# Sports Trauma

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Elinor Naor

## Noncontact Athletes

Athletes that participate in sports involving overhead include baseball, cricket, javelin, and tennis apply repetitive stress to their upper extremities. Within noncontact sports, overhead athletes are most likely to suffer an injury to the shoulder. In order to execute a throwing motion successfully, athletes must exercise coordinated muscle action at a velocity that is more rapid than the rest of the body. The biomechanical chain involved in overhead throwing includes the hip joint, trunk, scapula, and shoulder joint (Fig. 7.1). The shoulder maintains its stability during these actions via the glenohumeral capsular ligaments. A single kinetic chain failure can result in an overexertion injury to the muscles, ligaments, and tendons surrounding the shoulder [1].

E. Naor (✉)
Department of Rehabilitation & Human Performance, Icahn School of Medicine at Mount Sinai, Mount Sinai Health System, New York, NY, USA



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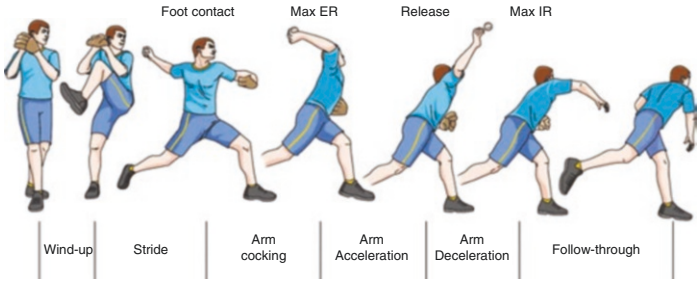


**Fig. 7.1** Schematic of the phases of throwing motion from windup to follow-through [1]

## Anterior Capsular Ligament Dysfunction

### Pathology

When athletes perform repetitive throwing motions, the anterior capsule may be stretched to the point of creating excessive ligamentous laxity. During active abduction, extension, and external rotation, the anterior portion of the inferior glenohumeral ligament (IGHL) is loaded, thus preventing anterior humeral movement. This can sometimes result in traumatic subluxation as the arm moves through space. Subluxation differs from dislocation in that it is an incomplete, transient displacement of the glenohumeral joint. Anterior capsular ligament dysfunction includes any pathology involving the anterior capsule of the glenohumeral ligament.

### Clinical Presentation

Athletes suffering from anterior shoulder instability present with shoulder pain elicited by end-range external rotation of an abducted shoulder. Anterior shoulder instability is typically seen in male athletes between the ages of 15 and 29 years old. This pathology is also associated with SLAP lesions as described later in this chapter.

## Physical Exam

During the physical exam, inspection of athletes suffering from anterior capsular ligament dysfunction, there typically are no noticeable masses, deformities, or asymmetries. Special tests are used to isolate the anterior capsule as the culprit. The anterior load-and-shift test applies anterior strain on a flexed and abducted shoulder, while the scapula is stabilized to assess for glenohumeral translation. To examine for inferior sulcus sign, the examiner applies inferior traction to the arm while observing for a space between the acromion and the humeral head. Another useful test is the Gagey hyperabduction test, which assesses the IGHL by abducting the shoulder to 105 degrees. If athletes have severe injuries which may need surgical intervention, the physical exam can be performed under anesthesia.

## Diagnostic Studies

To diagnose an athlete with anterior capsular ligament disorders, an X-ray (AP and axillary view) is initially obtained to rule out any bony deformities. The gold standard for labral and ligamentous evaluation is MR arthrography (Fig. 7.2). Ultrasound has not proven to be a useful modality for evaluation of deep ligaments of the shoulder. Direct visualization of ligamentous structures can be accomplished using surgical arthroscopy as well [3].

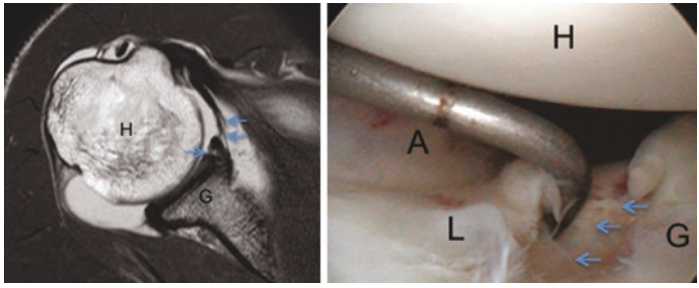


**Fig. 7.2** (Left) Anterior labral tear and scapular ligament elongation on MR arthrography. (Right) Arthroscopic view. A, anterior band of the inferior glenohumeral ligament; G, glenoid; H, humeral head; L, anterior labrum [2]

### Treatment

Injuries due to repetitive microtrauma, such as anterior capsular ligament dysfunction, typically resolve with conservative management. This includes pain management with oral anti-inflammatories (NSAIDs or acetaminophen) and a home exercise program. Therapeutic exercises should focus on strengthening the rotator cuff along with the core, trunk, and legs. These muscles are all involved in the kinetic chain and will be targeted in the rehabilitation process for other shoulder pathologies as well.

### Return to Play

Athletes may return to play once they can execute throwing motions without pain.

## Superior Labrum Anterior-Posterior (SLAP) Lesion

Overhead athletes stress their shoulders by straining the anterior capsule structures during the late cocking phase of throwing. This motion causes the posterior capsular structures to shorten and the anterior capsule structures to stretch, resulting in posterior-superior displacement of the humeral head. With repetitive irritation, this can progress into a posterior-superior impingement at the glenoid rim and an injury to the labral-biceps tendon complex. Labral pathologies are covered in detail in Chap. 4 (labral tears).

### Pathology

A superior labrum anterior-posterior (SLAP) lesion is localized to the superior labrum of the glenoid and travels from anterior to posterior in a circular manner. There are a total of ten different types of SLAP lesions documented; in this chapter, we will note the most common. Type 1 is the fraying and degeneration of the superior labrum and intact biceps tendon. Type 2 is avulsion of the superior labrum and biceps tendon from the supraglenoid tubercle. Type 3 is a superior labral tear (bucket handle) with intact biceps tendon anchor. Type 4 is a superior labral tear extending to the biceps tendon [4].

### Clinical Presentation

Typically, patients suffering a SLAP lesion will present with deep shoulder pain that is incited by throwing motions. They may also mention feeling popping, catching, or clicking of the shoulder. Athletes could also complain of "dead arm syndrome," stating that as they initiate the throw, they experience acute pain followed by a decrease in velocity with associated shoulder instability [5].

### Physical Exam

When assessing range of motion in patients with SLAP lesions, asymmetry may be noted when comparing both shoulders. The throwing arm may show evidence of muscular hypertrophy and present in a "rolled forward" position. If palpation along the biceps' tendon elicits pain, a superior labral injury is likely present. When moving the affected shoulder passively in abduction and flexion, apprehension or a catch can be appreciated. In addition, restricted passive internal rotation and extreme external rotation can be observed [6]. A number of special tests are positive in SLAP lesions – these include but are not limited to the anterior glide test, compression rotation, active compression (Obrien), modified dynamic labral shear test, and Crank test [7].

### Diagnostic Studies

While soft tissue injuries are not well visualized on plain films, X-rays (AP, scapular Y, and axillary views) are indicated in patients with shoulder pain to rule out associated fractures. MRI arthrography with the patient's arm in abduction and external rotation can be used to identify a SLAP lesion. If an MRI is contraindicated, a CT arthrogram can be done instead. For athletes under the age of 35, high complexity imaging modalities should only be ordered if it appears that surgical repair is needed, along with a referral to orthopedics [8].

### Treatment

Initial treatment for SLAP tears should include avoiding exacerbating activities and taking oral anti-inflammatory medications as needed. High-level athletes involved in sports with overhead

throwing are initially evaluated for SLAP lesions; after this, they can begin a home exercise program that includes rotator cuff strengthening exercises within a pain-free range of motion. Recreational athletes who have failed physical therapy may need to be re-evaluated for surgical repair. The therapy script should include graded eccentric and concentric rotational strengthening shoulder exercises along with range-of-motion and strengthening exercises of the spine. Surgery is indicated in patients who fail conservative therapy. There is a large variety of surgical options which include arthroscopic labral refixation and biceps tenodesis. However, patients over the age of 60 years old rarely benefit from labral repair surgery [4].

### Return to Play

One study showed that with physical therapy alone, 67% of athletes diagnosed with SLAP lesions were able to return to play, with an average timeline of 53 days for return to play. Studies have shown variable success rates in patients who have failed conservative therapy and have instead opted for arthroscopic SLAP repair [9]. Generally speaking, postoperative return to throwing has been shown to take as long as 6–12 months.

## Proximal Humeral Epiphysiolysis (Little League Shoulder)

### Pathology

Proximal humeral epiphysiolysis, also known as little league shoulder, is an injury to the growth plate (stress fracture) as a result of overuse [10]. In adolescents, the epiphyseal plate of the humerus is weaker than the surrounding tendons of the rotator cuff and is, therefore, at higher risk of injury with repetitive throwing motions. This recurring instance of microtrauma then does not get an opportunity to heal, ultimately leading to a proximal humeral stress fracture [1].

### Clinical Presentation

Proximal humeral stress fractures are typically seen in patients between the ages of 11 and 16 who play overhead throwing sports such as baseball. Athletes will complain of progressively worsening shoulder pain over the span of several weeks that is localized to the proximal humerus and worse with throwing.

### Physical Exam

When evaluating a patient with proximal humeral epiphysiolysis, a deformity may be noted along the surface of the proximal humerus. On physical exam, patients will have tenderness to palpation along the proximal lateral humerus. Patients may also complain of pain with active range of motion of the shoulder [11].

### Diagnostic Studies

Little league shoulder is diagnosed by visualizing widening of the proximal humeral epiphysis on ultrasound or X-ray (AP view, shoulder externally rotated). An MRI of the shoulder can also be used to confirm the diagnosis. Of note, widening is commonly found in asymptomatic athletes as well; so a clinical correlation is necessary [12] (Fig. 7.3).

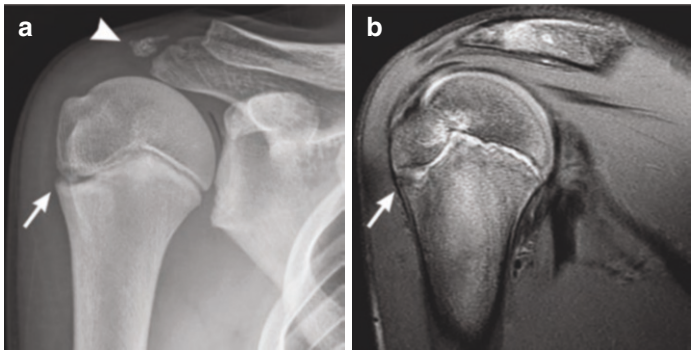


**Fig. 7.3** (**a**) AP radiograph: physeal widening and irregularity in the lateral proximal humeral physis are visualized (arrow). Note the well-corticated acromial ossification center (arrow head). (**b**) On MR arthrogram: Physeal widening and irregularity, bone marrow edema and effacement (arrow) [13]

### Treatment

When treating little league shoulder, anti-inflammatory medications and ice can be used to assist tolerance of motion and pain control. If diagnosed early, this condition can heal with physical therapy alone. Physical therapy exercises include core and both upper and lower extremity strengthening exercises to improve the kinetic chain. Patients should refrain from performing activities that exacerbate their symptoms. With relative rest, symptoms will resolve within 2–3 months.

### Return to Play

Athletes may return to play after completing a "return to throwing" therapy program. During this program, therapists review proper biomechanics of throwing and provide 3–5-min rest breaks between throwing activities. Once full strength has been regained and the athlete is asymptomatic, they may return to full activity [12].

## Contact Athletes

Contact and collision sports, such as ice hockey, football, basketball, and soccer, put players at high risk of musculoskeletal injuries. The shoulder is the most common musculoskeletal injury seen. More specifically, acromioclavicular (AC) joint trauma is the most common injury (32–41%), and shoulder dislocation is the most common cause of playtime loss [13].

### Acromioclavicular Joint Trauma

### Pathology

The AC joint is formed by the acromion (of the scapula) and the clavicle. This joint is stabilized by the AC ligament in the AP plane and the coracoclavicular (CC) ligaments (in the vertical plane [13]. Typically, traumatic injury to the superior lateral

shoulder while the arm is adducted can cause an AC joint sprain or, in more severe cases, AC and CC rupture. AC separation secondary to trauma will typically cause the acromion and scapula to move inferior to the clavicle, increasing AC ligamentous strain. This pressure causes the AC ligament to stretch and can eventually lead to CC ligament sprain [14].

The Rockwood classification system is used to classify AC injuries: Type 1 is a sprain isolated to the AC ligament. Type 2 is a torn AC ligament and a sprain of the CC ligament [13]. Types 1 and 2 are considered incomplete AC joint injuries and are twice as likely to occur than the more severe AC joint injuries [15]. Type 3 involves complete injury to both AC and CC ligaments, causing the scapular end of the clavicle to displace superiorly. Type 4 results from posterior displacement of the clavicle through the trapezius along with AC and CC ligament disruption. This level of injury is typically the result of a high-energy collision as in snowboarding or direct trauma as from a baseball bat. Type 5 occurs when both AC and CC ligaments are torn along with muscular and fascial damage.

### Clinical Presentation

Patients with an AC joint injury will typically present after sustaining a trauma directly to the area. AC joint pain is localized to the top of the shoulder, but it can also refer to the base of the neck and trapezius [16].

### Physical Exam

On inspection of the shoulder, bony deformity may imply AC joint separation or clavicular fracture. Tenderness to palpation along the AC joint is expected. Athletes with AC joint injuries will have limited shoulder flexion, adduction, and internal rotation. Touching the unaffected shoulder will elicit the pain due to pressure placed on the AC joint. There are several special tests to be performed on physical examination to confirm AC joint dysfunction. These include the cross-body adduction test, the AC shear test, and the active compression test [17].

In type 1 injuries, there may be mild swelling on exam, and moving the affected arm across the body will elicit pain. Type 2 injuries will show substantial swelling of the acromion. A visible AC joint deformity along with CC tenderness will be seen in type 3 injuries. In type 4 injuries, the posteriorly displaced clavicle is palpable and may affect a patient's breathing. Type 5 AC injuries cause a severe disruption to the supportive structures of the shoulder which cause the shoulder to translate inferiorly. The superior displacement of the clavicle may even break the skin or cause ischemia [15].

## Diagnostic Studies

When AC joint pathology is suspected, shoulder radiographs are ordered and should include AP shoulder, AP glenoid, scapular Y, and Zanca views. The Zanca view is specific for the AC joint. It is taken with the arm in an abducted position, and the beam is directed at a 30-degree cephalic tilt (pointing down the AC joint) [18]. Another diagnostic tool that can be used is a bone scan, on which a painful joint will show increased metabolic activity. If surgical treatment is indicated, an MRI of the shoulder is also obtained to rule out other pathologies. Finally, a diagnostic ultrasound-guided lidocaine injection can be used. While an injection can be done by palpation of bony landmarks, ultrasound-guided injections have shown improvements in symptoms and range of motion for up to 6 months longer than palpation-guided injections [19].

## Treatment

Type 1 and type 2 injuries are treated conservatively with immobilization, oral anti-inflammatories, ice, and physical therapy. Physical therapy will include range-of-motion exercises and strengthening of the muscles of the rotator cuff. If conservative treatment initially fails in type 3, surgery is indicated. In types 4 and 5 injuries, surgery is always indicated. Of note, glucocorticoid injections can be used for chronic pain management.

### Return to Play

Physical therapy alone should lead to complete healing within 6–10 weeks in type 1 and type 2 injuries and 6–12 weeks in type 3 injuries. Exercise is increased gradually over the weeks, initially starting with gentle range-of-motion exercises and ending with full-strength training without pain. Return to play is limited by pain [20].

## Shoulder Dislocation

### Pathology

Even though only 25% of the humeral head articulates with the glenoid, the glenohumeral joint provides the shoulder with a large range of mobility. The shallow joint, along with the small point of contact, increases the chances of shoulder dislocation. A dislocation may occur in the anterior, posterior, or inferior direction. Anterior dislocation results from a trauma sustained to an abducted, externally rotated, and extended shoulder. Posterior dislocation results from a blow from the anterior to the posterior direction or from strong muscle contraction (electrocution or seizure). Finally, an inferior dislocation is typically caused by axial loading of an abducted shoulder or hyperabduction [21]. Sports that involve the action of arm tackling a player that is running, like in football or blocking a basketball shot, may cause an inferior dislocation [22].

Anterior shoulder dislocations are associated with fractures known as Hill-Sachs deformities, Bankart lesions, and greater tuberosity fractures. The Hill-Sachs lesion appears on X-ray as a cortical depression deformity along the humeral head [23]. Bankart lesions occur due to bony fragment avulsion within IGHL complex [24]. Posterior shoulder dislocations are associated with tuberosity and surgical neck fractures, reverse Hill-Sachs lesions, and labral and rotator cuff pathologies [25].

### Physical Exam

The appearance of a dislocated shoulder on physical examination varies depending on the direction in which the humerus is avulsed. If the shoulder is dislocated anteriorly, the arm at rest will be in an abducted and externally rotated position. On examination, the AC joint may be more prominent in thinner individuals. Shoulder dislocation in any direction may affect the axillary nerve, which can manifest as abnormal sensation along the superior lateral surface of the shoulder and may progress to deltoid weakness [26]. When the shoulder is posteriorly dislocated, exam findings will include a notable prominence posteriorly with a flattened anterior surface. At rest, the patient will hold the arm in the adducted and internally rotated position. If the shoulder is dislocated inferiorly, the athlete will have difficulties with shoulder adduction; therefore, at rest, the forearm is usually draped over the head [27].

### Diagnostic Studies

When a shoulder dislocation is suspected, an initial X-ray (AP, scapular Y, and axial views) should be ordered to confirm the diagnosis and rule out fractures. An anteriorly dislocated shoulder will show a humeral head in the subcoracoid position on the AP view and positioned medial to the "Y" in the scapular view. The axillary view is useful in distinguishing a true dislocation from a false dislocation (when the humerus is fractured) [27]. In a posteriorly dislocated shoulder, AP X-ray will show the humeral head internally rotated, hiding the tuberosities resulting in a circular appearance known as "light bulb sign." In addition, the distance between the medial humeral head and the anterior glenoid rim may be widened in posterior dislocations. Of note, an inferior dislocation will show the humeral head below the coracoid as well; however, the shoulder will be in an abducted position [28]. If the location of the humeral head cannot be identified on X-ray, a CT scan can be used. In addition, a CT scan can be used to visualize associated fractures. If there is a suspicion for axillary artery injury, a CT angiogram is indicated. MRI arthrograms can be used to visualize associated lesions, such as the Bankart tear (Fig. 7.4),

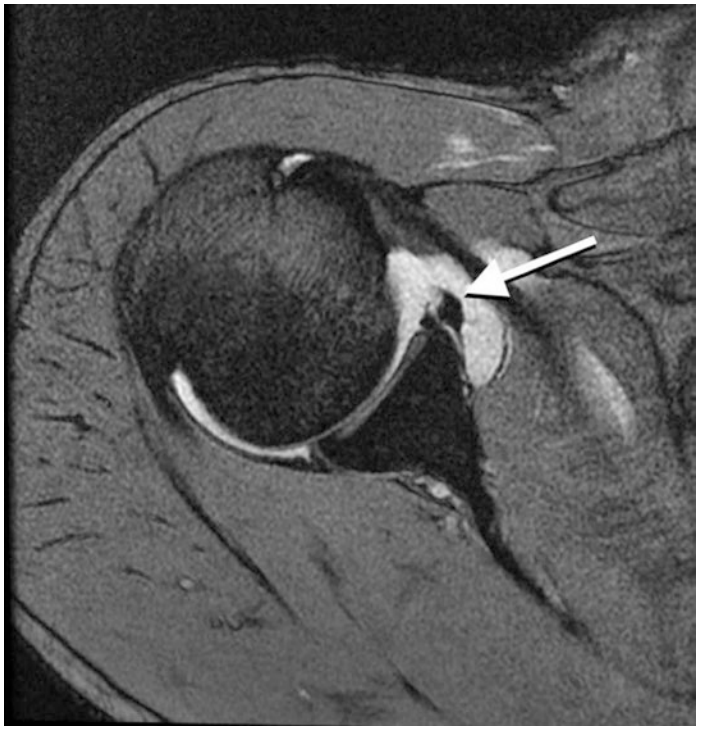

**Fig. 7.4** Bankart lesion (a tear that happens in the lower rim of the labrum) on MRI arthrogram (white arrow) [13]

the reverse Bankart lesion, and the reverse Hill-Sachs lesions (Fig. 7.5) [29].

## Treatment

Shoulder reduction is the first-line treatment for a dislocation. The risks are rare, but they can include humeral fracture, rotator cuff injury, or axillary neurovascular injury. In elderly patients, the risks are higher, and therefore, an orthopedic consultation may be

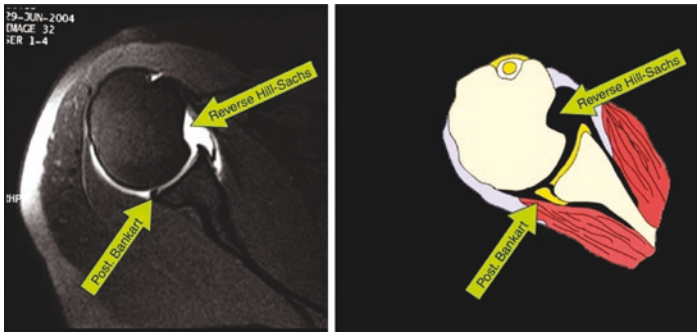


**Fig. 7.5** Reverse Bankart lesion (detachment of the posteroinferior labrum with avulsion of the posterior capsular periosteum) and reverse Hill-Sachs lesion (fracture of the anteromedial aspect of the humeral head) on MRI arthrogram [13]

indicated. Acute, anterior, recurrent, or nontraumatic shoulder dislocations can be reduced without anesthetic. Following the reduction, X-rays are done to determine success [30]. Once the shoulder is in place, it is immobilized using a sling, and the patient is instructed to follow up within 1 week. Length of immobilization depends on age; if they are under the age of 30 years old, immobilization should last 3 weeks. If the patient is over the age of 30 years old, early mobilization at 1 week is recommended to avoid stiffness. Irreducible dislocations and Bankart fractures are indications for surgery.

## Return to Play

During the immobilization phase, gentle range-of-motion exercises are advised while avoiding abduction and external rotation. Eventually, active-assisted range-of-motion and isometric exercises are initiated by therapists. Barring any complications, athletes may return to play with limitations at 12 weeks and may return to full activity at 16 weeks [31].

## Acute Brachial Plexus Injury

### Pathology

In contact sports, injury to the shoulder can result in neurovascular injury. There are three known mechanisms of upper trunk (C5, C6) brachial plexopathies in relation to the shoulder. One is a traction injury, where the neck is forced laterally away from a depressed shoulder. The second is a direct trauma to the supraclavicular fossa. The third is neck hyperextension and side bending, resulting in nerve compression [32]. This last brachial plexus injury, also known as a "burner" or "stinger," is a result of neck or shoulder trauma.

### Clinical Presentation

Athletes with burner syndrome will typically present following neck and shoulder trauma with pain radiating down the affected arm. The pain is typically described as burning and surrounding the entire arm. Patients may also complain of numbness, heaviness, or sensitivity in the limb. Most often, these symptoms resolve within minutes.

### Physical Exam

Patients suffering from an acute brachial plexus injury on inspection may hold their arm adducted at rest or may shake the affected limb. Over several weeks, deltoid atrophy is observed due to denervation. The cervical spine should be examined to rule out injury. Special tests such as the Spurling compression test can be used to rule out cervical nerve root compression. Multiple muscles can be affected by an injury to the upper trunk of the brachial plexus leading to weakness in shoulder abduction (deltoid, supraspinatus) and external rotation (infraspinatus), elbow flexion and supination (biceps brachii), and forearm pronation (pronator teres). Of note, the timeline of weakness development can vary from minutes to days [33].

### Diagnostic Studies

Patients suspected of having nerve injury affecting the upper extremity should initially have a cervical spine X-ray (AP; lateral, in flexion and extension; and oblique views) to rule out spinal stenosis or cervical instability. An MRI should be done as well to rule out spinal cord dysfunction or nerve root compression. The gold standard for diagnosing peripheral nerve injury is an electrodiagnostic study. In an upper trunk brachial plexopathy, the electromyography findings can include fibrillation potentials, prolonged latencies, and positive sharp waves in C5 and C6 innervated muscles [34].

### Treatment

Once diagnosed, burners are initially treated with physical therapy. Exercises include neck, shoulder, and upper extremity active range of motion and stretching. Stiffness and weakness predispose patients to burners, thus emphasizing the value of strength and mobility training. In addition, "chest-out" posturing is recommended to decrease nerve compression [34]. In contact sports, brachial plexus injuries can be prevented by using protective equipment such as shoulder pads.

### Return to Play

Before an athlete can return to play, steps should be taken to educate them on proper biomechanics to avoid repeat injuries. Once symptoms have resolved and the range of motion of the neck and shoulder are back to baseline, they may return to contact sport. However, players cannot participate if neurological symptoms persist. Of note, recovery can take months and may even become a chronic issue [35].

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