CHAPTER 29

# Nerve Injuries

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## *In This Chapter*



## INTRODUCTION

- An increased awareness of peripheral nerve injuries about the shoulder and their effect on athletic function is reflected in the growing body of published reports on the subject.
- These injuries have a varied presentation, with associated acute trauma demanding on-field decision making or athletes with chronic symptoms presenting in the clinic after failure of previous diagnostic attempts.
- These injuries present a significant challenge to medical personnel attempting to provide athletes with full and safe participation in competitive activities.
- In this chapter, we discuss the presentation, diagnosis, and management of commonly encountered nerve injuries about the shoulder. These include the burner/stinger syndrome, suprascapular nerve entrapment and surgical techniques used in its treatment, and axillary, long thoracic, spinal accessory, and musculocutaneous nerve injuries.
- Less commonly encountered conditions, such as the Parsonage-Turner and thoracic outlet syndromes, are beyond the scope of this chapter but are mentioned in the context of a complete diagnostic workup.

## PERIPHERAL NERVE INJURY

The pathophysiology of peripheral nerve injury has been studied in great detail. Seddon[1] developed the classification system most commonly used today, defining three progressive patterns of injury severity. This has been further modified by Sunderland[2] to include five levels of injury. The mildest form, neurapraxia, involves an interruption of axonal function without frank disruption of the axon. The prognosis for recovery is favorable, with complete functional return expected within weeks to months. Axonotmesis involves loss of continuity of the axon, with varying degrees of injury to the endoneurium and perineurium. Prognosis for recovery varies greatly due to varying degrees of nerve tissue injury. Wallerian degeneration takes place, and the nerve must regenerate from the site of injury at the rate of 1 mm/day, with recovery of end-organ function possibly taking months. Neurotmesis involves complete disruption of the nerve, including the axon, endoneurium, perineurium, and epineurium, although the outermost nerve sheath may or may not be intact. The prognosis for recovery is very poor, and nerve repair or grafting may be indicated.

The differential diagnosis of peripheral nerve injury about the shoulder includes cervical spine instability, cervical spine fracture, herniated cervical disk, cord concussion/contusion, transient quadriplegia, acute brachial plexitis (Parsonage-Turner syndrome), rotator cuff tear or tendonitis, clavicular fracture, acromioclavicular joint injury, glenohumeral subluxation/dislocation, glenohumeral arthritis, adhesive capsulitis, thoracic outlet syndrome, scapular fracture, and proximal humerus fracture. Each of these must be considered in the evaluation of the athlete with shoulder-related complaints.

## TRANSIENT BRACHIAL PLEXOPATHY (BURNER/STINGER SYNDROME)

### Clinical Features and Evaluation

The "burner" or "stinger" is one of the most frequently encountered conditions evaluated by athletic team medical personnel. The majority of these injuries occur in American football, in which as many as 65% of collegiate squad members have reported one or more episodes during a 4-year career.[3,4] The syndrome is so frequently encountered by and familiar to athletes that it may often go unreported to team staff.

An athlete with a burner usually presents after a traumatic event with a complaint of pain, numbness, burning, tingling, or stinging pain radiating from the shoulder down the arm, possibly into the hand, most often unilaterally. The athlete may also complain of weakness in the shoulder, elbow, or hand of the affected upper extremity. He or she may be holding the affected extremity by his or her side or be noticed to shake the hand or arm as if it is "asleep" or "dead." More ominous signs may include holding the neck in a flexed position to alleviate pressure on the cervical nerve roots or a complaint of bilateral or lower extremity symptoms. This may suggest the possibility of spinal cord involvement instead of nerve root or plexus injury. Pain localized to the trapezius may be present, but neck pain is usually not a complaint, and its presence, especially if severe, requires medical personnel to initiate spinal precautions and to perform a detailed workup for spinal injury.

The physical examination should focus on the spine and affected extremity of the athlete. Careful attention to the results will help differentiate a relatively benign condition from a more severe injury. Most athletes will have a normal physical examination by the time they arrive on the sideline. Clinical observation of the athlete is followed by palpation for tenderness and deformity along the spine, shoulder, and extremity, facilitated by removal of clothing and protective gear as needed. Spinal examination should then test active flexion, extension, lateral bending, and rotation and, if normal, may include provocative tests such as Spurling's compression maneuver or axial manual traction. The shoulder/extremity examination should concentrate on sensation, motor testing, and reflexes. The upper trunk of the brachial plexus, most often involved in burner syndrome, is evaluated by sensory examination of the C5 and C6 dermatomes, and strength testing of the deltoid, biceps, and rotator cuff. Weak shoulder abduction may be present, even after pain cessation. Deep tendon reflex testing of the biceps (C5), brachioradialis (C6), and triceps (C7) should then be performed. The lower trunk is less frequently involved. Sensory examination is performed with attention to the C7, C8, and T1 dermatomes, and motor testing should concentrate on the intrinsic muscles of the hand, including grip strength and finger abduction.

## Relevant Anatomy and Pathophysiology

The exact mechanism of burner syndrome is debated and likely represents varying levels of injury location and severity. The injury location can vary from nerve root, which is thought to be less common in athletic injuries,[5] to peripheral nerve injury, as described previously. The injury level likely is a function of the position of the neck, arm, and shoulder at the time of impact. It is thought to result from a compression or traction (pinch-stretch) injury to either the cervical nerve root or the brachial plexus, most frequently the upper trunk.[6]

There are three commonly described mechanisms of injury in burner syndrome, occurring in isolation or combination. Forceful neck extension and lateral bending can cause neural foraminal narrowing, leading to compression of the cervical nerve roots.[6,7] A traction injury may occur from forceful depression of the ipsilateral shoulder, as occurs in blocking, tackling, or wrestling, with the nerve roots fixed proximally.[3] This injury mechanism may be enhanced with lateral bending of the neck to the contralateral side. A third mechanism may be a direct blow to the anterolateral neck at Erb's point (Fig. 29-1), located superior and deep to the clavicle, lateral to the sternocleidomastoid. At this point, the brachial plexus is most superficial and susceptible to injury.

The relationship of cervical stenosis to burner syndrome has been extensively reviewed. The Torg ratio is determined by measuring the distance from the midpoint of the posterior aspect of the vertebral body to the nearest point on the corresponding spinolaminar line and dividing this value by the anteroposterior diameter of the vertebral body on a lateral radiograph.[8] Meyer et al[6] concluded that there was a relationship between cervical stenosis, defined as a Torg ratio less than 0.8, and the occurrence of stingers or nonparalyzing extension/compression injuries, although the clinical significance of the Torg ratio continues to be debated.[9]

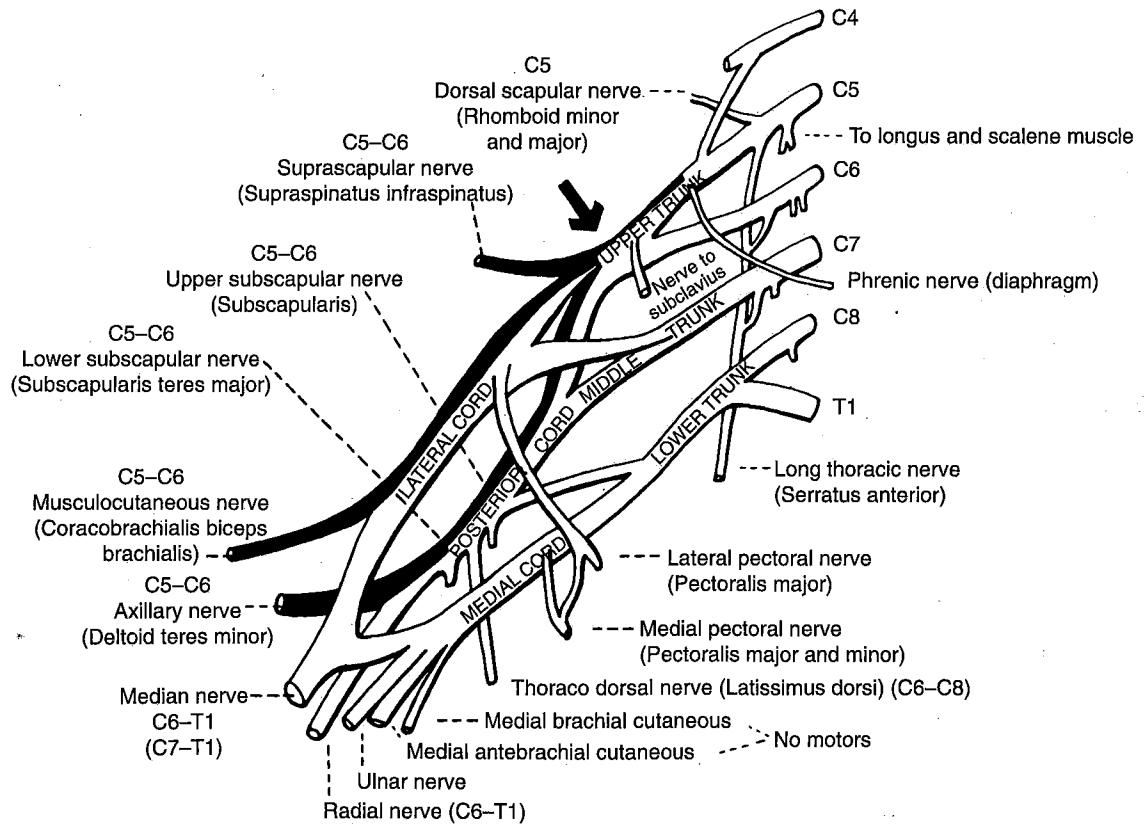



**Figure 29-1** Diagram of the brachial plexus demonstrating the location of Erb's point *(arrow)*. Brachial plexus stretch injuries may result from traction at this point. (From Torg JS: Athletic Injuries to the Head, Neck and Face, 2nd ed. St. Louis, Mosby-Year Book, 1991.)

### Criteria for Return to Sports

If the athlete's sensory and motor symptoms resolve within seconds or minutes and there is no associated neck pain, range-of-motion limitation, or findings consistent with other more significant injuries to the neck or shoulder, then the player may safely return to competition. Full motor strength is an absolute requirement for return to sports. Paresthesias usually resolve within seconds to minutes and motor symptoms within 24 hours. Persistence of symptoms, including paresthesias, weakness, limited range of motion of the neck or extremity, or pain, requires removal from participation and further evaluation. Persistent or recurrent episodes require complete neurologic workup, including cervical spine radiographs and possibly magnetic resonance imaging (MRI) or computed tomography myelography to assess for cord or root compression. If symptoms persist for more than 2 to 3 weeks, electromyography (EMG) may be useful in determining the extent of injury. However, electromyographic changes may persist for several years after injury and should not be used as a criterion for return to sports. Abnormal findings on these studies require a case-by-case evaluation for return to sports.

A physical rehabilitation program that emphasizes neck and trunk strengthening should be instituted on return to competition. The use of a neck roll, collar, or molded thermoplastic neck-shoulder-chest orthosis,[4] in conjunction with well-fitted shoulder pads, has been shown to decrease the recurrence and severity of episodes in athletes with a history of stingers.

## SUPRASCAPULAR NERVE ENTRAPMENT

### Clinical Features and Evaluation

Injury to the suprascapular nerve has been associated with multiple sports, including baseball, football, tennis, swimming, volleyball, and weight lifting.[10] Direct trauma to the neck or scapula may cause injury to the suprascapular nerve, and crutch use has been implicated,[11] as has heavy labor. The athlete with suprascapular nerve palsy may present with an often vague range of symptoms or even be asymptomatic.[12] Pain over the posterolateral shoulder or easy fatigability with overhead activities may be reported, or painless weakness of external rotation with or without spinati muscle atrophy may be noted. Compression of the nerve at the suprascapular or spinoglenoid notch is a commonly reported mechanism of injury in the athlete and is discussed in detail.

The physical examination plays a critical role in discerning the site of suprascapular nerve injury. Clinical observation of the athlete's shoulder girdle is important. More proximal injury, as seen with suprascapular notch compression, may result in atrophy of both the supraspinatus and infraspinatus, whereas more distal compression at the spinoglenoid notch will result in isolated infraspinatus weakness and atrophy (Fig. 29-2). Tenderness over the course of the nerve may be present but is often difficult to localize. Weakness of shoulder abduction or external rotation with vague posterolateral shoulder pain may be the only significant examination finding, although a decreased range of motion, specifically adduction, may be noted due to pain.

Plain radiographs of the shoulder are routinely negative. EMG and nerve conduction velocity (NCV) studies play a particularly useful role in the diagnosis and localization of a suspected suprascapular nerve injury. As with most nerve injuries, these studies are generally more useful if obtained in the subacute phase of injury, at least 3 to 4 weeks after onset of symptoms. However, careful clinical correlation with study results must be used, as both false-negative and false-positive nerve findings have been described.[13] MRI may be useful in demonstrating atrophic muscle degeneration of the spinatii or to reveal the presence of a compressive lesion along the course of the nerve. Most commonly, this will be a ganglion cyst, often seen in association with a superior labral tear (Fig. 29-3).

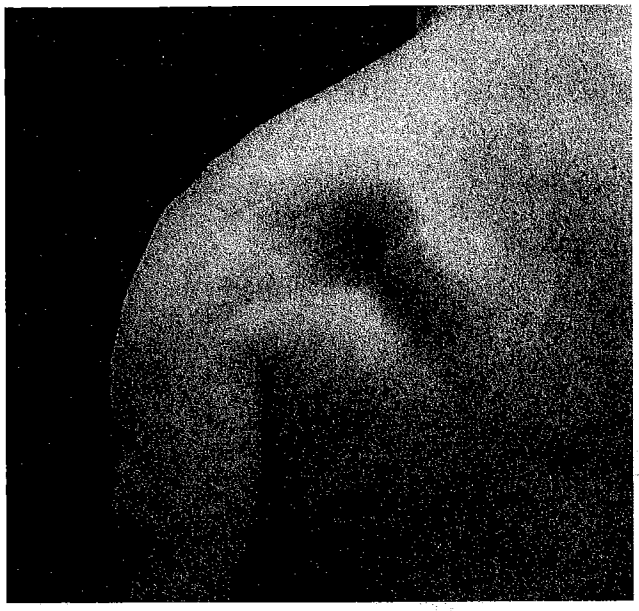


**Figure 29-2** Suprascapular neuropathy resulting in infraspinatus atrophy. (From Jobe FW: Operative Techniques in Upper Extremity Sports Injuries. St. Louis, Mosby, 1996.)

### Relevant Anatomy and Pathophysiology

At Erb's point, the suprascapular nerve branches from the upper trunk of the brachial plexus, with contributions from C5 and C6. The nerve then travels below the transverse scapular

**Figure 29-3** Magnetic resonance imaging of the right shoulder demonstrating a ganglion in the spinoglenoid notch compressing the infraspinatus branch of the suprascapular nerve.

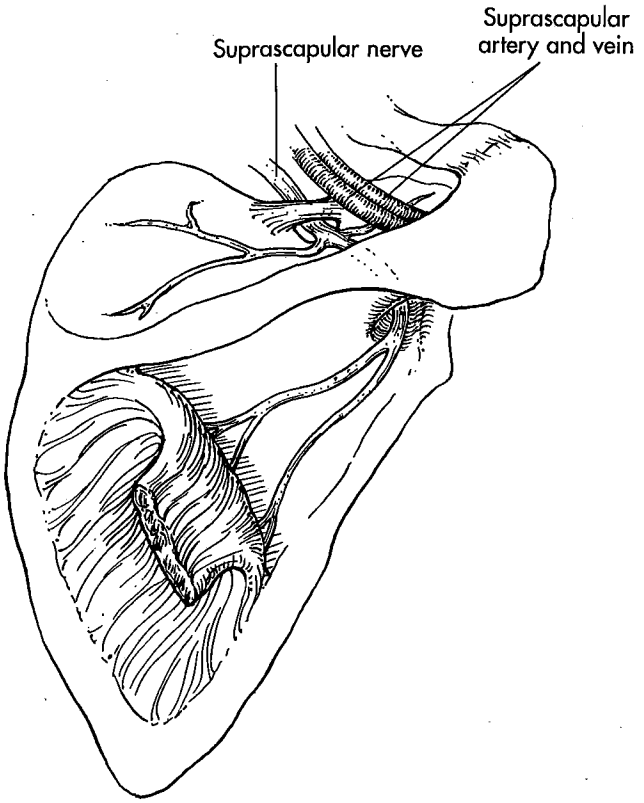



**Figure 29-4** Anatomy of the suprascapular nerve. (From Jobe FW: Operative Techniques in Upper Extremity Sports Injuries. St. Louis, Mosby, 1996.)

ligament as it crosses the suprascapular notch to enter the supraspinatus fossa (Fig. 29-4), while the suprascapular artery usually travels above the ligament. The nerve traverses the supraspinatus fossa, giving motor branches to the supraspinatus, with variable minor sensory contributions to the glenohumeral and acromioclavicular joints and occasionally to the skin.[14] The nerve then angles around the spine of the scapula at the spinoglenoid notch, traveling with the artery under the spinoglenoid ligament.[15] The motor branches to the supraspinatus are approximately 3 cm from the origin of the long head of the biceps, while the motor branches to the infraspinatus average 2 cm from the posterior glenoid rim.[16]

Like other nerves, the suprascapular nerve is susceptible to injury from compression, traction, or direct trauma. Vascular microtrauma has also been postulated to cause nerve dysfunction. The most commonly reported mechanism of injury is compression by a ganglion cyst, usually at the suprascapular or spinoglenoid notch. A thickened or calcified ligament may also compress the nerve. A ganglion cyst is often associated with a tear in the glenohumeral joint capsule or labrum, with fluid being forced through the tear and then being trapped outside the joint.

## Treatment Options

Treatment of the acute injury to the suprascapular nerve is similar to that for most nerve injuries about the shoulder. Relative rest and pain control are followed with progressive range-of-motion and strengthening exercises as tolerated. More chronic cases are managed depending on the duration of symptoms and the mechanism of injury, although the exact duration of symptoms is frequently difficult to determine. MRI can be used to evaluate for a compressive lesion. If a compressive lesion or cyst is noted on imaging, the patient can be observed for 2 to 3 months, followed by surgical decompression if symptoms continue (see "Surgery"). An athlete with symptoms associated with repetitive overhead activity, as seen with volleyball, tennis, or baseball players, should be followed for 6 to 12 months with observation, activity restriction, and periscapular therapy, after confirming the absence of a compressive lesion. Periodic EMG/NCV studies can follow the electrophysiologic nerve recovery. Surgical intervention with this overuse mechanism of injury has demonstrated variable results at best,[17] and function usually returns by 12 months. As with other painful nerve injuries about the shoulder, Parsonage-Turner syndrome (acute brachial neuritis) must be considered and, if present, should be managed conservatively with pain control, observation, and therapy.

## Surgery

The suprascapular nerve can be approached either with an open technique or arthroscopic technique. If the lesion is proximal and both the supraspinatus and infraspinatus are involved, then the entire nerve should be released, but most importantly the transverse scapular ligament must be released. If only the infraspinatus is involved or if there is a structural lesion in the spinoglenoid notch such as a paralabral cyst, then the nerve may be simply decompressed at the spinoglenoid notch. Associated labral tears should be repaired using standard techniques.

### *Open Decompression*

The suprascapular nerve can be approached either by the direct approach, splitting the trapezius, or by an extensile approach, elevating the trapezius from the spine of the scapula. The transverse scapular ligament is found 2.5 to 3 cm medial to the acromioclavicular joint at the medial border of the coracoid process. With a direct superior approach, the skin is incised in line with Langer's lines medial to the acromioclavicular joint in a typical Saber style. The trapezius muscle is split in line with its fibers for approximately 5 cm. The supraspinatus muscle is retracted posteriorly, and the suprascapular notch and transverse ligament are palpated. The suprascapular artery can either be retracted out of the way or ligated and the transverse scapular ligament is then released. A neurolysis can then be performed. If the ligament is ossified, which can be seen on computed tomography scan, then a small rongeur can be used to remove the bone and decompress the nerve. This approach is cosmetic but limits access to the posterior course of the nerve at the spinoglenoid notch.

For open suprascapular nerve decompression, the authors prefer to use the extensile approach. This allows access to the entire nerve if necessary. An incision is made along the spine of the scapula and the trapezius is elevated and reflected anteriorly. This gives access to the entire supraspinatus fossa. The supraspinatus muscle is retracted posteriorly and the transverse scapular ligament is palpated, visualized, and released as described. By working on either side of the supraspinatus muscle belly, the suprascapular nerve can be visualized over most of its course and can be followed to the spinoglenoid notch. By extending the incision inferiorly and splitting the posterior deltoid, the suprascapular nerve can be traced to its terminal arborization into the motor branches that supply the infraspinatus muscle. The suprascapular nerve runs just at the base of the scapular spine in the spinoglenoid notch. Often there is a thickened band of connective tissue called the spinoglenoid ligament

that can tether the nerve in this region. If present, this should be released as well. Since this approach uses extensile, internervous planes, closure is simply done by repairing the trapezius back to the spine of the scapula using nonabsorbable sutures.

### Arthroscopic Decompression

An arthroscopic approach is a more sophisticated way of addressing the suprascapular nerve and is our preference when there is an associated intra-articular lesion, such as a SLAP (superior labrum anterior to posterior) tear or labral tear. It is our preferred method for treating spinoglenoid neuropathy due to paralabral cysts, and, furthermore, it is becoming our preferred method for decompressing the nerve at the suprascapular and spinoglenoid notches. It does require advanced arthroscopic skills but offers a less invasive and more cosmetic approach with better overall visualization and access. Moreover, concomitant intra-articular pathology can be addressed easily.

### Arthroscopic Release at the Suprascapular Notch

We prefer to use the beach chair position. The arthroscope is placed in an anterolateral portal and accessory anterior and posterior portals are used. The view is initially into the subacromial space. The coracoid process must be visualized and the dissection is then carried medially. Arthroscopic retractors are helpful to retract the supraspinatus muscle belly posteriorly. The dissection is carried along the posterior aspect of the coracoid process. The coracohumeral and coracoclavicular ligaments are identified and at the base of the coracoid the suprascapular notch is identified. The artery is cauterized using radiofrequency ablation, and the ligament is released using hand-held arthroscopic tissue punches (Fig. 29-5). The nerve can be probed to ensure there is no compression. It can be seen passing deep to the supraspinatus.

### Arthroscopic Release at Spinoglenoid Notch or Cyst Decompression

This is our preferred technique for treating paralabral cysts. Again the beach chair position is used. Standard anterior and posterior portals are created. A transrotator cuff portal as used for SLAP repairs is created. The arthroscope is placed laterally through the transcuff portal. This gives excellent visualization. If there is a labral tear, it is repaired with suture anchors using standard technique. Some have advocated working through the labral tear to access the cyst, but we have found this to be quite difficult and furthermore it is virtually impossible to visualize the suprascapular nerve. Therefore, we have gone to performing a capsulotomy, releasing the posterosuperior capsule at the periphery of the labrum until the fibers of the supraspinatus are identified. The supraspinatus muscle is then elevated superiorly using a retractor, which is placed from our anterior portal. With careful and meticulous dissection, the cyst itself can invariably be demonstrated and resected. The typical ganglion cyst fluid is seen when the cyst is perforated (Fig. 29-6). The suprascapular nerve runs 2.5 to 3 cm medial to the superior aspect of the glenoid at the base of the supraspinatus fossa (Fig. 29-7). It can be traced posteriorly from there until it passes through the

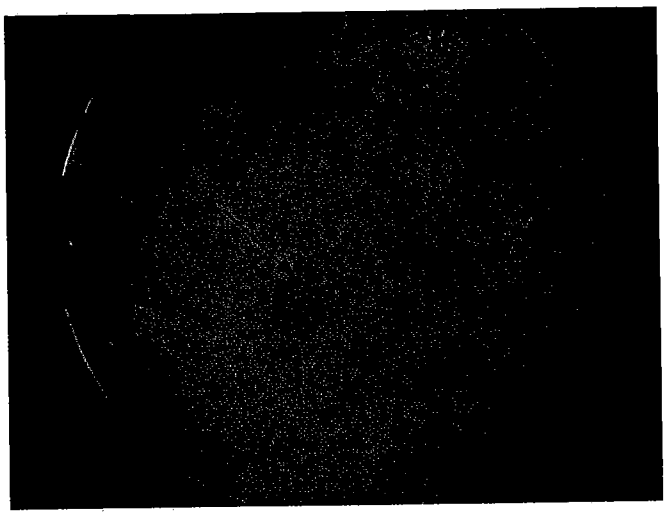


**Figure 29-6** Arthroscopic view of right shoulder spinoglenoid notch cyst immediately following perforation *(arrow)* and decompression. The suprascapular nerve is deep and medial to the cyst wall.

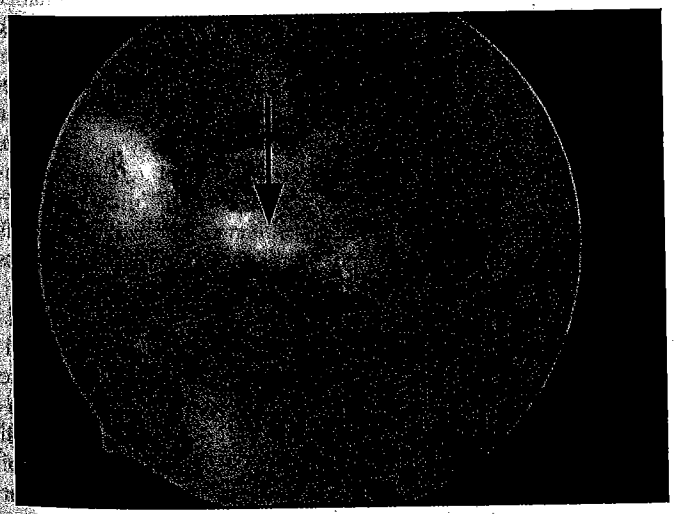


**Figure 29-5** Arthroscopic view of right shoulder suprascapular notch demonstrating the transverse scapular ligament *(large arrow)* traveling over the suprascapular nerve *(small arrow)*. The suprascapular artery above the ligament has been coagulated.

**Figure 29-7** Arthroscopic view of right shoulder spinoglenoid notch demonstrating the infraspinatus branch of the suprascapular nerve *(arrow)* after débridement of the compressive cyst.

spinoglenoid notch. Using hand-held basket punches and arthroscopic probes, a careful neurolysis can be performed.

### Results and Outcomes

The results of both operative and nonoperative treatment of suprascapular nerve injuries are not easily interpreted. The duration of symptoms is often difficult to assess, and the diagnosis may be incorrect or incomplete with respect to associated intra-articular pathology. Several studies have reported on the results of both operative and nonoperative treatment.[10,13,17] In a recent meta-analysis of the literature, Zehetgruber et al[18] found suprascapular nerve entrapment to be rare, occurring mainly in patients younger than 40 years of age. Isolated infraspinatus atrophy was most often associated with a ganglion cyst, whereas a history of trauma was usually associated with ligamentous compression of the nerve. Surgical treatment seems to give reliable pain relief, with persistent atrophy of the spinatii muscle, a common but well-tolerated finding.

### Postoperative Rehabilitation

Postoperatively patients are immobilized in a sling for comfort. Early motion is encouraged. If a labral tear was repaired, then the athlete is protected for 4 weeks before resuming active motion. Strengthening begins at 6 weeks. Throwing and overhead activities generally commence at 4 to 5 months postoperatively.

### Criteria for Return to Sports

While the athlete remains symptomatic, full athletic function should be avoided, especially when the injury mechanism is one of overuse. Patients undergoing surgical intervention for persistent symptoms demonstrate excellent pain relief, and although the spinatii often demonstrate persistent atrophy, return to full competitive activity can still be expected.[19]

## AXILLARY NERVE INJURY

### Clinical Features and Evaluation

Axillary nerve injury is a relatively common peripheral nerve injury in the athlete, particularly in contact sports.[20] Shoulder dislocation or direct trauma to the deltoid muscle can result in axillary nerve injury and subsequent deltoid or teres minor muscle paralysis. When injury does occur, the athlete often presents not with an obvious motor deficit, but rather may complain of easy fatigability of the shoulder with overhead activity or resisted shoulder abduction.[21] However, the athlete may note weakness of shoulder external rotation, forward flexion, or abduction. Sensation over the lateral aspect of the shoulder may or may not be intact, even in the face of motor weakness.

The quadrilateral space of the shoulder may be a site of compression of the axillary nerve[22] and posterior humeral circumflex vessels, with subsequent injury and dysfunction (Fig. 29-8). The athlete may complain of a vague, poorly localized ache over the lateral or posterior shoulder, often aggravated by activity, especially forward flexion, abduction, and external rotation, as seen in overhead sports such as throwing. A history of unsuccessful shoulder surgery for the pain is not uncommon.

The physical examination should, as stated previously, concentrate on the cervical spine, shoulder, and extremity involved. Observation of the shoulder girdle may demonstrate deltoid and/or teres minor atrophy if the injury is long-standing. A detailed neurovascular examination should always be performed, with special attention paid to sensation to light touch over the lateral shoulder. Point tenderness is often present over the quadrilateral space[22] if neurovascular compression is present, and this may be accentuated by testing in the FABER (forward flexion, abduction, and external rotation) position.[22] Weakness of external rotation due to teres minor involvement may be present, and deltoid dysfunction may be noted in testing shoulder abduction, forward flexion, or extension.

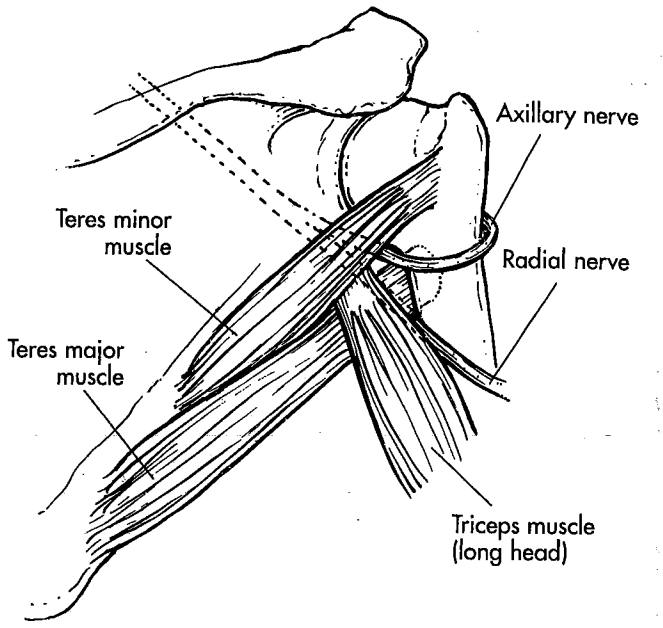



**Figure 29-8** The boundaries of the quadrilateral space as viewed from behind. (From Jobe FW: Operative Techniques in Upper Extremity Sports Injuries. St. Louis, Mosby, 1996.)

With respect to diagnostic testing, plain radiographs of the shoulder are a necessity to rule out associated bony injury, especially in the traumatic injury setting. Cervical spine radiographs may also be indicated. EMG and NCV studies are useful to confirm the diagnosis and determine the severity of injury but will likely not be positive until 3 or more weeks after injury. The intermittent compression of quadrilateral space syndrome may result in normal EMG and NCV studies. Magnetic resonance imaging may demonstrate muscle substance changes in chronic cases.

With regard to quadrilateral space syndrome, associated arterial occlusion of the posterior humeral circumflex artery can be diagnosed with arteriography.[22] Historically, the study will be normal with the affected shoulder in adduction but will demonstrate a filling defect with the shoulder in the FABER position (Fig. 29-9). However, magnetic resonance arthrography has demonstrated positive findings in asymptomatic patients, and its value is unclear.[23]

### Relevant Anatomy and Pathophysiology

The axillary nerve originates from the posterior cord of the brachial plexus, directly behind the coracoid process and conjoined tendon, with contribution from the C5 and C6 cervical nerve roots. It courses along the anterior inferolateral border of the subscapularis tendon and then passes near the inferior shoulder capsule,[24] receiving a sensory branch from the anterior capsule. The nerve then passes with the posterior humeral circumflex artery through the quadrilateral (quadrangular) space, formed by the long head of the triceps medially, the humeral

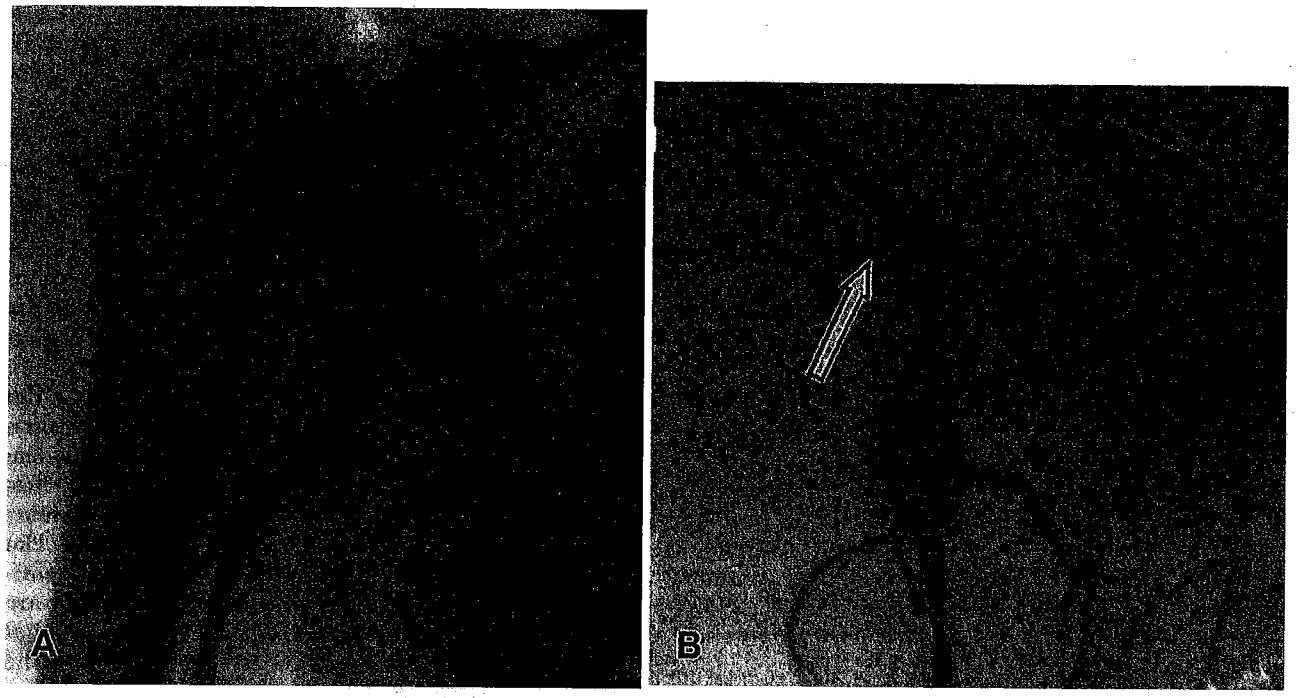



**Figure 29-9** An angiogram of a patient with quadrilateral space syndrome. **A,** Digital subtraction angiogram with arm in adduction reveals patent posterior humeral circumflex artery. **B,** Angiogram of same patient with the arm in abduction reveals complete occlusion of the posterior humeral circumflex artery *(arrow)*, confirming the diagnosis. (From Safran MR: Nerve injury about the shoulder. Am J Sports Med 2004;32:803–819, 1063–1076.)

shaft laterally, the teres minor superiorly, and the teres major inferiorly. At this point, it branches into an anterior and posterior branch along the posterior humeral surgical neck. The anterior branch innervates the middle and anterior deltoid, traveling an average of 6 cm distal to the lateral edge of the acromion.[5] The posterior branch divides into the upper lateral brachial cutaneous sensory branch and the nerve to teres minor. The posterior deltoid is variably innervated by the anterior, or less frequently, the posterior branch.[5]

The axillary nerve is relatively fixed at the posterior cord and the deltoid, thus leaving it susceptible to traction injury in anterior shoulder dislocation or proximal humeral fracture. The proximity to the shoulder capsule also makes the nerve susceptible to injury during arthroscopic or open shoulder surgery. Direct injury to the nerve from impact to the anterolateral shoulder has also been reported.[21] The factors that may increase the likelihood of axillary nerve injury with shoulder dislocation include age older than 40 years, unreduced dislocation longer than 12 hours, or higher energy mechanisms of injury.[20]

### Treatment Options

The treatment of an axillary nerve injury is a function of the mechanism of injury. Timely shoulder reduction and management of bony injury must be addressed when present, and the athlete should be reassured that the prognosis for recovery of function is good. Even with persistent weakness of the deltoid, return to competitive sports can be expected,[20] although athletes with significant overhead demands may note decreased function. Nonoperative treatment is the mainstay of management of these injuries, particularly in the first 3 to 6 months after injury.[25]

### Surgery

In the symptomatic athlete with incomplete clinical or EMG/NCV evidence of recovery after 3 to 6 months, surgery may be indicated. This may include decompression of the quadrilateral space in the presence of a positive arteriogram, neurolysis, or nerve grafting and results in more predictable functional return if undertaken within the first year after injury. Tendon transfer may also be considered for refractory cases, but return to competitive activity may not be possible.

### Criteria for Return to Sports

As with other injuries about the shoulder, maintenance of motion is key during the recovery period. Passive, active-assisted, and active range-of-motion exercises should be instituted early. Sport-specific rehabilitation begins when symptoms allow. Residual weakness of the deltoid and teres minor is often well tolerated but may result in easy fatigability of the shoulder. Therefore, a maintenance program of posterior capsular stretching and rotator cuff and periscapular strengthening should be instituted.

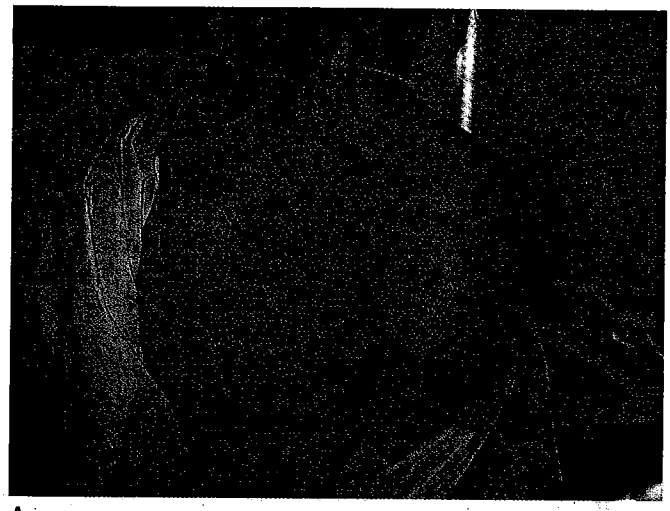
## LONG THORACIC NERVE INJURY (MEDIAL SCAPULAR WINGING)

### Clinical Features and Evaluation

Although relatively uncommon, traction injury to the long thoracic nerve has been recognized in athletes participating in numerous sports. Some of the activities previously associated with this injury include archery, backpacking, baseball, basketball, bowling, football, golf, gymnastics, hockey, rifle sports, shoveling, soccer, tennis, volleyball, weight lifting, and wrestling.[26] The athlete may present with medial winging of the scapula during shoulder forward flexion but more often may note only vague shoulder pain or easy fatigability, especially with overhead activity. Onset of symptoms is often insidious but may be associated with trauma, often a result of depression of the shoulder girdle from a direct blow to the top of the shoulder or a traction injury to the arm.[27] Symptom onset may follow the

trauma by several weeks. Acute brachial neuritis should be considered when significant pain precedes the onset of dysfunction, as the long thoracic nerve is often involved in Parsonage-Turner syndrome.

As with any complaint of shoulder pain or dysfunction, the physical examination should include evaluation of the cervical spine, shoulder, and extremity involved. Observation of the shoulder girdle may demonstrate medial winging of the scapula at rest. This involves medial and posterior translation of the inferior angle of the scapula (Fig. 29-10), which can be accentuated with resisted forward flexion of the shoulder, as demonstrated by the wall push-up. Forward flexion may be weak, and serratus anterior muscle atrophy may be noted in the thin, muscular patient. Scapular dyskinesia will be evident,[28] with possible associated impingement symptoms. Relief of the impingement symptoms may be noted with stabilization of the medial scapular border by the examiner while testing forward flexion and abduction. Complete serratus anterior paralysis may limit forward flexion to 110 degrees.[29] Confirmation of the diagnosis with EMG and NCV studies may useful to determine the severity of injury.



A

B

**Figure 29-10** Photographs of patient with right long thoracic neuropathy demonstrating medial scapular winging, as seen from behind **(A)** and laterally **(B)**.

### Relevant Anatomy and Pathophysiology

The long thoracic nerve originates from the ventral rami of the C5, C6, and C7 cervical nerve roots. There are variable contributions from the intercostal nerves and, less frequently, the C8 cervical nerve root. The individual contributing roots variably pass through or between the middle and anterior scalene muscles, before joining and traveling anterior to the posterior scalene muscle. The nerve then travels deep to the clavicle and variably the first or second rib before exiting the thoracic wall in the midaxillary line. The nerve innervates the serratus anterior muscle slips. The serratus anterior muscle arises from the anterolateral surface of the first eight ribs and inserts into the medial scapular border, functioning to stabilize and protract the scapula during abduction or forward flexion of the shoulder.

In sports, repetitive stretching of the nerve, as may occur in overhead activity, has been implicated in the dysfunction of the serratus anterior muscle.[29] As with brachial plexus injuries, shoulder depression and contralateral neck bending may further contribute to neurapraxia of the long thoracic nerve. Compression from multiple locations along the nerve as well as direct trauma to the anterolateral chest wall may also contribute to injury.

### Treatment Options

As with many sports-related nerve injuries about the shoulder, conservative treatment should be the mainstay. The aggravating activity must be curtailed to allow recovery, which can be expected usually within 9 months.[29] Application of a canvas brace may stabilize the scapula enough to prevent stretching of the serratus anterior during recovery but it is insufficient to allow full return to activity.[30]

### Surgery

Surgical treatment of isolated long thoracic nerve injury is rarely necessary and is aimed at restoring scapular stability. For severe dysfunction of 6 months' duration or longer, neurolysis may play a role.[31] For refractory cases of longer than 12 to 24 months' duration, transfer of the sternal head of the pectoralis major to the scapula has been shown to provide excellent restoration of scapular function.[32] Scapulothoracic fusion may stabilize the scapula but has been shown to result in significantly decreased function.[33]

### Criteria for Return to Sports

Exercises to maintain range of motion should be instituted early, followed by progressive strengthening of the rotator cuff and periscapular muscles. Maintenance of motion is vital during the recovery period, with passive, active-assisted, and active range-of-motion exercises playing a key role. Sport-specific rehabilitation begins when symptoms allow, usually within 6 months of injury. A maintenance program of rotator cuff and periscapular strengthening should be instituted, as with other shoulder injuries.

## SPINAL ACCESSORY NERVE INJURY (LATERAL SCAPULAR WINGING)

### Clinical Features and Evaluation

The diagnosis of an injury to the spinal accessory nerve in the athlete is often missed due to its rarity, thus potentially delaying its treatment.[34] A history of surgery in the area of the posterior neck, such as a cervical lymph node biopsy, or of penetrating trauma may lead to consideration of the diagnosis.

Blunt trauma to the posterior neck or traction may also result in injury to the accessory spinal nerve.[35] The most common presentation is a painful shoulder or neck, especially with activities that involve using the involved extremity above eye level. Loss of motion or early fatigue may be a secondary complaint. The athlete may note shoulder asymmetry, and rotator cuff impingement symptoms are often present.

Examination of the athlete with a spinal accessory nerve injury will reveal a depressed, or sagging, shoulder on the involved side. The supraclavicular recess may be relatively deepened due to trapezius atrophy. Lateral winging of the scapula, involving lateral rotation of the inferior scapular angle, may be elicited with resisted forward flexion but will not be as dramatic as the medial winging of long thoracic nerve palsy. Inability to elevate the acromion with a shoulder shrug may also indicate trapezius dysfunction. This may result in examination findings of rotator cuff tendonopathy. The levator scapulae and rhomboids may be prominent and palpable due to spasm in their effort to compensate for the weak trapezius. As with many nerve injuries about the shoulder, EMG and NCV studies may be useful in confirming the diagnosis and determining the severity of injury after 4 to 6 weeks of observation.

### Relevant Anatomy and Pathophysiology

The spinal accessory, or 11th cranial, nerve exits the skull through the jugular foramen, innervating the sternocleidomastoid and traveling across the posterior cervical triangle to innervate the trapezius. The trapezius arises from the ligamentum nuchae to the lower thoracic vertebrae and inserts into the lateral clavicle, the acromion, and the scapular spine. It functions to stabilize, elevate, and retract the scapula. The trapezius receives innervation not only from the spinal accessory nerve but also the ventral rami of the C2, C3, and C4 spinal nerve roots, possibly preventing complete denervation atrophy after accessory nerve injury. Scapulohumeral dyskinesia may result in depression of the acromion, with resultant subacromial impingement symptoms.

### Treatment Options

The treatment of spinal accessory nerve injury depends on the mechanism history. A closed injury, either from a direct blow or trauma, can be observed for a minimum of 6 months. If the patient remains symptomatic with continued pain, sagging of the shoulder, or weakness on forward flexion, surgical exploration with neurolysis, direct repair, or nerve grafting can be considered, especially if EMG/NCV findings confirm dysfunction. In the face of penetrating or operative trauma to the nerve, consideration of surgical exploration should be given after 6 weeks, with the best results reported for surgical intervention within 6 months.[34] It is imperative that shoulder range of motion be maintained during the observation period.

### Surgery

As stated previously, local surgical exploration may be beneficial with associated "open" trauma. When symptomatic trapezius weakness continues for more than 12 months, regardless of the injury mechanism, reconstructive surgical intervention should be considered. Tendon transfer procedures, most notably the Eden-Lange procedure with transfer of the levator scapulae and rhomboids, have a good prognosis for return of functional activities of daily living.[36] Prognosis for return to sports, however, is less favorable. Scapulothoracic fusion is an acceptable salvage procedure and may be considered the primary reconstructive option in patients with heavy demands on the shoulder. Prognosis for return to competitive athletic activity is very poor, however.

### Criteria for Return to Sports

Full functional return of trapezius strength is a prerequisite for return to vigorous overhead athletic activity. Many patients may be able to compensate for mild to moderate weakness of the nondominant shoulder, allowing adequate daily activity function and return to less demanding athletic activity. Although shoulder range of motion and strengthening exercises can maximize available function, it is unlikely that the other periscapular muscles can compensate for significant trapezius paralysis, especially if the dominant extremity is involved. Shoulder function may not be sufficient to allow return to competitive activity with persistent trapezius weakness, even after reconstructive surgery.[36]

## MUSCULOCUTANEOUS NERVE INJURY

### Clinical Features and Evaluation

Isolated musculocutaneous nerve injury in the athlete is rare. It has been reported in weight lifters[37] and rowers[38] and has been associated with strenuous, sustained physical activity. The athlete presents with paresthesias of the lateral forearm, with or without painless weakness of the biceps. The history may often reveal recent surgery to the anterior shoulder, or a direct blow to the anterior chest in the area of the coracoid. Rarely, history of a recent anterior glenohumeral dislocation may be elicited.

The examination must differentiate between isolated musculocutaneous nerve dysfunction and injury to the brachial plexus or C5 or C6 nerve roots. Observation may reveal an atrophied or flaccid biceps, and reflex testing should demonstrate an absent biceps reflex with an intact brachioradialis reflex. The sensory changes will be isolated to the lateral and radial forearm, with sparing of the C6 dermatome of the radial hand. Relative weakness of elbow flexion and forearm supination may also be present.

### Relevant Anatomy and Pathophysiology

The musculocutaneous nerve arises from the posterior cord of the brachial plexus, with contributions from the C5 and C6 nerve roots. It enters the coracobrachialis approximately 5 cm distal to the coracoid,[13] although smaller branches may enter earlier. It then exits the tendon approximately 7 cm distal to the coracoid before entering the biceps and brachialis muscles, providing motor innervation to these.[39] The nerve leaves the brachialis and enters the deep brachial fascia above the elbow crease to continue as the lateral antebrachial cutaneous nerve, providing sensory innervation to the anterolateral forearm.

The most common mechanism of injury is associated with anterior shoulder surgery, usually due to vigorous medial retraction of the conjoined tendon near the coracoid, although anterior arthroscopic portal placement may also injure the nerve.[40] This combined motor-sensory dysfunction may be differentiated from the isolated dysesthesias in the lateral forearm that may occur with compression of the musculocutaneous nerve as it enters the deep brachial fascial compartment at the elbow.

### Treatment Options

Since most injuries are related to stretching of the nerve, observation of the athlete for a period of 4 to 6 weeks usually results in evidence of recovery. However, continued weakness or

paresthesias after 4 weeks can be further evaluated with EMG/NCV studies to determine the level and severity of injury.

### Surgery

If clinical and/or electrophysiologic recovery is not noted, surgical exploration within the first 6 months after injury may be indicated. Surgical treatment may include decompression, neurolysis, and nerve grafting or may include nerve transfer using branches of the proximal ulnar nerve. For cases evaluated more than 1 year after injury, tendon transfer procedures may be indicated to supplement weak elbow flexion.

### Criteria for Return to Sports

Return to sports-related activity should be customized to the individual athlete. The prognosis for return of full function after postsurgical traction injury or direct blow trauma to the nerve is good, and athletic participation can be allowed. However, if the nerve injury is associated with repetitive or sustained sport-specific activity, modification of the athlete's mechanics may be necessary to prevent recurrence.

## CONCLUSIONS

An athlete presenting with pain about the shoulder can pose a significant diagnostic challenge to the athletic medical staff. The etiologies of the symptoms vary from minor to career ending. The examination of the athlete includes a detailed examination of the spine, shoulder, and upper extremity, and nerve injuries must be considered in the wide differential diagnosis. A thorough understanding of the presentation, anatomy, and pathophysiology of nerve injuries about the shoulder of the athlete is imperative for accurate and timely diagnosis and treatment. Prompt management of both bony and soft-tissue injuries may prevent or minimize the long-term impact of these injuries on the athlete.

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