Conservative Management of Shoulder Impingement Syndrome in the Athletic Population

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Shoulder pain is a common complaint among overhead athletes. Oftentimes, the cause of pain is impingement of the supraspinatus, bicipital tendon, and subacromial bursa between the greater tuberosity and the acromial arch. The mechanisms of impingement syndrome include anatomical abnormalities, muscle weakness and fatigue of the glenohumeral and scapular stabilizers, posterior capsular tightness, and glenohumeral instability. In order to effectively manage impingement syndrome nonoperatively, the therapist must understand the complex anatomy and biomechanics of the shoulder joint, as well as how to thoroughly evaluate the athlete. The results of the evaluation can then be used to design and implement a rehabilitation program that addresses the cause of impingement specific to the athlete. The purpose of this article is to provide readers with a thorough overview of what causes impingement and how to effectively evaluate and conservatively manage it in an athletic population.

Key Words: rehabilitation, anatomy, secondary impingement evaluation

Because of the shoulder joint’s unique anatomy, impingement syndrome is a common disorder that plagues the athletic shoulder, often requiring medical attention by an orthopedist (52). Neer (46), who was the first to use the term impingement syndrome, described it as an impingement of the rotator cuff beneath the coracoacromial arch. Since then, however, it has been reported that additional anatomical structures are involved. Because of their orientation inside the subacromial space, the supraspinatus, bicipital tendon, and subacromial bursa can become impinged between the greater tuberosity of the humeral head and unyielding acromial arch when dysfunction is present at the glenohumeral or scapulothoracic joint. In order to effectively evaluate and manage impingement, the therapist must understand not only the biomechanics of the shoulder but also how alterations and abnormalities anatomically and biomechanically cause this debilitating syndrome. The purpose of this article is to provide readers with a thorough overview of what causes subacromial impingement and how to effectively evaluate and conservatively manage it in an athletic population.

Anatomical Basis of Impingement Syndrome

Understanding the pertinent anatomy of the shoulder is imperative to fully understand impingement syndrome. The pertinent anatomy includes the acromial arch, subacromial space, subacromial bursa, supraspinatus, bicipital tendon, and scapulothoracic musculature.

The acromial arch consists of the acromioclavicular joint, coracoacromial ligament, and acromion (23). The coracoacromial ligament originates on the coracoïd process and migrates posteriorly toward insertion on the anterior acromion (43). The acromial arch aids in protecting the humeral head and the contents of the subacromial space, as well as providing superior stability to the glenohumeral joint (35).

The subacromial space consists of the region just inferior to the acromial arch and superior to the humeral head. Termed the impingement interval, the subacromial space houses the structures commonly involved with impingement syndrome (19). Flatow et al. (18) report that the subacromial space is approximately 1-1.5 cm on radiographic evaluation. Because there are soft tissue structures in this space, the subacromial space is significantly narrower than the width reported (18). Present within this narrow space are the subacromial bursa, supraspinatus tendon, and bicipital tendon.

The subacromial bursa is located inferior to the acromial arch and superior to the supraspinatus tendon. Its main function is joint nutrition and lubrication during glenohumeral movement (43). Inferior to the subacromial bursa in the subacromial space is the supraspinatus. The supraspinatus originates at the supraspinous fossa of the scapula, travels through the narrow subacromial space, and inserts on the greater tuberosity. The supraspinatus functions to aid in shoulder abduction and draws the humeral head into the glenoïd, thereby assisting with dynamic stabilization (2, 5, 55).

The biceps brachii has two points of origin. The short head originates at the coracoïd process of the scapula and the long head originates inside the subacromial space at the supraglenoid tuberosity. The biceps brachii muscle acts as an elbow and shoulder flexor, aids in humeral head depression, and provides anterior stabilization in a position of abduction (8, 37, 57).

In addition to the glenohumeral joint, motion of the scapulothoracic articulation is essential for fluent, coordinated movement of the shoulder. The role of the scapulothoracic articulation is twofold. First, it provides a firm base of support for movement of the humerus at the glenoid. Second, as the head of the humerus moves...
on the glenoid fossa, the scapula simultaneously moves to keep the glenoid fossa and humeral head in good alignment.

This movement of the scapula in relation to the humerus is termed scapulohumeral rhythm (11, 26). Its purpose is to maintain optimal humeral head position on the glenoid, thereby maintaining the proper length-tension relationship between the rotator cuff muscles and the humeral head. By maintaining the proper length-tension relationship, both active and passive insufficiency are limited (50). As the humerus abducts at the glenoid, the scapula simultaneously upwardly rotates. During the first 30° of abduction, scapular motion is limited (setting phase). Once the humerus reaches 30°, the scapula rotates at a 1–2° ratio. For every 2° of humeral abduction, the scapula rotates upward 1°. Once the humerus reaches 120°, the scapular and humerus rotate simultaneously at a 1–1° ratio (26). Normal shoulder-abduction range of motion is between 160 and 180° (50). Of that 160–180°, 120° results from glenohumeral motion, and the remaining range of motion results from scapulothoracic motion (50). The upward scapular rotation necessary for full glenohumeral abduction results from a force couple by the trapezius (upper, middle, and lower portions) and the serratus anterior (27, 43). Shoulder flexion elicits scapulohumeral motion similar to abduction.

**Causes of Shoulder Impingement Syndrome**

Impingement syndrome results from the greater tuberosity of the humerus impinging the supraspinatus tendon, bicipital tendon, and subacromial bursa on the anterior third of the acromion and coracoacromial ligament (14, 23, 46, 47, 60). During abduction, the humerus must externally rotate to clear the greater tuberosity; otherwise, the tuberosity will impinge on the subacromial structures. Wueke, Piitz, and Roetman (70), using a dynamic model of the shoulder joint, reported that during 85–136° of elevation, peak force was exhibited on the subacromial structures. This increase in force corresponds to the painful arc exhibited by an athlete with an impingement syndrome. Burns and Whipple (9), in cadaveric studies, forward-flexed the shoulder to 90° and forcibly internally rotated the humerus. This caused direct contact by the supraspinatus and bicipital tendon on the coracoacromial ligament (9). This motion is similar to the repetitive follow-through motion experienced by overhead athletes. Cullito (14) reported that continued impingement of the subacromial bursa and supraspinatus leads to fibrotic and hypertrophic changes accentuating impingement by narrowing the subacromial space.

Impingement can be categorized as either primary or secondary. Primary impingement is caused by mechanical narrowing of the subacromial space. Causes of primary impingement include bone spur development, thickening of the rotator cuff, fibrosis of the subacromial bursa, os acromion (unfused acromial apophysis), calcification of the coracoacromial arch, a hooked acromion, and acromioclavicular joint degeneration (1, 4, 19, 20, 24, 46). Often times conditions such as these warrant surgical intervention. Because primary impingement most often occurs in an older population (1), the causes warrant mention, but the focus of this article is on the causes of secondary impingement.

In contrast to primary impingement, which generally involves the older population, secondary impingement is most common in the overhead-athletic population less than 25 years of age (1). Secondary impingement results from a functional narrowing of the subacromial space. Common causes include glenohumeral instability, biceps tenosynovitis, muscle weakness and fatigue, and posterior capsular tightness (3, 12, 19–21, 35, 42, 47, 48, 51).

Glenohumeral instability often causes secondary impingement (1, 21, 35, 52, 63). Glousman (21) reported that instability often goes unrecognized with persistent shoulder pain and symptoms. Parker and Seitz (52) call this combination of impingement and instability shoulder impingement/instability overlap syndrome. Warner, Micheli, Arslanian, Kennedy, and Kennedy (63) reported that 68% of their test population with anterior instability also presented signs of impingement syndrome.

Because of its lack of osseous stability, the shoulder joint relies on both static (fibrous capsule and glenohumeral ligaments) and dynamic (rotator cuff and biceps brachii) stabilizers to provide much-needed stability. If the static and/or dynamic structures fail, instability results. The most common type of instability leading to secondary impingement is anterior instability (21). Anterior instability—or glenohumeral atraumatic subluxation, as termed by Glousman (21)—results from anterior capsular laxity. Arroyo, Herson, and Bigliani (1) reported that repeated microtrauma to the glenohumeral joint leads to excessive laxity of the static structures. Because this anterior laxity exists, the rotator cuff muscles must compensate to provide stability, thus leading to fatigue from overuse.

The main function of the rotator cuff muscles is centralization and approximation of the humeral head within the glenoid fossa (51). During abduction or flexion of the shoulder, the natural tendency is for the humeral head to migrate upward, disrupting joint congruency and causing impingement. If the cuff muscles, as well as the biceps brachii, are fatigued or weak because of excess workload, the humeral head migrates upward and impinges on the structures in the subacromial space (3, 4, 19, 20, 35, 51). Sharkey and Marder (58), using cadaveric models, reported that abduction without the subscapularis, teres minor, and infraspinatus caused significant superior migration of the humeral head. This supports the notion that glenohumeral instability can lead to impingement syndrome.

Shoulder pain is a common complaint in the overhead athlete (1, 10), who is susceptible to injury and dysfunction because of the repetitious, high-velocity mechanical stresses placed on the shoulder (1). Anterior instability causing secondary impingement is often diagnosed in the overhead-athletic population (1). During the late cocking phase of pitching, excessive stress is placed on the anterior capsule, leading to microtrauma and eventually instability (21). As a result of this instability, as well as the repetitive stress placed on the shoulder joint, pathological changes occur in the overhead athlete that can contribute to secondary impingement syndrome (1, 21).
Overhead athletes often demonstrate abnormalities at the scapulothoracic articulation (37). These include temporal lags in scapular protraction and lateral scapular rotation in the overhead position (37). Abnormalities such as these cause changes in the length–tension relationship between the humeral head and the scapula, as well as alter scapulothoracic rhythm, leading to impingement.

Several pathological changes, including posterior capsular tightness, surface during the evaluation of overhead athletes. Overhead athletes often exhibit limited internal rotation caused by posterior capsular tightness (37). This tightness leads to anterior–superior translation of the humerus, causing a disruption of joint congruity and interfering with the rotator cuff length–tension relationship, leading to impingement syndrome (12, 19, 20, 25, 37).

Fatigue or weakness of the scapular-stabilizing muscles disrupts gleno-humeral–scapulothoracic alignment and the coordinated movement provided by the scapulothoracic joint. Oftentimes, secondary impingement is a result of poor action by the upward rotators during shoulder abduction or flexion. As the humerus abducts or flexes, the scapula must upwardly rotate in order to maintain joint congruity (50). If upward rotation is disrupted because of weakness or fatigue of the trapezius and serratus anterior, elevation of the humeral head causes the greater tuberosity to impinge on the structures in the subacromial space. In addition to disrupting upward rotation, serratus anterior weakness causes winging of the scapula away from the thorax, which leads to malalignment between the gleno-humeral joint and scapulothoracic articulation. This malalignment increases the likelihood of greater tuberosity impingement by disrupting length–tension relationship between the rotator cuff muscles and the humerus (35). In addition to scapular stabilizer weakness, Kamkar, Irgang, and Whitney (35) reported that shoulder pain from impingement could lead to protraction of the scapular muscles, leading to atrophy and thereby accentuating the problem.

Bicipital tendinitis and tenosynovitis can cause impingement. Inflammation of the biceps tendon as it travels through the transverse humeral ligament and bicipital groove causes decreased biceps activity (47). Decreased bicipital activity leads to decreased humeral head depression, which leads to impingement.

Although it is the focus of this article, impingement in the athletic population is not limited to subacromial impingement syndrome. Throwing athletes can also exhibit internal or posterior–superior impingement syndrome (10, 28, 29).

Classification of Impingement Syndrome

Several clinicians have developed classifications for impingement syndrome, with the most commonly used ones being reported by Jobe and Myynes (31) and Neer (47). Jobe and Myynes (31) developed a four-stage classification system that quantifies the amount of rotator cuff pathology. Edema and inflammation, with temporary thickening of the rotator cuff and bursa, characterize the first stage. The second stage involves some cuff fiber disruption, along with permanent thickening of the rotator cuff and bursa. Stage 3 is characterized by a rotator cuff tear less than 1 cm in length. The final stage of this system consists of a partial cuff tear exceeding 1 cm in length.

Neer (47) developed the most widely used and accepted system of classification for impingement. The edema and hemorrhage stage, or Stage 1, is characterized by acute bursitis, subacromial edema, point tenderness on the greater tuberosity and anterior edge of the acromion, and a painful arc between 60 and 120° of abduction. The rotator cuff lesion associated with Stage 1 is reversible and is most common in people less than 25 years of age. Stage 2 (the fibrosis and tendinitis stage) is characterized by all the signs and symptoms of Stage 1, with the addition of tendinitis of the rotator cuff, fibrosis or thickening of the subacromial bursa, a partial thickness cuff tear, crepitus with movement, and “catching” with lowering of the arm at approximately 100° of abduction. Unlike the first stage, the cuff lesions associated with Stage 2 are not reversible. The symptoms associated with Stage 2 are most commonly found in individuals between the ages of 25 and 40. The final stage (Stage 3) of the Neer classification is known as the bone spur and tendon rupture stage. This stage is characterized by an amalgamation of Stages 1 and 2. In addition to the symptoms of those stages, Stage 3 includes either incomplete or complete rotator cuff ruptures, biceps long head lesions, and bone spur development at the anterior acromion. The changes involved with the third stage of impingement are not reversible and are most commonly found in individuals over 40 years of age.

Evaluation of Impingement Syndrome

In order to implement a conservative treatment regimen for an athlete with impingement syndrome, it is important to ascertain what is causing impingement. Impingement syndrome is difficult to evaluate: Because of the number of contributing factors, its clinical presentation can be confusing (3). A thorough evaluation of the athlete is essential for setting rehabilitation goals and implementing a program that fills the athlete's needs.

As with any evaluation, it is important to begin with a thorough history. Oftentimes, individuals with impingement syndrome complain of pain with overhead activities. They report discomfort on the anterior and lateral aspects of the acromion, superficial to the subacromial space, and some complain of weakness or stiffness or describe the sensation of “dead arm” (3). Night pain sometimes plagues individuals with impingement syndrome (60). In more severe cases, there might be complaints of crepitus, clicking, or popping with shoulder motion (60).

After taking the history, the clinician must assess both active and passive range of motion. Bilateral comparison of internal rotation is vital. Posterior capsular tightness manifests as limited internal rotation, and through posterior humeral glides. Evaluation of abduction and forward flexion can provide valuable information implicating impingement syndrome. As an individual abducts at the shoulder, he or she might
exhibit decreased range of motion and complain of a painful arc. A painful arc is
discomfort with shoulder elevation between 70 and 120° of motion and is indicative
of impingement syndrome (30). This painful range corresponds to the greater
tuberosity impinging on the subacromial structures. After approximately 120°, the
humerus externally rotates, clearing the tuberosity and alleviating the discomfort.

Examination for muscle asymmetry is also warranted. In the late stages of
impingement syndrome, disuse of the infraspinatus and supraspinatus can yield visible
atrophy in their respective fossa (10). The therapist should look for asymmetries in
motion at the glenohumeral joint and scapulothoracic articulation. One can recognize
asymmetric scapular humeral rhythm, as well as increased shoulder elevation by the
upper trapezius muscle to compensate for a loss in glenohumeral motion.

A thorough evaluation includes strength assessment of the glenohumeral sta-
bilizers, including manual muscle testing of the biceps brachii, infraspinatus, teres
minor, subscapularis, and supraspinatus. In regard to supraspinatus manual muscle
testing, many athletes with impingement syndrome exhibit weakness secondary to
pain, a sign of impingement.

In addition to strength assessment of the rotator cuff musculature, scapular
stabilizer strength must be ascertained. Because weakness of the scapular stabil-
izer, specifically the upward rotators, can lead to alterations in scapulohumeral
rhythm and the length-tension relationship between the rotator cuff muscles and
their insertion, it is important to determine whether and where weakness occurs. In
addition to the manual muscle test for the serratus anterior described by Daniels
and Worthingham (15), a simple wall push-up can reveal serratus anterior weak-
ness. As an athlete with serratus anterior weakness performs the push-up, the scapula
protrudes away from the thorax. This scapular winging disrupts the normal rela-
tionship between the glenohumeral joint and scapulothoracic articulation, causing
impingement. In synergy with the serratus anterior, the upper and lower portions
of the trapezius muscle, both of which must be assessed, upwardly rotate the scapula.

As with any evaluation, palpation of the anatomic landmarks provides valuable
information as to region of pain. Palpation of the anterior lateral of the acromion,
the bicipital tendon, and the supraspinatus tendon sometimes elicits pain in an athlete
with impingement syndrome. Although the biceps tendon is somewhat superficial
and simple to palpate, the supraspinatus tendon, because of its orientation under the
acromion, is more difficult. In order for the clinician to palpate the greater tuberosity
and supraspinatus tendon, the athlete must fully extend at the shoulder while the
therapist palpates just anterior to the acromion (7). By fully extending the humerus, the
supraspinatus tendon rotates anterior on the humeral head.

The literature presents several provocation tests for evaluating impingement
syndrome. Impingement provocation tests work on the premise of recreating the
impingement mechanism, eliciting pain to signal a positive sign of impingement.
Neer (47) described the traditional impingement test. The athlete stands with his
or her elbow in full extension and the shoulder in neutral rotation. The therapist
then forcibly elevates the limb, causing pain by impinging the subacromial structures
(47). A doctor then injects the subacromial bursa with 1% lidocaine and repeats the
test. If pain is absent after the injection, this indicates a positive impingement test.

Hawkins and Kennedy (24) describe an impingement test that places the athlete’s
shoulder at 90° of flexion. The therapist then passively internally rotates the humerus,
causing the greater tuberosity to impinge on the acromial arch (24). Discomfort in
the subacromial space indicates a positive impingement test. The Speeds test signals bi-
cipital tendinitis involvement in impingement syndrome. The athlete stands or is seated
with his or her elbow in extension and forearm supinated. The athlete then flexes the
shoulder against resistance provided by the therapist (24). If pain is present in the
bicipital groove, it indicates bicipital tendon involvement.

Finally, Corso (13) described an impingement relief test. The athlete per-
forms three to five impingement tests as described by Neer (47). The therapist
records when pain occurs in the range of motion, as well as the athlete’s pain
ingrating, and then repeats the test while applying a gentle inferior or posterior infe-
terior glide to the humerus just prior to the position of pain. Pain relief with the
humeral glide indicates a positive finding of impingement (13).

Assessment of glenohumeral stability is a key component in any impinge-
ment syndrome evaluation. Anterior instability is assessed with the apprehension-
relocation test, the clunk test, and the anterior drawer test, and posterior instability
is assessed with the posterior drawer test, the jerk test, and the posterior apprehen-
sion test (39). Inferior instability can be assessed using both the sulcus test and the fegi
test (39). Even though stability assessment is a necessary component of an evalua-
tion for impingement syndrome, a description of these tests is beyond the
scope of this article. Bublik and Hawkins (7) and Wilk, Andrews, and Arrigo (64)
provide insight into evaluation of the shoulder complex, with emphasis on gleno-
humeral instability and impingement syndrome.

Finally, no examination of the shoulder is complete without ruling out cervical
spine pathology. Because of proximity, cervical pathology can refer pain to the
shoulder region. In addition, radiculopathy at the C5–C6 vertebral level can affect
the supraspinal nerve, causing atrophy of the infraspinatus and supraspinatus
and thereby leading to impingement (10).

Taking the time to fully evaluate both glenohumeral joint and scapulothoracic
articulation, as well as the cervical spine region, is key to implementing a rehabili-
tation program that fills the needs of an athlete with impingement syndrome.

Conservative Management of Impingement Syndrome

Conservative management is advocated for treatment of impingement syndrome,
especially for Neer’s first- and second-stage impingement (60). The goal of con-
servative management is to restore pain-free function in the shortest time possible,
allowing for maximized performance and avoidance of future injury. When de-
signing a conservative management approach, the therapist must set objectives
specific to the athlete’s needs. These goals include decreasing pain and inflamma-


addressing range-of-motion deficits, increasing glenohumeral and scapular stabilizer strength, reestablishing neuromuscular facilitation through functional activities, and facilitating return to athletic participation.

Pain and Inflammation Reduction

The initial objective of conservative management is to decrease subacromial inflammation and pain, allowing for healing of the compromised subacromial structures (10, 44). Richardson (36) advocates active rest as the initial stage in conservative treatment. Actively resting means avoiding the aggravating activity while still maintaining a fitness level suitable for competition (36). Hawkins and Kennedy (24) reported that rest is necessary if pain is strong enough to affect performance. Although rest is advocated for an athlete out of season, in-season athletes might not be afforded that luxury. In cases of in-season athletes, simply reducing the number of repetitions (pitches, serves, strokes, etc.) during practice, in combination with the management techniques described next and summarized in Table 1, might provide a sufficient compromise to rest for the athlete.

Table 1  Conservative Management of Impingement Syndrome

<table>
<thead>
<tr>
<th>Purpose</th>
<th>Technique</th>
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<tbody>
<tr>
<td>Pain and inflammation reduction</td>
<td>modality use anti-inflammatories (NSAIDs and corticosteroid injection)</td>
</tr>
<tr>
<td>Range-of-motion restoration</td>
<td>posterior capsule joint mobilizations posterior cuff stretching</td>
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<tr>
<td>Glenohumeral musculature</td>
<td>“empty can” exercises</td>
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<tr>
<td>strengthening</td>
<td>prone supraspinatus exercises</td>
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<td></td>
<td>prone external rotator exercises</td>
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<tr>
<td></td>
<td>biceps curls</td>
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<tr>
<td></td>
<td>shoulder flexion exercises</td>
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<tr>
<td></td>
<td>external rotation exercises (rubber tubing)</td>
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<tr>
<td>Scapular stabilizer strengthening</td>
<td>push-ups with a plus</td>
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<tr>
<td></td>
<td>shoulder shrugs</td>
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<tr>
<td></td>
<td>press-ups</td>
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<tr>
<td></td>
<td>prone lower trapezius exercises</td>
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<tr>
<td>Functional activities</td>
<td>wide-grip seated rowing exercises (rubber tubing)</td>
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<tr>
<td></td>
<td>closed kinetic chain exercises (unstable surfaces)</td>
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<tr>
<td></td>
<td>slide board push-ups</td>
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<td></td>
<td>rhythmic stabilization exercises</td>
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<td>D2 PNF patterns</td>
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<td></td>
<td>stretch-shortening exercises (plyometrics)</td>
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</table>

In addition to active rest or reduction of repetitions, cryotherapy, transcutaneous nerve stimulation, and ultrasound are beneficial (42). Cryotherapy treatment after activity is commonly prescribed in the literature to aid in reducing pain and decreasing inflammation (14, 23, 33, 56). Ultrasound is effective in treating individuals with impingement syndrome, but one must be cautious with its use (24): In situations of excessive intensity, both unstable cavitation and exacerbation of inflammation can result (56, 59). Clinicians should begin with application for nonthermal effects and progress to thermal application as tolerated.

Use of both nonsteroidal anti-inflammatory drugs (NSAIDs) and corticosteroids such as cortisone is prescribed in the literature for treating impingement syndrome (1, 3, 10, 19). NSAIDs such as ibuprofen, naproxen sodium, and aspirin act systemically to limit inflammation in the subacromial joint (1, 3). For athletes who are in season, a combination of lidocaine and corticosteroid can be injected into the subacromial bursa to decrease inflammation and pain. Although it is effective, care must be taken with injection. It should be limited to the bursa and not involve the compromised tendon, because corticosteroid injection can compromise a potentially weak tendon (10, 16, 56). Cavallo and Speer (10) recommend no more than three injections at least 6 weeks apart.

Range-of-Motion Restoration

Restoration of both physiological and arthrokineic motion at the shoulder joint should begin immediately in the rehabilitation process. Restoring range of motion includes using mobilizations and simple physiological stretching exercises.

Joint mobilizations are advocated for addressing capsular tightness and pain relief (25, 35, 38, 42, 49). They provide a precise stretch specific to a part of the joint capsule while reducing the load on periarticular structures and decreasing the compressive forces on articular structures, as compared with physiological stretching (38). Nicholson (49) studied the effect of mobilization treatment on adhesive capsulitis. The participants were treated three times per week for 4 weeks. They exhibited both pain relief and increased function (49). Conroy and Hayes (12) reported similar results in individuals with impingement syndrome, finding that mobilizations were effective in decreasing pain.

To address posterior capsular tightness using joint mobilizations, the athlete lies supine with the scapula stabilized by the table and the shoulder suspended over the edge. The therapist stabilizes just medial to the shoulder with one hand and applies a dorsal glide of the humerus with the other (see Figure 1). Variations in the humerus position stress different portions of the joint capsule.

In addition to mobilizations, physiological stretching is advocated. A simple stretch consisting of pulling the arm across the body can stretch the posterior cuff and glenohumeral musculature. It is important to stay below the level of the shoulder because elevation above it might impinge the subacromial structures. To address the lack of internal rotation exhibited by overhead athletes, the athlete lies supine
Glenohumeral Musculature Strengthening

A vital component of conservative management is to implement a glenohumeral stabilizer-strengthening program. Whether the cause of impingement is muscle weakness and fatigue or instability resulting from overhead activity, a strengthening program focusing on both the rotator cuff musculature and the biceps brachii should be implemented. Initially, the strengthening exercises can be performed with isometrics, progressing to isotonic exercises once full, pain-free, active range of motion is achieved (1, 31, 58). The goal of glenohumeral stabilizer strengthening is to increase the efficiency of the humeral head depression for dynamic stabilization (10). Parker and Seitz (52) reported that rotator cuff strengthening decreased the pain associated with impingement syndrome in individuals with anterior instability.

An effective glenohumeral-strengthening program for an athlete is based on three premises. First, the rotator cuff muscles not only act as humeral movers but also as humeral head compressors vital to stabilization. The rotator cuff must constantly contract to maintain dynamic stability. As a result, the athlete should perform high repetitions with low resistance when strengthening the rotator cuff musculature. Second, an effective program consists of both concentric and eccentric exercises. Jobe, Tibone, Perry, and Moynes (32) reported that eccentric contraction of the subscapularis in the late cocking phase of pitching limited external rotation and that eccentric contraction of the infraspinatus, teres minor, supraspinatus, and deltoit decreased the limb during the follow-through phase of pitching. Because the rotator cuff musculature of the shoulder contracts eccentrically to decelerate the limb during overhead activities, eccentric training of the rotator cuff muscle better prepares an athlete for the stress experienced during athletic competition (31, 37). Finally, glenohumeral exercises should be performed in the scapular plane (31, 34, 37), which is defined as 30°-45° anterior to the frontal plane (54). Johnston (34) reported that the scapular plane is more functional for movement and places less stress on the shoulder joint. Townsend, Jobe, Pink, Perry, and Tibone (61) provide a thorough EMG analysis of glenohumeral rehabilitation exercise for strengthening the dynamic stabilizers of the shoulder.
There are several dumbbell exercises that are effective in strengthening the dynamic stabilizers of the glenohumeral joint. The first exercise, the “empty can exercise,” is performed in the scapular plane. In this exercise, proposed by Jobe and Moynes (31), the athlete internally rotates and elevates the humerus in the scapular plane. It is important to stay below the level of the shoulder to prevent impingement of the subacromial structures. EMG analysis of elevation in the scapular plane with humeral internal rotation has revealed increased activity in the supraspinatus, anterior deltoid, and middle deltoid (40, 61, 68).

Blackburn, McLeod, White, and Wofford (5) suggested a different position for training the supraspinatus. They recommended the prone position, with the elbow extended and the arm abducted to 100° and externally rotated (5). Malanga, Jend, Cogswell, and An (40) analyzed both the scapular and the prone positions for training the supraspinatus using EMG. They reported that both positions resulted in significant activity of the supraspinatus (40). The scapular position produced greater activation of the anterior deltoid and pectoralis major, whereas the prone position produced significant activation of the middle deltoid (40). Malanga et al. (40) concluded that both positions can be used to strengthen the supraspinatus. Worrall, Core, York, and Santiestaban (69) reported that the prone position produced significantly greater activity, but the standing position produced the greatest force. They concluded that the increased force generation in the standing position resulted from muscle substitution and recommended the prone position for rehabilitation of the supraspinatus. With either position, the athlete might have to flex the elbow early in the rehabilitation process. Performing the exercises with elbow flexion decreases the length of the lever arm, placing less stress on the shoulder joint (1, 37, 65).

To strengthen the external rotators of the shoulder (infraspinatus and teres minor), Blackburn et al. (5) reported increased external rotator activation with horizontal abduction exercises. The athlete lies prone with the humerus abducted to 90° and externally rotated. The exercise is performed by horizontally abducting the humerus. Because the humerus is externally rotated, clearing the greater tuberosity, the risk of impingement syndrome is minimal. EMG analysis of horizontal abduction exercises has revealed significant activity in not only the external rotators but also the posterior deltoid (5, 61).

An effective, conservative program must also include strength training of the biceps brachii. The biceps brachii aids in depression of the humeral head, is active during the late cocking phase (external rotation), and acts as a decelerator during the follow-through phase of pitching (8, 22, 37, 62). Both the traditional biceps curl using standard dumbbells and shoulder flexion exercises strengthen the biceps brachii. Shoulder flexion exercises should be performed in elbow extension to stress the biceps brachii. Staying below the level of the shoulder and externally rotating the humerus minimize impingement. In addition to training the biceps brachii as a shoulder flexor, Townsend et al. (61) reported increased EMG activity in the anterior and middle deltoid with shoulder flexion exercises.

As an adjunct to the dumbbell exercises, rubber tubing provides a convenient, portable way of training the glenohumeral musculature (37). With minor modifica-

tion, all of the dumbbell exercises described above can be performed with rubber tubing, which applies a constant load to the entire musculotendinous unit (65). McCann, Wooten, Kadaba, and Bigliani (41) reported that rubber tubing exercises performed below the level of the shoulder elicited moderate EMG levels of activity for both the rotator cuff and the deltoid, thereby providing an effective means of addressing muscle weakness. The athlete performs humeral rotator exercises in the scapular plane, progressing to a position of vulnerability at 90° of abduction (see Figure 3).

Scapular Stabilizer Strengthening

As previously stated, the scapular stabilizers play a significant role in impingement syndrome. Strengthening these stabilizers (especially the serratus anterior and trapezius) is repeatedly advocated in the literature (1, 31, 33, 35, 37). Although emphasis should be placed on the upper rotators of the scapula, all of the scapular muscles are responsible for scapular motion and stabilization and should be addressed (35). The scapular stabilizers have a postural function; as such, training of these muscles should focus on fatigue-resistant exercises using low resistance and high repetitions (6, 35).

Five exercises are presented here that include all of the scapular-stabilization musculature:

- The first exercise is a push-up with a plus (45): The patient performs a normal push-up, with the exception of adding maximum protraction at the scapula in the extension phase (see Figure 4). The push-up with a plus stresses the serratus anterior and pectoralis minor (45).
- The shoulder shrug trains the upper trapezius, levator scapulae, and rhomboids. The athlete stands with bodyweight on both hands and then elevates and rolls his or her shoulders posteriorly, with emphasis on full scapular retraction (37).
- The athlete performs the press-up by sitting on the edge of a treatment table (see Figure 5) and applying a downward force to it, lifting his or her buttocks. The press-up elicits muscle activity in the latissimus dorsi and pectoralis major (61).
- To train the lower trapezius, the athlete lies prone, abducted to 110° with neutral rotation, and then elevates his or her humerus, eliciting significant lower trapezius activity (45).
- Litchfield, Hawkins, Dillman, Atkins, and Hagerman (37) recommended the wide-grip seated row using rubber tubing. After securing the rubber tubing to a stationary object, the seated athlete pulls the tubing toward his or her body, being careful not to cross the level of the shoulder. The wide-grip seated row addresses the trapezius, rhomboids, and serratus anterior (37).

Functional Activities

Up until this point, the rehabilitation activities discussed in this article address strength and range-of-motion deficits. Although these activities are important, they
Figure 4 — Push-up with a plus for strengthening the serratus anterior.

Figure 5 — Press-up for the latissimus dorsi and pectoralis major.
do not truly prepare an individual for athletic activity. An athlete also needs to restore the neuromuscular mechanisms to promote coordinated movement patterns and functional stability when extreme loads are placed on the joint (36). After sufficient healing, range of motion, and muscular strength and endurance are achieved, functional rehabilitation activities are implemented to restore the dynamic mechanisms specific to athletic activity (36). The traditional rehabilitation exercises already described should be continued during functional rehabilitation. The goals of functional rehabilitation are to minimize the difficult transition from rehabilitation to full activity (10), restore neuromuscular mechanisms, and restore sport-specific motor skills.

Lehman and Henry (36) reported that both closed kinetic chain and open kinetic chain exercises that mimic the athlete’s sport are recommended for restoring neuromuscular mechanisms. Exercises such as tripod balancing on an unstable surface (wobble board, foam, multiaxial devices; see Figure 6) and push-ups on a slide board facilitate cocontraction of the force couples at the shoulder, in addition to stimulating scapular stabilization (see Figure 7). Wilk and Arrigo (66) described a scapular neuromuscular control exercise in which the patient is side-lying on the uninvolved limb. The patient’s shoulder is flexed to 90° with the humerus internally rotated and fixed on the table. The therapist then resists elevation, depression, protraction, and retraction, using rhythmic stabilization techniques (66). Exercises such as press-ups and tripod exercises, which stimulate both the glenohumeral and the scapular muscles, establish the synchrony necessary for coordinated shoulder motion.

Several exercises are recommended for restoring sport-specific motor patterns. It is important to implement exercises that are specific to athletic participation. Diagonal 2 (D2) proprioceptive neuromuscular facilitation exercises with rubber tubing mimic the functional plane of throwing and serving (see Figure 8). Their use facilitates functional movement patterns and reestablishes the neuromuscular mechanisms important for smooth, coordinated movement. Stretch-shortening drills (plyometrics) are also beneficial in preparing a thrower to return to activity. They can mimic throwing movement patterns and reestablish neuromuscular mechanisms by increasing the speed of the myotatic reflex, improving neural efficiency through adaptation, and enhancing muscle coordination (53, 67). In addition, plyometrics imparts the type of eccentric stress experienced by the limb during athletic competition. Athletes can progress from activities below the level of the shoulder to a position of vulnerability, which prepares them for return to competition. Stretch-shortening drills can only be implemented after full range of motion and strength are achieved through rehabilitation (67). Wilk, Arrigo, and Andrews (67) and Pezzullo, Karas, and Irrgang (53) provide descriptions of upper extremity plyometrics, as well as progression of rehabilitation.

Return to Play
In order for an athlete to return to activity after suffering from impingement syndrome, he or she must meet certain criteria. The athlete must be pain free and have

Figure 6 — Closed kinetic chain exercises on a wobble board (A) or therapy ball (B) facilitate cocontraction of the shoulder force-couple musculature.
full range of motion, sufficient strength, suitable glenohumeral and scapular stability, and the ability to confidently perform athletic activity at a competitive level.

In addition, the athlete must be educated as to correct training technique for skill performance (12). Exercises such as the shoulder press, bench press, triceps pullover, latissimus dorsi pull-down, and pull-up behind the head should be avoided or modified because of their impinging mechanisms (17, 37). Fees, Decker, Snyder-Mackler, and Axe (17) recommend avoiding the wide-grip bench press to minimize the torque placed on the shoulder joint, and they suggest narrowing the grip width in instances of shoulder injury. In addition, behind-the-head shoulder presses should be avoided because of the impinging position; they can be replaced with rear deltoid raises, seated rows, or dumbbell rows (17). Isometric shoulder presses in front of the head at varying angles can also act as a substitute (17). Finally, the latissimus dorsi pull-down behind the head should be modified to a front pull-down in a 30° reclined position (17).

Last of all, a vital aspect of returning to play is implementing a prophylactic training program. This program should include continued glenohumeral and scapular stabilizer strengthening both eccentrically and concentrically and stretching exercises for the posterior capsule and external rotators as described in this article. The exercises can be self-administered by the athlete and only take approximately 10 min to perform.
Conclusion

Because of the anatomic geometry of the shoulder and the stresses placed on it during athletic activity, it is not surprising that shoulder pain caused by impingement of the supraspinatus, bicipital tendon, and subacromial bursa in the subacromial space is commonly seen in the field of sports medicine. There are a number of mechanisms responsible for impingement, including muscle fatigue and weakness, tightness of the glenohumeral and scapulothoracic musculature, glenohumeral instability, and changes that result from repetitive overhead activities, such as posterior capsular tightness. This wide array of mechanisms makes it difficult to evaluate. To thoroughly evaluate an athlete with impingement syndrome, a firm understanding of the anatomic and biomechanical characteristics of the shoulder joint is needed. In addition, an understanding of how to differentiate among the mechanisms causing impingement syndrome in the athlete is beneficial. The information obtained from an evaluation is used to implement a rehabilitation program designed to return the athlete to a competitive level.

References


