Radial Nerve Injury in Humeral Shaft Fracture

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KEYWORDS

• Radial nerve injury • Humeral shaft fracture • Nerve repair

KEY POINTS

- Orthopedic surgeons should understand the principles of radial nerve injuries in the context of humeral shaft fractures in order to facilitate optimal treatment of this injury constellation.
- Expectant management of nerve injury in conjunction with nonoperative treatment of fracture includes appropriately timed clinical follow-up with detailed clinical examinations and serial electromyograms.
- Surgical exploration (with possible reconstruction) is recommended at 3 to 4 months from injury if there is no evidence of improvement.
- Treatment of radial nerve injuries identified at the time of operatively treated fractures includes direct repair under no tension when possible, reconstruction with graft if repair is not possible, and tagging the nerve ends under physiologic tension with referral for specialist care when acute reconstruction is not appropriate.
- Irreparable proximal radial nerve injuries benefit from distal nerve or tendon transfers.

INTRODUCTION

Fractures of the humeral shaft are common, accounting for 1% to 3% of all fractures.[1] Concurrent radial nerve injury has been reported in approximately 10% of these fractures.[2] Certain fracture patterns may carry an even higher incidence of radial nerve injury. The classic example of this is a spiral fracture at the distal third of the humeral diaphysis, otherwise known as the Holstein-Lewis fracture.[3]

At the time of initial presentation, the extent to which the nerve is damaged is often unknown, but the mechanism itself can provide clues as to the nature of the nerve injury. However, any injury pattern is capable of creating the whole spectrum of nerve injuries from contusion to complete transection. For example, open injuries with a sharp penetrating mechanism are classically thought to have a higher likelihood of nerve transection secondary to the laceration of soft tissues within the field of injury. Ballistic injuries are classically thought to have a higher likelihood of nerve contusion secondary to the shock-wave and soft tissue contusion caused by the high-velocity projectile. Closed humeral shaft fractures with simple fracture patterns can result in stretching of the nerve by displacement of the soft tissues at the time of injury, contusion of the nerve from entrapment between mobile fracture ends, or complete laceration of the nerve over the sharp edges of the fractured bone.[4,5]

Treatment algorithms are designed around reducing the overall time to appropriate treatment in the context of this uncertainty. An understanding of the treatment for these injuries is enriched by an understanding of the anatomy, classification, and prognosis for peripheral nerve injuries generally.

Peripheral Nerve Injury

The microstructural anatomy of the peripheral nervous system is most usefully conceptualized as a series of concentric layers from the inside

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to the outside (Fig. 1A).[6] The basic unit of the peripheral nervous system is the single axon, which conducts action potentials, as a means of neurotransmission. An interrupted myelin sheath surrounds the axon, accelerating conduction velocity. The endoneurial lining surrounds these 2 elements and is the peripheral analogue to the "blood-brain barrier" in the central nervous system. Functional axonal units with similar function are clustered into fascicles by the perineurium, from which peripheral nerves derive their tensile strength. Fascicles for specific anatomic regions are clustered into named peripheral nerves by the epineurium, which houses the vasonervosum (the blood supply for the peripheral nervous system).

There are 2 classification systems for peripheral nervous system injury, which are both based on this underlying anatomic arrangement (Fig. 1B). The Sunderland classification system is based on the microstructural level of injury.[7] The 5 levels of peripheral nervous system microstructure include the following: I, mylin sheath; II, axon; III, endoneurium; IV, perineurium; V, epineurium. Ascending levels of injury in the Sunderland classification system represent disruption of the corresponding microstructural element listed above, along with all of the preceding microstructural elements.

The Sedon classification system consists of 3 levels (neurapraxia, axonotmesis, and neurotmesis) and is based on function and macroscopic anatomy (see Fig. 1B).[8] Neuropraxia represents a transient disruption in nerve conduction most commonly thought to be secondary to temporary disruption of the myelin sheath (Sunderland grade I). Axonotmesis is any injury disrupting the axon, without disrupting the macroscopic continuity of the peripheral nerve as seen from the outside (Sunderland grade II, III, and IV). Neurotmesis is an injury causing frank discontinuity of the peripheral nerve (Sunderland grade V).[9]

Recovery of appropriate action potential conduction across a region of injury depends on the severity of the injury. Neuropraxia has an excellent prognosis, as no axonal regeneration is required. The prognosis of axonotmetic injury depends largely on the level of microstructural disruption. Axonotmetic injuries preserving the microstructure allow regenerating axons to reliably find their original target and have good prognoses without surgical intervention. Axonotmetic injuries with disruption of microstructure do not allow for successful nerve regrowth and have poor nonsurgical outcomes. The exercise of trying to determine the difference in severity of axonotmetic injuries is largely intellectual, as this is practically indeterminable outside of research laboratories. Last, neurotmetic injuries will not recover without surgical intervention.

A good clinical outcome after nerve injury depends on the likelihood that nerve conduction will be restored from its origin to its intended target before that intended target undergoes atrophy, loss of function, and cellular death. This means that prognosis for recovery of native nerve function depends on the nature of injury according to the injury classification system above, as well as other features of the host and injury (Fig. 1C).[10,11] Younger patients with greater regenerative potential, both centrally and peripherally, carry a better prognosis. Injuries occurring distally in an extremity carry a better prognosis because axonal growth cones have a shorter distance to traverse before finding their target and successful reinnervation of target end plates. Injuries requiring surgical intervention that are repaired earlier carry a better prognosis because early intervention reduces the time to reinnervation. Injuries that can be repaired directly (end to end) carry a better prognosis because regenerating nerves only have 1 coaptation to navigate, rather than having 2 coaptation sites as is the case with any grafting technique.

PATIENT EVALUATION AND CLINICAL DECISION MAKING

Each of the patient and injury factors affecting prognosis do so by affecting the time to

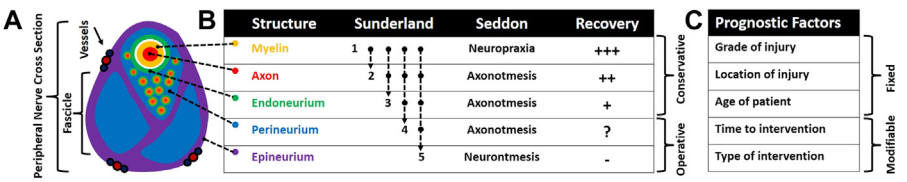

Fig. 1. Peripheral nerve injury primer. (*A*) Overview of peripheral nerve anatomy as mapped onto (*B*) Sunderland and Sedon injury classification systems and association with prognosis. (*C*) Additional factors driving outcomes in peripheral nerve injury.

reinnervation or extent of reinnervation. A good clinical outcome depends on the likelihood that nerve conduction will be restored from its origin to its intended target before the intended target dies. In the case of the neuromuscular junction, end organ death is thought to occur approximately 12 months from denervation (Fig. 2).[11] This means that selecting the most appropriate treatment pathway requires a timely decision to proceed with surgery when it becomes necessary. It is the surgeon's obligation to identify patients for whom expectant management is unlikely to succeed and indicate them for early corrective intervention.

Expectant Management

Expectant management consists of serial examinations (detailed physical examination and electromyogram [EMG]), timed in such a way to catch the leading edge of clinical recovery (see Fig. 2C). Evidence of early returning function rules out neurotmetic injuries that would benefit from early exploration with reconstruction. Radial nerve injuries in the context of humeral shaft fractures are expected to recover without intervention in more than 70% of cases, and approximately 90% of cases, including those that underwent procedural intervention.[12] In the context of closed fractures, recovery with expectant management is even more likely,[4,5,13] with recovery rates consistently greater than 90%.

Physical examination

The pattern of muscle innervation of the radial nerve is reproducible (Fig. 3).[14] The most common site of laceration is at the lateral margin of the humerus, where the radial nerve passes from the posterior compartment to the anterior compartment (see Fig. 3, point E). Nerve mobility is limited here secondary to tethering by the intermuscular septum, making it especially prone to neurotmetic injury. The most proximal motor endplates occur in the brachioradialis (BR) and extensor carpi radialis longus (ECRL; 10 cm and 12 cm from the lateral intermuscular septum, respectively). With an average regeneration rate of 1 mm/d, this implies that clinical evidence of recovery consisting of activation of the BR and ECRL should be observable by approximately 3.5 to 4.5 months from injury. After this point, reinnervation occurs in a reproducible pattern based on injury consisting of recovery of wrist extension, then extension over the ulnar-sided digits, and finally, extension of the thumb and index finger. Once reinnervation has started down this pathway, it generally restores good function in all muscle groups. However, very distal muscles, such as the extensor indicis proprius, with a reinnervation distance of 30 cm, are likely to take up to 10 months to recover. These calculated estimations of activity recovery are echoed in clinic observations of recovery in Sedon neuropraxic- and axonotmetic-type injuries, with early recovery noted by 12 months, and full recovery taking up to 1 year.[13] Additional early findings indicating that the nerve injury is not complete include preservation of sensory function. Motor function is the first to be lost and last to be regained after neuropraxic injury; incomplete injuries that preserve sensory function have an excellent prognosis. Similarly, advancing Tinel sign at the lateral aspect of the humerus during their initial visits carries an excellent prognosis, as it indicates regeneration down the distal portion of the nerve has already begun. Documentation of the exact location of the Tinel sign (single

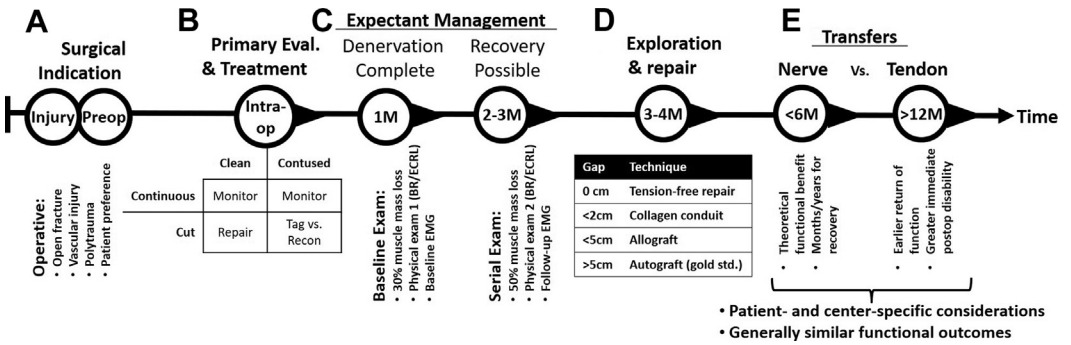

Fig. 2. Timeline of possible interventions with key points: decisions to proceed with intervention occur at multiple timepoints, including (*A*) primary surgical indications for fracture fixation, (*B*) intraoperative decision making regarding associated nerve injury, (*C*) expectant management, which may progress to (*D*) secondary exploration and reconstruction, and the possibility of (*E*) late nerve or tendon transfers. Eval., evaluation; Intra, intraoperative; Preop, preoperative; Postop, postoperative; Recon, reconstruction; Std., standard.

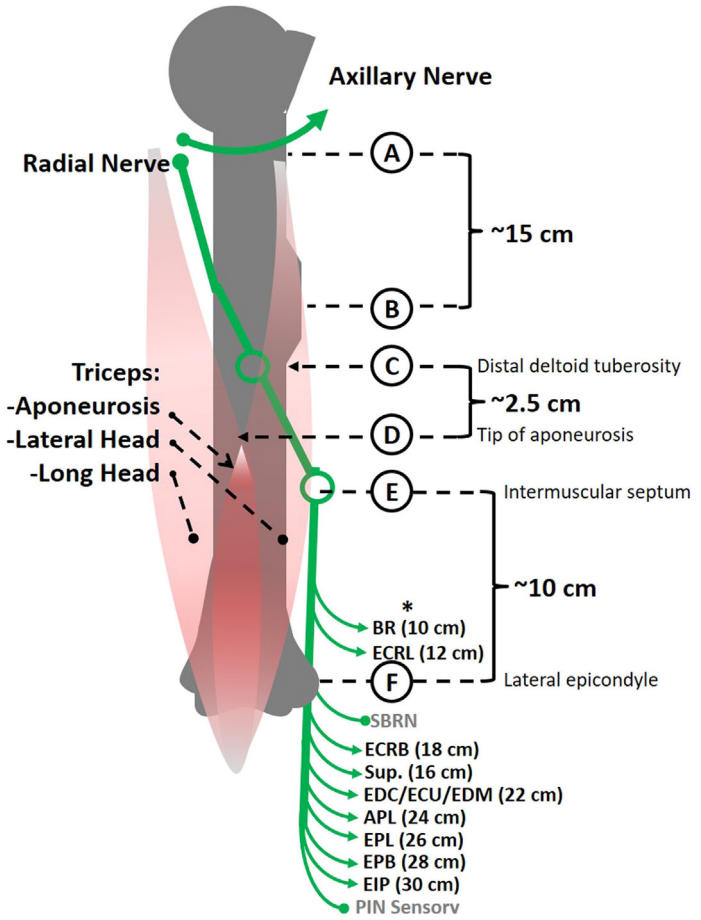

Fig. 3. Radial nerve schematic with landmarks: radial nerve anatomy with relevant landmarks. APL, abductor pollicis longus; ECU, extensor carpi ulnaris; EDM, extensor digiti minimi; EIP, extensor indicis proprius; EPB, extensor pollicis brevis; EPL, extensor pollicis longus; PIN, posterior interosseous nerve; Sup., supinator. * indicates that the starting point of reinnervation after injury at the intermuscular septum.

spot with maximum effect) relative to the lateral epicondyle will help differentiate between patients with progressive recovery versus those with static neuroma formation.

Electromyogram

In cases whereby neurotmesis has occurred, no functional recovery will be observed. Clinically, this will appear simply as the absence of functional recovery over serial examinations. However, changes occur on the level of muscle cell function that may become evident while awaiting clinical improvement. Because the grade of the injury in unexplored cases cannot be known before initiating expectant management, serial EMGs are a useful way of providing additional quantitative measures of nerve function to supplement serial clinical examinations. A review of the EMG changes exceeds the scope of this review. However, these EMGs should be performed in the radial nerve innervated muscle groups listed previously specifically to assess for progressive reinnervation or its absence. It is useful to have serial studies performed by the same provider and in the same location in order to allow for direct comparison of serial examinations. EMG changes, such as the development of abnormal spontaneous activity, are expected to evolve during the first 4 weeks after injury. EMG studies before this point are not recommended. A second EMG study at 3 to 4 months from injury will allow adequate time for most injuries to reach the proximal ECRL and BR in the case of axonotmetic injuries. In medicolegal cases, more frequent EMGs starting before this point may help identify the most likely timing of nerve injury, but this is not routinely recommended.[15,16]

Primary Exploration and Treatment of Nerve Injury

Indications for acute surgical exploration, with possible repair, are similar to indications for operative treatment of humeral shaft fractures;

these are (1) open fractures, (2) fractures complicated by vascular injury, (3) global patient injury constellations requiring fracture fixation with the theoretic benefit of earlier mobilization of the extremity, or (4) strong patient preference for operative treatment and an understanding of the associated risks and benefits (see Fig. 2A).

In the case of radial nerve injury, open reduction with internal fixation of the humeral shaft may be preferred over closed intramedullary nailing when the skeletal injury is amenable to both, for the simple reason that the nerve should be directly examined in these cases. There is a risk that the nerve palsy is secondary to incarceration in the fracture site, in which case the act of reaming and passing an intramedullary nail could cause secondary injury to the nerve.[17] In addition, minimally invasive surgical treatment is a missed opportunity to characterize the nerve injury, which assists with downstream clinical decision making. Surgical exploration of the fracture with visualization of the radial nerve should be at least considered when surgical fixation is indicated, regardless of skeletal fixation tactic.[13]

If a transected radial nerve is encountered during surgical fixation of a fracture, treatment of the radial nerve at that time depends on the continuity of the nerve (cut vs continuous), and the quality of the nerve (contused vs clean) (see Fig. 2B).

Continuous and clean

In cases whereby the nerve is in continuity and healthy-appearing, conservative management is expected to yield an excellent result, with recovery of full manual strength by 1 year from injury in 98% of cases.[13] In cases of nerve discontinuity or significant contusion, the prognosis is less clear, and there is little definitive evidence guiding clinical decision making.

Cut and clean

Cases whereby the nerve is cut and the ends are clean appearing may best be described as simple lacerations. The gold-standard treatment of any simple nerve laceration is direct, tension-free repair. Direct nerve repair falls generally into 2 categories: epineurial versus fascicular repair. Epineurial repairs consist of reapproximating of the epineurium using blood vessels and other surface landmarks to grossly restore alignment. The goal is to preserve fascicular alignment while limiting intraneural suture in an effort to reduce foreign body reaction and disordered scar formation at the coaptation site. Fascicular repair is the direct repair of individual fascicles, achieving direct fascicular realignment at the expense of increased manipulation of the nerve tissue and intraneural placement of sutures. In nerve repairs, as in nerve transfers, it is generally agreed that fascicular alignment is important.[18,19] However, the literature has not demonstrated the superiority of either of the above techniques.[20]

Regardless of the technique used, nerve repair should always be performed under no tension. The simplest way of achieving a tension-free repair is immobilization along the length of the proximal and distal nerve ends. Additional length can be achieved through anterior transposition of the nerve through the fracture site if fracture morphology and soft tissue injury patterns are amenable to this.

Continuous and contused

Nerves in gross continuity, but with a contused appearance, have the most variable prognosis. The epineurium is intact, but the internal extent of microstructural disruption is highly variable. In some cases, there is little disruption, and recovery progresses well. In other cases, the internal derangement prevents any effective axonal regrowth through the zone of injury and results in a neuroma-in-continuity. The long-term outcome is unknowable based on features available during visual inspection. Acute resection with grafting may be beneficial in a well-demarcated, short-segment crush injury. Otherwise, results with resection and grafting of a contused nerve in continuity may not yield better results than expectant management alone.

Cut and contused

Cases whereby the nerve is lacerated and the nerve ends appear contused are the most controversial. The first step generally requires the debridement of the injured-appearing nerve back to the level of healthy-appearing nerve fascicles. Many nerve surgeons advocate for performing this acutely. However, there has yet to be an objective way to identify and differentiate between contused portions of nerve that will recover versus a contused portion of nerve that will not recover. Some surgeons therefore advocate for tagging the nerve with the intention of performing a subacute reconstruction after the zone of nerve injury has been demarcated.

If definitive management of the nerve ends is not being performed at the initial surgical setting, classic teaching recommends tagging the free nerve ends proximally and distally with suture for easier identification later by the treating surgeon. It is the authors' viewpoint however

that this technique is of limited utility. Simple suture tags do not prevent retraction of the nerve ends, and retraction of ~1.5 cm at each nerve end can be expected over the following weeks, resulting in a large nerve gap (Fig. 4C, blue bracket). In addition, free suture tags do not facilitate retrieval during secondary procedures, as they are difficult to identify within their surrounding scar tissue (see Fig. 4C, blue arrowheads). The authors' preferred tactic is suturing the nerve ends to one another under physiologic tension in order to prevent further retraction, preserve their alignment, and maintain the nerve's anatomic position to facilitate identification during subsequent reconstructive surgery. Some surgeons even recommend tagging the nerve ends with a collagen nerve tube. Staining the nerve tube with methylene blue to facilitate location of the nerve ends during a secondary procedure is another described approach. Regardless of the management choice, of critical importance are clear communication and documentation of the manner in which the nerve was addressed. A clear description of the

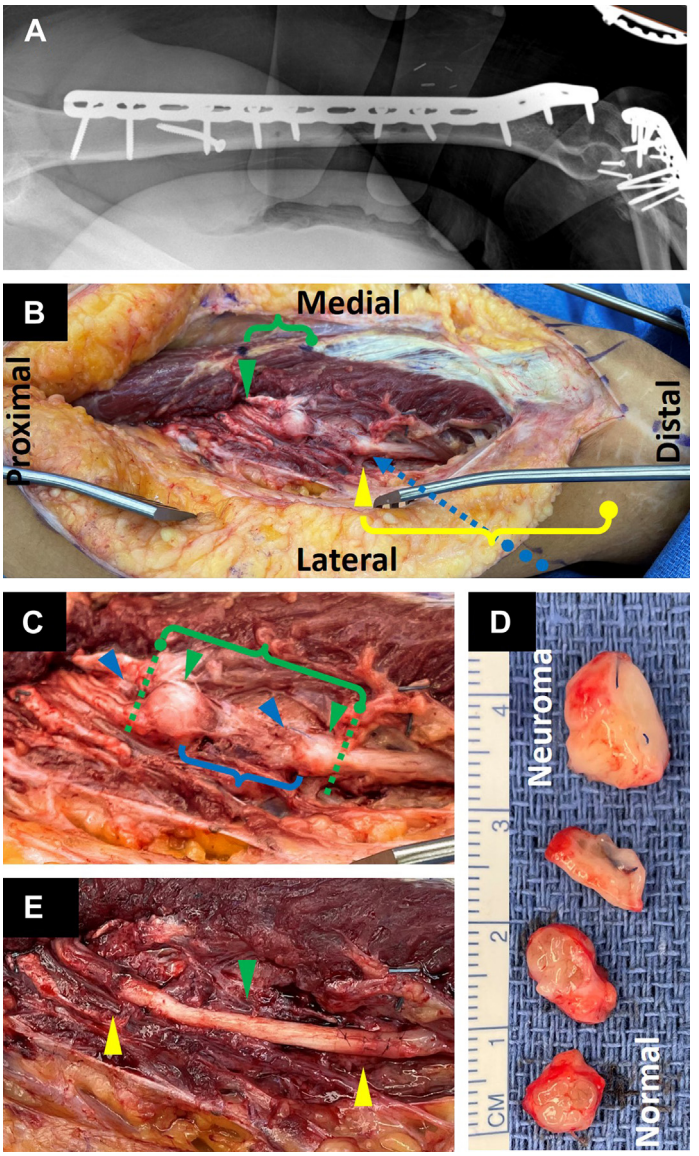

Fig. 4. Case example, delayed radial nerve reconstruction: A 38-year-old woman sustained an open humeral shaft fracture and transolecranon fracture dislocation in conjunction with a closed head injury complicated by ischemic stroke. She was medically stabilized, and a traumatic radial nerve transection was noted during index fixation of her humeral shaft fracture (*A*). The ends were tagged with 6-0 Prolene suture and left. The patient was referred for specialist care ~3 months after fixation for evaluation and treatment of her radial nerve injury. Surgical exposure consisted of a revision Gerwin approach consisting of elevation of the atretic lateral head of the triceps. The proximal portion of the radial nerve (*B*; *green arrowhead*) was found crossing the midpoint of the humerus 2 cm proximal to the tip of the triceps aponeurosis (*B*; *green bracket*). The distal portion of the nerve was found crossing the lateral aspect of the humerus (*B*; *yellow arrowhead*), 10 cm proximal to the lateral epicondyle (*B*; *yellow bracket*), where the proximal extension of the anterior forearm musculature had been marked on the skin with the elbow flexed 45° (*B*; *blue dashed arrow*). The nerve ends had formed neuroma (*C*; *green arrowheads*), which obscured the tagging suture (*C*; *blue arrowheads*). The unsecured nerve ends had retracted ~2 cm (*C*; *blue bracket*), almost doubling the total nerve gap after neuroma resection (*C*; *green bracket*). The proximal and distal neuromas were resected in sections until healthy-appearing fascicles were visualized (*D*). The resultant 4.5-cm gap was bridged using acellular allograft (*E*; *green arrowhead*) rather than autograft at the patient's request, and to coaptation sites were reinforced with a nerve wrap (*E*; *yellow arrowheads*).

location of the laceration and the fate of the discontinuous nerve ends, as well as a description of the approximate location of the nerve relative to any fixation can additionally be useful to receiving surgeons, especially if the nerve was left in an extra-anatomic position.

There is no evidence-based guidance on the timing of secondary nerve reconstruction, with recommendations ranging from 3 days to 3 weeks.[21] It is the authors' experience that early reoperations within 1 week of the index surgery simplify the process of isolating the nerve ends, whereas delayed reoperations performed more than 3 weeks from injury simplify identification of the zone of injury. These treatment decisions should be made by the surgeon who will ultimately perform the surgery, however, so referral to a specialist as soon as possible is critically important in optimizing patient care.

Secondary Nerve Exploration and Reconstruction

Secondary surgical exploration of radial nerve injuries, which have not improved with expectant management, is most commonly performed between 3 and 5 months from injury. Indication for surgery includes the absence of any clinical or EMG evidence of recovering function (see Fig. 2D).[12] This time period allows for the development of any early observable clinical recovery in nerves that will recover native function and allows for the earliest possible exploration of the zone of nerve injury in nerves that will not. If an injury requiring proximal reconstruction is found and repaired, this timing allows for 8 months of regeneration following repair before reaching the 12-month deadline at which loss of neuromuscular junctions becomes irreversible. Secondary reoperation in cases of known transection should be performed as soon as the soft tissues and patient are amenable to a second surgery.

Operating through healthy tissue planes is always ideal but is frequently not possible in the context of secondary exploration. A knowledge of the previous approach used for skeletal fixation (ie, if a Gerwin approach vs a triceps splitting approach was used) may help establish an expectation of which tissue planes may remain useful during reexposure of the radial nerve.

The authors recommend using a Gerwin approach, including elevation of the lateral head of the triceps off the intermuscular septum.[22] This approach is advantageous because the location for nerve injury is often at its transition point from the posterior to anterior compartment (see Fig. 3, point E). In cases whereby a triceps-splitting approach was used for skeletal fixation, the lateral tissue plane may even be preserved, facilitating the dissection and exposure of the radial nerve. The lower lateral cutaneous sensory nerve can be followed through the lateral head of the triceps to the radial nerve. However, during revision exposures, this anatomic landmark may not be as readily identifiable. In cases where the Gerwin approach was used at the index procedure (Fig. 4A), it may be possible to find motor branches to the lateral head of the triceps during its reelevation, which can be followed to the proximal stump of the radial nerve. If the location of injury is more proximal or the lateral triceps cannot be safely reelevated, the radial nerve can be found proximally between the long and lateral head of the triceps in the triangular window (see Fig. 3, points A–B).

If clean tissue planes cannot be established and followed to the region of nerve injury, gross anatomic musculoskeletal landmarks can be used. The proximal segment of the radial nerve most frequently crosses the midpoint of the humeral shaft at the level of the distal end of the deltoid tuberosity radiographically (see Fig. 3, point C). This same point can be located immediately deep to a point 2 cm proximal to the proximal-most tip of the triceps aponeurosis (see Fig. 3, points C–D; see Fig. 4B, green bracket).

The distal segment of the radial nerve crosses the humerus laterally at a point 10 cm proximal to the lateral epicondyle (see Fig. 3, point E; see Fig. 4B, yellow bracket). The same point can be identified by placing the elbow at 45° of flexion and marking a line indicating a proximal extension of the anterior aspect of the forearm musculature (see Fig. 4B, blue line and blue dots). This line approximates the anterior edge of the BR, and its intersection with the humerus indicates where the radial nerve passes between the BR and the brachialis muscle bellies at the lateral aspect of the humerus.

Once the nerve segments have been found proximally and distally, they can be followed reliably into the zone of injury, exposing the neuromata at their ends (see Fig. 4C, green arrowheads). The neuroma tissue prevents ordered nerve regrowth and therefore needs to be trimmed to the level of healthy-appearing fascicles in order to facilitate a functional repair. The "bread-loafing" technique is commonly described in this setting. Thin cross-sections are taken in the direction of diseased tissue toward healthy tissue and examined at each section for the appearance of the fascicles (see Fig. 4D). Sectioning is stopped when normal-appearing fascicles are reached. Healthy

fascicular ends are an absolute requirement for nerve regeneration. However, reducing the amount of resected tissue to only the required amount to reach this level is important in limiting the net nerve gap. The final nerve gap (see Fig. 4C, green bracket) is the sum of the gap created by nerve end retraction (see Fig. 4C, blue bracket) and additional proximal and distal tissue loss from neuroma resection (see Fig. 4C, green arrowheads). The size of the defect can be mitigated through proximal and distal mobilization of the nerve (or even nerve transposition, as mentioned previously) in order to simplify subsequent reconstructive steps.

The technique used to bridge the resultant gap between cleaned nerve ends depends on the gap size. Optimal techniques to facilitate nerve regeneration across the gap, especially with regards to autograft versus the increasingly popular option of acellular allograft, remain controversial. However, classic teaching would dictate the following general guidelines: nerve gaps less than 3 cm can be bridged effectively using a collagen conduit; nerve gaps less than 5 cm can be bridged effectively using acellular allograft (see Fig. 4E, green arrowhead); and nerve gaps greater than 5 cm require autogenous graft.[23] Adjunct techniques consisting of a variety of nerve wraps or other neuroprotective implantables (see Fig. 4E, blue arrowheads) are becoming increasingly popular; however, data supporting any technique over any other technique remain limited.

Tendon and Nerve Transfers

In cases whereby the proximal defect is irreparable or the time window for direct repair has been missed, distal nerve transfers or tendon transfers offer an extra-anatomic set of reconstructive tools. The outcomes for these procedures are consistently good in the context of radial nerve injuries,[24] prompting some groups to promote nerve and tendon transfers over primary repair as the treatment of choice regardless of the time or nature of injury. In addition, combination surgeries using both nerve and tendon transfers to provide a mix of their respective benefits are becoming popular, blurring the lines between these 2 treatment avenues.[25]

All transfer surgeries require removing functionality from one source in order to replace functionality that has been lost someplace else. The details of each transfer sequence are designed to reduce the donor site morbidity and expedite the postoperative return of function to the recipient site by matching magnitude and direction of activity (synergistic transfers). An overview of the unique pros and cons of a nerve transfer surgery versus tendon transfer surgery is discussed briefly in later discussion. There is continued debate regarding outcomes from these procedures, with some case series reporting superiority of the more recently developed nerve transfers,[26] and some reporting equivalence.[25] Interpretation of these results is complicated by the limited number of centers publishing on this topic, the tremendous selection bias in these retrospective studies, and the differences in technique and reporting used between studies.

Nerve transfers

Donor nerves are selected that are redundant (have multiple motor pedicles or multiple muscle groups with overlapping function), are size matched to the recipient nerves, are locally available to the recipient nerve, and exist in a host with excellent neuroregenerative capacity (young age). Nerve transfers for high radial nerve injury require the sacrifice of nerve branches from the median nerve to supplement radial nerve branches. The most commonly used is transfer of a flexor digitorum superficialis branch to the extensor carpi radialis brevis (ECRB) motor branch to restore wrist extension, and a flexor carpi radialis motor branch to posterior interosseous nerve branch to restore digital extension.[27]

There are 2 primarily theoretic benefits to nerve transfer surgery: (1) because the extensor digitorum communis (EDC) tendons are left free, there is potential for return of individual digital extension; (2) because most anterior interosseous nerve innervated motor units receive motor innervation at multiple points, there is limited loss of function of the donor motor units. Similar to other procedures that depend on regenerating nerves reaching motor endplates, however, there is a finite window during which this procedure should ideally be performed within 6 months from injury in order to allow for successful reinnervation of distal endplates in less than 12 months, although many surgeons use 12 months from injury as the procedural cutoff. In addition, patients will not notice a difference in function immediately following surgeries because functional return depends on successful regeneration of nerves to their motor endplates. This generally takes more than 6 months and may ultimately be unsuccessful, contributing to patient frustration.

Tendon transfers

Donor tendons are selected with redundant function, where tendon excursion is appropriate

for their intended target, and activation during stereotyped functional activities is synergistic with the intended target. The only available functioning donor tendons are flexors. A commonly used tactic for tendon transfers is the pronator teres to the ECRB for wrist extension, the FCU to the EDC (conjoined) for digital extension, and the palmaris longus to the EPL for thumb extension.[28] However, a wide variety of alternatives and modifications exist with similarly good outcomes.

The primary benefit of tendon transfers is their early effect. Extensive physical therapy to provide guidance during a protected return to activities is generally recommended, which means there is a postoperative period of several months during which the patient will have activity restrictions and splint requirements. In addition, these tendon transfers depend on the side-to-side tenorrhaphy of the EDC tendons, sacrificing individual digital extension. However, the mechanical link between joints and powered motor units is immediately restored at the time of surgery, yielding immediate results, so patient satisfaction tends to be high.

SUMMARY

Radial nerve injuries are a common complicating factor in the treatment of humeral shaft fractures. Expectant management of nerve injury during closed treatment of fracture is standard of care. Serial physical examination of BR and ECRL function along with targeted EMGs at ~4 weeks and ~8 weeks from injury facilitates early differentiation between neuropraxic injuries (which will recover by themselves) and high-grade axonotmetic and neurontmetic injuries (which require surgical intervention). Early exploration and repair for nonrecovering injuries should be performed ~3 to 4 months from injury (earlier if there is a documented transection) using a reconstructive technique based on the size of the nerve gap. In cases whereby direct repair is unlikely to result in a good outcome, nerve transfers, tendon transfers, or a combination of both can be performed with expectation of a favorable outcome.

CLINICS CARE POINTS

- Expectant management of appropriately selected radial nerve injuries in conjunction with humeral shaft fracture requires serial examinations for early identification of nonrecovering injuries.
- Nerve transections identified during fracture fixation and not treated with acute reconstruction should be tagged under physiologic tension to prevent retraction of the nerve ends and worsening of the nerve gap, then referred for early specialist care.
- Knowledge of anatomic landmarks, including the proximal radial nerve crossing the midpoint of the humerus 2 cm proximal to the triceps aponeurosis, and the distal radial nerve crossing the lateral aspect of the humerus 10 cm proximal to the lateral epicondyle, can assist in revision nerve surgeries.
- Secondary intervention for nerve injury should be performed as early as a diagnosis of a nonrecovering nerve injury is suspected, to maximize the time for regeneration to motor endplates before loss of endplates becomes irreversible (~12 months).

DISCLOSURE

The corresponding author has a consulting agreement with DePuy Synthes.

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