

AXILLARY NERVE PALSY AND DELTOID MUSCLE ATONY

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Abstract

» Axillary nerve palsy presents in a variety of ways and can be a substantial source of dysfunction about the shoulder. It is important for the orthopaedic surgeon to recognize axillary nerve injuries in the setting of acute trauma. The surgeon should also be aware that such injuries are part of the complication profile of both open and arthroscopic surgery involving the shoulder.

» Early recognition and treatment is important as full recovery becomes less likely in association with chronic axillary nerve injuries. However, diagnosis is often difficult and can be masked by compensatory mechanisms of the adjacent shoulder muscles.

» Several treatment options are available, depending on the extent and type of injury, including neurolysis, neurorrhaphy, nerve-grafting, and nerve transfer. This article highlights the etiology, diagnostic pathways, and treatment of both traumatic and iatrogenic axillary nerve injuries.

Peripheral nerve injuries about the shoulder are a cause of pain and dysfunction and have a variety of etiologies and presentations. Among these, axillary nerve injury is one of the most common^{1,2}, representing >50% of all infraclavicular brachial plexus injuries in a recent study of 101 patients³. Axillary nerve injury can occur in cases of trauma, with 1 study demonstrating a rate of 16% (38 of 240) among patients with anterior shoulder dislocation⁴. It is also an increasingly well-known and devastating iatrogenic complication of shoulder surgery, with the diagnosis being frequently delayed or missed^{5,6}. A delay in diagnosis may be attributable to absent or delayed symptomatology due to compensation by the muscles of the rotator cuff or due to an occult presentation in the setting of severe associated injuries, such as proximal humeral fracture or shoulder dislocation⁷.

Axillary nerve injury also may occur during other forms of glenohumeral

trauma, with or without associated subluxation of the humeral head, resulting in compression, traction, or contusion of the nerve along the deep surface of the deltoid muscle. Such mechanisms of injury frequently have been reported in association with motor-vehicle accidents and contact sports (e.g., skiing, football, and rugby)^{8,9}. Additionally, atraumatic and chronic forms of injury may occur secondary to repetitive microtrauma (e.g., in overhead throwing athletes such as baseball pitchers), brachial neuritis, quadrilateral space syndrome, blunt trauma, and neural entrapment secondary to space-occupying lesions⁷.

A delay in diagnosis is concerning as clinical outcome is strongly dependent on the time from the injury to treatment⁷, with a worse prognosis for injuries that remain undiagnosed for more than 6 to 12 months^{7,8}. Therefore, it is critical to recognize the clinical settings that demand a high index of suspicion for axillary nerve injury and to understand corresponding evidence-based treatment options. To this

end, we present an updated review of the anatomy, pathophysiology, diagnosis, treatment, and prevention of axillary nerve injury and dysfunction.

Anatomy

The axillary nerve, also known as the circumflex nerve, is a mixed motor and sensory nerve that originates from the ventral rami of the fifth and sixth cervical nerve roots⁵. Most commonly, the axillary nerve arises from fibers of the posterior cord of the posterior division of the brachial plexus¹⁰; however, variants have been reported in as many as 20% of patients, in whom the nerve arises from the posterior division of the upper trunk¹¹. The axillary nerve travels obliquely along the anterior surface of the subscapularis muscle and posterior to the axillary artery before diving inferiorly into the quadrilateral space alongside the posterior humeral circumflex artery, in close proximity to the inferomedial capsule (Fig. 1)^{5,12}.

The quadrilateral space is bound by the long head of the triceps muscle medially, the humeral shaft laterally, the teres major and latissimus dorsi muscles inferiorly, and the subscapularis muscle anteriorly. After passing through the quadrilateral space, the nerve travels around the posterolateral aspect of the humeral neck, where it divides into anterior and posterior branches. The anterior branch courses around the surgical

neck of the humerus, approximately 4 to 7 cm inferior to the anterolateral corner of the acromion, and terminates in the anterior portion of the deltoid^{12,13}, supplying collateral sensory and motor branches to the anterior and lateral segments of this muscle (Fig. 2). The posterior branch provides motor innervation to the posterior portion of the deltoid muscle and teres minor muscle and provides sensory cutaneous branches to the skin overlying the posterior portion of the deltoid muscle before terminating as the superior lateral brachial cutaneous nerve¹⁴. During arthroscopic surgery, Yoo et al.¹⁵ found that the axillary nerve typically lies at or near the most inferior position of the glenoid (6 o'clock position), approximately 10 to 25 mm from the inferior glenoid rim, and is farther away from the operative field when the arm is in abduction and neutral rotation.

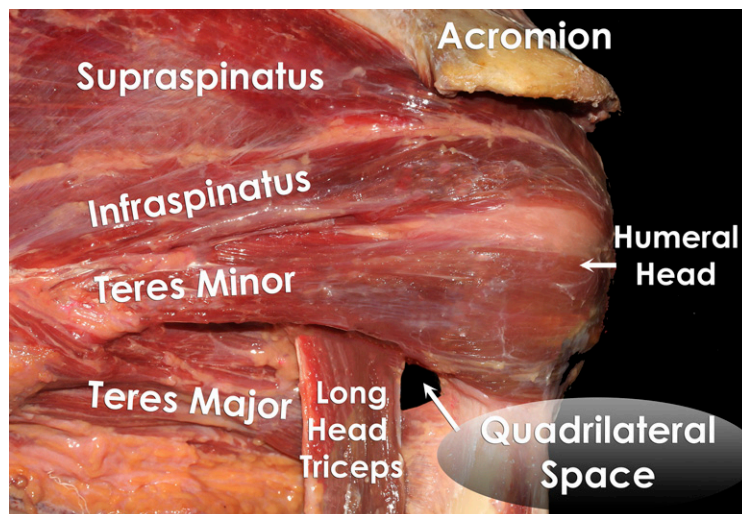
Pathophysiology of Nerve Injury

The severity of peripheral nerve injuries is graded primarily on the basis of the amount of axonal discontinuity observed. The 2 most commonly referenced systems that are used to describe nerve injury are the Seddon classification system¹⁶ and the Sunderland classification system¹⁷ (Table I). First described in 1943, the Seddon classification system involves 3 categories that correspond to the degree of axonal

discontinuity: neurapraxia, axonotmesis, and neurotmesis. Neurapraxia is the mildest of the 3 categories and typically is caused by excessive compression or traction of the nerve. It is characterized by conduction abnormality in the absence of structural deformity; that is, there is no axonal loss or interruption of the epineurium, perineurium, or endoneurium. Axillary nerve injury caused by glenohumeral dislocation or blunt trauma is commonly neurapraxic in nature¹⁸. These injuries carry an excellent prognosis, with complete resolution of symptoms typically occurring within days to weeks without surgical intervention. In contrast, axonotmesis is characterized by an injury to the epineurium and perineurium. Unlike neurapraxia, axonotmesis involves damage to the axon and the myelin sheath but spares Schwann cells involved in initiating the process of Wallerian degeneration. Although operative treatment is usually not required, axonotmetic injuries may take weeks to months to heal as governed by the rate of Wallerian degeneration (maximum rate, ~1 mm per day), and nerve recovery may be incomplete. Neurotmesis comprises the most severe form of injury, representing a complete transection of all 3 neural layers. Neurotmesis typically requires surgical nerve-grafting for the restoration of nerve functionality (as described in later sections).

Fig. 1

Posterior-view photograph of a cadaveric right shoulder, demonstrating the quadrilateral space. The quadrilateral space is bounded by the long head of the triceps muscle medially, the humeral shaft laterally, the teres major and latissimus dorsi muscles inferiorly, and the subscapularis muscle anteriorly. The axillary nerve travels through this space and around the posterolateral part of the humeral neck, where it divides into anterior and posterior branches.



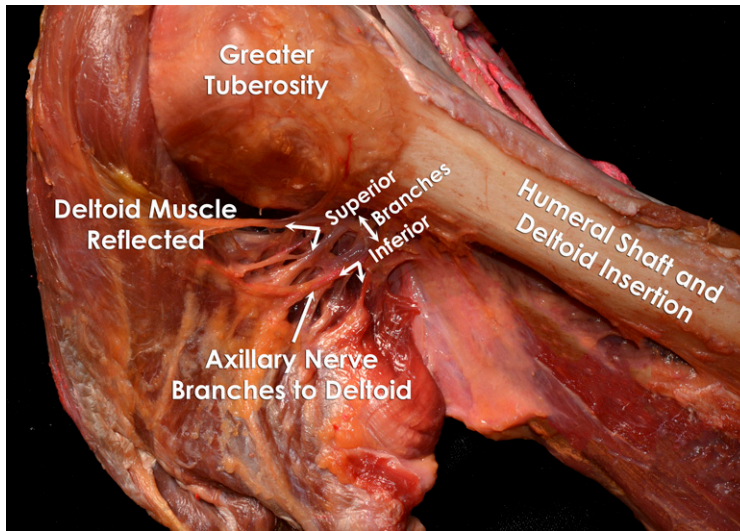


Fig. 2
Photograph of a cadaveric right humerus with the deltoid muscle belly reflected, revealing the axillary nerve as it branches to the deltoid muscle.

Unfortunately, iatrogenic axillary nerve injuries often involve axonotmesis or neurotmesis¹⁸.

Patient Evaluation

The diagnosis of axillary nerve injury can be challenging as a result of asymptomatic or subclinical presentation. While a complete and thorough physical examination should detect the identifying patterns of nerve injury, deltoid dysfunction initially may be masked in young patients with excellent compensatory mechanisms or those with difficulty complying with the examination¹¹. Injuries resulting in incomplete paralysis may not be obvious when only the anterior or posterior portion of the deltoid muscle is affected. Additionally, in some cases, the patient may retain nearly full abduction of the shoulder secondary to supraspinatus compensation.

Clinical Examination

Evaluation begins with a thorough history. A recent history of blunt trauma or shoulder dislocation makes an axillary neurapraxia more likely, whereas penetrating trauma or recent surgery may point toward laceration of the nerve. It is important to note that a patient age of >50 years and a lack of concentric glenohumeral reduction for >12 hours are known risk factors for axillary nerve injury following shoulder dislocation^{5,18}.

A focused but comprehensive physical examination of the shoulder should be performed bilaterally. Following inspection and palpation of the shoulder, sensory examination of the terminal branch of the axillary nerve (the superior lateral brachial cutaneous nerve), which supplies sensory innervation to the lateral aspect of the arm near

the distal two-thirds of the deltoid muscle, can reveal sensory changes that are suggestive of axillary nerve damage. Despite its utility, this sensory testing does not reliably exclude mixed brachial plexus injuries that may be associated with axillary nerve injury. Because of the incompleteness of sensory testing, a thorough distal strength examination is paramount as brachial plexus injuries may prolong or limit the resolution of symptoms, require intensive physiotherapy, or require additional or separate surgical interventions^{2,19}.

While the testing of deltoid muscle strength can be used to determine axillary nerve function, patients who have had a recent operation or traumatic episode may have difficulty performing such testing, which may present a challenge when pain prevents a comprehensive physical

TABLE I Seddon and Sunderland Classification Systems for Peripheral Nerve Injuries ^{16,17}			
Seddon	Sunderland	Injury Type	Neurological Deficits
Neurapraxia	I	Intrafascicular edema with conduction block, possible segmental demyelination	Neuritis, paresthesias
Axonotmesis	II	Severed axon, intact endoneurium	Paresthesia, episodic dysesthesias
	III	Severed axon, disruption of endoneurium	Paresthesia, dysesthesia
	IV	Disruption of endoneurium and perineurium	Hypoesthesia, dysesthesia, neuroma
Neurotmesis	V	Complete nerve discontinuity	Anesthesia, intractable pain, neuroma
	VI	Combination of above	Mixed presentation

examination. Special physical examination techniques have been described to aid in the identification of isolated deltoid muscle dysfunction. The swallow-tail test (Fig. 3) is performed by having the patient bend forward at the waist and maximally extend the shoulders, first with elbows extended and then with elbows flexed. A difference in shoulder extension between the normal and affected sides of $>20^\circ$ indicates a positive test²⁰. The deltoid extension lag test involves the examiner maximally extending the shoulder and elbows of the patient and then asking the patient to hold that position. A lag in shoulder extension on the affected side as compared with the contralateral side is considered a positive test²¹.

The abduction in internal rotation test²² (Fig. 4) is our preferred method for evaluating deltoid muscle function as it has been shown to be superior to both the deltoid muscle extension lag test and swallow-tail test²¹. It is performed by placing the patient's shoulder in full internal rotation with the elbow flexed

and asking the patient to abduct the shoulder. If the patient is unable to fully abduct the shoulder in this position, the examiner assists with abduction and asks the patient to maintain this position. Any abduction lag indicates a positive test. The internal rotation of the shoulder in this maneuver displaces the supraspinatus tendon insertion anteriorly, decreasing its ability to aid in shoulder abduction and increasing the sensitivity of the examination.

Further clinical examination following the identification of axillary nerve injury often allows for determination of the location of the lesion as it relates to the quadrilateral space. If the axillary lesion spares the posterior portion of the deltoid and teres minor muscles, the lesion is distal to the quadrilateral space⁸.

Imaging and Other Testing

Standard shoulder radiographs should be made for patients with suspected deltoid muscle atony. Conventional radiographs often will be normal outside the setting of dislocation or fracture.

However, slight inferior displacement, or pseudosubluxation, of the humeral head may be evident, indicating deltoid muscle dysfunction. Advanced cervical spine imaging may assist in ruling out a more proximal lesion^{23,24}. Advanced imaging studies, including computed tomography (CT) and magnetic resonance imaging (MRI), also can be performed, depending on the mechanism and clinical suspicion of the underlying injury. As in other regions of the body, MRI typically is superior for the evaluation of soft-tissue lesions such as nerve injuries. MRI scans traditionally have been useful when space-occupying lesions or fibrotic scars are compressing the nerve; however, recent advances in MRI have allowed for detailed evaluation of nerve injuries resulting from both traumatic and surgical causes. Magnetic resonance neurography also has been proposed as a method for visualizing the axillary nerve.

When clinical presentation and/or imaging studies are concerning for an axillary nerve injury, electromyography and nerve conduction studies (EMG/NCS)

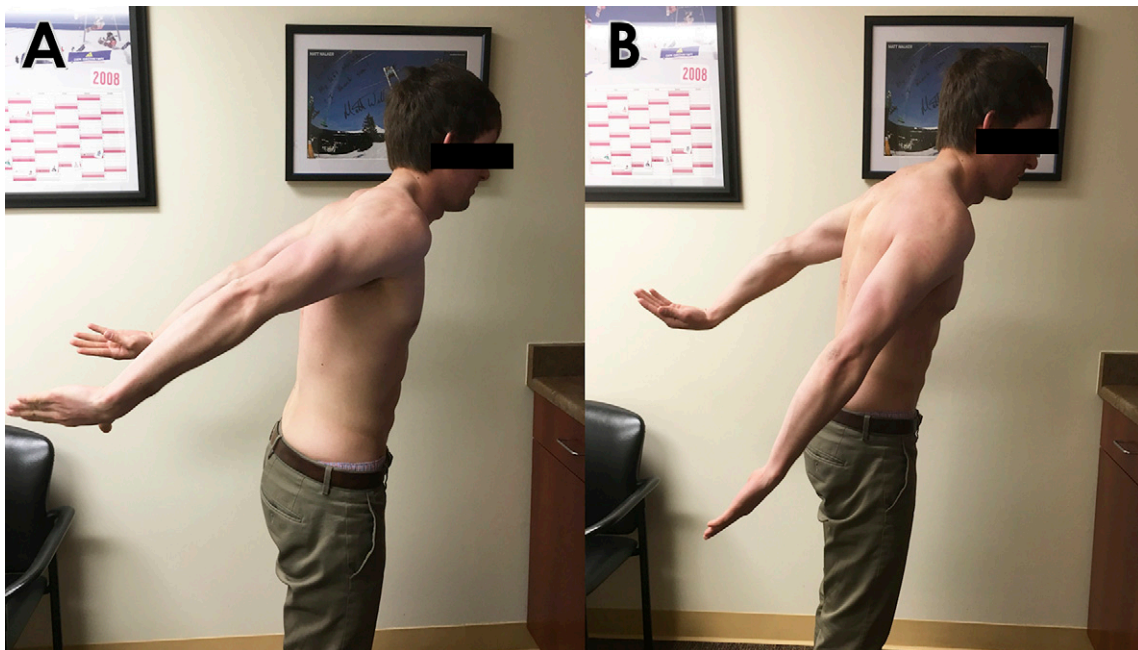


Fig. 3
Photographs illustrating the swallow-tail test. In a normal test (Fig. 3-A), both shoulders are fully extended to the same level. However, in an abnormal test (Fig. 3-B), the posterior fibers of the deltoid muscle fail to fire, thus limiting full extension of the affected shoulder.

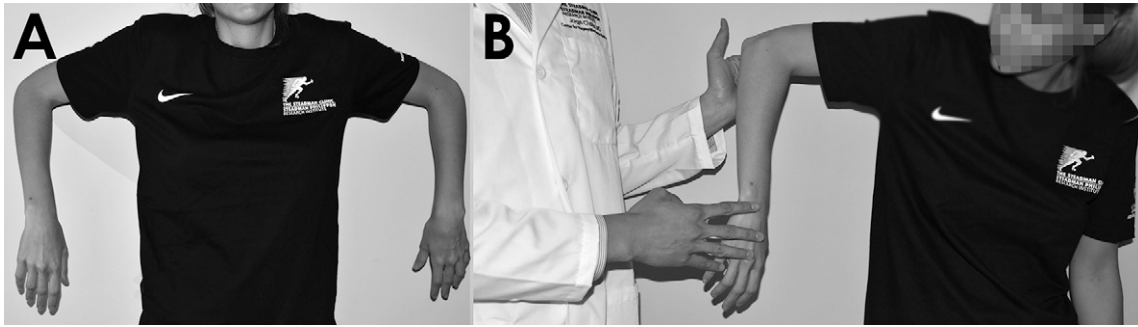


Fig. 4

Figs. 4-A and 4-B Photographs illustrating the abduction in internal rotation test. **Fig. 4-A** The patient is instructed to abduct and internally rotate the shoulder actively with both elbows flexed. **Fig. 4-B** If any discrepancies are noted, the examiner repeats the maneuver (passively) and the patient is instructed to maintain the position. If the patient is not able to do so, the test is positive.

can be used to confirm the diagnosis. One to 4 weeks typically are required for changes to manifest on EMG/NCS following an axillary nerve injury²⁴. Therefore, delaying EMG/NCS until approximately 4 weeks can be helpful for establishing a post-injury baseline for the patient and can help to better delineate the full extent of the injury.

Etiologies

Non-Surgical Injuries

Understanding the etiology or mechanism of injury is of paramount importance as the treatment of an axillary nerve injury is based on the severity and type of injury that has been sustained. A detailed clinical history is vital for treatment decision-making as each mechanism has a characteristic injury pattern. Operative treatment typically is not indicated for low-energy, non-penetrating traumatic injuries. Such injuries are usually traction, torsion, or compression neurapraxias in which the axon is not disrupted. Rather, neurological symptoms occur following trauma as a result of a conduction block due to transient demyelination or intrafascicular edema in the nerve.

Shoulder dislocation is one of the leading causes of traction injury of the axillary nerve, with the reported rates of nerve injury following dislocation ranging between 5% and 54%^{5,23}. Injuries to the nerve occur in association with anteroinferior shoulder subluxation, frank dislocation, and Bankart lesions²⁵. When nerve damage occurs in

association with acute shoulder dislocations, complete spontaneous recovery usually occurs, with reported rates in the literature ranging from 87.5% to 100% of cases^{3,24}. In the study by Visser et al.²⁴, 4 of 32 patients in whom a severe axillary nerve injury was confirmed on the basis of both clinical examination and EMG findings failed to recover full function. However, only minor residual symptomatology remained. Interestingly, those authors did not find an increasing severity of nerve injury, but they did find that the risk of nerve injury in association with dislocation increased with age ($p < 0.007$). Results have been similarly favorable in patients with low-energy torsional and compression-type injuries. Guerra and Schroeder²⁶ reported on 5 patients with torsional nerve injury, all of whom had good or excellent restoration of motor function after a mean duration of follow-up of 33 months.

Compression of the axillary nerve can occur in association with blunt, non-penetrating trauma in the absence of fracture or dislocation¹¹. These mechanisms typically involve a direct blow to the anterolateral aspect of the deltoid muscle. If compression occurs at the site where the nerve exits the quadrilateral space, isolated posterior deltoid muscle paralysis and sensory loss along the superior lateral brachial cutaneous distribution are observed. This presentation also can occur in cases of quadrilateral space syndrome, in which the axillary nerve and circumflex humeral artery

exiting the quadrilateral space are compressed by surgical adhesions, glenoid paralabral cysts, ganglion cysts, muscle hypertrophy, or displaced scapular fractures^{14,27}. Quadrilateral space syndrome also can occur after prolonged lying in the lateral decubitus position²⁸. Patients who have sustained blunt trauma to the axillary nerve distal to the quadrilateral space often present with soft-tissue swelling throughout the anterolateral aspect of the shoulder associated with full or partial deltoid muscle paralysis and variable sensory loss. In some cases, sensation may be normal in the presence of complete deltoid muscle paralysis.

In cases of blunt trauma, outcomes have been less favorable. Perlmutter et al.²⁹ reported on 11 athletes who sustained axillary nerve injury as a result of direct blunt trauma. At the time of the latest follow-up (mean, 73.8 months) all patients were still experiencing clinically apparent motor and sensory deficits of the axillary nerve. However, 10 of the 11 patients returned to the preinjury level of athletics. Similarly, Berry and Brill³⁰ reported on 13 patients who experienced axillary nerve injury as a result of blunt trauma without fracture or dislocation. Only 6 patients had complete or nearly complete resolution of neurological symptoms. Certainly, more studies are needed to determine the natural history of, and best treatment for, these types of injuries.

Torsional peripheral nerve injuries are exceedingly rare and involve rotation

or twisting of the axillary nerve on itself, resulting in a so-called hourglass-like deformity. While the underlying etiology of these injuries remains unclear, it is generally accepted that posttraumatic cases of axillary nerve injury occur secondary to inflammation and fibrosis of epineurial and perineurial tissue while spontaneous cases of nerve injury occur secondary to forceful movements that cause stretching of the axillary nerve over the scapulohumeral joint of the medial aspect of the shoulder. A clinical hallmark of axillary nerve torsion injury is the abrupt onset of pain, often associated with pronounced motor deficits. Clinical and electrophysiological recovery often do not occur even months after the initial injury. Ultimately, persistent ischemia of the axillary nerve may result in exudation of proteins into the epineurium and subsequent fibrosis that may be detectable on MRI scans²³.

Other comorbidities or medical conditions also can confound the diagnosis of axillary nerve injury. Diabetes mellitus, thyroid disease, renal disease, autoimmune disorders, or chronic liver disease may lead to peripheral neuropathies that can symptomatically mimic isolated injury to the axillary nerve^{31,32}. Furthermore, cervical compression of either the C5 or C6 nerve roots may cause isolated axillary symptomatology or may contribute to a so-called double-crush phenomenon in cases of both cervical compression and distal axillary nerve injury³⁰. A thorough history and physical examination are required to appreciate these comorbidities and to avoid misdiagnosis.

Surgical Injuries

Iatrogenic injury is a frequent and preventable cause of axillary nerve palsy. Open injuries are more likely to result in axonotmesis or neurotmesis of the nerve, which require operative repair. Mechanisms of intraoperative nerve injury include limb malpositioning, inadvertent suturing, heat penetration, aggressive retractor placement, and excessive inferior dissection of the shoulder capsule^{15,33-37}. Attention also should be

paid to factors that increase the predisposition to iatrogenic injury, including current or previous use of methotrexate, a history of shoulder surgery, a history of radiation treatment, a preoperative passive external rotation range of motion of $<10^\circ$, and decreased operative time^{38,39}.

Several surgical procedures have been implicated in axillary nerve injury as well. Among these, the Latarjet procedure, which is a means of glenohumeral joint stabilization that includes transfer of the coracoid process to the anterior aspect of the glenoid, has become increasingly popular³⁴. While the Latarjet procedure is effective, a recent systematic review of 1,904 such procedures demonstrated a relatively high rate of neurovascular injury (1.8%), including 6 axillary nerve injuries⁴⁰. The cadaveric study by Freehill et al. demonstrated consistent postoperative overlap between the musculocutaneous and axillary nerves as well as medial displacement of both nerves relative to their preoperative positions during the Latarjet procedure⁴¹. Delaney et al. used intraoperative neuromonitoring during the procedure and found the nerve to be most at risk during glenoid exposure and graft insertion⁴². Additionally, 79.4% of the nerve alerts occurred when the arm was in external rotation, indicating that while the axillary nerve is under the least amount of tension in external rotation, that position also may bring the axillary nerve closest to the surgical field⁴². In order to avoid injury, those authors advised against excessive retraction and a more superior placement of the coracoid graft.

Axillary nerve injury is also a well-documented complication of total shoulder arthroplasty. Neurological injuries occur in association with about 1% of total shoulder arthroplasty procedures, with the axillary nerve being most commonly affected⁴³⁻⁴⁷. In a systematic review, Bohsali et al.⁴³ found that 10 (77%) of 13 axillary nerve injuries resolved spontaneously without surgery while 1 patient with a transected axillary nerve required

trapezius tendon transfer to restore deltoid functionality. Nagda et al.³⁸ found that 57% of 30 patients demonstrated EMG findings consistent with impending intraoperative compromise of nerve function during positioning of the involved extremity at the time of total shoulder arthroplasty. Fifty percent of the nerve events occurred while the extremity was placed in abduction, external rotation, and extension, and the other 50% occurred during preparation of the glenoid. While these changes may have been related to retractor tension on the nerve, repositioning of the arm into a more neutral position, rather than retractor removal alone, was more likely to return EMG readings to baseline. Limb lengthening associated with reverse total shoulder arthroplasty increases the native tension across the entire brachial plexus, placing these structures at risk of peripheral nerve palsies and other neurological complications⁴⁸. A recent cadaveric study quantified the proximity of the axillary nerve to the implant after reverse shoulder arthroplasty⁴⁹. The principal finding of that study was that the main anterior branch of the axillary nerve was approximately 5 mm away from the humeral prosthetic implant. Additionally, the nerve was never closer than 15 mm to the glenosphere, which constituted a safer implant with respect to the nerve.

The open inferior capsular shift, which reduces capsular redundancy and restores anterior, inferior, and posterior stability, is another procedure in which damage to the axillary nerve can occur⁵⁰. During the procedure, excessive inferior dissection or thermal injury resulting from electrocauterization can cause damage to the axillary nerve as it courses across the operative field beneath the glenohumeral ligament. This type of injury also can occur in association with arthroscopic or open Bankart lesion repair⁵¹. Neer and Foster reported postoperative axillary neuropathy in 3 (8%) of 40 patients undergoing open inferior capsular shift procedures⁵².

A limited body of literature has described cases of axillary nerve compromise after proximal humeral fixation. One cadaveric study showed that the axillary nerve could be elevated almost 14 mm from the bone without becoming taut⁵³. Nonetheless, the nerve could be exposed to greater tension forces during manipulation of the fracture fragments, fibrosis, or osteosynthesis materials. Park and Jeong, in a consecutive series of 21 patients with proximal humeral fractures that were treated with minimally invasive percutaneous plating, reported 1 case of axillary nerve palsy⁵⁴. Additionally, humeral nail fixation is not without risks. A cadaveric study demonstrated that the axillary nerve can be damaged during the insertion of the locking screws despite the use of protection sleeves and trocars⁵⁵.

Given our understanding of the complications related to open surgical procedures about the shoulder, the majority of primary stabilization procedures are currently being performed arthroscopically. As a result, it is important for surgeons to consider the close proximity of the axillary nerve even during arthroscopic procedures. Several reports have described the close proximity of the axillary nerve to the inferior axillary pouch during the placement of arthroscopic capsular plication sutures for the treatment of multidirectional instability, for anteroinferior capsulolabral repair, or for complex arthroscopic reconstruction^{15,33-37}. However, recent reports also have shown that the nerve may be as close as 7 mm during bicortical drilling at the time of biceps tenodesis procedures or during the placement of standard or accessory anterior or posterior portals or posteroinferior (7 o'clock) arthroscopic portals⁵⁶⁻⁵⁹. While axillary nerve injuries resulting from arthroscopic procedures remain rare, the reporting on such complications is limited and therefore these injuries may be more frequent than presently recognized. Surgeons must maintain a high level of attention to anatomic landmarks

and surgical precision in order to avoid possible iatrogenic injury.

Reverse shoulder arthroplasty is becoming more common, and limb lengthening associated with this procedure may increase the native tension across the entire brachial plexus and the axillary nerve, thus posing a risk of peripheral nerve palsies and other neurological complications⁴⁸. A recent cadaveric study also quantified the location of the axillary nerve in relation to the implant following reverse shoulder arthroplasty and suggested that nerve damage can occur secondary to the proximity of the axillary nerve during the placement of a reverse prosthesis. The principal finding of that study was that the main anterior branch of the axillary nerve was approximately 5 mm away from the humeral prosthetic implant, placing it at risk for irritation, abutment, or injury. However, the nerve was never closer than 15 mm to the convex hemispherical glenoid prosthesis (glenosphere)⁴⁹.

Injury involving the axillary nerve also may occur in association with proximal humeral fractures or during the reduction and fixation of such fractures. A limited body of literature, however, has described cases of axillary nerve compromise following proximal humeral fixation. As noted above, cadaveric laboratory dissection has demonstrated that the axillary nerve can be elevated nearly 14 mm from the bone without being placed under tension⁵³. Tension may, however, be placed on the nerve during reduction or manipulation of fracture fragments if the nerve is tethered by fibrosis or if the nerve is abutting fixation implants. Park and Jeong, in a consecutive series of 21 patients with proximal humeral fractures that were treated with minimally invasive percutaneous plating, reported 1 case of axillary nerve palsy⁵⁴. In addition, humeral nail fixation is not without risks. A cadaveric study demonstrated that the axillary nerve can be damaged during the insertion of the locking screws despite the use of protection sleeves and trocars⁵⁵.

Natural History

The vast majority of axillary nerve injuries are temporary neurapraxias, which typically resolve within 6 to 12 months after the index injury. Nevertheless, permanent axillary nerve deficit also has been reported^{30,60}. Regardless of the fact that nerve damage can be devastating for the patient, previous reports have described nearly normal shoulder function after closed axillary nerve injury (although none of those reports documented permanent nerve damage)^{29,61-64}. It also has been established that a delay in diagnosis is strongly correlated with the final outcome and that a less-than-optimal prognosis can be expected if the diagnosis is made >6 months after the index injury.

Nonoperative Treatment

With the exception of certain cases of quadrilateral space syndrome, neurapraxic injuries usually are treated nonoperatively. Several case series involving patients who have had complications after shoulder surgery have shown high rates of spontaneous recovery following axillary nerve neurapraxia^{18,39,43}; however, comparative studies with operative treatment are lacking. Nonoperative treatment should include a rehabilitation program emphasizing passive and active range of motion as well as strengthening of the rotator cuff, deltoid muscle, and periscapular musculature^{7,65}. Shoulder contracture typically is not associated with axillary nerve injury unless an extensive ligamentous or osseous injury is present³⁰. If no apparent clinical improvement is evident at 3 months following the initial insult, repeat EMG/NCS should be performed, and serial EMG studies may be performed to monitor the progression of recovery³⁹. If there is no improvement in terms of the clinical and/or electrophysiological recovery status within 3 to 4 months after the injury, operative treatment should be considered¹⁸. Operative treatment typically is recommended between 3 and 6 months after the injury for patients lacking evidence of clinical or

electrophysiological improvement^{11,18}. We support surgical exploration of the axillary nerve within 3 to 4 months after the injury if testing shows a lack of electrophysiological recovery; these specific interventions will be discussed in later sections. The strength of this recommendation is graded as weak on the basis of the limited available literature, and comparative studies between expectant management and operative repair are needed.

Operative Treatment

The results of operative treatment of axillary nerve injuries are optimal when surgery is performed within 3 to 6 months after the injury⁷. Outcomes of surgical treatment for axillary nerve injury are worse beyond this time frame, with only marginal benefits seen when surgery is performed ≥ 12 months after the injury⁶⁶⁻⁶⁸. These recommendations are based on case series, and further comparative studies are needed to optimize the timing of surgery. Four standard modalities of operative treatment are generally utilized: neurolysis, neurorrhaphy, nerve-grafting, and neurotization⁸. The choice between treatment modalities is contingent on the findings observed during operative exploration.

Neurolysis

Neurolysis is a procedure that involves excision and debridement of scar tissue around the nerve (external neurolysis) or removal of scarred or fibrosed nerve tissue within the perineurium (internal neurolysis). In cases of axillary nerve palsy, external neurolysis is indicated if the nerve is found to be intact but entrapped^{69,70}. This type of entrapment is seen in patients with a history of shoulder surgery as well as overhead athletes in whom fibrosis develops as a result of repetitive microtrauma⁶⁹.

An anterior approach with the patient in a modified beach-chair position or the lateral decubitus position can be used to achieve direct access to the nerve⁷¹. If the area of fibrosis is suspected to be in the quadrilateral space, visualization can be attained through a

posterior approach between the long head of the triceps and teres major muscles. However, both anterior and posterior approaches may be required to achieve adequate debridement or decompression^{69,71,72}. For this reason, preoperative MRI is extremely helpful for determining the etiology and location of neurological symptoms.

Outcomes following neurolysis for the treatment of stretch or contusion injuries have been favorable. Kline and Kim found that, even in patients with complete clinical nerve loss, axillary nerve recovery was possible with isolated neurolysis as long as intraoperative nerve action potentials were observed⁷¹. However, clinical and patient-reported outcomes are poorer when concomitant distal peripheral nerve injury in the arm or hand, or associated mixed brachial plexus injuries, are present⁷³.

Neurorrhaphy

Primary repair of the axillary nerve with suture, also known as neurorrhaphy, is indicated for the treatment of acute transections or lacerations of the nerve and in cases in which the nerve is found to be cleanly bisected with minimal scarring. Primary repair is particularly advantageous for the treatment of the axillary nerve (as compared with other peripheral nerves) because of its simple fascicular structure and close proximity to effector muscles⁷³. For this reason, primary repair is preferred to nerve-grafting or neurotization. In patients with acute injuries, it is important to perform the repair as soon as possible as fibrosis will lead to increased dissection and blunted or distorted repair ends, which may negatively impact outcomes^{71,73}. If the nerve cannot be mobilized intraoperatively, or if a tension-free anastomosis cannot be achieved, nerve-grafting may be necessary.

Focal, isolated transection lesions of the axillary nerve are rare, and mobilization is often difficult because of retraction of the nerve ends or surrounding musculature. As a result, there is a scarcity of literature on the outcomes

of direct repair. Kline and Kim reported on 3 patients who underwent direct end-to-end suture repair of the axillary nerve that resulted in clinical recovery of deltoid muscle strength⁷¹. Terzis and Barmptsioti also performed direct repair of the axillary nerve in a larger study but did not report on outcomes specific to those patients⁷⁴.

Nerve-Grafting and Neurotization

In some cases of axillary nerve injury or transection, scarring and retraction have occurred, making repair or other less-invasive procedures difficult. In such cases, nerve reconstruction by nerve-grafting or nerve transfer, known as neurotization, is recommended. Although typically recommended in chronic settings, neurotization also may be recommended in cases of severe or segmental traumatic axillary injuries, especially those associated with a root or cord avulsion, because of the need for rapid regeneration of nerve fibers⁷⁵⁻⁷⁷.

Neurotization involves the transfer of autologous nerves from redundant or less-used areas of the body. Common harvest sites include the thoracodorsal, phrenic, spinal accessory, and intercostal nerves, with promising results for the correction of axillary nerve palsy^{75,76}. Reports of transferring the medial triceps branch of the radial nerve to the axillary nerve (triceps-to-axillary transfer) have demonstrated reasonable outcomes^{77,78}. Triceps-to-axillary transfers have been particularly successful for the treatment of acute axillary nerve palsy^{77,78}. However, other studies have shown that outcomes following the transfer of the medial triceps branch of the radial nerve are less predictable, and negative prognostic factors include delayed time to surgery, increased age, and increased body mass index (BMI)⁷².

Nerve-grafting also is an option for the treatment of large, traumatic nerve lesions or in cases in which considerable neuroma resection is required. Short nerve grafts have been associated with better outcomes than long grafts²³, which initially showed disappointing results⁷⁹. However, a recent study by

Wolfe et al. showed no difference between long nerve grafts and triceps-to-axillary nerve transfers in adult patients with axillary nerve palsy⁸⁰.

Prevention

Several measures can be routinely followed to prevent iatrogenic axillary nerve injury. Proper understanding of local anatomy is crucial for the avoidance of injury to the axillary nerve, particularly when planning surgical incisions over the lateral portion of the deltoid muscle, placing arthroscopic portals, or performing capsular plications or releases, as previously discussed. In addition, arm positioning during the operation is vital. A cephalad-directed force on the flexed elbow helps to eliminate inferior translation of the humerus and decreases the risk of compression or traction injury of the axillary nerve^{81,82}. We recommend a mobile arm positioner with the patient in the beach-chair position. The use of shoulder-specific operating tables should be considered in order to improve patient comfort, body alignment, and safety.

In order to preserve the function of the deltoid muscle, the location and course of the axillary nerve should be carefully identified, particularly during open capsular releases, arthroplasty, or arthroscopic procedures near the inferior capsule. In procedures involving open anterior approaches to the shoulder, the axillary nerve courses across the operative field, and the course of the nerve can be delineated with use of the “tug” test⁸¹. This test is performed by simultaneously palpating the axillary nerve both medial and lateral to the humeral shaft with both index fingers. Once the surgeon has located the nerve medially as it courses inferiorly on the subscapularis and laterally on the undersurface of the deltoid muscle, a gentle “tug” is applied by either finger and can be felt by the contralateral index finger. This maneuver allows for direct palpation of the axillary nerve in order to assess its continuity and to identify its location for protection throughout the

procedure. However, this tactile maneuver does not provide direct information regarding compressive or conductive injuries to a nerve in continuity. Alternatively, deltoid muscle atrophy can be minimized by using an extended deltopectoral approach, which preserves both the origin and the insertion of the deltoid muscle.

Several studies have demonstrated that the position of the axillary nerve varies as a function of abduction, extension, and rotation of the humerus^{12,82}. Adduction of the humerus moves the axillary nerve closer to the operative field and glenohumeral joint, whereas external rotation at 45° of horizontal glenohumeral flexion moves the axillary nerve away from the field. The arm should be in the latter position during deep dissection and retractor placement near the insertion of inferior capsule¹⁰. During arthroscopic surgery, the axillary nerve is farther away from the field with the arm in abduction and neutral rotation¹⁵. Additional studies are required to determine how the position of the axillary nerve varies according to surgical approach, type of procedure, and stage of procedure. Extreme ranges of arm positioning, particularly external rotation, should not be maintained for extended periods of time⁴².

Because the optimal balance between visualization and safety of the axillary nerve is difficult to assess, intraoperative neuromonitoring may be helpful for identifying impending or subclinical nerve damage secondary to retractor placement or limb malpositioning. While multiple studies have found that this technique may prevent iatrogenic nerve damage, cost-benefit analyses are needed to assess the effectiveness of this practice^{33,83}. Currently, intraoperative neuromonitoring is best considered for patients with anticipated risk factors for nerve injury, as previously discussed.

Overview

Axillary nerve injuries may present as sensory deficits, deltoid muscle

dysfunction, motor weakness, or a combination of these symptoms. An understanding of the pathophysiology of nerve injury aids in clinical decision-making, which may result in early operative intervention before irreversible nerve damage occurs. In addition, an understanding of axillary nerve anatomy is important for diagnosis and treatment as well as for reducing the risk of intraoperative iatrogenic injury. Additional studies are needed to provide both guidelines for treatment and future directions as the indications and types of shoulder procedures continue to expand.

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