



Grand Rounds from HSS

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Innovative Options for Patients with Devastating Upper Extremity Injuries

Edward C. Jones, MD, MA
Editor

The HSS Center for Brachial Plexus and Traumatic Nerve Injury epitomizes the surgical expertise, collaboration, and multimodal techniques necessary to properly diagnose and treat very complex conditions. The 3 cases presented in this issue highlight teamwork as an essential aspect of ensuring good outcomes for patients with devastating injuries to the brachial plexus and upper extremity.

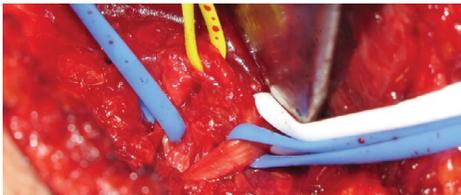
A meticulous diagnostic process begins with physical examination and the identification of subtle, elusive physical signs. These findings serve to direct a purposeful, multidisciplinary patient evaluation using sophisticated electrodiagnostic testing, nerve-specific high-resolution magnetic resonance imaging (MRI), and ultrasonography. Surgical planning involves collaboration among specialties and in some cases 2 surgical teams operating simultaneously, using multiple approaches.

All 3 cases were authored by **Drake G. LeBrun, MD, MPH, Darryl B. Sneag, MD, Joseph H. Feinberg, MD, Ogonna K. Nwawka, MD, Steve K. Lee, MD, and Scott W. Wolfe, MD**. In the first case, multiple surgical windows were used for neurolysis and nerve transfer for iatrogenic axillary nerve injury following the arthroscopic repair of a humeral avulsion of the glenohumeral ligament. In the second case, high-resolution MRI was used to pinpoint anterior interosseous fascicles of the median nerve and to guide surgeons in decompressing fascicular constrictions using micro-internal neurolysis to treat anterior interosseous nerve syndrome resulting from Parsonage-Turner syndrome. In the third case, 2 surgical teams used multiple approaches to achieve triple nerve transfer and sural nerve grafting to restore function to a patient with an extensive brachial plexus injury.

We hope you find these cases to be of interest and the principles presented informative. Comments are always welcome at complexcases@hss.edu.

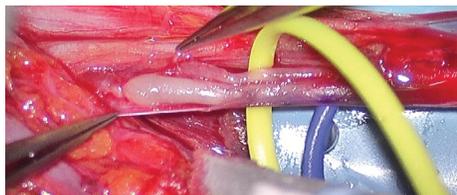
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Triple Nerve Transfer and Long Nerve Grafts Restore Function in a Devastating Brachial Plexus Injury

Case 1

Case presented by Drake G. LeBrun, MD, MPH, Darryl B. Sneag, MD, Joseph H. Feinberg, MD, Ogonna K. Nwawka, MD, Steve K. Lee, MD, and Scott W. Wolfe, MD

Neurolysis and Nerve Transfer for Iatrogenic Axillary Nerve Injury After Humeral Avulsion of the Glenohumeral Ligament Repair

Case Report A 39-year-old left-handed man presented with 2 years of numbness and weakness in the left shoulder following arthroscopic repair of a humeral avulsion of the glenohumeral ligament (HAGL) in his home country. He had previously undergone electrodiagnostic testing (EDX) and a magnetic resonance imaging (MRI) scan of the brachial plexus, which showed moderate-to-severe denervation of all 3 heads of the deltoid muscle and normal appearance of the brachial plexus.

Physical examination revealed marked deltoid atrophy and sensory deficits within the autonomous zone of the axillary nerve. Strength testing demonstrated an isolated British Medical Research Council (BMRC) [4] M4 strength deficit in left shoulder abduction. Active range of motion was full and symmetric. EDX demonstrated low discrete motor unit recruitment (1 to 2 motor units) in the anterior and middle heads of the deltoid, decreased recruitment (substantial but not full motor unit recruitment) and nascent motor unit action potentials (MUAPs) (evidence of axonal regeneration) in the posterior deltoid, and full motor unit recruitment in the teres minor.

Brachial plexus MRI showed the axillary nerve following an aberrant course between the intermuscular fat planes of the subscapularis and coracobrachialis, extending in a cephalocaudal direction toward the anteroinferior aspect of the glenohumeral joint (Fig. 1). The nerve appeared in continuity but tethered to the inferior glenohumeral ligament (IGHL) by a suture (Fig. 2). Diagnostic ultrasound further showed the axillary nerve extending superiorly toward the glenohumeral joint. Because the nerve remained in continuity and EDX demonstrated partial axillary nerve function, the patient was indicated for exploration and release of the axillary nerve.

In the operating room, the patient was placed in the modified beach chair position to allow for anterior access. A multiwindow deltopectoral approach [5] was used to provide adequate anterior and posterior exposure of the axillary nerve. The deltopectoral groove was opened, and the axillary nerve, identified on the subscapularis, was traced to its division into anterior and posterior branches (Fig. 3). The nerve was found tethered at the 6 o'clock "blind zone" [3] position by capsular sutures

that were raised and divided, freeing the nerve (Fig. 4). Electrical stimulation of the posterior branch demonstrated contraction of the posterior deltoid and teres minor; however, the anterior branch was severely constricted, and stimulation yielded no response. The wound was irrigated and closed to allow for repositioning of the patient in the lateral decubitus position. A posterior approach was subsequently employed to microsurgically transfer the long head of the triceps branch of the radial nerve to the anterior division of the axillary nerve (Fig. 5 and Fig. 6).

Nine months following neurolysis and nerve transfer, the patient had M5 strength and full range of motion in his left shoulder, with residual diminished sensation over the autonomous region of the axillary nerve. EDX showed nascent MUAPs (axonal regeneration) in all 3 deltoid heads, decreased recruitment pattern in the middle and posterior heads, and discrete (limited) recruitment in the anterior head.

Discussion This case highlights several important points about iatrogenic nerve injury following HAGL repair. First, although outcomes of arthroscopic and open HAGL repair are comparable [1], the risk and degree of impairment from axillary nerve injury may outweigh the perceived benefits of an arthroscopic approach [2].

Additionally, it is important for surgeons to know how to identify and manage patients with postoperative nerve injuries. A combination of diagnostic tools, including meticulous physical examination, EDX, and advanced, nerve-sensitive, high-resolution MRI scanning, can aid in the prompt identification and localization of iatrogenic nerve injury. In particular, MRI should be focused on the specific nerves in question to ensure the highest diagnostic yield. As this patient's initial MRI showed, a conventional brachial plexus MRI may include a field of view encompassing only the takeoff of the terminal branches from the cord level and may not extend laterally enough to visualize the course of the axillary nerve as it traverses inferior to the glenohumeral joint and enters the quadrilateral space.

Lastly, the use of different surgical windows for exposure and treatment of axillary nerve injuries [5] may help overcome the

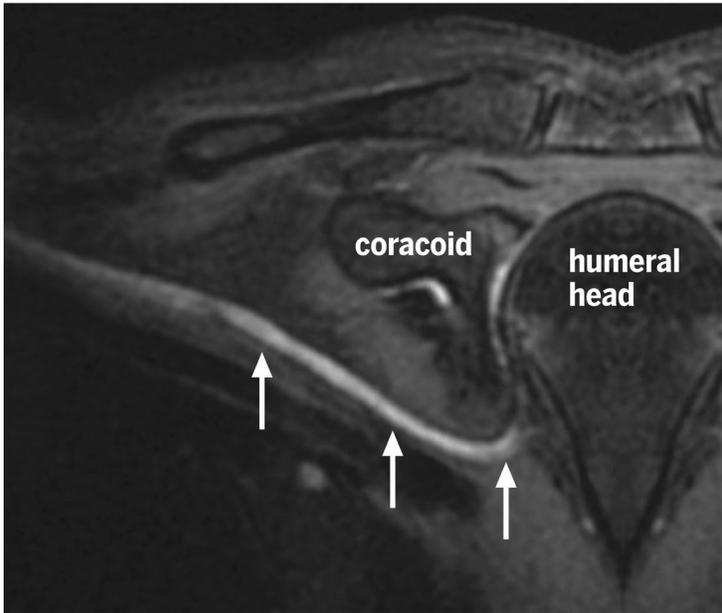
difficulty of accessing the "blind zone" [3]. This area of the axillary nerve cannot be reached using standard shoulder or plexus approaches and may be the site at which a suture anchor can tether the axillary nerve during HAGL repair. ■

Images on the next page

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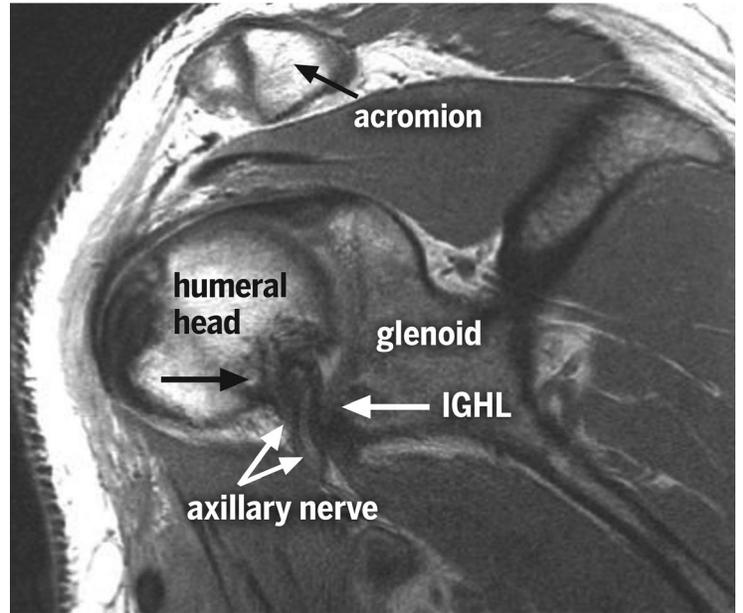
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Figure 1



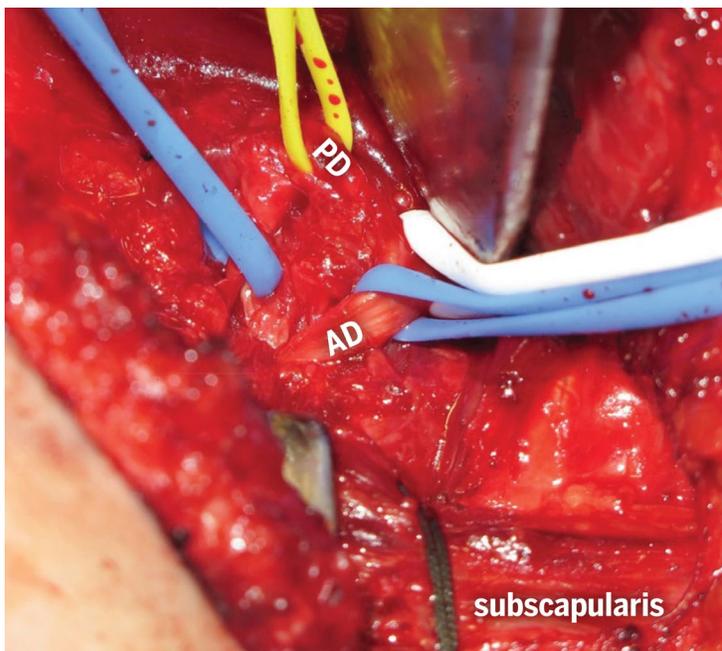
Curved, multiplanar reformatted T2-weighted inversion recovery brachial plexus MRI shows the axillary nerve (white arrows) following an aberrant course cephalocaudally toward the anteroinferior aspect of the glenohumeral joint.

Figure 2



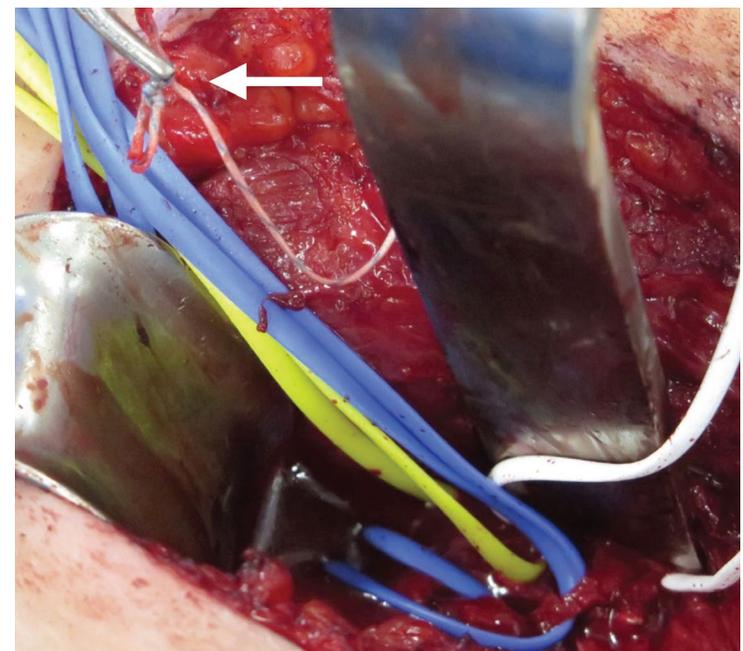
Oblique sagittal proton density-weighted MRI shows the axillary nerve tethered by suture (lower black arrow) to the inferior glenohumeral ligament (IGHL).

Figure 3



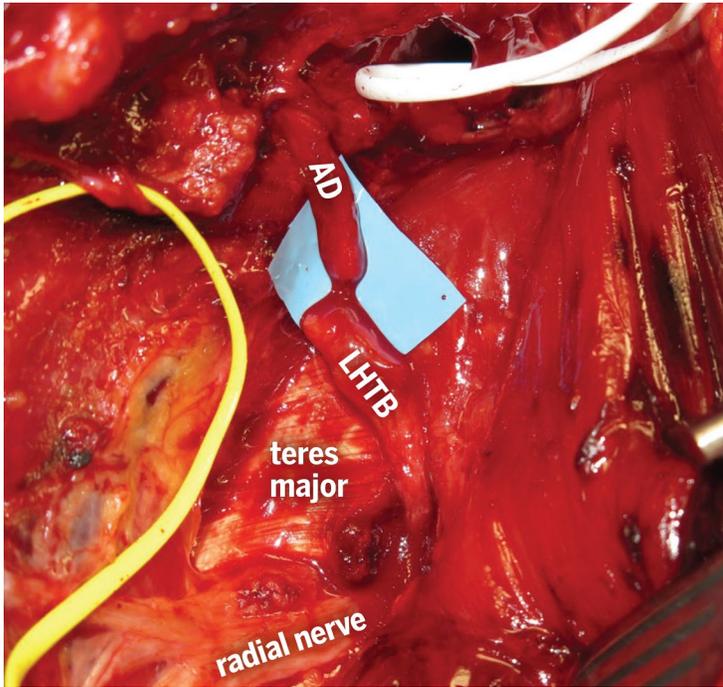
The subscapularis tendon is retracted with green suture, allowing the anterior division (AD) of the axillary nerve to be traced proximally to its tether point. The posterior division (PD) is retracted with a yellow vessel loop.

Figure 4



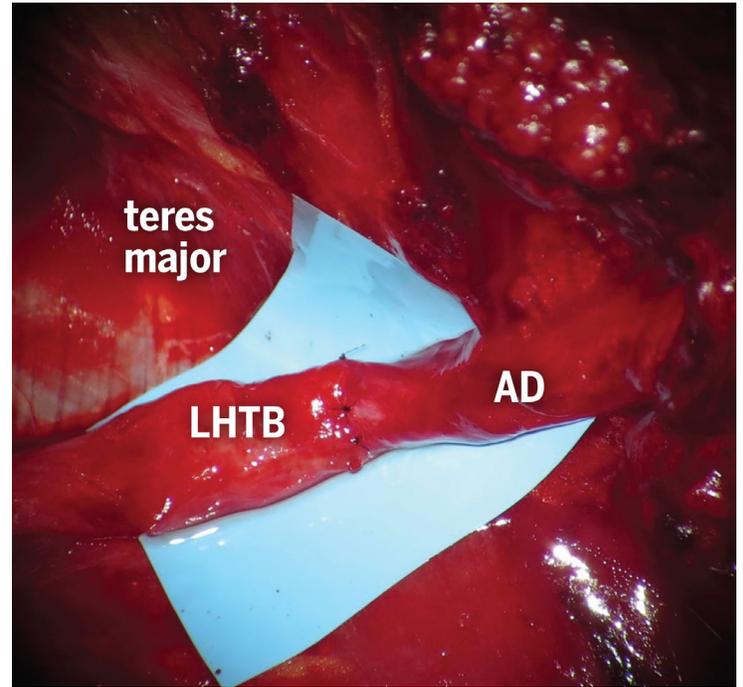
The anterior division of the axillary nerve is freed from tethering, with capsular sutures cut and removed (white arrow).

Figure 5



Long head of triceps branch (LHTB) reflected and prepared for nerve transfer to the AD of the axillary nerve, with a slit in the teres major extending the swing distance of the nerve. The PD of the axillary nerve is held by the white vessel loop.

Figure 6



Microneurotomy of the LHTB to the AD of the axillary nerve, demonstrating a good size match without tension.

Microneurolysis to Treat Anterior Interosseous Nerve Syndrome Resulting from Parsonage–Turner Syndrome

Case Report A 52-year-old right-handed woman presented with 12 months of paralysis of her right thumb. Her symptoms started 1 week following an episode of septic shock while hospitalized for an acute small bowel obstruction. She did not recall antecedent arm or forearm pain, although she found it difficult to recall specific symptoms she endured during her severe illness.

Physical examination showed proximal forearm atrophy, with a positive Kiloh–Nevin sign [1]. British Medical Research Council (BMRC) strength grading [2] was notable for M0 strength in the flexor pollicis longus (FPL), M4 strength in the index finger flexor digitorum profundus (FDP), and M5 strength in the long finger FDP. The patient otherwise had full strength of all right upper extremity muscles and no sensory deficits.

Electrodiagnostic testing (EDX) was notable for moderate to severe spontaneous activity, with no voluntary motor activity in the FPL or pronator quadratus (PQ), diagnostic of complete denervation. The patient also had incomplete denervation of the index FDP, marked by mild abnormal spontaneous activity, nascent voluntary motor unit action potential, and decreased recruitment pattern. Magnetic resonance imaging (MRI) of the right brachial plexus demonstrated signal hyperintensity in 2 posteriorly positioned fascicles of the median nerve, approximately 7 cm proximal to the medial epicondyle. These fascicles showed focal caliber changes suggestive of fascicular constrictions at the level of the medial epicondyle and within 2 cm proximal to the medial epicondyle [4] (Fig. 1). Right forearm MRI demonstrated no extrinsic compression of the median nerve. Right forearm ultrasound demonstrated no thickening or contour lobulation of the anterior interosseous nerve (AIN), with some denervation effect seen in the PQ and FPL.

Based on these findings and the lack of improvement in the patient's symptoms, she was diagnosed with AIN syndrome (AINS) [3]. Operative and non-operative treatments were discussed, and the patient elected to undergo microneurolysis of the median nerve.

A medial supracondylar incision was made from the medial mid-arm to a point just distal and anterior to the medial epicondyle. The median nerve was identified and determined to have an excellent perineural

vascular pattern. Under loupe magnification (3.5× power), the nerve was rotated to identify the posterior fascicular groups, which appeared pale and swollen. The epineurium was opened, and posterior fascicles were separated by intraneural dissection under the operative microscope (10× power). The flexor carpi radialis and pronator teres fascicles were identified and confirmed by handheld electrical stimulation at 0.5 mA. The dissection was continued proximally in the parent nerve to identify and stimulate additional fascicular groups. The posterior fascicles stimulated the flexor digitorum superficialis (FDS) strongly, but the posteromedial fascicular group, which did not respond to stimulation, was determined to represent the AIN fascicular group (Fig. 2).

Each fascicular group was traced proximally and distally, and a meticulous search for fascicular constrictions was performed. The FPL fascicle of the anterior interosseous nerve fascicular group had an acute constriction at the level of the medial epicondyle. Under the microscope, the constriction was noted to have the appearance of a “nerve torsion” [4] (Fig. 3). Perineurolysis was performed, which revealed spiral perineural fibrous bundles surrounding and constricting the nerve fascicle, which was swollen proximal and distal to the fascicular constrictions in an “hourglass” configuration (Fig. 4). After division of the perineural bands, the fascicle was identified to be translucent; within minutes it swelled to near normal caliber with a healthier appearance (Fig. 5). There was no torsion of the nerve or the fascicles themselves. The wound was closed, and the patient was allowed gentle range of motion of the elbow, progressing to activity as tolerated over the next several weeks.

Eleven months after micro–internal neurolysis, the patient had M4 strength in the FPL, with 50° active range of motion, and M4+ strength in the index FDP, with full range of motion (Fig. 6). She had M5 strength in pronation. EDX demonstrated mild abnormal spontaneous activity and decreased recruitment in the FPL and the PQ, suggesting axonal regeneration and near complete motor unit recovery.

Discussion This case shows the utility of microneurolysis in a patient with characteristic clinical, electrodiagnostic, and imaging findings of AINS, an idiopathic

axonopathy of the median nerve that causes an acute motor palsy of the muscles innervated by the AIN. AINS is a subtype of neuralgic amyotrophy, also called Parsonage–Turner syndrome (PTS) [5]. Although AINS was conventionally considered an idiopathic forearm neuropathy, it is now recognized as a disorder of the median nerve, characterized by fascicular constrictions at or proximal to the elbow [3].

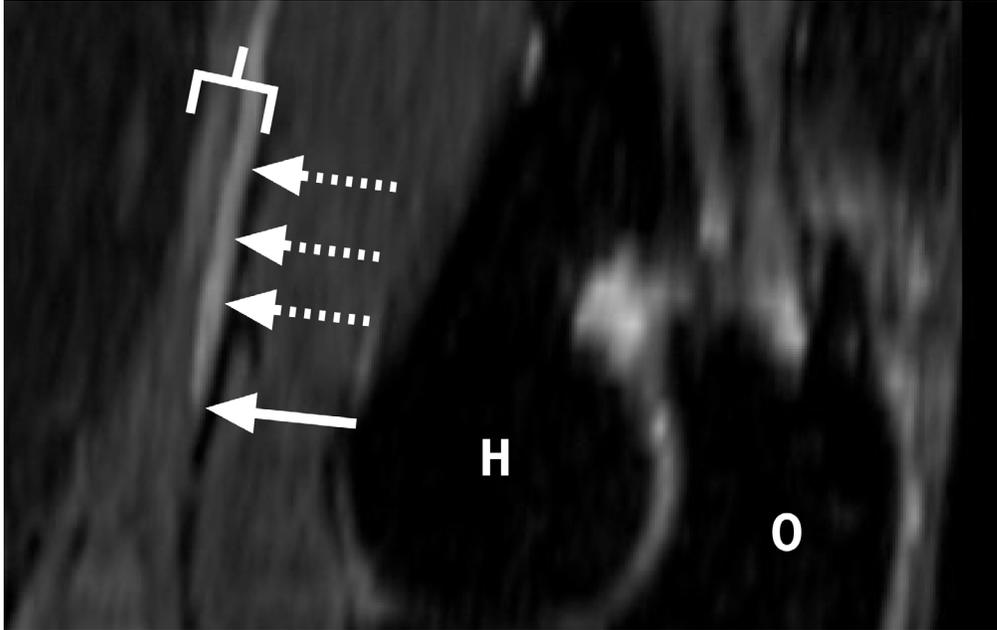
Furthermore, this case highlights the importance of a multimodal diagnostic strategy involving EDX and targeted, nerve-specific imaging to identify the fascicular constrictions characteristic of AINS and PTS [3]. Previously, MRI and ultrasound served a secondary role in diagnosing PTS by showing muscle denervation and evaluating for causes of external compression in the forearm. As in this case, high-resolution diagnostic MRI can pinpoint localization of anterior interosseous fascicles of the median nerve and assist the surgeon in localizing and decompressing fascicular constrictions. ■

[Images on the next page](#)

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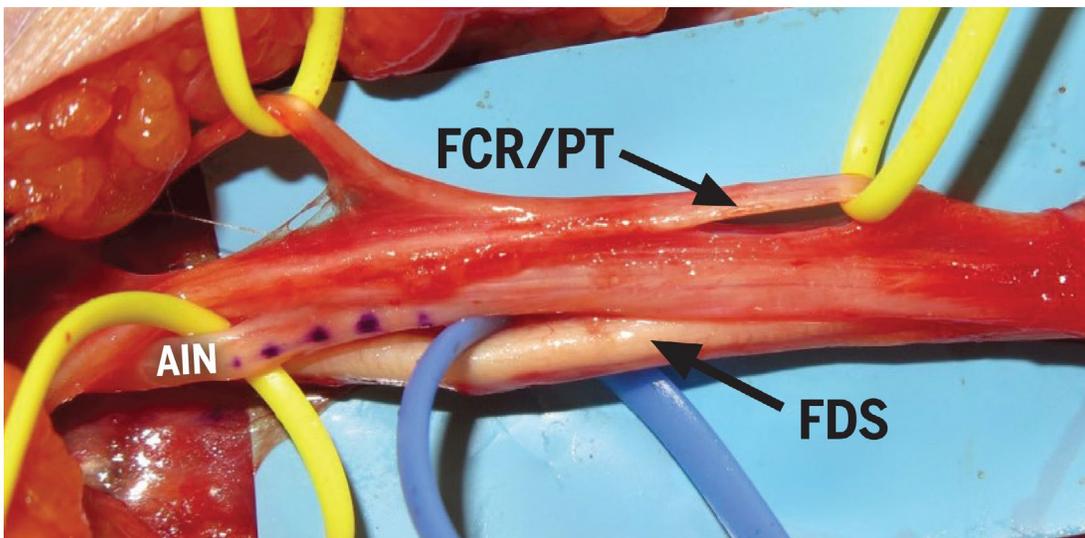
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Figure 1



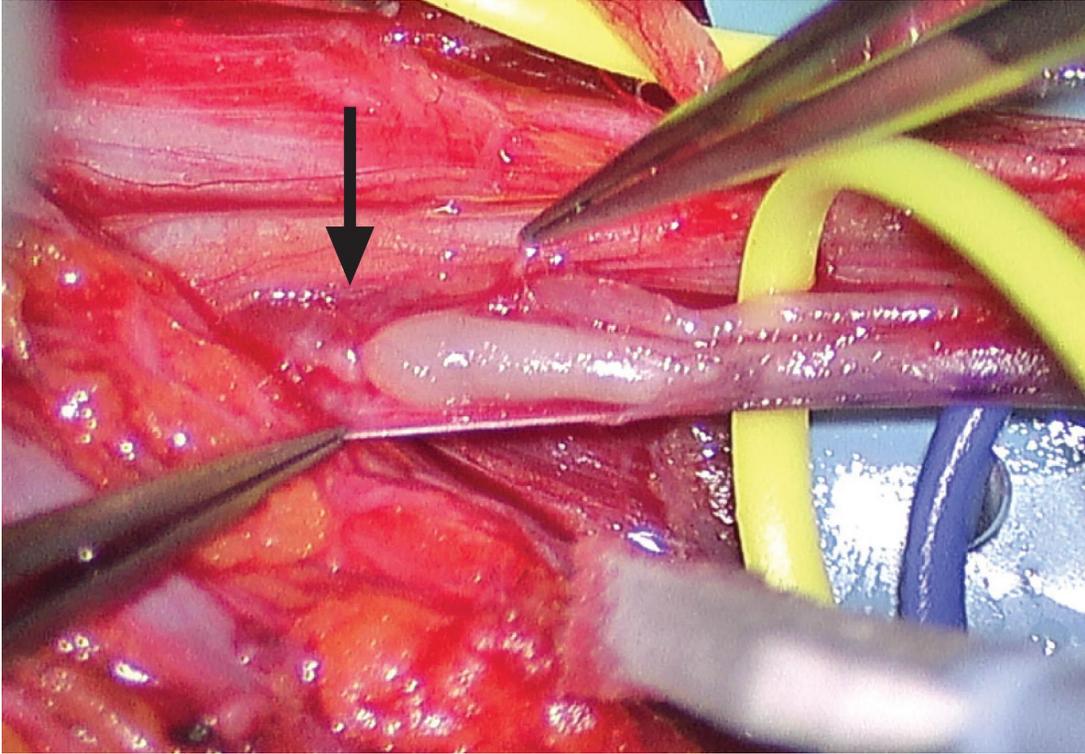
Curved, multiplanar reformatted sagittal projection T2-weighted fat suppressed image demonstrates abnormal signal hyperintensity of a posteriorly positioned fascicular bundle (dashed arrows) of the median nerve proper (bracket) within the distal arm. Note the sharp tapering of the fascicle as it approaches the elbow joint, compatible with an intrinsic constriction (solid arrow). Distal humerus (H), olecranon (O).

Figure 2



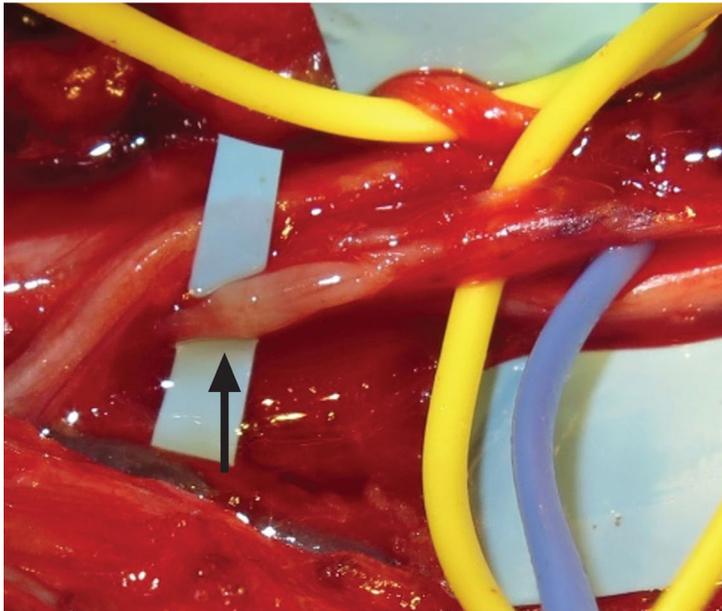
Anterior and posterior fascicles of the median nerve following intra-neural dissection. Flexor carpi radialis (FCR) and pronator teres (PT) fascicles stimulated strongly and are held by yellow vessel loops on the top portion of the image. The posterior fascicle to the flexor digitorum superficialis (FDS) stimulated strongly and is held by blue vessel loops at the bottom of the image. The posteromedial fascicles (dotted) did not stimulate and were determined to represent AIN fascicles.

Figure 3



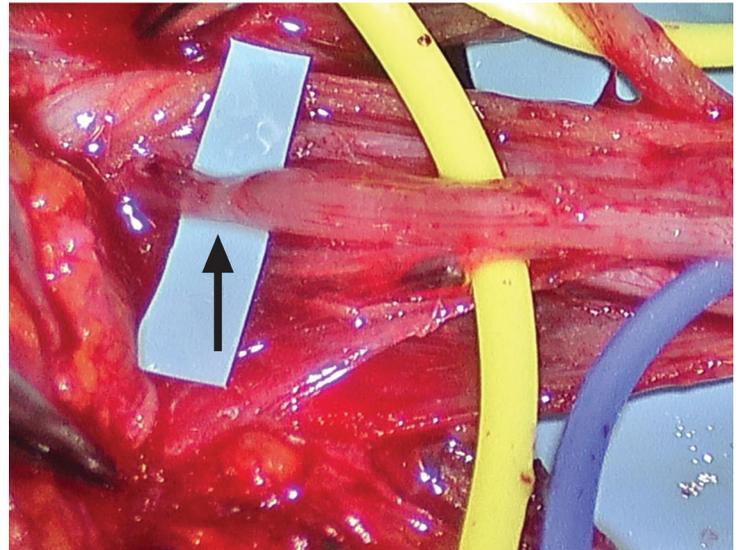
Anterior interosseous nerve fascicle with pre-stenotic dilatation and spiral bands (black arrow).

Figure 4



Perineurolysis of the AIN branch revealing an hourglass constriction (black arrow).

Figure 5



AIN fascicle 30 minutes after microneurolysis, showing increased vasculature and translucency at the site of the prior hourglass constriction (black arrow).

Figure 6A



Figure 6B



Figure 6: The patient, 1 year postoperatively, showed improved ability to flex at the thumb interphalangeal joint and index finger distal interphalangeal joint.

Case 3

Case presented by Drake G. LeBrun, MD, MPH, Darryl B. Sneag, MD, Joseph H. Feinberg, MD, Ogonna K. Nwawka, MD, Scott W. Wolfe, MD, and Steve K. Lee, MD

Triple Nerve Transfer and Long Nerve Grafts Restore Function in a Devastating Brachial Plexus Injury

Case Report An 18-year-old left-handed woman presented with right upper extremity weakness and numbness 6 weeks after a motor vehicle collision (she had hit a tree as an unrestrained driver). She could not fully move her right shoulder, flex or extend her elbow, or extend her wrist and digits.

Clinical examination demonstrated complete palsy of her supraspinatus, infraspinatus, deltoid, biceps, brachialis, triceps, wrist extensors, and digital extensors. She had no Horner syndrome, no scapular winging, and no sensation in the C5 and C6 dermatomes. The patient was diagnosed with a complete upper trunk and partial C7 brachial plexus palsy.

Electrodiagnostic (EDX) studies at 3 months post-injury demonstrated no voluntary motor unit action potentials (MUAPs) in the supraspinatus, infraspinatus, deltoid, triceps, biceps, brachioradialis, or wrist extensors.

EDX findings were consistent with a predominately postganglionic and severe brachial plexopathy of C5 through C7. A multidisciplinary brachial plexus case conference was held to discuss findings of all preoperative studies. Given her lack of spontaneous recovery, we recommended surgical reconstruction to restore shoulder and elbow function, and the patient agreed.

The patient underwent surgery at 4 months post-injury. She was positioned supine with the neck, right upper extremity, and both lower extremities prepared and draped. Two teams operated simultaneously, using multiple approaches to the right supra- and infraclavicular brachial plexus and right sural nerve area (posterior calf to the lateral ankle).

In the supraclavicular region, a large upper trunk neuroma was excised and thin slices of the C5 and C6 root contributions sent for frozen section histologic analysis. The C6 nerve root demonstrated well-defined myelinated axons in more than two-thirds of the cut section, while the C5 root had less than 50% axonal viability. The C6 root demonstrated intact axonal continuity by somatosensory evoked

potential (SSEP), while the C5 root demonstrated no response. Based on these data, we elected to use the C6 root for nerve grafting.

In the infraclavicular region, the axillary, musculocutaneous, median, ulnar, radial, pectoral, and thoracodorsal nerves were identified and neurolysed. A double fascicular nerve transfer was performed to restore elbow flexion by first isolating the motor branches to the biceps and brachialis and dividing these from the parent musculocutaneous nerve. A double Oberlin transfer to the arm was used to restore elbow function [3]. Microscopic dissection and electrical stimulation were used to identify individual nerve fascicles of the median and ulnar nerves, which control wrist flexion and forearm pronation. These fascicles were divided distally, with enough length to allow rotation and microscopic nerve suture to the biceps and brachialis motor branches [3] (Fig. 1).

Simultaneously, the supraclavicular team had prepared the distal branches of the spinal accessory nerve for microscopic transfer to the suprascapular nerve, which had been divided from the injured upper trunk. This transfer was performed to restore shoulder abduction and external rotation via the rotator cuff (Fig. 2). Lastly, 45 cm of sural nerve graft was harvested, and a 15-cm cable graft was interposed between the C6 nerve root proximally and the axillary nerve distally. All grafts and transfers were sewn with 8-0 and 9-0 nylon sutures and reinforced with fibrin glue. Given its partial involvement, the C7 nerve root was kept intact in anticipation of possible spontaneous recovery of elbow, wrist, and digit extension.

Postoperatively, the patient started to recover active elbow flexion at 4 months, elbow extension and shoulder function at 6 months, and wrist and digit extension at 9 months. EDX at 9 months showed evidence of significant axonal regeneration (decreased recruitment with nascent potentials or di- or triphasic configurations) to the extensor carpi radialis, triceps lateral and medial heads, middle and posterior deltoid, brachioradialis, biceps, brachialis, flexor carpi radialis, pronator teres, supraspinatus, and infraspinatus.

There was also axonal regeneration to the extensor pollicis longus and anterior deltoid, but this was limited (discrete recruitment with nascent potentials). At her most recent follow-up visit 6 years after surgery, she had M5 strength in shoulder abduction (100°), external rotation (50°), elbow flexion (150°), finger flexion (full), wrist extension (60°) (Fig. 3), and the intrinsic muscles. She has excellent upper extremity function and has returned to a full workout regimen.

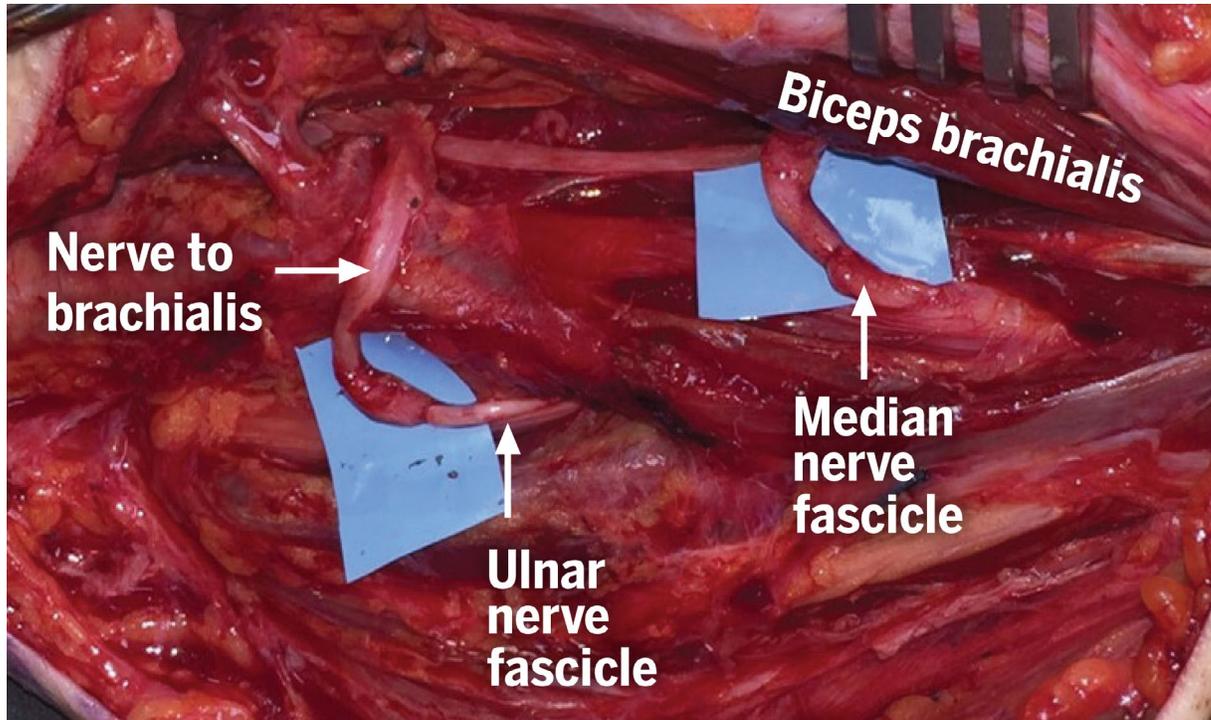
Discussion Traumatic brachial plexus injuries require comprehensive preoperative evaluation and planning, including meticulous physical examination and EDX, to determine the optimal strategies for neurolysis, nerve transfer, and grafting [1]. This case highlights the transfer of intact portions of pure motor nerves including the spinal accessory nerve and selective motor fascicles of the median and ulnar nerves [2, 3] to restore shoulder abduction, external rotation, and elbow flexion. The case also illustrates the successful use of long nerve grafts to restore axillary nerve function, a prior technique that had been largely abandoned but that HSS physicians have resurrected and refined [4]. ■

[Images on the next page](#)

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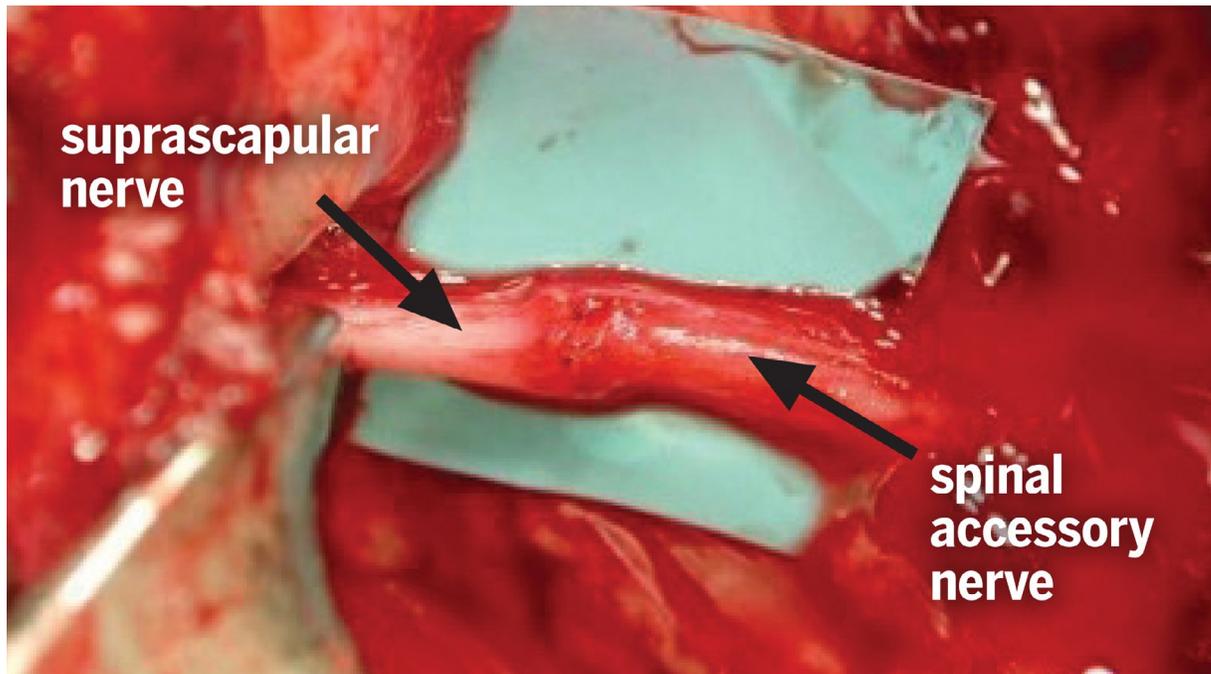
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Figure 1



A double Oberlin transfer to the arm to restore elbow function. Nerve fascicles from the median and ulnar nerves are transferred to the nerves to the biceps and brachialis, respectively.

Figure 2



Spinal accessory nerve transfer to the suprascapular nerve to restore shoulder function.

Figure 3A



Figure 3C



Figure 3B

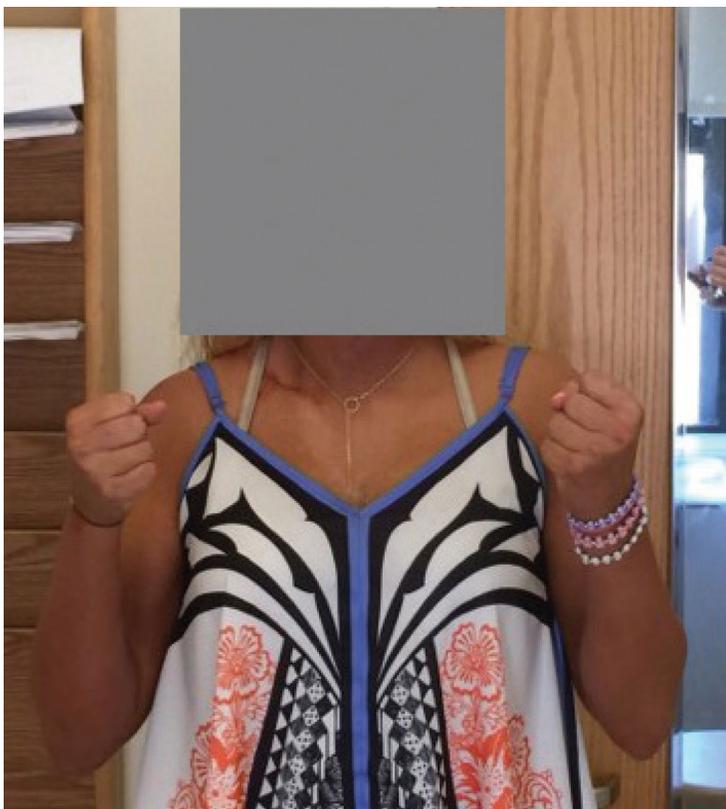


Figure 3: The patient showed significantly improved active range of motion at 6 years postoperatively, abducting the shoulder to 100° (A), flexing the elbow to 150° (B), and externally rotating the shoulder symmetrically (C).

View videos from the Center for Brachial Plexus and Traumatic Nerve Injury: bit.ly/BrachialPlexus.

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