Understanding and Treating Iatrogenic Nerve Injuries in Shoulder Surgery

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Abstract

As surgical techniques and technology continue to advance in shoulder surgery along with the increased use of regional anesthesia, it is important to remember that iatrogenic nerve injuries remain a possible complication. Iatrogenic nerve injuries associated with shoulder surgery lead to patient disability and distress, increased healthcare costs, and possibly additional procedures. To obtain the best possible outcome for the patient after the nerve injury has been discovered, a timely appreciation of the management options is necessary rather than expectant management. Early recognition, appropriate neurodiagnostic testing, and prompt treatment or referral are mandatory for optimal outcomes.

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Nerve injuries associated with shoulder surgery are devastating and lead to patient disability and distress, including functional deficits, intractable neuropathic pain, increased costs, additional procedures, and possible physician litigation. This article discusses the causes of iatrogenic nerve injuries associated with shoulder surgery, prevention techniques, and the diagnosis and management of iatrogenic nerve injuries to achieve optimal patient outcome.

Iatrogenic Nerve Injuries Associated With Shoulder Surgery

Nerve Injuries Associated With Regional Anesthesia

Regional anesthesia such as interscalene, supraclavicular, infraclavicular, and suprascapular nerve blocks and peripheral nerve catheters are routinely used in shoulder surgery. Nerve blocks and peripheral nerve catheters can improve patient satisfaction, decrease postoperative opioid use, and may even allow faster functional recovery of the operated limb.[1] The administration of regional anesthesia, however, has the potential to cause iatrogenic nerve injuries. The frequency of peripheral nerve injuries after peripheral nerve blocks varies from 0% to 5%.[2–4] Transient paraesthesia may occur in up to 15% of patients after a peripheral nerve block, with 99% resolving within 1 year.[5] The incidence and prognosis of more permanent neurologic deficit remain unclear. Potential complications of peripheral nerve blocks include direct puncture of peripheral nerves, compressive hematoma, or local neurotoxicity of local anesthetics.[3,6]

Direct puncture or transection of nerves with needles may lead to mechanical damage to the nerve as well as increased neurotoxicity in intrafascicular injections due to the exposure of the nerve to higher concentrations of local anesthetic. In addition, intrafascicular injection has also been shown to be associated with higher injection pressure and risk of nerve injury compared with perineural injections in experimental studies.[7] Neurotoxicity has been shown to be both dose and time dependent.[8] Although cases of nerve injury have been reported with the use of continuous peripheral nerve catheters, it remains unclear whether this is due to neurotoxicity from prolonged exposure to local anesthetic.[9] Iatrogenic vascular injury surrounding the peripheral nerves during insertion of the needle may cause compressive hematoma, leading to nerve ischemia. Ultrasonography-guided injection technique allows practitioners to visualize nerves and their adjacent structures and the location of the needle tip and to potentially use lower volume of anesthetic to achieve anesthesia.[6,10]

Phrenic nerve palsy and hemidiaphragmatic paresis are not uncommon occurrences after interscalene block; however, the respiratory consequences remain questionable as the vast majority of patients in the literature require no specific treatment of this complication.[11] Although most phrenic nerve palsy is transient, persistent phrenic nerve palsy has an estimated incidence of up to 1 in 100.[12] Modifications of interscalene blocks have been developed to decrease the rate of phrenic nerve palsy, which include reduction in local anesthetic volume and concentration.[13,14] Superior trunk, suprascapular, and axillary nerve blocks have also been advocated to mitigate the risk of phrenic nerve palsy.

Thorough physical examination before, during, and after regional anesthesia is important for early recognition of nerve injuries and to avoid confusion regarding the etiology of nerve injury. Regional nerve blocks of the shoulder generally block the brachial plexus at the nerve root or trunk level. Therefore, it is highly unlikely that an isolated branch of the brachial plexus (ie, axillary nerve) is injured from regional anesthesia. Isolated sensory and/or motor dysfunction in a terminal branch of the brachial plexus is concerning for iatrogenic nerve injury. Examination of individual terminal branches must be done postoperatively and in the first postoperative visit to determine the presence and etiology of an iatrogenic nerve injury. A recent study by Horneff et al[15] discussed the presence of preoperative distal mononeuropathy in patients undergoing arthroscopic rotator cuff repair, highlighting the importance of preoperative history and physical in the diagnosis of nerve injuries. In addition, special attention should be paid to elicit preoperative history of cervical radiculopathy, diabetic neuropathy, or brachial plexopathy.

Nerve Injuries From Patient Positioning

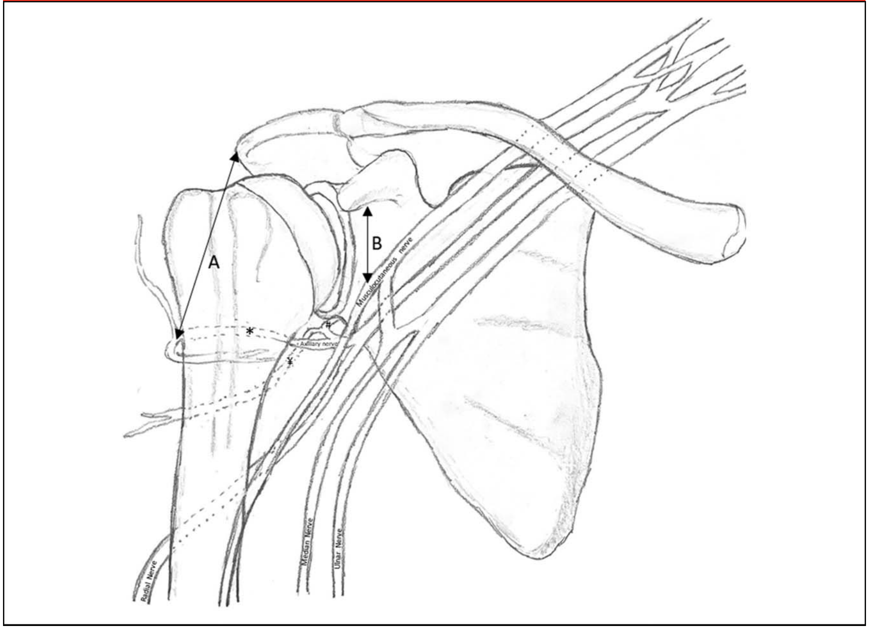
Intraoperative patient positioning during shoulder surgery may lead to iatrogenic nerve injuries from prolonged compression and/or traction of vulnerable nerves, whether the patient is positioned in beach chair or lateral decubitus. Although lateral decubitus positioning allows for improved visualization and access to the glenohumeral joint, there is risk when lateral and axial traction is applied for improved visualization when surgical time increases as traction injuries to peripheral nerves and the brachial plexus have a reported incidence up to 10% to 30%.[16] In a study of 120 patients, Andrews et al[17] reported two ulnar nerve neurapraxias and one musculocutaneous neurapraxia. Similarly, in a study of 439 patients undergoing shoulder arthroscopy in the lateral decubitus, Ogilvie-Harris et al reported one musculocutaneous nerve injury.[18] Maximum visibility with minimal strain on the brachial plexus has been shown to be achieved with 45° forward elevation and either 0° or 90° of abduction.[19] Compression nerve injuries of the down-side/dependent extremities may also occur in the lateral decubitus position. An axillary roll should be placed to prevent compression of the dependent brachial plexus. The patient's lower extremities should also be well padded to avoid common peroneal nerve compression.

Although some surgeons advocate the beach chair position due to the belief that this may lead to decreased rates of neurologic injury, other complications such as cerebral desaturation events have been reported. By contrast, beach chair positioning may risk greater auricular nerve neurapraxia or lesser occipital nerve neurapraxia due to head positioning as well as lateral femoral cutaneous nerve neurapraxia from the belt restraint.[20] It is important to recognize that sciatic nerve compression is usually avoided by flexing the knees to 30°. Currently, no consensus exists about the increased rate of neurologic injury in the lateral decubitus or beach chair position, and the surgeon should choose the position he or she believes will allow him or her to perform the surgical procedure in an expeditious manner while paying attention to padding the patient's extremities and the patient position.

Nerve Injuries From Retractors

As retractor placement, even when carefully placed, may lead to traction or crush injuries to the peripheral nerves, the operating surgeon must have a keen awareness of the location of the nerves intraoperatively. Traumatic retraction may be reduced using carefully placed flat/blunt retractors in areas of risk (Figure 1). In the deltopectoral approach, the musculocutaneous nerve is to be protected by avoiding vigorous retraction of the conjoint tendon medially. The musculocutaneous nerve pierces the coracobrachialis and short head of the biceps 3 to 8 cm from the coracoid. If a surgeon chooses to use the deltoid splitting approach, then he must be careful to protect the axillary nerve as prolonged soft-tissue retraction can place the axillary nerve at risk being approximately 5 to 7 cm from the acromial edge.

Figure 1



Anatomic relationships of the axillary and musculocutaneous nerves. Nervous anatomy of the shoulder. Line A represents distance from the anterolateral corner of the acromion to the axillary nerve, which is between 5 and 7 cm. Line B represents distance from the coracoid to the point at which the musculocutaneous nerve pierces the short head of the biceps and coracobrachialis, which is between 3 and 8 cm. "*" represents the anterior division of the axillary nerve, which innervates the anterior and middle deltoid. "#" represents the branch of the axillary nerve to the teres minor. "¥" represents the posterior division of the axillary nerve, which innervates the posterior deltoid and terminates as the superior lateral cutaneous nerve.

Nerve Transection Injuries

Nerve transection injuries may occur after sharp transection with a surgical instrument such as a blade, spinning drill or sharp retractor placement, aberrant portal placement during arthroscopy, or screw penetration.[21] Although it is intuitive, it bears reminding that surgeons must have a detailed three-dimensional understanding of the anatomy about the shoulder and must perform careful dissection with adequate visualization, identification, and protection of the peripheral nerves. Transection injuries of the peripheral nerves should be recognized and managed early as primary repair and early nerve transfers lead to better results.

In shoulder arthroscopy, the posterior portal is the primary viewing portal of the glenohumeral joint. This portal is usually located approximately 3 to 4 cm superior to the quadrangular space; therefore, care must be taken to avoid improper portal placement and trajectory to avoid injury to the axillary nerve. Passing an 18- or 20-gauge spinal needle into the glenohumeral joint before placement of the trochar helps confirm proper placement of the posterior portal. The suprascapular nerve is also at risk with the Neviaser portal and with the passage of instruments along the lateral border of the scapular spine. The axillary nerve is at particular risk when performing an arthroscopic humeral osteoplasty of a large inferior humeral osteophyte. Although an accessory posteroinferolateral portal made under direct visualization has been advocated to minimize the risk of iatrogenic axillary nerve injury, this does not eliminate the risk even in the hands of experienced shoulder surgeons.[21,22]

Screw or drill penetration may also cause peripheral nerve injury, particularly from screws placed into the glenoid. Posterior glenoid drilling for fixation of the glenoid baseplate in reverse shoulder prosthesis must be placed with care to avoid injury to the suprascapular nerve in the spinoglenoid notch.

Electrocautery/Coblation Injuries

Thermal injuries to the nerve may also occur in shoulder surgery, particularly in the management of shoulder instability and arthroplasty. Though no longer routinely performed, injury to the axillary nerve has been reported with thermal capsulorrhaphy in the inferior axillary recess.[23] In addition, thermal injury to the axillary nerve may occur when dissecting the shoulder capsule during shoulder arthroplasty. Injury is most likely caused by heat penetration from the radiofrequency probe through the capsule to the nerve, as the axillary nerve is between 1 and 8 mm inferior from the inferior extent of the shoulder capsule. Purely sensory and both motor and sensory injuries to the axillary nerve have been described.[24] To reduce the risk of axillary nerve injury, reducing the temperature of the probe and minimizing the time in the inferior axillary recess have been advocated.

Diagnosis

Intraoperative Nerve Monitoring/Stimulation

Continuous intraoperative nerve monitoring is used regularly in spinal surgery; however, the literature regarding nerve monitoring in shoulder surgery is not as robust. In 2007, Nagda et al used continuous intraoperative nerve monitoring during shoulder arthroplasty. Of the 30 patients studied, 17 had episodes of nerve dysfunction during surgery. Intraoperative compromise of nerve function was signaled by sustained neurotonic electromyographic activity or greater than 50% amplitude attenuation of transcranial electrical motor evoked potentials (or both). They concluded that routine nerve monitoring may be considered for patients with decreased motion and history of open shoulder surgery.[25]

Esmail et al[26] used intraoperative axillary nerve monitoring for patients undergoing arthroscopic shoulder stabilization and noted change in heat application in 4 of 11 patients undergoing thermal capsulorrhaphy. Warrender et al[27] used intraoperative nerve monitoring in proximal humerus fracture fixation and suggested that transcranial electrical motor evoked potentials were sensitive indicators of impending iatrogenic nerve injury during open surgical management of proximal humerus fractures, particularly in patients with underlying cervical spine disease, low body mass index, diabetes mellitus, and/or surgical delay in surgical treatment greater than 2 weeks. In 2016, Parisien et al[28] used intraoperative nerve monitoring for reverse total shoulder arthroplasty and total shoulder arthroplasty and concluded that clinical utility of routine nerve monitoring remains in question due to the high level of nerve alerts and lack of persistent postoperative neurologic deficits.

Most shoulder surgeons do not routinely use intraoperative nerve stimulation to identify nerve injuries, but this is a tool that should be considered in difficult situations such as revision open shoulder surgery with scarring. Failure of the stimulation of the nerve to elicit motor response may indicate conduction block or transection. For precise localization of nerve injury, stimulation at multiple points along the nerve may be helpful. DC stimulators only result in single muscle twitches and have the potential to cause chemical changes in the surrounding tissue possibly leading to nerve damage. Contrarily, pulsing AC allows for frequent and continuous stimulation without diminished nerve response, allowing for more definitive nerve identification. Overall, neurologic monitoring may be effective for certain patients; however, the literature does not support its routine use for the prevention of intraoperative nerve injuries.

Postoperative Diagnosis

A comprehensive postoperative physical examination is necessary for the timely recognition of potential iatrogenic nerve injuries. If there is delayed presentation of the patient from a surgery performed by another surgeon, a detailed history including date of surgery, postoperative course, and obtaining surgical reports is important.

Electrophysiologic testing for the diagnosis and characterization of nerve injuries includes nerve conduction studies (NCSs) and needle electromyography (EMG). Overall, NCS is more valuable for nerve injuries as EMG may remain normal and are largely qualitative in nature. Conventional teaching usually holds that electrodiagnostic studies should not be performed until at least 3 weeks as this is when fibrillation potentials indicating denervation are seen on EMG. Earlier studies, however, do help determine whether the injury is electrophysiologically incomplete or complete because even a single motor unit under voluntary control indicates that the nerve injury is not complete.[29] Studies performed at 3 to 4 weeks still remain the benchmark and ideal initial timepoint. Studies done approximately 3 months after an injury may detect early reinnervation with the presence of nascent potentials. Generally, surgical management of nerve injuries can be considered if no clinical or EMG/NCS evidence of recovery is present by 3 months.

The primary end points of NCS are compound muscle action potentials (CMAPs) for motor nerves and sensory nerve action potentials (SNAPs) for sensory nerves. In neurapraxias, CMAPs and SNAPs elicited on stimulation distal to the lesion are maintained indefinitely, whereas stimulation proximal to the lesion reveals conduction block (focal demyelination), loss of CMAP amplitude, change in CMAP configuration, and slowing of conduction velocity. When remyelination is complete, these findings generally improve or disappear. In axonotmesis and neurotmesis, CMAPs and SNAPs elicited distal to the injury decrease in amplitude in rough proportion to the degree of axon loss. After Wallerian degeneration is complete, conduction in the affected axon or axons is lost.

Treatment Options

Acute Injuries

Nerve continuity determines the method of management of acutely recognized iatrogenic peripheral nerve injuries. Nerve injuries with the nerve still in continuity (neurapraxia) resulting from stretch, crush, thermal injury should be managed as closed nerve injuries with close follow-up as the zone of initial injury may not be immediately discernible and spontaneous recovery may still be possible. Intraoperatively, early recognition is imperative with cessation of the offending maneuver, instrument placement, or patient positioning to prevent further damage.

The benchmark for acute small gap nerve injuries remains primary end-to-end neurorrhaphy. If an acute nerve injury is recognized, it should be primarily repaired within 72 hours by a surgeon facile with microsurgery technique. Delayed repair after this period up to approximately 7 days will require freshening of the proximal and distal ends before coaptation. If the nerve is not repaired acutely, the proximal and distal stumps retract, increasing the need for grafting which is more likely to have a worse outcome. Nerves should be repaired with 8-0 or 9-0 nonabsorbable microsutures under operating microscope with an epineural repair as the preferred method of coaptation with attempt to realign fascicles using the least amount of sutures possible to achieve a tension-free repair. Perineural repair may be performed but may increase the potential for fibrosis and require longer duration without evidence of superior outcomes.[30] Fibrin-based glue for coaptation is an alternative to suture repair that has been explored; however, no strong human evidence exists comparing its efficacy with conventional suture methods.[31] Nerve injuries with larger gaps (>2 cm) that do not allow for tension-free primary end-to-end neurorrhaphy require alternatives such as nerve grafting, nerve conduit, or nerve transfer.

Chronic Injuries

Chronic peripheral nerve injuries resulting from shoulder surgery are difficult clinical situations. Detailed history and physical regarding the patient's medical and surgical history as well as review of surgical reports and electrophysiologic studies are important to determine the etiology, chronicity, treatment options, and prognosis of the patient's nerve injury. With all suspected nerve injuries, electrophysiologic studies should be performed approximately 3 weeks after the injury so as to establish a baseline. In cases of neurapraxic nerve lesions, spontaneous clinical recovery is likely and electrophysiologic studies will provide objective evidence to the nature of the injury so that expectant management is appropriate. On the contrary, injuries involving discontinuity result in abnormal electrophysiologic findings with distal stimulation as discussed earlier, including changes in CMAP, SNAP, fibrillation potentials, and lack of spontaneous recovery.

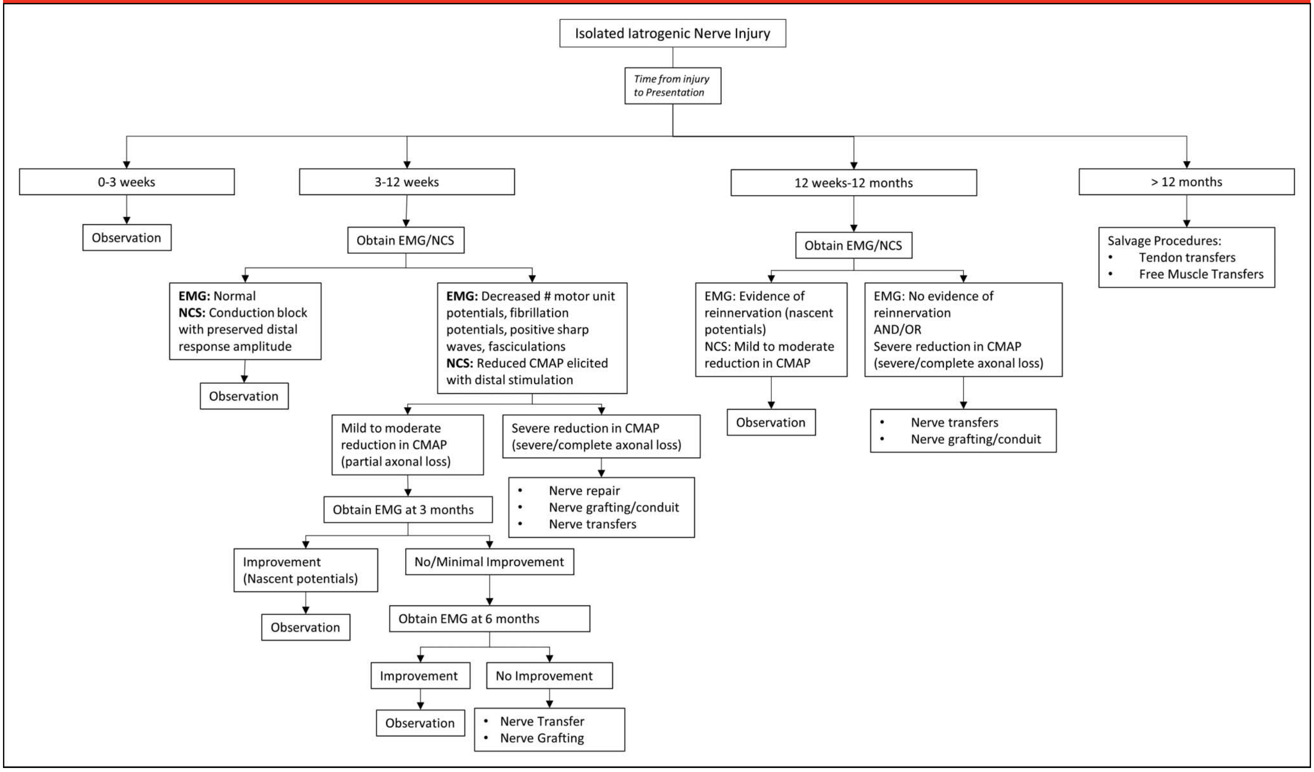
In the setting of chronic iatrogenic peripheral nerve injuries with unclear etiology, surgical intervention should only be performed if there is no improvement clinically or on EMG/NCS over a period of at least 3 months (Figure 2). Options for chronic reconstruction of peripheral nerve injuries include autogenous interpositional nerve grafts, allografts, nerve transfers, and muscle transfers. It is important to note that attempts at reinnervation of motor neurons may be limited by motor end plate viability, deteriorating stromal environment for regeneration, and progressive structural changes in muscle including atrophy and fibrosis after nerve injury. Therefore, functional reinnervation is unlikely after 12 months. The timeframe for successful sensory reinnervation is longer, but the maximum timeframe remains uncertain.[32]

Autogenous nerve grafts are bridging scaffolds that provide neurotrophic factors and Schwann cells allowing for directed axonal growth from the proximal to distal stumps. Allograft nerve graft may also be used; however, there is potential immunogenicity and have not shown the same recovery as autogenous grafts. Whereas the authors have not had this experience, the literature suggests that long-nerve grafts may be comparable with nerve transfers for axillary nerve injuries.[33]

Nerve transfers, or neurotization, essentially convert a proximal nerve injury to a distal nerve injury by the transfer of a redundant nerve near the target to the distal nerve stump. Nerve transfers about the shoulder include among others: spinal accessory to suprascapular nerve, medial triceps branch of radial nerve to axillary nerve, ulnar nerve to biceps branch of the musculocutaneous nerve, spinal accessory nerve to subscapular nerve, and medial pectoral nerve to proximal spinal accessory nerve. Nerve transfers allow for earlier reinnervation before degeneration of the neuromuscular junction due to the proximity of donor nerves to target motor end plates. In addition, in cases of severe scarring or long gaps, nerve transfers may be more reliable than nerve grafting.

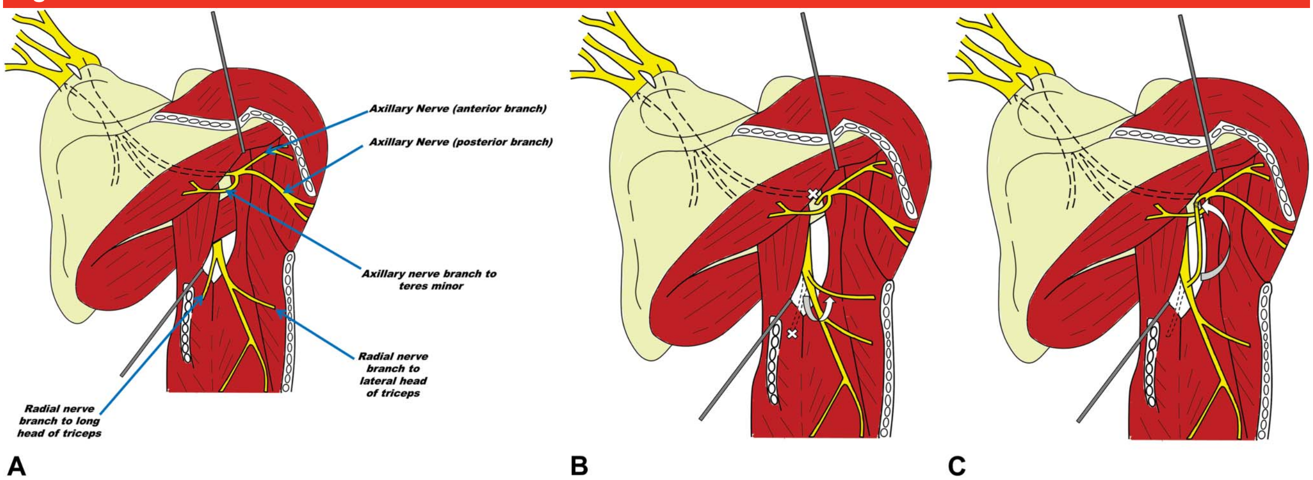
The partial radial nerve to axillary nerve transfer (Figure 3) is particularly useful in the management of chronic axillary nerve injuries as the radial nerve has nearby motor fascicles that are transferrable with minimal functional loss. Transfer of the radial nerve to the axillary nerve may improve dynamic stability of the shoulder, strength, abduction, and external rotation.[34,35] Intraoperative use of a nerve stimulator is vital to isolate the proper fascicle for radial nerve transfer. Care must be taken to avoid sacrificing the radial nerve proper to prevent downstream motor/sensory dysfunction. With intraoperative nerve stimulation of the branches of the radial nerve, elbow extension rather than wrist extension is grossly

Figure 2



Management protocol for isolated, postoperatively discovered iatrogenic peripheral nerve injuries.

Figure 3



Radial to axillary nerve transfer. **A**, Relevant surgical anatomy. **B**, Demonstrates radial nerve branch to long head of triceps being isolated as well as neurolysis of the anterior branch of the axillary nerve. **C**, Demonstrates coaptation of the radial nerve branch (donor) to the axillary nerve (recipient). Drawing by Dr. Antony Hazel.

appreciated. The anterior/deep division of the axillary nerve is the recipient nerve, as the anterior and middle deltoid provide much of the shoulder abduction function compared with the posterior deltoid, which is innervated by the posterior division of the axillary nerve.

For chronic irreparable musculocutaneous nerve injuries, the ulnar nerve to musculocutaneous nerve transfer (Oberlin) may be used to restore elbow flexion.[36] A single, large fascicle of the ulnar nerve can be identified with a portable nerve stimulation unit with maximal contraction of the flexor carpi ulnaris without contraction of the ulnar intrinsic muscles. This fascicle is then divided distally as close to the musculocutaneous nerve branch to the biceps and coapted.

A spinal accessory to suprascapular nerve transfer may be used in the setting of chronic irreparable suprascapular nerve injury. For this transfer, it is important to preserve branches to the upper and middle trapezius when using the spinal accessory nerve as a donor.

After motor unit degeneration, attempts to restore neurologic function such as nerve grafts or nerve transfers are no longer indicated. Muscle transfers are an option for chronic reconstruction in these situations. The triple tendon transfer, an Eden-Lange variant, has been described for trapezius paralysis.[37] Options for chronic deltoid paralysis include bipolar latissimus dorsi transfer, pedicled pectoralis transfer, and trapezius transfer.[38,39] Triceps to biceps transfer has been described for restoration of elbow flexion.[39] Free muscle transfers, such as gracilis free muscle transfer, are another option for shoulder reconstruction in chronic nerve injuries with shoulder dysfunction.

Medical Management

Neuropathic pain resulting from iatrogenic nerve injuries may be medically managed with tricyclic antidepressants, antiepileptics such as pregabalin and gabapentin, duloxetine, venlafaxine, lamotrigine, and in severe cases with opioids.[40]

Referral

Early referral to a surgeon comfortable with the complex management of peripheral nerve injuries is important to achieve the best possible outcomes for patients who unfortunately suffer from an iatrogenic nerve injury. Delayed presentations and care decreases the chance of successful outcomes in an already difficult situation.

Summary

Iatrogenic nerve injuries resulting from shoulder surgery are rare but are potentially devastating injuries that require prompt diagnosis, treatment, or referral. Care must be taken in the preoperative phase with anesthesia and patient positioning, intraoperatively with careful dissection and appropriate usage of surgical instruments, and postoperatively with thorough examination and high index of suspicion for iatrogenic complications. Strategies to improve nerve injury treatment allowing for successful reinnervation and return of function should be the focus of future work.

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