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COMPRESSIVE NEUROPATHIES IN THE UPPER EXTREMITY (E SHIN, SECTION EDITOR)

Severe Cubital Tunnel Syndrome: Considerations for Nerve Transfer Surgery

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Abstract

Purpose of Review Cubital tunnel syndrome is the second most common compressive neuropathy, next to only carpal tunnel syndrome in its incidence. Severe states of disease do not respond to nonoperative management. Likewise, functional outcomes of cubital tunnel surgery decline as the disease becomes more severe. The relatively long distance from site of nerve compression at the elbow to the hand intrinsic muscles distally makes it a race between reinnervation of the muscle and irreversible motor endplate degeneration with muscle atrophy. Loss of intrinsic function can lead to severe functional impairment with poor dexterity and clawing of the hand. While decompressing the nerve at the site of compression is important to prevent further axonal injury, until recently, the only option to restore intrinsic function was tendon transfers. Tendon transfers aim to restore thumb side pinch and control clawing with addition surgery. They also require the sacrifice of wrist extensors or finger flexors. In the past decade, nerve transfers to the distal portion of the ulnar nerve innervating these intrinsic muscles, originally described for proximal ulnar nerve injury or transections, have become increasingly popular as an adjunct procedure in severe cubital tunnel syndrome. Physicians treating severe ulnar neuropathy must be aware of these nerve transfers, as well as their indications and expected outcomes.

Recent Findings The so-called supercharged anterior interosseous nerve (AIN)–to–ulnar motor nerve transfer has become a mainstay for distal nerve transfers for ulnar neuropathy and/or injury. Ideal patients to undergo such a procedure demonstrate severe ulnar neuropathy on nerve conduction and electromyography studies, with reduced compound muscle action potential (CMAP) amplitude and fibrillations at rest. Recent studies demonstrate nerve transfers to be superior in intrinsic muscle reinnervation compared with nerve graft in the setting of large segmental nerve defects. Likewise, compared with decompression alone, patients undergoing the supercharge procedure are more likely to regain intrinsic function and less likely to need secondary tendon transfer surgeries. Finally, initial results for sensory nerve transfer to recover sensation in the ulnar-sided digits in severe cubital tunnel are more advantageous than for decompression alone.

Summary Distal nerve transfers offer a reliable, reproducible treatment option for the restoration of intrinsic hand function and protective sensation in the setting of severe cubital tunnel syndrome.

Keywords Severe cubital tunnel · Nerve transfers · AIN-to-ulnar · Intrinsic weakness · Ulnar neuropathy · Ulnar nerve dysfunction

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Introduction

Chronic ulnar nerve compression within the cubital tunnel at the elbow may result from variety of causes that result in compression and/or traction on the ulnar nerve. Common causes include frequent flexion of the elbow, external forces secondary to positional placement at rest or occupation, anomalous musculature of the medial elbow (e.g., anconeus epitrochlearis muscle), or post-traumatic adhesions and scarring due to prior injury or surgery. Multiple studies have confirmed decreased intra-compartmental volume within the cubital tunnel as well as increased intraneural pressure with

elbow flexion compared with extension [1]. While ulnar nerve subluxation has not been directly associated as a cause of chronic ulnar neuropathy, it can lead to inflammation within the cubital tunnel and make the nerve more prone to inadvertent external injury or neuritis.

Cubital tunnel syndrome is a very common condition, with an incidence of 30 per 100,000 person-years [2]. It is the second most common compression neuropathy found in humans, behind only carpal tunnel syndrome. Its incidence increases with age and has a slight predominance in males. In a study by Osei et al., 41.3% of patients diagnosed with cubital tunnel syndrome were eventually treated surgically in their study period of 6 years [2].

Clinical Presentation

Patients with ulnar nerve compression at the elbow may present with a variety of clinical symptoms. Classically, those with cubital tunnel complain of numbness and tingling in the small finger and ulnar side of the ring finger. These symptoms are often exacerbated at night when the elbow is flexed for prolonged periods during sleep. In more long-standing or severe cases, patients may complain of weakness of their hands with difficulty manipulating small objects or grasping. Also, patients may eventually report more constant paresthesias regardless of elbow position. Patients frequently complain of numbness and tingling when talking on the phone as cell phone use requires elbow flexion. Driving with the elbow resting on center console or door frame can also be associated with symptoms.

Physical Examination

Patients with severe cubital syndrome may display decreased 2-point discrimination or other threshold testing abnormalities in the small and ring fingertip pads. First dorsal webspace muscle atrophy as well as hypothenar eminence atrophy may also be appreciated. Patients may be unable to cross their fingers due to interossei weakness. Loss of grip strength may result from weakness of the intrinsic muscles to the small and ring finger. Classic ulnar nerve palsy tests like Froment's and Wartenberg's signs may also be positive, as a result of weakness in the adductor pollicis and third palmar interossei, respectively (see Fig. 1). A positive Froment's sign is seen with thumb inter-phalangeal joint flexion with attempted thumb-to-index side pinch secondary to compensatory flexor pollicis longus activity via median nerve. Wartenberg's sign is demonstrated by an abduction of the small finger from the hand at rest. It also should be documented if the ulnar nerve subluxates or dislocates out of the cubital tunnel during dynamic flexion of the elbow. Additionally, gentle percussion of the ulnar nerve within the cubital tunnel results in an "electrical-like" sensation along the course of the nerve to the small and ring fingers, known as a Tinel sign.

Electrodiagnostic Studies

In severe cubital tunnel syndrome, transient ischemia seen in earlier phases of the disease progresses into permanent ischemia and intraneural damage. This first leads to demyelination of the ulnar nerve. Motor nerve conduction velocity across the elbow is slowed (< 50 m/s). Untreated compression of the nerve may eventually lead to axonal loss. At this point, symptoms become permanent, and progressive motor loss will occur. Amplitude will be decreased on nerve conduction studies (NCS) and electromyography (EMG) will show aberrant activity during the insertional phase, positive sharp waves/fibrillations during the resting phase, and overall reduced compound muscle action potential (CMAP) volitional activity within ulnar-innervated intrinsic muscles (i.e., first dorsal interosseous muscle). It is important to remember that electrodiagnostic studies may reveal the level of axonal loss but may not correlate to patient symptoms and clinical strength (see Fig. 2). Gordon et al. have demonstrated that muscle function is preserved until 80% of the motor unit has been lost [3].

A skilled electromyographer is needed for proper evaluation of a patient's neuropathy. He or she may also study other nerves of the upper extremity if poly-neuropathic involvement is suggested as well for evaluation of the viability of possible nerve or tendon transfer donors.

Surgical Management

Nerve Decompression

The severity of cubital syndrome can be broadly divided into the nerve injury classification schemes created by Sunderland and Seddon [4]. Patients with early disease, where there is no axonal loss and only nerve conduction slowing across the elbow, represent nerve demyelination from a site of compression. This is neuropraxia or Sunderland first-degree injury. Patients with this lesser form of neuropathy will benefit from nonsurgical management or removal of the offending site of ischemia by compression and/or traction. These treatment modalities include therapy, elbow extension bracing, anti-inflammatory medications, and activity modification.

In severe cubital tunnel syndrome, as confirmed by axonal loss on EMG and conduction velocity < 40 m/s on NCS [1], there is a very low likelihood that nonoperative management will improve symptoms. Symptoms may become permanent if left untreated at this time. Decompression of the nerve is

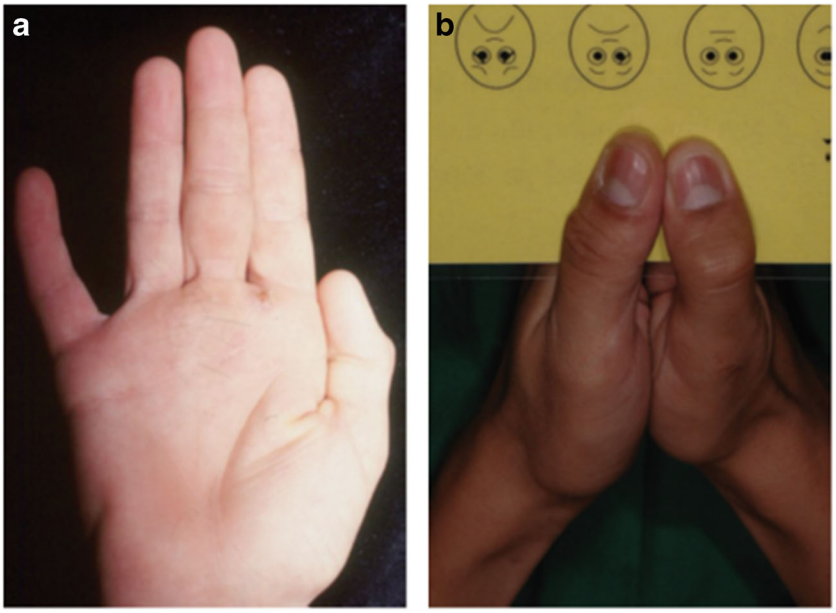

Fig. 1 Clinical signs of ulnar neuropathy. **a** Wartenberg's sign is demonstrated in the clinical photograph. In severe ulnar neuropathy, loss of intrinsic function, specifically the third palmar interosseous muscle, leads to weakness in finger adduction. The EDM, which is innervated by the radial nerve, and inserts on the dorsal ulnar base of the small finger proximal phalanx, becomes unopposed. This extends and abducts the fifth digit. **b** Froment's sign. In this test, the patient is asked to grip a piece of paper between the thumb and index finger. The paper is pulled away from the patient. Patients with severe ulnar neuropathy and intrinsic weakness will have a weakened adductor pollicis muscle. To compensate for their weakened adduction ability, these patients will fire their FPL muscle, causing flexion at the IP joint of the thumb thereby pinching at the thumb tip instead. Adapted from Sebastin and Chung (2012) [23]

important to prevent further nerve damage. Multiple surgical techniques for decompression at the elbow are described such as in situ decompression, medial epicondylectomy, or anterior nerve transposition. An analysis of the merits of one technique over others is controversial and beyond the scope of this paper.

Regardless, nerve decompression alone can be an unreliable treatment option in severe cases due to the axonal loss.

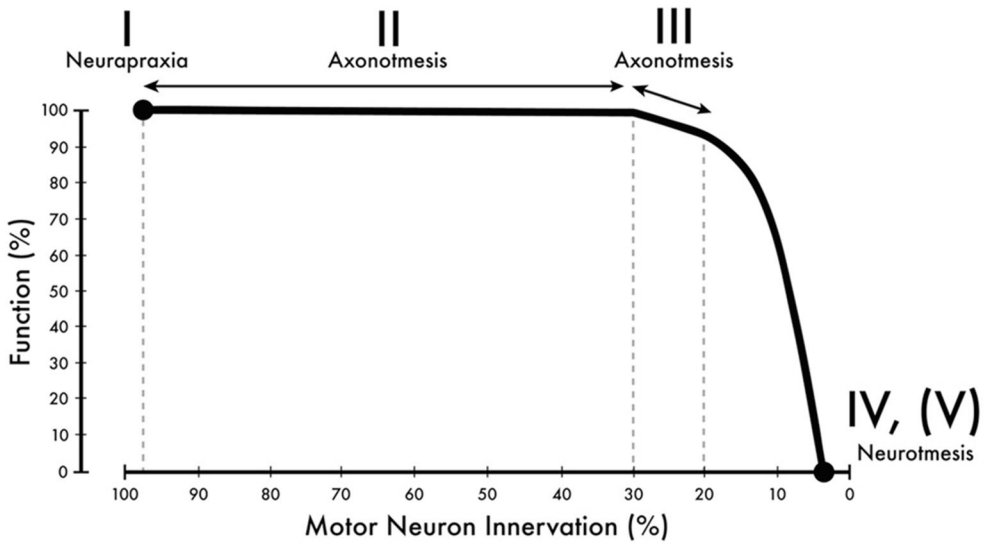

Fig. 2 Graphical representation of muscle function and intact neurons. While the association of intact motor neurons and muscle function is known, the relationship between nerve injury, as described by Sunderland and Seddon classification, and functional muscle strength has not been fully elucidated. In compression neuropathies, long-standing pressure on the nerve leads to neuropraxia, then varying degrees of axonotmesis. As the compression persists, the damage to a nerve's axons increases. In earlier stages, recovery is complete and full if the offending compression is relieved. However, as injury severity progresses (Sunderland IV, V), there is a essentially no ability for functional recovery. Sunderland IV and V injury patterns are very unusual in strictly compressive neuropathies. Adapted from Power et al. (2019) [14]

High ulnar nerve palsies present a challenge in restoring distal intrinsic muscle reinnervation given the distance from the site of nerve injury to the motor endplates of the intrinsic muscles [5,6]. There is essentially a race between axonal reinnervation of the muscles and irreversible motor endplate degeneration. Because of the lengthy distance that nerve regeneration must overcome to reach the intrinsic muscles, some have adopted the use of more distal nerve transfers to the ulnar-innervated intrinsics to speed up the reinnervation process and avoid irreversible motor endplate atrophy.

The AIN-to-Ulnar Motor Nerve Transfer

All patients seeking surgical management of their severe cubital tunnel syndrome should have decompression of their cubital tunnel and Guyon's canal [7]. Additionally, to speed recovery of nerve fibers to the distal hand intrinsics, the use of the most distal branches of the anterior interosseous nerve (a branch of the median nerve) for nerve transfer to the ulnar nerve has been proposed. First described by Mackinnon et al. in 1991 to restore intrinsic function in complete ulnar nerve injury, in an end-to-end fashion, it was later described by Mackinnon et al. in 2009 for use in severe cubital tunnel syndrome in an end-to-side manner [7,8]. The advantage of the so-called supercharged AIN-to-ulnar motor nerve transfer is that it allows for earlier neuro-regeneration of the target muscles while preserving the injured nerve as it heals (which was sacrificed in the end-to-end transfer) [9]. Studies have demonstrated the growth of nerve fascicles through this "reverse end-to-side" coaptation distally to the end-target muscles. Ultimately, these nerve growth cones reach the neuromuscular junctions of the muscle fibers and maintain their viability during the time it will take the native nerve to reach distally [10•].

The supercharged AIN-to-ulnar motor nerve transfer has gained popularity since its inception. In fact, in a recent paper by Domeshek et al., of 670 survey respondents, nearly 72% state they perform nerve transfer surgery. In the same study, when asked how frequently they would use nerve transfer surgery for ulnar motor nerve injury, 33% of respondents reported that they would "always" or "usually" perform a distally based motor nerve transfer [11].

The procedure involves isolation of the motor fascicles of the ulnar nerve roughly 9 cm proximal to the wrist crease at the division of the nerve into the dorsal cutaneous branch, the deep motor branch, and the sensory branch to the ring and small finger. The distal AIN nerve, a branch of the median nerve, is then localized as it enters the pronator quadratus muscle and its branches are isolated. This nerve is ideal as a donor as the pronator teres can compensate for forearm pronation, it is close to the ulnar motor branch, is a motor nerve itself, and obviates the need for interposition graft [7]. The branches of the AIN to the pronator quadratus contain between 500 and 700 nerve fibers while the deep branch of the ulnar motor contains around 1200 at this level [7]. The distal AIN is divided at the pronator quadratus and mobilized towards the deep ulnar motor bundle. Once adequate length of the AIN has been achieved, it is coapted to the side of the ulnar motor bundle through a small epineural window and secured with micro-sutures and/or fibrin glue. As a result, the procedure has earned the nickname of "supercharging" the ulnar nerve (see Fig. 3).

Davidge et al. examined the efficacy of supercharged AIN-to-ulnar motor nerve transfer for ulnar nerve injury in continuity. In their study's cohort, 42% of patients had the diagnosis of compressive neuropathy. Additional diagnoses included traction injuries, gunshot wounds, and motor neuropathy. The study evaluated preoperative and postoperative pinch/grip strength as well as Disabilities of the Arm, Shoulder, and Hand (DASH) questionnaire scores. Mean key pinch (7.5 ± 5.1 preoperatively versus 9.7 ± 4.5 postoperatively; $p < 0.012$) and grip strength (35.7 ± 23.8 preoperatively versus 46.3 ± 20.2 postoperatively; $p < 0.001$) improved postoperatively. Additionally, postoperative DASH scores improved compared with preoperatively (48.2 ± 20.4 versus 38.3 ± 19.1; $p < 0002$). The authors then looked for predictors of intrinsic muscle recovery. They concluded that absent CMAPs preoperatively predicted poor functional outcome of the intrinsic muscles [8].

In a matched-cohort study by Baltzer et al., patients with a high ulnar nerve injury (defined as above the junction of middle and proximal 1/3 of the forearm) who received supercharged end-to-side transfer were compared with matched patients with ulnar nerve injuries, treated with conventional treatment (i.e., primary repair of transected nerve or decompression alone). Both traumatic and compressive etiologies were included. The primary outcome was the return of intrinsic function, with at least 1 year of follow-up. Thirteen patients were included in the cohort group. Results of their research demonstrated that, while initial recover of intrinsic function first appeared at similar times postoperatively (2.9 months for the supercharged group, 3.8 months for the conventional group; $p > 0.2$), the supercharged group had a statistically significant higher rate of improvement of intrinsic function compared with the conventional group (11 of 13 versus 5 of 13, respectively; $p < 0.05$). This finding was more pronounced in transection injuries as opposed to compression injuries. Certainly, multiple factors can account for differences in these patient populations, but a beneficial trend is noted with the use of supercharged end-to-side transfer [12].

To refine candidates that would benefit from such distally based nerve transfers, NCS and EMG are important in guiding the effectiveness of such treatment options in severe cubital tunnel syndrome. NCS and EMG should demonstrate fibrillations of the ulnar nerve–innervated intrinsic muscles (e.g., first dorsal interosseous or abductor digiti minimi) with reduced

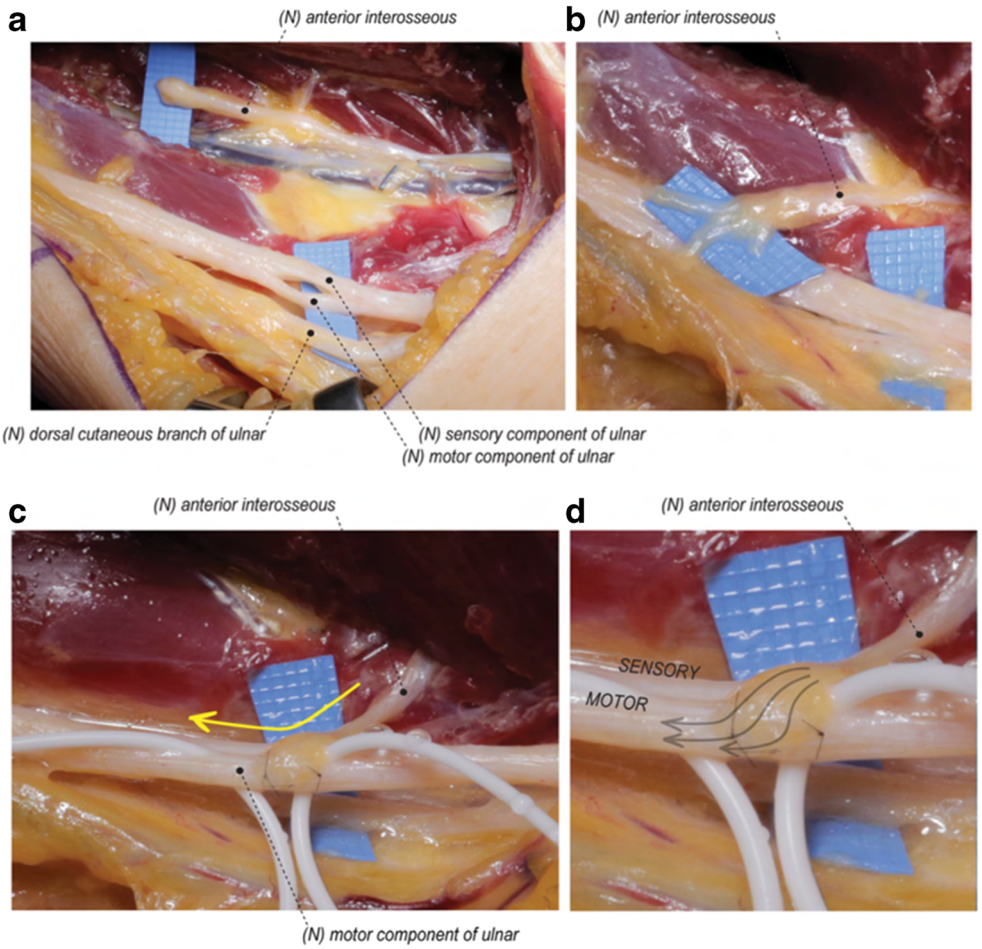

Fig. 3 Intraoperative photographs of distal nerve transfer for severe ulnar neuropathy. **a** The topography of the ulnar nerve at the level of wrist is identified, and the terminal branches of the AIN to the PQ are transected. **b** The AIN is seen lying on top of the ulnar nerve, demonstrating adequate excursion for a tension-free coaptation. **c** The AIN is coapted to the ulnar nerve motor fascicle in an "end-to-side" fashion after creating an epineural window in the ulnar nerve. **d** The AIN fibers are draped over the ulnar motor group, and not the sensory branch. Adapted from Power et al. (2020) [13]

CMAPs. This corresponds to axonotmesis or Sunderland second- or third-degree injury [13••]. In a study by Power et al., CMAP amplitude was found to be a sensitive indicator of axonal loss of the ulnar nerve and predictor of severity of cubital tunnel syndrome [14]. Isolated conduction velocity slowing was not found to correspond to severity. Some have hypothesized that NCS measures the velocity at which nerve signals travel through the fastest remaining functional nerve fibers, whereas motor amplitude is a better assessment of remaining functional axons [15]. Fibrillations on EMG indicate that denervation of the ulnar nerve–innervated muscles has occurred but that the motor endplates remain receptive to reinnervation given the spontaneous activity. In patients without fibrillations and absent CMAPs on electrodiagnostic studies, the disease process is too severe for nerve transfers to be beneficial as the endplates have already been lost (see Fig. 4).

Nerve Transfer for Pinch

In a recent article by Bertelli et al., the authors described an additional nerve transfer to specifically restore thenar punch after proximal ulnar nerve injury. The authors cite that reinnervation of the first dorsal interosseous (FDI) muscle and adductor pollicis (ADP)—which together combine for 75% of the thumb's power for adduction—is unlikely to occur with proximal ulnar nerve repair [16]. Tendon transfers, including sacrificing the extensor carpi radialis brevis and flexor pollicis brevis, offer the surgeon the ability to restore adduction; however, it is often half as strong as the unaffected side [17]. The authors cite that Sallam et al. noted poor reinnervation of the FDI muscle after AIN-to-ulnar motor branch nerve transfer for proximal ulnar nerve lesions [18]. A possible explanation for this poor reinnervation is the relative sparsity of motor nerve fibers in the AIN at the level of the transfer compared with the normal number of nerve fibers in the deep motor branch of the ulnar nerve (500–700 versus 1200 nerve fibers). The authors therefore recommend transfer of the opponens pollicis motor branch from the thenar motor branch of the median nerve to the terminal division of the deep branch of the ulnar nerve. The opponens pollicis branch of the median nerve and the terminal division of the deep branch to the ulnar nerve are then coapted in an end-to-end fashion (see Fig. 5). After surgery, the authors observed reinnervation of the first dorsal interosseus and 80–90% of improvement in pinch strength [16]. Injury to the median nerve from trauma or compression would preclude the use of this transfer.

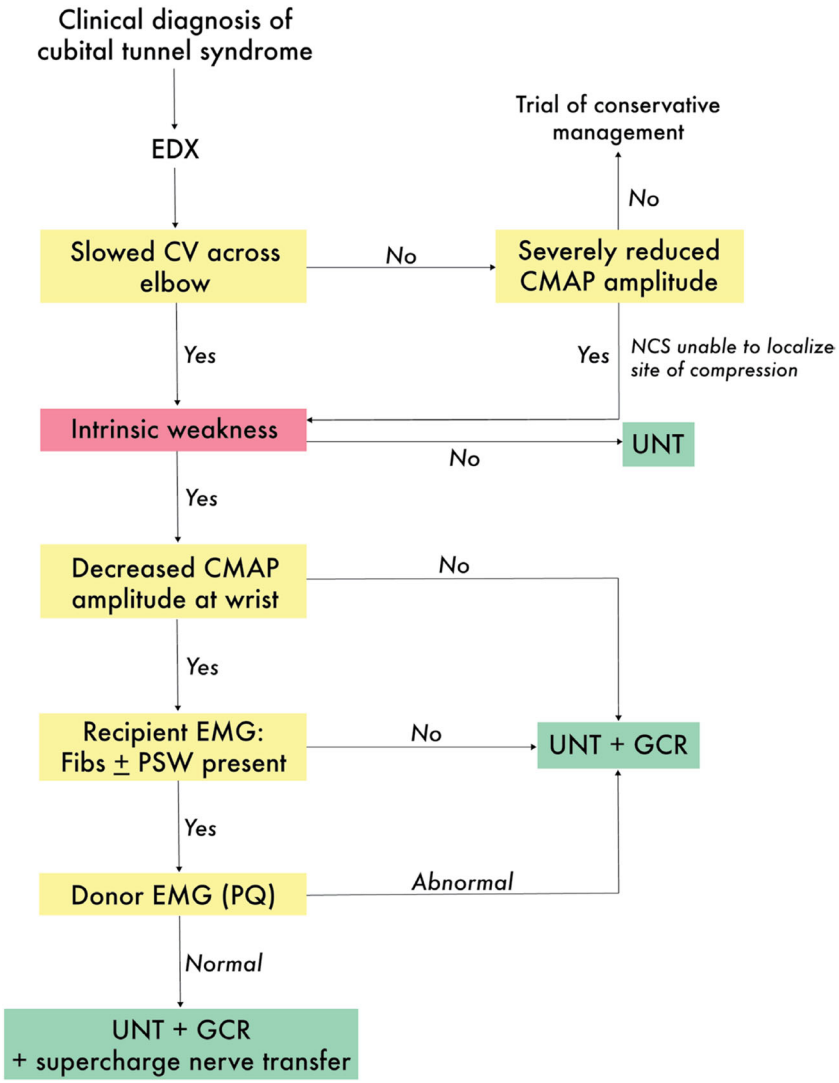

Fig. 4 Algorithm for management of severe cubital syndrome with intrinsic weakness. Developed by Mackinnon and colleagues, the flowchart reveals the importance of electrodiagnostic studies (EDX) combined with physical exam findings in the management of severe compressive ulnar neuropathy. CV, conduction velocity; CMAP, compound muscle action potential; UNT, ulnar nerve transposition; Fibs +/− PSW, fibrillations +/− positive sharp waves; GCR, Guyon's canal release; EMG, electromyography. Adapted from Power et al. (2020) [13••]

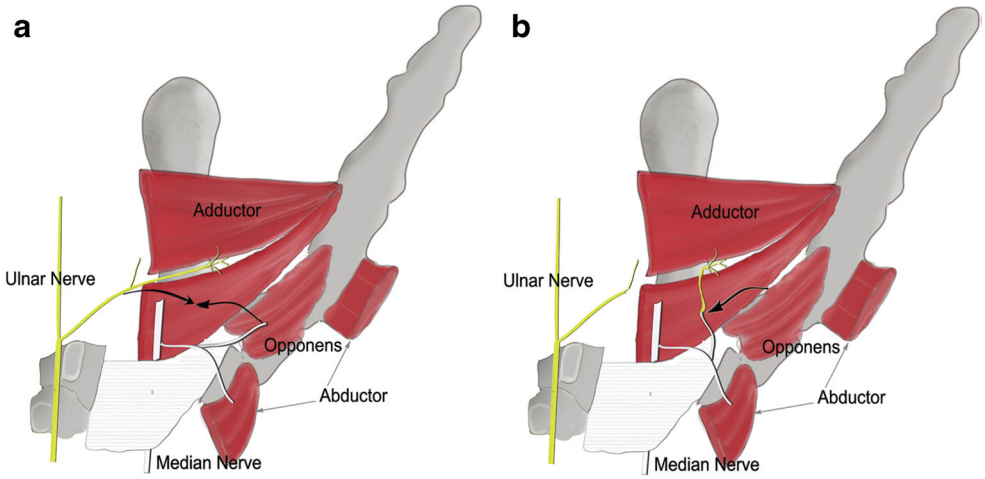

Fig. 5 Schematic representation of the motor branch of the opponens pollicis to the adductor pollicis. **a** The terminal division of the deep branch to the ulnar nerve (TDDBUN) is seen traveling to the transverse and oblique heads of the adductor pollicis muscle. The recurrent branch of the median nerve is seen traveling to the opponens pollicis and abductor pollicis brevis. Following a cadaveric study, it was discovered that the TDDBUN contributed a single motor branch to the transverse head of the adductor and 1 or 2 branches to the oblique head. The recurrent branch contributed 1 to 2 fascicles to the opponens pollicis muscle. **b** The TDDBUN is transected and transferred to the branch of the opponens pollicis muscle. Adapted from Bertelli et al. (2019) [16]

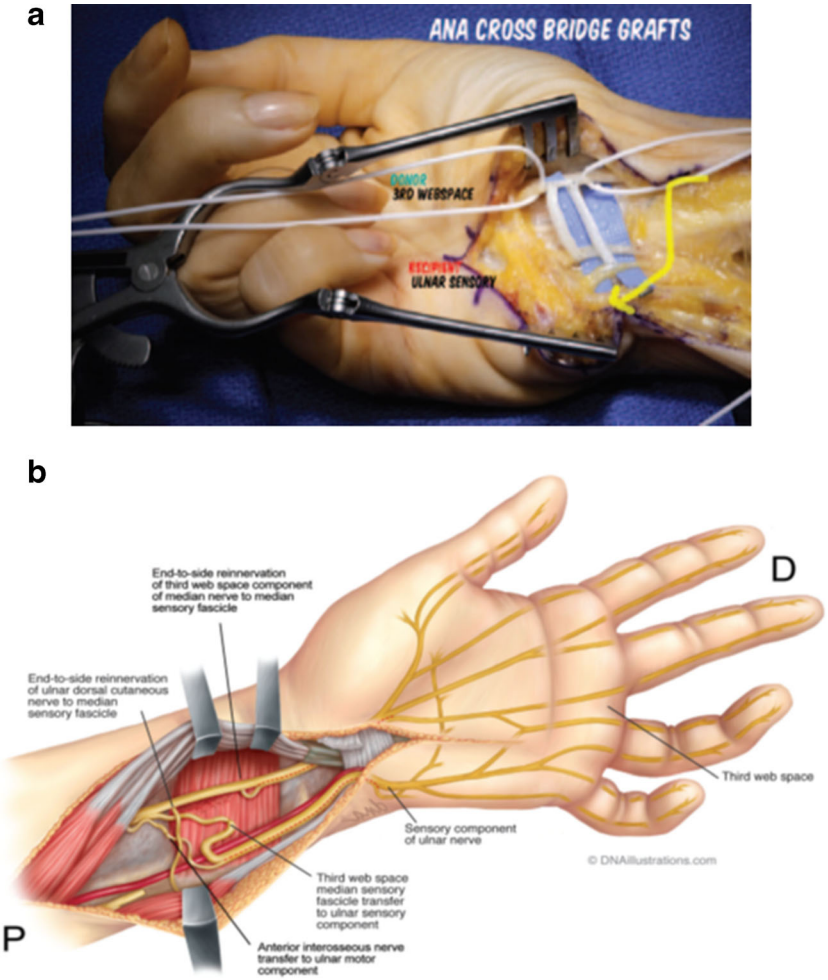

Fig. 6 Intraoperative photographs and Schematic representation of distal nerve transfers to restore ulnar sensory function. **a** Intraoperative photograph demonstrating the use of the 3rd webspace median sensory fascicle transfer to ulnar sensory fascicles. The 3rd webspace fascicles are located distally and traced proximally. Two perineural windows are then created in the nerve at the level of the carpal tunnel as well as in the ulnar sensory branch in Guyon's canal. The nerves are then bridged across the palm using autograft (e.g., MABC nerve) or acellular allograft. **b** Restoration of dorsal ulnar protective sensation. The dorsal cutaneous branch of the ulnar nerve is identified, usually branching 6–8 cm proximal to the ulnar styloid, and mobilized. The nerve is transected proximally and coapted in an end-to-side fashion to the median nerve in the distal forearm. This procedure can be done in conjunction with other distal median-to-ulnar transfers, but the authors of this study stress the importance of first addressing restoration of intrinsic function and regaining protective palmar sensation of the ulnar-sided digits [7]. Adapted from Felder et al. (2019) [21•] and Brown et al. (2009) [7]

Sensory Nerve Transfers

Advanced ulnar neuropathy from cubital tunnel syndrome of course also affects sensory nerves resulting in paresthesias or anesthesia in the ring and small fingertips. Patients frequently lack protective sensation to the ulnar border of their hand as well. While motor function restoration to the hand intrinsics remains the primary goal in nerve transfer surgery for such patients, there do exist options for sensory nerve transfer in the severe cubital tunnel patient. For those with irrecoverable nerve injury, sensory donor nerve autograft can be taken from the lateral antebrachial cutaneous nerve, the palmar cutaneous nerve, or the third webspace component of the median nerve. However, this obviously leaves donor site numbness. If there is a possibility of ulnar sensory recovery, a side-to-side procedure has been shown to be possible [19]. This was first demonstrated in humans by Yüksel et al. in a traumatic ulnar nerve lesion case in a report published in 2004. In their patient, a large portion of the ulnar was necrosed requiring sural nerve cable grafting to fill the void. Then, a side-to-side neurorrhaphy of the median and ulnar nerve was performed distal to the autograft to expedite the recovery of the ulnar nerve. Their patient regained protective sensation in the ulnar nerve distribution 9 months postoperatively [20]. The technique was later refined as described by Felder et al. The median and ulnar nerves are exposed at the palm. Epineural windows are created in two separate locations in the portion of the median nerve to the third webspace at the level of the carpal tunnel [21•]. Two additional windows are created in the ulnar sensory nerve at the level of Guyon's canal. The two windows

on each nerve are connected to one another by nerve grafts, either allograft or autograft if available [7].

In a 2019 study by Felder et al., outcomes for 24 patients who underwent the cross-palm nerve graft for ulnar sensory recovery were examined. All patients had preoperative severe ulnar neuropathy, with loss of protective sensation to 2-point discrimination > 8 mm or Semmes-Weinstein monofilament testing > 4.56 on the ulnar-innervated digits. All patients underwent procedures to repair or decompress the ulnar nerve as well as side-to-side cross-palm nerve grafting, as described above. Twenty-one of the 24 patients had return of protective sensation, 16 had return of diminished sensation to light touch, and 6 had return to normal sensory function. DASH scores improved to 30.1 postoperatively from 60.1 preoperatively. No patients experienced a worsening of their ulnar nerve sensation, and there were no reported cases of loss of median nerve sensory loss [21•].

Brown et al. also describe transferring the dorsal ulnar cutaneous nerve to the median nerve in an end-to-side fashion to restore dorsal ulnar sensation (see Fig. 6). The dorsal ulnar cutaneous nerve is localized and mobilized. It is then transected proximally. An additional epineural window is made on the ulnar side of the median nerve to a sensory fascicle. The dorsal cutaneous branch of the ulnar nerve is coapted in an end-to-side fashion with nylon suture and fibrin glue. It is noted in the paper that the supercharged AIN-to-ulnar motor branch and third webspace cross-palm transfers should be completed first as they are more beneficial to patient recovery and function. It is believed that sprouting of the median nerve occurs and regenerates down the dorsal ulnar cutaneous to restore sensibility to the ulnar hand. This would preclude any chance of regeneration originating from the native ulnar nerve proximally [7, 22].

Despite the promising results of nerve transfers for ulnar sensory loss, in Domeshek's study, surgeons were less likely to use sensory nerve transfers than for motor transfers in cases of proximal ulnar nerve injury. Only 12% of surveyed surgeons reported to "always or usually" using sensory nerve transfers for proximal ulnar injuries compared with 33% for ulnar nerve motor dysfunction [11].

Conclusion

Nerve transfers have been used for proximal ulnar nerve injuries for some time with reproducible results, especially in the reinnervation of hand intrinsic muscles. More recently, such techniques have been used on severe compressive ulnar neuropathy at the elbow. The rationale of such nerve transfers is founded on the concept that nerve regeneration from the elbow to the ulnar-innervated hand intrinsic is too slow and thereby allows for nerve endplate loss before the nerve reaches its target, causing irreversible muscle atrophy. By transferring a healthy donor nerve such as terminal branches of the AIN, the distance or nerve regeneration to occur is significantly decreased and allows for reinnervation before muscle atrophy occurs. Ideal candidates for such a procedure should have electrodiagnostic evidence of ulnar neuropathy at the elbow and decreased CMAP amplitude in ulnar-innervated muscles, as well as fibrillations at rest. These findings suggest denervation of the target muscle, but not complete loss of motor endplates, indicating that the muscle can still be reinnervated.

Although most nerve transfers performed for severe cubital tunnel syndrome are focused on recovery of motor function, there are sensory nerve transfers that exist for restoration of sensibility in the affected distribution.

More studies are required looking into long-term outcomes of nerve transfers. However, at present time, they offer promising results for both motor and sensory nerve regeneration without sacrificing significant pronation strength or sensation to a crucial portion of the hand. Studies have confirmed that, compared with ulnar nerve decompression alone or nerve grafting of a diseased portion of nerve, nerve transfers offer improved functional results.

Compliance with Ethical Standards

Conflict of Interest The authors have reported no conflicts 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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- •• Of major importance

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