etiologies may mimic cubital tunnel syndrome, making electrodiagnostic studies prudent in all cases.

Physical examination

Systematic assessment of the muscles and sensory distributions innervated by the ulnar nerve is helpful in localizing the level of compression of the ulnar nerve. Compression at the cubital tunnel from Guyon's canal can be differentiated with specific comparison of intrinsic versus extrinsic motor strength, as well as comparison of sensation in the volar versus dorsal ulnar nerve sensory distributions. The motor examination includes evaluation of the flexor carpi ulnaris, ulnar-innervated flexor digitorum profundus, finger abduction, and finger adduction, while the sensory examination includes subjective responsiveness to sensation to light touch (Ten Test) [6] and two-point discrimination in the distributions of the dorsal cutaneous branch and volar main sensory branch of the ulnar nerve. In cases of advanced ulnar neuropathy, further inspection may reveal atrophy in the hypothenar eminence and in the first dorsal interosseous muscle bulk, as well as clawing of the ring and small fingers. Eponymous findings such as Froment's sign (overt flexion of the thumb interphalangeal joint while attempting resisted pinch) and Wartenburg's sign (persistent abduction posture of the small finger due to unopposed action of the radial-innervated extensor digiti minimi) may also be present. We also recognize a "pseudo-Froment" sign, in which the interphalangeal joint of the involved thumb may not have overt flexion but has a distinctly different appearance than the uninvolved side. This finding may especially be noted in patients with ligamentous laxity and hypermobile joints.

Provocative testing of the ulnar nerve classically includes percussion (Tinel's sign) and direct pressure applied over known compression points of the nerve. The most sensitive (91 %) provocative test for ulnar nerve compression at the elbow is direct pressure over the ulnar nerve posterior to the medial epicondyle while the elbow is in flexion [7]. We use a modification of the scratch collapse test [8] to identify distinct or concurrent points of ulnar nerve compression [9•]. An area of nerve compression is identified by weakness in resisted shoulder external rotation (while arm is adducted at the side and the elbow is flexed 90°) after that area has been stimulated by the examiner's scratch. Additional points of compression can be solicited by applying a topical anesthetic to the area(s) where a positive result was found. We have found this examination maneuver to correlate well with electrodiagnostic and intraoperative findings in patients with ulnar neuropathy [10]. The identification of distinct and/or concurrent points of ulnar nerve compression is especially helpful in evaluation of patients with failed primary surgery. The hierarchy of the collapse points helps in determining whether the ulnar nerve is primarily irritated at the cubital tunnel or at Guyon's canal or at other areas of compression (such as the arcade of Struther's in the mid-brachium). Frequently, the scratch collapse test will be used to identify associated compression at Guyon's canal and/or the arcade of Struthers in patients with "failed primary cubital tunnel surgery." Compression at both of these sites is amenable to surgical treatment without "revisiting" the previously operated decompression about the elbow.

Stability of the ulnar nerve within the groove posterior to the medial epicondyle should also be assessed. Hypermobility of the ulnar nerve was present in 37 % of a healthy volunteer population and may be associated with increased nerve irritability [11]. Although not supported by uniform consensus or a strong evidence base, traditional teaching indicates that patients with clinically apparent subluxation of the ulnar nerve should undergo anterior transposition (rather than in situ decompression) to decrease the chances of persistent symptoms, recurrent symptoms, or postoperative worsening of nerve subluxation. At the time of surgery, any associated subluxation of the triceps muscle must be evaluated and treated, if present [12].

Classification and interpretation of electrodiagnostic studies

The histopathology of chronic compression neuropathy spans a breakdown in the blood–nerve barrier, followed by subperineurial edema, localized (and then diffuse) demyelination, and finally axonal loss. Compressive ulnar neuropathy presents along a similar predictable spectrum of disease: dynamic ischemia, demyelination, and axonal loss. These stages can be differentiated by careful history from the patient,

clinical examination, and interpretation of electrodiagnostic studies.

During *dynamic ischemia*, compression of the ulnar nerve is the result of transient decreases in perfusion of the ulnar nerve. Symptoms are largely position-dependent and resolve when blood flow to the nerve is restored. Patients with dynamic ischemia typically improve with nonoperative measures (such as protective padding, night positioning in extension with a towel roll, and ergonomic adjustments) to avoid positions that provoke symptoms. Electrodiagnostic studies (nerve conduction studies and electromyography) are likely to be negative, as conduction velocity has not yet slowed in the fastest-conducting nerve fibers. If symptoms warrant surgical decompression, the patient is likely to experience near-immediate relief after surgery. As the disease process progresses, prolonged ischemia will cause *demyelination* of the ulnar nerve. Conduction velocity, which reflects the speed of conduction along the fastest-conducting nerve fibers, is slowed. Symptoms become more pronounced and less intermittent. Relief after surgery is reliably expected within a period of 3–4 months after surgery as remyelination occurs. Long-standing or severe compression will lead to the development of *axonal loss*. Symptoms are constant, and changes in two-point discrimination, motor weakness, and muscle atrophy are evident. Nerve conduction studies show a decrease in amplitude, which reflects an overall decrease in the number of functioning nerve fibers. Electromyography shows abnormal activity during the insertional phase (indicating muscle denervation), fibrillations during resting phase (a sine qua non for motor axon loss), and the presence of motor unit action potentials during the recruitment phase (indicating attempted reinnervation by either collateral sprouting or axonal reinnervation). Recovery after surgery is much more prolonged, as axonal regrowth occurs at a rate of 1 mm per day. Collateral sprouting from adjacent, unaffected motor nerve fibers to sprout collateral branches to the neighboring denervated muscle fibers may expedite the reinnervation process.

Nonoperative management

We have found that patients with dynamic ischemia are most likely to respond to nonoperative treatments for ulnar nerve compression at the cubital tunnel. A dedicated regimen of eliminating elbow flexion at night and activity modifications for 3 to 6 months can lead to improvement of cubital tunnel syndrome in patients with mild to moderate symptoms [13]. Padua et al. demonstrated clinical and electrodiagnostic improvement at 1-year follow-up in approximately half of their patients treated with postural education [14]. Patient education by the surgeon and therapist regarding the positional nature of the neuropathy is helpful to ensure maximal effectiveness of activity modifications and ergonomic adjustments. Protective soft padding over the medial aspect of the elbow can decrease irritability of the ulnar nerve. At night, a soft towel is wrapped around the elbow to decrease elbow flexion and is more likely to be tolerated by patients than rigid splints. A neutral wrist splint decreases stretch on the ulnar nerve in Guyon's canal. We reserve nonoperative treatments for patients with mild to moderate symptoms (suggesting dynamic ischemia) and motor nerve conduction velocity >40 m/s across the elbow. If patients do not see improvement after several months of nonoperative treatment, we recommend surgical management of the ulnar nerve. Patients with significant axonal loss (as reflected by decreased amplitudes on nerve conduction studies) are not likely to be responsive to conservative management and are considered candidates for surgery.

Surgical management

Selection of procedure

Controversy exists regarding the appropriate surgical technique to treat ulnar neuropathy at the elbow. The many techniques described to treat cubital tunnel syndrome reflect options available to treat the ulnar nerve at the level of medial epicondyle. They can be generally classified as in situ decompression (open, mini-incision, or endoscopic) or anterior transposition (accomplished via medial epicondylectomy, subcutaneous transposition, transmuscular transposition, or submuscular transposition). In situ decompression has experienced a rise in popularity among surgeons in the USA [15], although more recent reports suggest significant recurrence rates. Appraisal of the available evidence indicates that the best treatment for cubital tunnel syndrome remains unclear [1–3].

In situ decompression of the ulnar nerve can provide benefit in the carefully selected patient, but extensive counseling is recommended prior to surgery to discuss the frequency of recurrent symptoms, the frequency of reoperation, and the outcomes after revision surgery if required. The theoretical advantages of in situ decompression include faster recovery due to less extensive dissection and lower risks of wound-related complications [16]. The theoretical advantage of preserving intrinsic and extrinsic vascularity has been refuted, as blood flow is maintained for a diameter-to-length ratio of 1:63 within the ulnar nerve when only proximal intrinsic vascularity is intact [17]. Furthermore, a recent clinical study showed no difference in motor or sensory outcomes whether or not the extrinsic vascular pedicle to the ulnar nerve was preserved [18]. The frequency of persistent symptoms and eventual anterior transposition has been reported as low as 7 % at 1-year follow-up [19]. However, the same center has more recently reported a less favorable revision rate of 19 % [20•], with 77 %

of the revisions performed within 2 years of the in situ decompression. Risk factors for revision surgery after in situ decompression included prior elbow fracture or dislocation and surgery performed for patients with mild symptoms and no motor weakness [20•]. When evaluating the outcomes after revision cubital tunnel surgery in a case–control investigation, the patients who had revision surgery were more likely to have persistence of constant symptoms (53 % of patients) and worse patient-reported outcomes than those undergoing primary surgery [21•]. The findings of this study are particularly salient to those patients undergoing in situ decompression of the ulnar nerve, as 93 % of the patients in the revision cohort had previously undergone an in situ decompression [21•]. The recent reports regarding in situ decompression leave us less optimistic regarding its role in management of cubital tunnel syndrome, particularly since the outcomes after revision are worse than doing an anterior transposition initially.

We prefer a specific transmuscular anterior transposition technique that has provided reliable and durable clinical improvement [12, 22]. With the assistance of a sterile tourniquet, the surgeon makes a long, longitudinal incision in line with the course of the ulnar nerve that is centered over the posterior aspect of the medial epicondyle. Branches of the medial antebrachial cutaneous nerve (MABC) are identified and carefully protected during the procedure. Rarely, if these branches are inadvertently injured, the MABC is crushed well proximal to the neurotemetic site with a hemostat and transposed proximally and deep to muscle to minimize the occurrence of a painful neuroma. The known points of compression of the ulnar nerve are identified and released. Visual inspection of the ulnar nerve at points of compression may reveal pseudoneuroma formation just proximal to Osborne's ligament and the absence of the normal bands of Fontana along the external epineurium. During decompression, particular attention is directed to excision of the proximal intermuscular septum and distal decompression of the flexor carpi ulnaris fascia. A distal intermuscular septum (between the ulnar-innervated flexor carpi ulnaris and the median-innervated flexor/pronator mass) should be excised [22]. Experience with revision cubital tunnel surgery indicates that this distal entrapment point is the most commonly neglected point of remaining compression, as the nerve is moved from its native position medial and below this septum to its new anteriorly transposed position [23]. While the proximal intermuscular septum is well recognized, the distal medial intermuscular septum is not (Fig. 1a). Without excision of this distal septum, kinking of the ulnar nerve as it moves to its transposed position is almost certain to occur. A Z-lengthening flap of the fascia overlying the flexor-pronator mass is created. Compressive fascial bands within the flexor-pronator muscle mass are excised. The decompressed ulnar nerve is transposed into the transmuscular muscle bed, checking multiple times for any residual points of compression or kinking proximally and distally. The Z-lengthened flexor-pronator fascia is very loosely reapproximated with one or two sutures, intentionally leaving redundancy to avoid a new iatrogenic site of compression (Fig. 1b). The tourniquet is deflated and removed from the surgical field. The surgeon palpates in the proximal apex of the wound for an arcade of Struther's fascial band in the midbrachium. It presents as a discrete tendinous band just posterior and below the ulnar nerve, inserting into the triceps muscle. If it is present, the incision is extended proximally and the fascial band is divided under direct visualization. The senior author has noticed the arcade of Struther's to be more proximally located than generally appreciated. After meticulously obtaining hemostasis, the senior author prefers to place a surgical drain and an indwelling anesthetic pump. Subcutaneous local anesthetic is applied and the wound is closed in two layers. A rigid posterior splint is applied with the wrist in neutral position, the elbow flexed, and the forearm pronated. The splint is removed 2 days after surgery and active elbow range of motion is started. We have found that the most important principles of this procedure are to preserve the MABC branches to avoid painful neuroma formation, thoroughly release proximal and distal compression sites, excise compressive fascial bands within the flexor-pronator mass, loosely reapproximate the flexor-pronator fascia, repeatedly check for proximal and distal compression or kinking after anterior

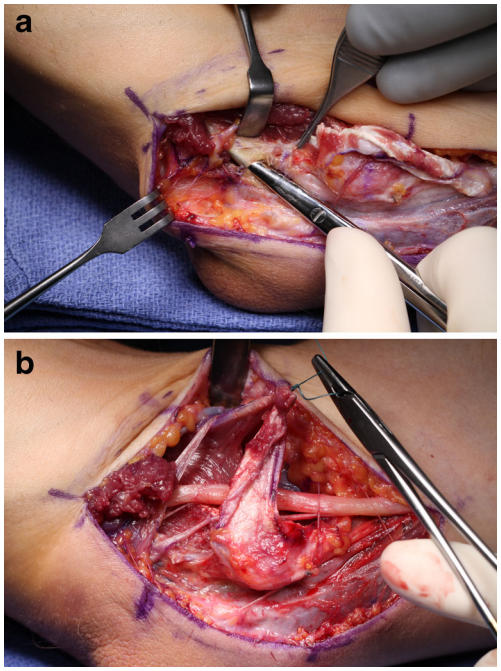

Fig. 1 **a** Distal intermuscular septum between the flexor/pronator mass (anteriorly) and the flexor carpi ulnar (posteriorly). If not divided, this septum (overlying the tenotomy scissors) is a potential point of new compression after anterior transposition of the ulnar nerve. **b** Completed anterior transmuscular transposition of the ulnar nerve. Note the loose reapproximation of the Z-lengthened flexor-pronator fascia—there is intentional redundancy to avoid creating a new iatrogenic site of compression

transposition of the nerve, obtain meticulous hemostasis, and early postoperative motion. Lack of adherence to any of these principles will compromise the clinical result.

We believe that two of the most common (and commonly unrecognized) errors with subcutaneous and submuscular transpositions are (1) kinking of the transposed nerve on the distal intermuscular septum between the flexor-pronator mass and the FCU and (2) failure to advance these muscles distally. We advance them a full inch so that the nerve is not kinked distally in its transposed position.

With regards to surgical treatment of ulnar nerve compression at Guyon's canal, there is less controversy than management of cubital tunnel syndrome. However, we believe it to be under-recognized in patients with concurrent cubital tunnel syndrome. While effective decompression is accomplished by adhering to surgical principles, the senior author's experience in performing revision surgery has provided several "pearls" that are helpful to ensure appropriate decompression. One underappreciated point of compression proximally is the distal antebrachial fascia. This compressive layer cannot be released without extending the incision proximally across the wrist crease. Another critical component of a thorough release is the decompression of the deep motor branch of the ulnar nerve. The deep motor branch cannot be visualized until the leading edge of the hypothenar muscles is actually released. There are two ways to ensure that the deep motor branch has been completely decompressed: visualization of the leading edge of the hypothenar muscles by mobilizing the neurovascular bundle medially at the level of the hook of the hamate and visualization of the profundus tendon to the small finger at the distal end of the release. With adherence to these principles, the surgeon can be confident that Guyon's canal is adequately decompressed.

Careful preoperative evaluation using the hierarchical scratch collapse test, the presence of intrinsic wasting, inability to cross the fingers, positive Froment sign, and electrodiagnostic tests are useful to determine whether concurrent decompression of both the cubital tunnel and Guyon's canal is warranted. The senior author has noted an increase in concomitant release in her practice as her understanding of ulnar neuropathy has evolved, with more than half of her patients undergoing release at both sites [12].

For cases of advanced axonal loss associated with prolonged ulnar neuropathy, the senior author has reported success in supplementing ulnar nerve recovery with a "supercharged" end-to-side (SETS) nerve transfer [24, 25•]. In addition to decompression of the ulnar nerve at the cubital tunnel and Guyon's canal, the branch of the terminal anterior interosseous nerve to the pronator quadratus is transferred to the side of the deep motor branch of the ulnar nerve. Axonal regeneration attributable to the SETS nerve transfer can be expected within 4 to 7 months after surgery, which can be helpful to begin reinnervating the ulnar intrinsic muscles while waiting for axonal regeneration from the more proximal ulnar nerve at the elbow after decompression. Indeed, the greatest improvement in pinch and first dorsal interossei strength is seen during the earlier period of reinnervation in which SETS nerve transfer is expected to be most helpful [25•]. Careful review of preoperative electrodiagnostic studies is critical to ensuring appropriate selection of patients for SETS nerve transfer. Clinical and, if needed, electrodiagnostic evaluation of the median nerve and anterior interosseous nerve should ensure the presence of normal donor nerve. SETS nerve transfer is only performed if the EMG shows fibrillations in the ulnar-innervated intrinsics (indicating recent denervation, reflecting the muscle's ability to become for reinnervation, and suggesting the presence of some functioning ulnar nerve fibers). In addition to the presence of fibrillations, the ideal candidates for SETS nerve transfer have compound muscle action potentials (CMAPs) of low amplitude (reflecting the severity of axonal involvement). Absence of fibrillations and CMAPs for ulnar-innervated muscles was associated with no/poor recovery of clinically apparent motor strength after SETS nerve transfer, suggesting that the severity of disease was too far advanced in these patients for SETS nerve transfer to provide benefit [25•]. CMAPs with good amplitude even in the presence of severe conduction loss suggest that reinnervation is likely to occur after decompression or transposition surgery without supplementation by SETS nerve transfer. In these cases of ulnar intrinsic atrophy and weakness with good amplitudes, release at Guyon's canal will be performed concomitant with the cubital tunnel surgery. We have also had a favorable experience in performing a side-to-side tenodesis of the ulnar-innervated profundus tendons to the median-innervated third profundus tendon while awaiting recovery after decompression of ulnar neuropathy at the elbow.

Acute ulnar nerve injuries

Acute compression of the ulnar nerve due to adjacent soft tissue swelling or fracture displacement may occur following elbow trauma [26], ligamentous reconstruction surgery of the elbow [27], or fractures near Guyon's canal [28]. Postoperative position of the elbow in flexion also places the ulnar nerve at risk for compression [4]. We urge surgeons who treat patients with elbow and wrist to recognize the potential for acute ulnar nerve compression and treat this condition in a manner similar to urgent release of the median nerve in the carpal tunnel in the setting of distal radius and forearm

fractures. A favorable prognosis is expected if the acute compression is recognized and treated in an expedient manner.

Iatrogenic injury to the ulnar nerve may also occur during elbow surgery. Neurotmetic injury to the ulnar nerve has been reported during Tommy John medial ulnar collateral ligament reconstruction [29]. If the injury occurs at the cubital tunnel, we recommend a multi-faceted approach to optimize chances at functional recovery given the relatively poor prognosis associated with this injury pattern. In addition to repairing the ulnar nerve and performing an anterior submuscular transposition, we will perform a concurrent distal *end-to-end* (ETE) motor nerve transfer (pronator quadratus branch of anterior interosseous nerve transferred to deep motor branch of the ulnar nerve) to expedite motor recovery, ETE sensory nerve transfers of the third web space branch of the median nerve to the ulnar sensory fascicles and the palmar cutaneous branch of the median nerve to the dorsal cutaneous branch of the ulnar nerve, and tenodesis of the flexor digitorum profundus (FDP) tendons to maintain finger motion while waiting for motor recovery [30]. Patients with high ulnar nerve lacerations and a Martin–Gruber interconnection are treated with SETS nerve transfer (rather than ETE) to preserve these axons. For injuries distal to the cubital tunnel (within the forearm), we will perform a SETS nerve transfer procedure (as described above) in addition to the FDP tenodesis used for proximal acute neurotmetic injuries. Recently, we have used short acellular nerve allografts (2.5 cm) in a side-to-side fashion from the median nerve (in the carpal tunnel) to the ulnar nerve (in Guyon's canal) with the goal of bringing early sensation to the ulnar-innervated portion of the hand.

Conclusions

Ulnar neuropathy is commonly encountered, both in the general population and in athletes. Careful clinical evaluation and discerning evaluation of electrodiagnostic studies are helpful in determining the prognosis of recovery with nonoperative and operative management. The potential need for decompression at both the cubital tunnel and Guyon's canal must be appreciated. Adherence to the principles of surgical decompression and the "pearls" presented above will assist the surgeon in delivering a reliable and durable result. Supplementation of decompression with supercharged end-to-side nerve transfer can expedite motor recovery of the ulnar intrinsic muscles in the appropriately selected patient.

Compliance with ethical standards

Conflict of interest Christopher J. Dy and Susan E. Mackinnon declare that they have no conflict of interest.

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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