Management of Ulnar Nerve Injuries

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Injuries to the ulnar nerve result in both sensory and motor deficits within the hand. Functional outcomes following repair of this nerve have not performed as well as outcomes following repair of the median or radial nerves. Advances in imaging modalities may provide earlier means of identifying and diagnosing closed nerve injuries. Early neurorrhaphy of acute nerve injuries provides the best outcome, but consideration should also be given to performing distal motor nerve transfers to preserve hand intrinsic motor function when injuries occur at or above the proximal forearm. This article attempts to summarize the most recent trends within ulnar nerve repair. *(J Hand Surg Am. 2015;40(1):173–181. Copyright © 2015 by the American Society for Surgery of the Hand. All rights reserved.)*

Key words Ulnar nerve, nerve repair, nerve injury, outcomes.

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ULNAR NERVE INJURIES CAN result in paresthesia, dysesthesia, and muscle weakness in the affected hand. According to the Nationwide Inpatient Sample database from the Healthcare Cost and Utilization Project, ulnar nerve injury was the most frequent major upper extremity peripheral nerve injury resulting in hospital admission from 1993 to 2006 when compared with median, radial, and brachial plexus injuries.[1] The demographics of patients with ulnar nerve injuries are disproportionately male in the working age group (18–45 y) with a median income of $36,000.[1] It is estimated that the health care cost associated with ulnar nerve injury is $10,563 to $42,000 per individual, with higher costs associated with injuries requiring concomitant tendon repair.[1,2] An additional intangible cost to society may also be attributed to the loss of work productivity for the injured individual.[2]

For better outcomes, ulnar nerve injuries require early repair compared with other peripheral nerve injuries.[1] Unfortunately, repair of this nerve often results in incomplete recovery with functional results that are inferior to those achieved following radial nerve and median nerve repair.[3,4] This article attempts to summarize the most recent trends within the management of ulnar nerve repair.

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ANATOMY

The ulnar nerve is the terminal branch of the medial cord of the brachial plexus. This cord contains nerve fibers from C7 to T1 nerve roots. It lies medial to the brachial artery in the upper arm and exits the posterior compartment of the arm as it descends down the humerus to enter the anterior compartment through the medial intermuscular septum. It continues anterior to the medial head of the triceps brachii muscle to enter the cubital tunnel posterior to the medial epicondyle, medial to the elbow joint capsule and the medial collateral ligament. After exiting the cubital tunnel, the ulnar nerve gives off 2 or 3 muscular branches to the flexor carpi ulnaris (FCU) muscle. The ulnar nerve passes beneath the humeral and ulnar heads of the FCU muscle to enter the volar aspect of the forearm, where it continues deep to the flexor pronator aponeurosis. In the forearm, the nerve passes anterior to the flexor digitorum profundus (FDP) muscle to supply motor branches to the ulnar half of FDP. The ulnar nerve travels the remaining length of the forearm between the FDP and the flexor digitorum superficialis (FDS) muscles (Fig. 1).

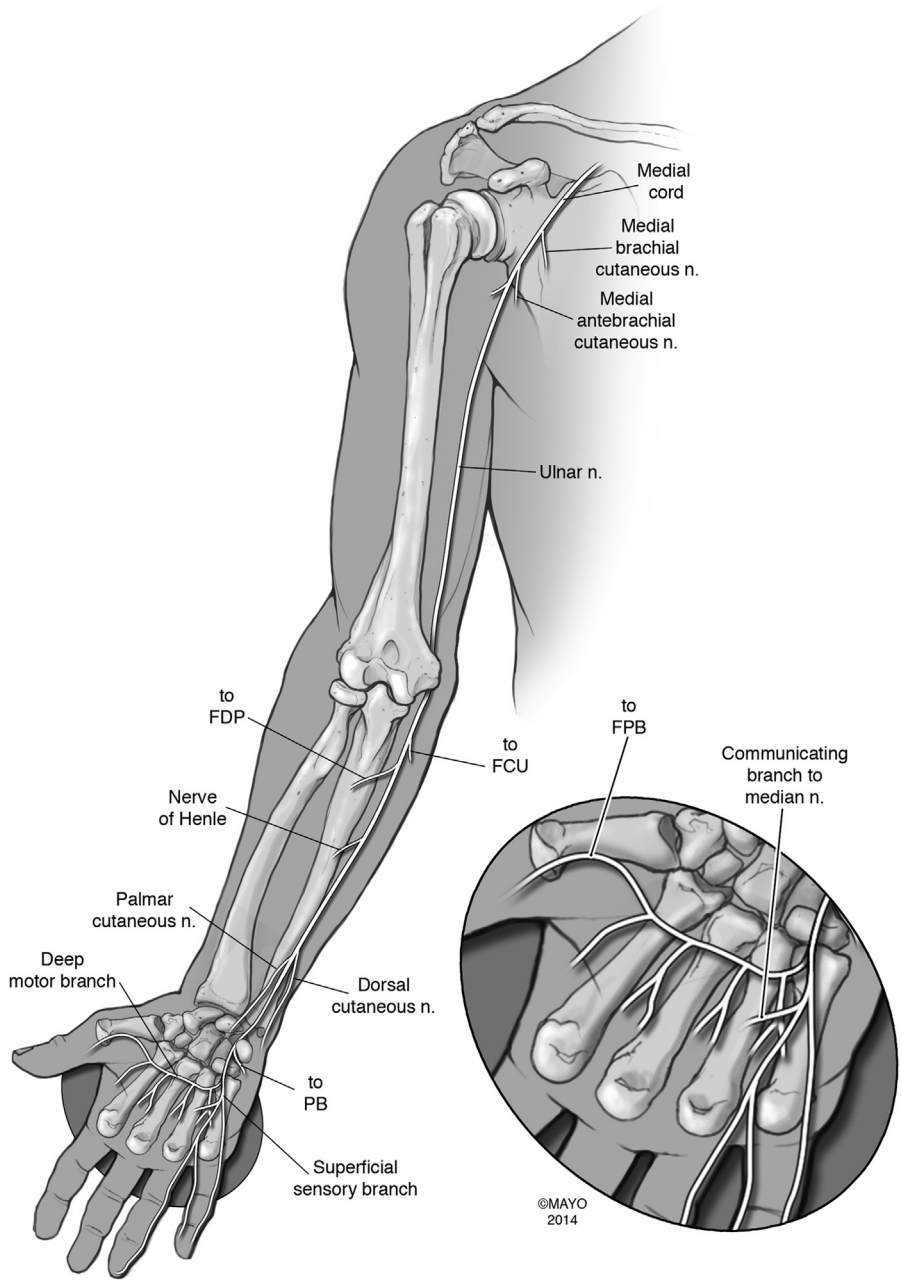

FIGURE 1: The ulnar nerve contains fibers from C7−T1. After traveling through the cubital tunnel, it supplies the FCU muscle and motor branches to the ulnar half of the FDP muscle. The ulnar nerve then travels the length of the forearm between the FDP and the FDS muscles; sympathetic innervation to the ulnar artery is supplied through the nerve of Henle. A palmar cutaneous branch may also exit the ulnar nerve within the distal forearm to supply sensation to the skin overlying the hypothenar area. PB, palmaris brevis; FPB, flexor pollicis brevis. Inset: The deep branch of the ulnar nerve innervates the interosseous muscles, adductor, and deep head of the flexor pollicus brevis muscle. (Copyright © Mayo Foundation 2014.)

The *dorsal cutaneous branch* of the ulnar nerve arises from the medial aspect of the ulnar nerve approximately 8 cm proximal to the pisiform bone. It provides dorsal branches to the small finger, the ulnar aspect of the ring finger, and the ulnar aspect of the carpus and hand. Palmarly at the level of the wrist, the ulnar nerve bifurcates into 2 main branches at the distal aspect of Guyon's canal; these branches are the *superficial sensory* and *deep motor* (Fig. 2). The deep motor branch passes deep between the flexor and the abductor digiti minimi muscles and continues laterally between the superficial and the deep layers of the opponens digit minimi. The deep motor branch then continues laterally through the deep palmar space to innervate the intrinsic and thumb adductor muscles.[5–7] The superficial branch of the ulnar nerve gives off 2

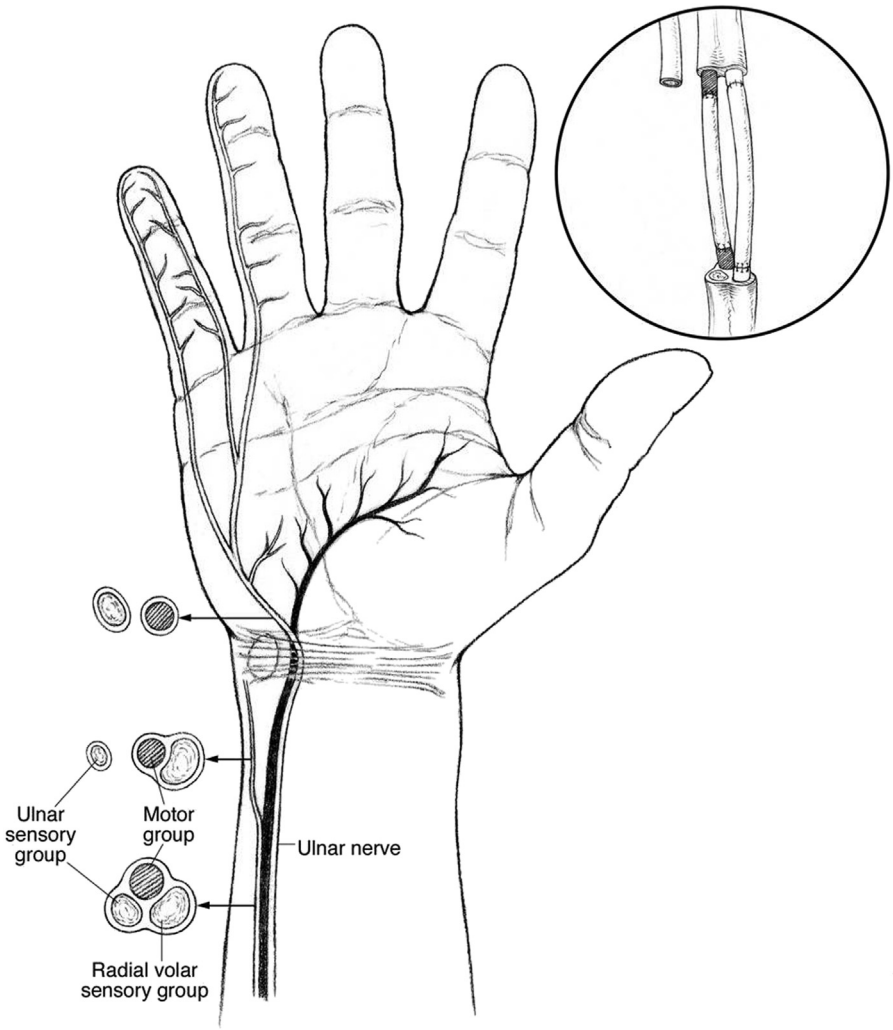

FIGURE 2: The ulnar nerve bifurcates into the superficial sensory and deep motor branches in the distal portion of the Guyon canal. Understanding of the topographical anatomy of the ulnar nerve is essential for adequate repair of injuries at the level of the wrist and for performing nerve transfers. Proper matching of the major braches provides the best chance for restoration of sensory and motor function. Inset: Cabled nerve grafts in this area should attempt to match like fascicles. (Copyright © Mayo Foundation 2007.)

small motor branches to the palmaris brevis muscle.[8] The superficial trunk gives off 2 sensory branches: the fourth common digital nerve and the ulnar proper digital nerve to the little finger. A communication may exist in the palm between the recurrent branch of the median nerve and the deep branch of the ulnar nerve, known as the Riche-Cannieu communication.[9]

Internal topography of the ulnar nerve is of particular importance when performing grouped fascicular repair and distal nerve transfers (Fig. 2). Internal topography has been most extensively studied in the proximal forearm and at the wrist. In the proximal forearm, the sensory group of fascicles that innervates the palmar ring finger and little finger is found on the radial side of the ulnar nerve. A smaller fascicular group for the deep motor branch of the ulnar nerve is found just ulnar to the sensory component; at the distal forearm, the motor fascicular group is at the ulnar-dorsal position and constitutes only 30% to 35% of the total fascicular cross-sectional area of the ulnar nerve.[10] The dorsal cutaneous branch lies just ulnar to the deep motor fascicles. A cleavage plane that can be easily identified under microscopic magnification separates these fascicular groups (Fig. 3).

CLINICAL FINDINGS

Ulnar nerve injuries can be broadly divided into low injuries and high injuries. In low ulnar nerve injuries, the nerve is damaged distal to the motor branch of the FCU and motor branch to the FDP of the ring and little fingers. In low injuries, sensibility to the palmar ulnar hand is lost and paralysis occurs usually to all 7 interosseous, the ulnar 2 lumbrical, the 3 hypothenar,

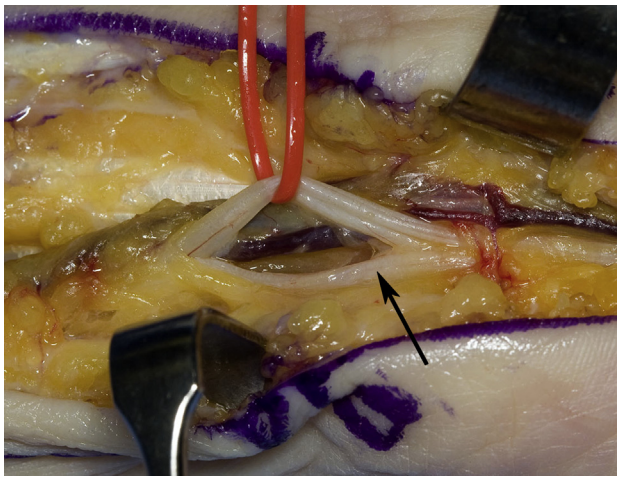

FIGURE 3: Cleavage plane within the ulnar nerve at the level of the distal forearm. The arrow points to the smaller ulnar motor branch, while the sensory branch is retracted radially.

the adductor pollicis, and the deep head of the flexor pollicis brevis muscles. Sensibility to the dorsum of the hand may be intact if the lesion has occurred distal to the takeoff of the dorsal ulnar sensory nerve. In high injuries, the nerve is damaged above the origin of the motor branch of the FCU and FDP muscles.[11]

The loss of intrinsic muscle function results in an inability to flex at the metacarpal (MCP) joints and extend the interphalangeal (IP) joints. This results in the development of the intrinsic-minus or claw posture of the ring and little fingers, where there is hyperextension at the MCP joints and flexion at the IP joints (Duchenne's sign). The development of clawing does require the presence of intact extrinsic extensor and flexor tendons function; thus, a high ulnar nerve injury results in less claw hand deformity because of the loss of FDP function.[12] The normal dynamics of finger flexion are substantially altered with the claw hand deformity as the affected IP joints flex before the MCP joints, resulting in objects being pushed out of the palm by the fingertips. The intact lubricales of the long and index fingers can initially prevent clawing in the radial digits; however, with progressive loss of MCP flexion strength and capsular laxity, clawing may develop in the entire hand over time.[13]

In addition, intrinsic weakness leads to decreased pinch and grip strength. With paralysis of the adductor pollicis, deep head of the flexor pollicis brevis, and first dorsal interosseus muscles, there is an 80% loss of pinch strength.[14] Thumb pulp to index pulp pinch leads to excessive flexor pollicis longus contraction, and development of the Froment sign and thumb MCP hyperextension (Jeanne's sign) if MCP joint laxity is present. Finger abduction is absent and loss of the palmar interosseus muscle function leads to abduction of the little finger, due to the unopposed pull of the extensor digiti minimi (Wartenberg's sign).[12]

Variation in ulnar innervation patterns is common and may have an effect on clinical presentation. Within the hand itself, variability may exist with regard to the innervation of finger sensation. Don Griot and colleagues[15] have noted, in a study of 43 patients with either complete ulnar or median nerve injuries, that the classic watershed area noted between the ulnar and the radial aspects of the ring finger was present in only 23% of hands. A transition zone of diminished, rather than lost, sensibility was noted at the radial side of the ring finger in 33% of patients who sustained a transection of the median nerve and an intermediate zone of sensibility was noted on the ulnar side of the ring finger in 61% of patients who had sustained a transection of the ulnar nerve.[15] Variation in classic nerve sensibility patterns can be explained by the presence of a communicating branch of ulnar to median or median to ulnar nerve occurring at the level of the midpalm. Such variation may produce diminished or normal sensation within portions of the little and ring fingers following ulnar nerve injury, which could potentially confuse the monitoring of postoperative recovery.[16]

In addition to distal sensory variation, approximately 15% of patients should be presumed to have a motor communication between the median and the ulnar nerves within the forearm (the Martin-Gruber).[9,17,18] Classically, this communication allows muscles normally supplied by the ulnar nerve to be innervated by the median nerve; however, combined motor and sensory median to ulnar connections have been reported.[19] Thus, injuries within the forearm can create a heterogeneous pattern of motor and sensory deficits within the hand, necessitating a detailed examination prior to surgical treatment.

DIAGNOSIS

The mechanism of ulnar nerve injury (excluding compression neuropathy) in order of frequency includes laceration, stretch, and contusion.[9] A careful history is essential, particularly in cases of blunt trauma, because it is critical to differentiate between neurapraxia and axonotmesis (which can be treated without surgery) and neurotmesis, which requires surgical intervention.[20] Electrodiagnostic testing is critical in determining the level of injury; however, nerve conduction velocity studies and the results of electromyography (EMG) can be limited in cases of severe axonal loss or early after

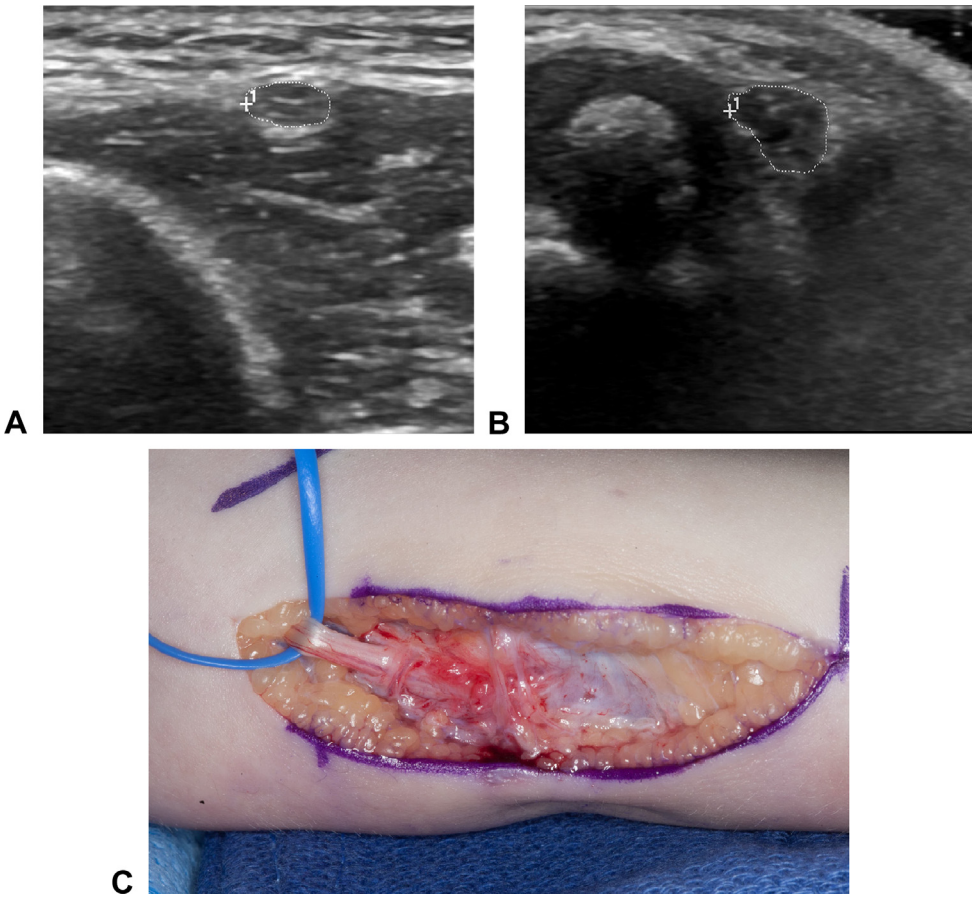

FIGURE 4: Images of a 5-year-old girl with persistent ulnar palsy 6 months following a closed reduction and percutaneous pinning for a supracondylar fracture. The ulnar nerve is imaged with cross-sectional ultrasound above **A** and within **B** the cubital tunnel. A change in the hypoechoic signal within the nerve indicates injury. **C** The epineurium is visualized and suggests the nerve may be in continuity. Surgical exploration found tethering of the nerve at the level of the fracture. Intraoperative nerve evaluation noted functional NAPs; thus, neurolysis was all that was required in this case.

injury, when neurapraxia cannot be discerned from neurotmesis. EMG can also be limited by pain and an inability to identify anatomical variability.[15]

Recently, high-resolution ultrasound (US) has been proposed as a diagnostic modality to aid in identification of peripheral nerve disruption. US imaging can now show individual nerve fascicules, and most peripheral nerves can now be depicted along their entire anatomical course (Fig. 4).[21] High-resolution US can reveal discontinuity of the nerve, perilesional scar tissue, and presence of a neuroma with an accuracy of up to 93%.[22] US is portable and economical and can be performed during surgery.[23] In more complex injuries, imaging with magnetic resonance imaging (MRI) or computed tomography (CT) myelogram may help identify etiology such as nerve root avulsion injuries, muscle denervation, nerve edema, sites of compression, and nerve disruption.[20,24]

Intraoperative testing of nerve action potentials (NAPs) can be a helpful modality in determining the status of injuries where the nerve is in continuity[4,9] (Fig. 3). When performing intraoperative NAP studies, stimulating and recording electrodes are placed on the nerve proximal to the lesion to assess the normal NAP; then the recording electrodes are moved into the region of injury, and then distal to the lesion. If an NAP is present through the area of trauma, external neurolysis, with or without internal neurolysis, is an appropriate treatment (Fig. 4).[25] When no NAP is recorded across a lesion in continuity, the area represents a nonfunctional neuroma; in these cases, resection and repair of the injured nerve is usually indicated.[4,25]

TREATMENT

In acute open ulnar nerve injury, immediate exploration and primary neurorrhaphy is recommended as long as the repair can be achieved under minimal tension. For contaminated wounds, for which immediate repair

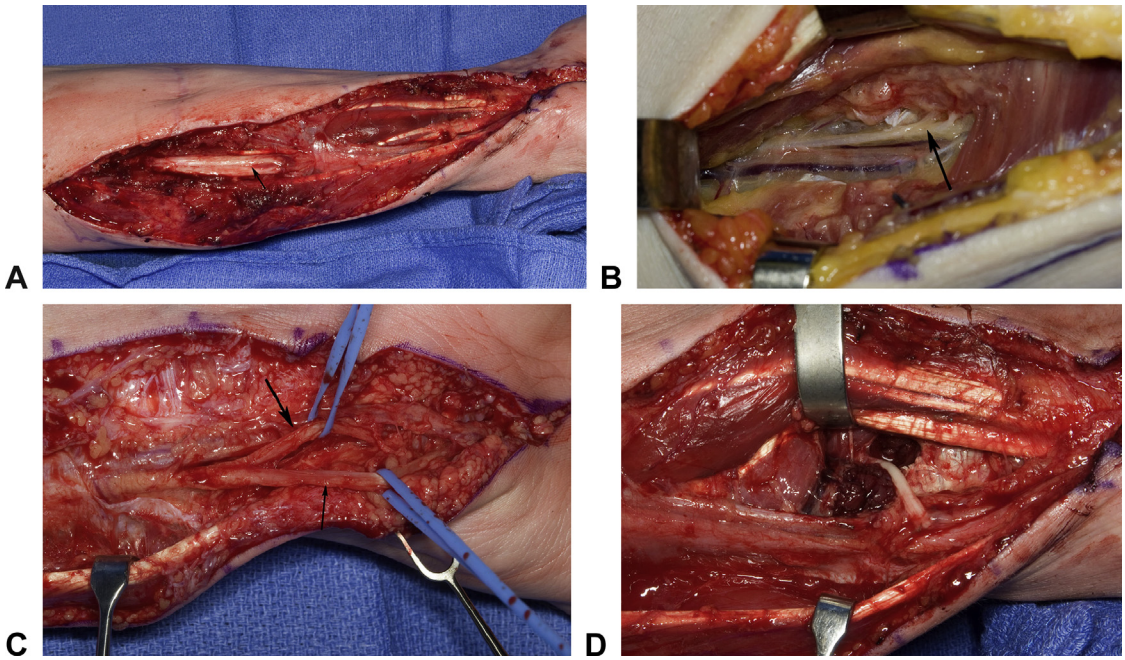

FIGURE 5: A 31-year-old firefighter suffered a crush injury to the ulnar aspect of the arm with development of ulnar palsy and clawing. The patient presented to our institution 4 months after the original injury with no improvement in function and no evidence of advancing Tinel. Urgent exploration and intraoperative NAP study revealed a large nonfunctional neuroma. **A** Resection of neuroma was performed with cable nerve grafting (arrow). Because of the time elapsed from the original injury and the location of the neuroma, an end-to-side AIN transfer was performed to the ulnar motor branch. **B** Image of the AIN nerve dissected through the pronator quadratus (arrow). **C** Intraoperative image of the motor branch (small arrow), which has been separated from the sensory branch (large arrow) in the forearm. **D** Intraoperative image shows the AIN mobilized and located next to the motor branch, allowing for end-to-side repair without interposition grafting. Video 1 (available on the *Journal's* Web site at www.jhandsurg.org) shows the patient's results at 1 year after surgery with recovery of interosseous function, loss of clawing, but a persistent Froment sign.

would be imprudent, a delayed repair can be performed ideally in less than 72 hours, but up to 7 days without detriment to outcome.[26–28] Delays in repair increase the likelihood for nerve grafting, neuron loss, and fibrosis of the distal stump.[26] It is essential that the zone of injury is resected to a normal nerve tissue prior to repair. Following preparation of the nerve stumps, tension may be reduced by judicious mobilization of the nerve ends. Excessive mobilization may lead to distal stump ischemia and compromise recovery.[29] Anterior ulnar nerve transposition has historically been suggested as a means of decreasing tension at the repair site for high injuries; however, recent studies by Abrams and colleagues[30] have suggested that the post-transposition nerve gap may not be considerably altered with anterior transposition in combination with elbow flexion to 45°.[31] In addition, transposition may create ischemia and scarring around the recovering nerve ends. Despite the controversy surrounding anterior transposition, wrist flexion has been shown to considerably lower gap distances for nerve repairs in the distal forearm.[30]

High ulnar nerve injuries traditionally have had poor outcomes with regard to intrinsic muscle recovery. Once the nerve has been injured, the motor end plates begin the process of degeneration. Functional recovery is determined by the time required for the motor end plate to be reinnervated and by the number of regenerated motor axons that can reach target muscle.[32] To decrease the time to reinnervation within the hand and preserve motor end plates in high injuries, distal nerve transfers have been recommended as an alternative to restore intrinsic function. The anterior interosseous nerve (AIN) transfer to the ulnar motor branch was described in the late 1990s.[33] Most recent reports have noted functional recovery in 70% to 100% of patients.[34–37]

Originally this motor nerve transfer was described as a direct end-to-end transfer, prohibiting any potential ingrowth from the proximal nerve; however, more recent techniques use the AIN as an end-to-side fashion, allowing for recovery of proximal fibers and augmentation from the end-to-side transfer. Experimental models have shown that axonal regeneration can occur across end-to-side nerve coaptation. Mackinnon and colleagues[38] have termed these transfers "supercharged end-to-side nerve transfers" (SETS) and perform them as a means of preserving distal motor

end plates until the native axons fully regenerate and to augment the regenerating nerve. A SETS augmentation with AIN motor branch can be considered for midlevel injuries near the elbow or in high ulnar nerve injuries with a Martin-Gruber communication.[38] Long-term and comparison outcomes studies for this method are lacking (Fig. 5).

Additional techniques have been designed to prevent intrinsic atrophy during proximal nerve recovery. Magdi-Sherif and Amr[39] describe the use of interpositional nerve grafts placed distally between the ulnar and the median motor branches. At 4 months following surgery, 3 of their 4 patients had evidence of intrinsic function.

Options for sensory recovery when primary repair has failed, or where reconstruction is impossible, are limited. Boyd and Mackinnon[40] have described the transfer of expendable sensory nerves from the median nerve to restore ulnar nerve function of critical areas. End-to-end nerve transfers can be performed between the third web space fascicles of the median nerve to the superficial sensory branch of the ulnar nerve at the level of the wrist. End-to-side transfer of the dorsal cutaneous nerve into the median nerve may be used to restore dorsal hand sensibility.[40]

Tendon transfer for ulnar nerve palsy is beyond the scope of this review but may be indicated for correction of clawing, weak thumb-index finger pinch and abduction deformity of the little finger and for correction of weak FDP flexion at the ring and little fingers. The time to perform tendon transfers is debatable. Historical results of distal motor recovery were so poor that some authors recommended tendon transfer at the time of proximal nerve repair.[41] Trevett and colleagues,[42] in their study of 50 isolated ulnar nerve injuries, found that tendon transfers are rarely indicated in sedentary workers. Patients should be reminded that improvement in hand function may be seen for up to 5 years following repair.[42,43] Tendon transfer should be reserved for manual laborers who have residual poor grip and key pinch 12 to 24 months following nerve surgery.[42]

OUTCOMES

Successful outcomes following ulnar nerve repair are defined by the recovery of M4 strength and S4 sensation. In evaluating outcomes, it is important to remember that function can improve for up to 5 years following repair.[43,44] Ongoing improvement may be due to continuing nerve regeneration and innervation but also psychological adaptation to the new patterns of innervation.[43] In general, more proximal injuries do worse, as do repairs requiring nerve grafts and those done in a delayed fashion.[44,45] Kim and colleagues,[25] in a study of 654 ulnar nerve lesions, have shown that functional recovery of grade M3 or better motor recovery is greatest when the injury requires only neurolysis (92% of patients), followed by primary repair (72% of patients), and then those injuries requiring nerve grafting (67% of patients). Ruijs and colleagues,[44] in their meta-analysis of 23 publications, found that ulnar nerve injuries gave a 71% lower chance of motor recovery than median nerve injuries.

Age at the time of repair has consistently shown to significantly affect the outcome.[44,46] Most recently, Chemnitz and colleagues[47] examined the 30-year outcomes of median and ulnar nerve repair performed in children and adolescents. Recovery was significantly better in those who had injuries repaired in childhood (< 12) as opposed to adolescence. On average, children recovered 87% of normal function compared with 67% in adolescents.[47] Reasons for improved outcome in children have been attributed to be shorter recovery distance, accelerated nerve growth, and increased brain plasticity allowing for better incorporation of aberrant sensory input.[48–50]

Earlier studies by Gaul[46] and others[51,52] have established outcomes against which newer studies must be compared. In 1982, Gaul[46] studied intrinsic motor recovery in 41 patients with ulnar nerve laceration. Mean key pinch returned to within 86% of normal in younger patients (< 18) and 82% of normal in older patients with low level injuries, whereas in adults with high-level lesions, no significant motor recovery was noted.[46]

Recently, Vordemvenne and colleagues[3] reported the outcomes in 65 patients with 71 complete median and/or ulnar nerve lacerations 8 years after injury. Seventy percent of the injuries occurred at the level of the wrist. Return of motor function was worse with ulnar repairs than with median repairs. Patients who had primary reconstruction of their ulnar nerves recovered S3+ sensibility in 20% of cases, S3 in 20%, and S2 in 55%. No patients recovered S4 sensibility; however, 55% of patients were able to recover M3 strength, 20% obtained M4, and 5% obtained M5. Average final Disorders of the Arm, Shoulder and Hand (DASH) score was 22 (DASH score ranges from 0 to 100, with lower scores being better).[3]

Galanakos and colleagues[53] also recently examined the prospective outcomes of 73 patients undergoing primary repair of sharp lacerations of the median and/or ulnar nerves. Patients were followed for over 3 years. Median nerve repairs fared better than ulnar nerve repairs for sensory and motor recoveries. Factors influencing outcome included the nerve injured followed by age, the level of injury, and concomitant injuries.[53]

Results of ulnar nerve repair can be maximized with both sensory reeducation and hand therapy. Sensory reeducation techniques have been widely applied in adults following nerve repair, and such therapy has been significantly favorably correlated with return to work.[54–56] In a recent study by Bruyns and colleagues,[57] 59% of workers were able to return to employment at an average of 30 weeks following isolated ulnar nerve injuries. White collar workers were able to return to work on average 10 weeks earlier than blue collar workers. Higher educational status, compliance with therapy, distal injuries, and better postoperative grip and pinch strengths tended to be positive predictors for earlier return to work.

In conclusion, functional outcomes after ulnar nerve injuries are difficult to achieve, partially because intrinsic muscle function is difficult to recover. Nerve lacerations should be repaired as soon as possible to maximize functional return and avoid the use of nerve grafts. In cases in which there is a neuroma in continuity at surgery, EMG, high-resolution US, and intraoperative NAP studies can help determine the degree of injury and guide treatment. Distal nerve transfers may be considered in settings in which the injury is proximal to the elbow to improve distal motor recovery. End-to-side "supercharging" of the motor branch may also prove to be an effective means of restoring intrinsic function. Full recovery from ulnar nerve injuries may take up to 5 years. Tendon transfer should be considered as a second-line treatment when motor loss is deemed permanent after nerve repair.

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