A Nonoperative Approach to Shoulder Impingement Syndrome

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ABSTRACT

Because the presentation of chronic shoulder pain from overuse injuries can be confusing, determining the underlying cause of the pain requires taking a complete history and conducting an appropriate physical examination. One of the more frequent causes of this pain is impingement of the rotator cuff tendons, also known as impingement syndrome. This article reviews the functional anatomy of the shoulder and provides illustrations of the maneuvers used in the physical examination, as well as concise definitions and descriptions of the subtypes of impingement syndrome. Successful treatment depends not only on an accurate diagnosis, but also on appropriate utilization of rehabilitation exercises, anti-inflammatory medications, corticosteroid injections, and, when appropriate, referral to a specialist. The authors provide a comprehensive approach to the prevention, diagnosis, and nonoperative treatment of the various stages of impingement syndrome, since a nonoperative approach is successful in the majority of individuals.


SHOULDER ANATOMY

The shoulder is a complex joint comprising 3 bones, 4 joints, and at least 15 muscles. The mobility of the shoulder exceeds that of any other joint in the human body. As a
result, it is subjected to a wide range of stresses, both in everyday activity and occupational and recreational pursuits. The glenohumeral articulation maintains a fine balance between mobility and stability, and in chronic overuse situations (i.e., throwing athletics), this balance is disrupted by the repeated microtrauma incurred in achieving peak performances. The bones of the shoulder girdle include the clavicle, the scapula, and the humerus.

The intrinsic muscles, which arise and insert on the shoulder girdle, include the supraspinatus, infraspinatus, teres minor, and subscapularis (Figure 1) and are collectively known as the rotator cuff. They surround the glenohumeral joint to perform two important functions as dynamic stabilizers of the shoulder. First, they act as humeral head depressors that prevent upward migration of the humerus, and second, they maintain the humeral head centered in the glenoid fossa during motion.3

GLENOHUMERAL JOINT

The glenohumeral joint is formed by the humeral head and the concave glenoid fossa. The glenoid surface is only about one-fourth of the size of the convex humeral head.4 Fortunately, the glenoid labrum, which is a band of dense, fibrous connective tissue, increases the concavity and surface area of the glenoid fossa, increasing the stability of the glenohumeral articulation (Figure 2). This glenohumeral articulation is held together by a glenohumeral capsuloligamentous complex (Figure 2). In addition to the joint capsule ligaments, the musculotendinous cuff formed by the rotator cuff muscles reinforces the anterior, superior, and posterior aspects of the glenohumeral joint.5

CORACOACROMIAL ARCH

When reviewing shoulder anatomy to understand impingement syndrome, the coracoacromial arch and the subacromial structures become the main anatomical foci. The coracoacromial arch is a tunnel formed by the acromion, the coracoacromial ligament, the coracoid process, and the scapula (Figure 2). Contained within this arch are the supraspinatus muscle, the subacromial bursa, the tendon of the long head of the biceps, the glenohumeral joint capsule, and the coracoacromial ligament (Figures 2 and 3). The coracoacromial arch protects these structures and the humeral head from direct trauma, while it prevents superior dislocation of the humeral head. In pathologic states, impingement of the rotator cuff and other subacromial structures may occur when they become entrapped between the greater tuberosity of the humerus and the coracoacromial arch.6 Impingement of the subacromial structures may occur at any point along this arch. Abnormal structures involving the acromion also can cause impingement of the subacromial tissue in the arch area. Examples of this include inferior osteophytes at the acromioclavicular (AC) joint in patients with AC joint degenerative joint disease and an unfused acromial epiphysis (os acromiale).7

The tendons of the rotator cuff muscles blend with each other and the joint capsule of the glenohumeral ligament and attach to the greater and lesser tuberosities. Movement of these tendons through the coracoacromial arch is facilitated by the interposition of the subacromial bursa (Figure 3), which acts as a lubricant.8 Although this area usually protects the structures that pass through it, because it has a fixed volume, it is a frequent site of injury when there is increased pressure within the arch or when there are anatomic abnormalities (i.e., a downward curve/type 2 or hooked/type 3 acromion) that limit the volume of the arch. Anatomic impingement of these structures within the coracoacromial arch can be caused by either repeated microtrauma from overhead arm motions or may be secondary to instability.9 In fact, when the rotator cuff is functioning properly, it allows only 4 mm of humeral head translation in any direction from the center of the glenoid fossa. With this in mind, it becomes much easier to appreciate how a small amount of abnormal motion in the glenohumeral joint can lead to a variety of pathologic processes, including impingement of the structures in the coracoacromial arch.

There is a critical zone in the supraspinatus tendon as it passes underneath the acromion, which is relatively less vascularized. This poorly vascularized region is more susceptible to injury from impingement.10 Thus, any condition that leads to inflamma-
tion, edema, swelling, and pain may be caused by anatomic impingement of these structures within the coracoacromial arch, and if allowed to continue, may eventually progress to rotator cuff tears.3

**Classifications of Impingement Syndromes**

There are several subtypes of impingement syndrome. For PCPs, it is important to understand the range of subtypes and to differentiate between them. Accurate subtyping will determine the course of conservative management as well as the surgical approach to treatment, should conservative therapy fail.

Impingement syndrome as first described by Neer in 1972 is a progressive degeneration of the rotator cuff tendon as a result of external compression from the acromion and the coracoacromial arch.9 This condition is now referred to as primary mechanical impingement.1,6 Neer noted that flexion of the shoulder in an internally rotated position led to the movement of the avascular zone of the rotator cuff under the coracoacromial arch. He also noted that bony changes and spur formation on the undersurface of the anterior acromion were due to repeated impingement of the rotator cuff tendon between the coracoacromial ligament and the humeral head. Neer divided impingement syndrome into 3 progressively advancing stages.6 Stage I usually occurs in patients younger than 25 and presents as edema and inflammation in the supraspinatus tendon and bursa. This stage is considered reversible and generally responds to rest and nonsteroidal anti-inflammatory medications. Stage II presents as a more advanced disease process in the 25- to 40-year-old age group and involves fibrosis and tendinitis, which may not necessarily be fully reversible by modification of activities. This group of patients often has activity-related pain and if there is no response to rehabilitation, treatment may be surgical.10 Stage III involves AC spurs, full-thickness rotator cuff tears, and biceps tendon ruptures and is usually seen in patients older than 40. These patients usually have progressive disability and are often candidates for surgery.10

Two additional classifications of impingement syndrome have recently been proposed in addition to the originally described primary mechanical impingement: secondary impingement (functional scapulothoracic instability) and internal impingement. Secondary impingement or functional scapulothoracic instability is defined as a relative decrease in the subacromial space with excessive anterior and superior humeral head translation, which is caused by instability of the glenohumeral joint.6 Accordingly, an association has been detected between impingement syndrome and glenohumeral instability, as seen in one study in which 68% of patients with anterior instability showed signs of impingement.11 Internal or glenoid impingement is used to describe a pathophysiologic process in which the undersurface of the rotator cuff (supraspinatus) is impinged against the posterior–superior surface of the glenoid. This internal impingement is a normal physiologic occurrence that becomes pathologic with repeated microtrauma. It often occurs during extension, abduction, and external rotation of the shoulder, as demonstrated in the late cocking stage of throwing.12 These classifications can be simplified by dividing impingement-type pain into 2 categories, that which occurs in patients younger than 35 years of age, and that which occurs in those older than 35.

The table lists the subtypes of impingement syndrome and their typical clinical presentations, which...
aid in appropriate classification. The table further emphasizes that certain shoulder conditions are age related. Thus, diagnostic probabilities change with increased age-related degeneration of soft tissue. For example, full-thickness tears of the rotator cuff are extremely rare in the young and usually result from significant trauma to the shoulder; however, the incidence of full-thickness rotator cuff tears dramatically increases after age 35.1

**Clinical Examination and Diagnosis**

With a better understanding of shoulder anatomy, it is easier to understand why impingement of the supraspinatus is usually associated with other conditions, including rotator cuff tendinitis, subacromial bursitis, and bicipital tendinitis. These conditions are exacerbated by motions that increase the encroachment of the humeral head into the coracoacromial arch, especially in overhead motion or in the acceleration phase of a throw. Both internal rotation of the humerus (which rotates the greater tuberosity under the acromion) and upward migration of the humeral head with rotator cuff (humeral head depressor) weakness tend to increase an individual's symptoms. Pain and tenderness can sometimes be localized inferior and lateral to the acromion or can occur at night when patients directly compress the injured structures by rolling on their sides or sleep in the “shoulder impingement position” with their hands under pillows or placed in an overhead position.13 Impingement signs (Neer and Hawkins tests, described later) are often positive, and a patients may have a painful arc of motion on abduction between 70 and 120 degrees as the inflamed and relatively avascular tendon passes under the coracoacromial arch.3

**History**

A thorough history that includes onset and symptoms, as well as the location, timing, and intensity of the pain is essential in delineating the cause. The PCP should use these clues to differentiate between primary and secondary impingement.6,12 In the early stages of impingement, pain often occurs only after strenuous shoulder activity. If that activity continues, pain can occur with any activity and may progress to constant pain, especially at night. Rotator cuff tendinopathy pain is usually described as anterior shoulder pain that is dull or achy and often presents as night pain. Pain associated with anterior instability is usually sharp and can present in the region of the posterior capsule during

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- = negative; + = positive; ++ = significantly or unequivocally positive; +/- = can be positive or negative; N SAID = nonsteroidal anti-inflammatory medications. Adapted from Cavallo RJ et al.12
the acceleration phase of a throw. In fact, the classic presentation of primary impingement is often one of pain that is exacerbated by overhead activities and occurs at night, making it difficult to lie on the affected shoulder. Though both secondary and internal impingement can also present in this manner, they are more often associated with pain on overhead throwing motions and less commonly associated with night pain. Further, the pain associated with internal impingement is usually localized more posteriorly. Neurologic symptomatology in the upper extremity, including "dead arm" syndrome, are more commonly associated with instability. (*Dead arm" syndrome is pain-induced subjective paresis, often described as a subjective event that causes the arm to suddenly "fall asleep" and usually is not associated with objective neurologic findings.) Unfortunately, primary impingement patients can also have referred pain down the lateral aspect of the upper portion of their arm, which can make an exact diagnosis difficult. For these reasons, patients with shoulder pain, especially if accompanied by neurologic symptoms, must have thorough cervical spine examinations.

A comprehensive history, along with an examination for signs of impingement and instability, should allow the practitioner to make a diagnosis of impingement and aid in distinguishing between the subtypes of impingement syndrome. Even though subacromial syndromes (including impingement and rotator cuff tendinitis and tears) are the most common causes of exertional shoulder pain, we must not overlook other potential causes. The differential diagnosis includes cervical radiculitis, shoulder instability, AC degenerative disease, thoracic outlet syndrome, and supra-scapular nerve entrapment. Common causes of subacromial syndromes include repetitive overhead activity, trauma, sleeping in the "impingement position," a hooked or curved acromion, os acromiale, shoulder instability, and improper technique during overhead shoulder sports.

**Physical Examination**

Although a detailed discussion of the diagnostic examination of the shoulder is beyond the scope of this article, the examination should include a comparative assessment of muscular and bony asymmetries, range of motion, location of tenderness, strength testing, and specific testing for signs of impingement and instability. For instance, on inspection of the shoulder, while muscle atrophy is abnormal, hemihypertrophy and shoulder girdle depression can normally be seen in a patient's dominant arm, especially if the patient is an overhead athlete. The scapulothoracic joint should be assessed for crepitation, winging, early protraction, or dyskinesia with loss of the normal smooth rhythm. Active and passive motion of the glenohumeral joint should be examined, looking for any associated pain, crepitation, clicking, or loss of motion. Increased external rotation with concomitant loss of internal rotation in the dominant arm is a common finding in normal throwing athletes. The location of tenderness can be an important clue to the diagnosis and can help differentiate between conditions such as AC arthritis, supraspinatus, bicep tendinitis, subacromial bursitis, and rotator cuff tears.

**Supraspinatus Testing**

Strength testing of the shoulder muscles must include the rotator cuff muscles, and most importantly, the supraspinatus. Testing of the supraspinatus is classically described as having the patient abduct an arm to 90 degrees, flex forward 30 degrees, and then to apply downward pressure with the patient's arm internally rotated so that the thumb points down. This is also known as the empty can test (Figure 4), and strength testing in this position puts the humerus in the plane of the supraspinatus muscle. More recent discussions of this test argue that the supraspinatus muscle strength is better isolated, and thus better assessed, with the thumb pointing upward instead. In either case, pain and weakness to downward pressure during this test is indicative of supraspinatus pathology. The rest of the cuff can be tested by resisted internal and external rotation, scapular push-off, modified push-off, and lag sign testing.

**Impingement Testing**

The 2 classic impingement tests used in this...
evaluated are the Neer and the Hawkins tests. In the Neer test, the patient's arm is internally rotated and then forward flexed to its limit while the scapula is stabilized (Figure 5). This maneuver rotates the greater tuberosity under the acromion, thereby limiting subacromial space and compressing the coracoacromial arch structures, with pain being elicited in a positive test. The Hawkins test is performed by forward flexing the arm to 90 degrees, flexing the elbow to 90 degrees, and then internally rotating the arm to its limit (Figure 6). Again, pain is indicative of a positive test. Magnetic resonance imaging (MRI) evaluation of the positions of these impingement tests have suggested that the Hawkins maneuver may test external shoulder impingement better than the Neer test. Examination and comparison to the unaffected side helps prevent false-positive test results by assessing the degree of difference in discomfort, if any, between the unaffected and affected sides.

Glenohumeral Instability

To help distinguish primary (mechanical) from secondary (functional instability) impingement, instability or glenohumeral translation testing is used, which includes the load and shift test, and the apprehension, augmentation, and relocation tests. These tests can be performed in either a seated or a supine position. Placing the patient in a supine position is beneficial because it provides scapular stability while helping the patient and the periscapular muscles to relax. For the load and shift test, the patient can be seated and the examiner can stabilize the limb girdle by placing 1 hand on shoulder and scapula. With the opposite hand the examiner helps load or direct the humeral head medially into the glenoid fossa while concomitantly palpating the anterior and posterior glenohumeral joint in order to assess the degree of anterior, posterior, and inferior translation of the humeral head (Figure 7). There is no standardized grading scale, although one widely used scale ranges from grades 1 to 3, where 1+ is increased humeral head translation, 2+ is increased translation to the glenoid rim, and 3+ is increased translation beyond the glenoid rim. Grades 1+ and 2+ may be considered normal, especially in the posterior direction, and if asymptomatic and symmetric with the opposite shoulder. This test is helpful in detecting gross or macroinstability but may not be as beneficial in detecting more subtle forms of microinstability. Some authors believe that the most sensitive means of detecting occult anterior microinstability is to perform the apprehension test with a relocation maneuver. The augmentation test (an anteriorly directed force during the apprehension
The apprehension, augmentation, and relocation tests are performed with the patient's shoulder in a position of abduction and maximal external rotation (Figure 8). A positive apprehension test result occurs when the patient becomes apprehensive or complains of pain during increased external rotation from a neutral position.

To perform the augmentation test, with the arm in the position described above, the examiner places an anteriorly directed force on the proximal humerus, thereby trying to anteriorly translate the humeral head out of the glenoid fossa. In a positive augmentation test result, the patient usually complains of pain or apprehension (impending dislocation) and experiences symptoms at a lesser degree of external rotation. Immediately, the examiner should next apply a posteriorly directed force to the humeral head while maintaining the patient's arm in the position of apprehension or pain. This relocation maneuver prevents anterior subluxation of the humeral head and may relieve the patient's apprehension or pain. Any reduction in symptoms is considered a positive relocation test. This test is considered the most sensitive clinical test for detecting forms of subtle instability.

One study found that the response of apprehension was actually much more specific for instability than a response of pain. Throwing athletes with occult instability and secondary impingement are more likely to complain of pain, while patients with posttraumatic instability are more likely to experience apprehension. Additionally, a negative apprehension test result in patients with impingement symptoms makes the diagnosis of primary impingement more likely, but patients with primary impingement can also experience pain on apprehension testing, making it more difficult to differentiate between a diagnosis of primary vs sec-
SHOULDER PAIN

Secondary impingement. While a positive relocation test result is most sensitive in detecting subtle instability, most throwing athletes will not exhibit this on clinical examination. Thus, an examiner should look for other possible signs of instability. Although excessive laxity does not necessarily imply the pathologic state of instability, excessive and/or generalized ligamentous laxity may predispose an individual to be more susceptible to secondary impingement syndrome. Signs of generalized ligamentous laxity include hyperextension at the elbows and thumbs. Excessive inferior glenohumeral translation and capsular laxity, as indicated by a positive inferior sulcus sign, can also signify multidirectional instability and abnormal translation as the cause of impingement symptoms.

**Impingement Test and Subacromial Injections**

An impingement injection test is sometimes needed to help differentiate between pain or weakness caused by the shoulder tests vs those caused by other conditions, such as full-thickness rotator cuff tears and adhesive capsulitis. This test is performed by injecting 10 mL of anesthetic into the subacromial space. The shoulder should be re-examined 5 to 10 minutes after the injection. Impingement pain and weakness due to tests will be relieved by the injection; however, weakness associated with a rotator cuff tear or pain associated with other conditions will persist. Interestingly, in patients who eventually go on to have surgery, some studies have indicated that a positive impingement test result is associated with higher success rates after surgery.

Injection into the subacromial space, whether for testing or treatment, can be done by an anterior or posterior approach. The posterior approach tends to be an easier way to enter the subacromial bursa. Also, injection via this route may be more comforting to patients, because they do not see the needle penetrate the skin. The entry point for the posterior approach is approximately 1 cm inferior from the posterior lateral corner of the acromion in the “soft spot” (the sulcus between the head of the humerus and the acromion). Once the soft spot (Figure 9) is identified and sterilely prepped, ethylene chloride can be used as a topical anesthetic to “freeze” the skin. The needle (22 gauge, 1.5 inch) should be parallel to the undersurface of the acromion and aimed towards the coracoid process (the examiner’s other hand can help palpate and identify this). The needle should be aimed upward at about a 10- to 15-degree angle to enter the subacromial bursa. If there is a problem finding the bursa, having an assistant apply downward traction on the patient’s arm may allow for more space in the subacromial area. Once in the bursa, there should be minimal resistance to the injection of the anesthetic/corticosteroid. Despite this technique, the accuracy of the needle in reaching the subacromial bursa has been questioned in a recent study. In this study, 30% of subacromial injections were actually into structures other than the subacromial space (the deltoid was the most common incorrect site), as determined by injected radiographic contrasted material (seen immediately on X-ray, postinjection).

**Goals of Managing Impingement Syndrome**

- Establish a specific diagnosis
- Control pain and inflammation
- Restore normal motion
- Improve rotator cuff function
- Strengthen scapular muscle/normalize scapulohumeral rhythm
- Correct form
- Resume normal function
- Prevent recurrence

Adapted from Marks, et al.

**Imaging**

In general, there is a large degree of crossover between the physical examination findings of instability, rotator cuff tendinopathy, and other shoulder disorders. Whereas many of the tests are sensitive for instability and rotator cuff pathology, they may not be specific. An experienced PCP must take into account the patient’s history, physical examination, and radiographic findings when formulating a diagnosis and treatment plan. In the early stages of impingement, X-ray findings are usually minimal, although a calcified tendon, acromial spurring, and...
increased acromial hooking may be seen. Later stages of impingement may show sclerosis and cyst-like changes at the greater tuberosity (pseudocyst formation) and sclerosis of the undersurface of the acromion (sourcil sign), which may indicate rotator cuff pathology. On the other hand, while these findings may be sensitive for impingement syndrome, they are not specific for rotator cuff pathology and impingement syndrome and are often considered normal variants in the absence of shoulder symptoms. A minimum of 3 views of the shoulder should be obtained: true anterior-posterior (AP) (Grashey), lateral in the scapular plane (outlet view), and axillary. An AC joint view should be included if that is the problem area as determined by history and/or physical examination.

Further radiologic workup, including computed tomography (CT) and MRI with contrast, may be necessary to make the appropriate diagnosis. In general, CT imaging can better delineate bony detail and thus, the findings of primary impingement. However, because the diagnosis of impingement is largely determined on a clinical basis, CT imaging is rarely indicated as a supplemental study. MRI also has been shown to be very sensitive and specific in the diagnosis of impingement syndrome. Although similar to CT imaging, it is rarely indicated solely for diagnosing impingement. MRI is a useful diagnostic tool when the cause of the shoulder pain is less clear or when a rotator cuff tear or labral pathology is suspected. Overall, we must understand that though sophisticated and expensive imaging techniques are very useful in obtaining a diagnosis, they are often unnecessary and are not cost effective in clear cases of impingement.

**Nonoperative Treatment**

The initial treatment of all subtypes of impingement syndrome should involve an early, aggressive rehabilitation program and should be a coordinated partnership between the PCP, the patient, and the physical therapist, even if this entails only 1 visit with the physical therapist. Initial treatment should focus on relief of pain through relative rest, modifying provocative activities, and avoiding aggravating movements (especially overhead work and athletic activities like swimming and throwing). Besides modifying work and athletic activities, if the aggravating factor is a sleeping position (eg, sleep with arms in an overhead “impingement position”), some clinicians and therapists will use either a shoulder immobilizer or have patients sleep with their arms against their bodies under a nightshirt to reduce sleep disturbance and to minimize shoulder impingement pain. The next step is an effort to further decrease inflammation and pain through the use of cryotherapy techniques, including ice massage along with an appropriate trial of nonsteroidal anti-inflammatory medications.

The subsequent focus should be restoration of normal flexibility. An individualized stretching program should be performed in a gentle, sustained manner several times per day. This can be enacted by an experienced PCP; however, in most instances we recommend using an experienced therapist to better ensure patient understanding and compliance. Once pain-free range of motion is attained, the next goal is restoration of strength through a rotator cuff and scapulothoracic stabilizing rehabilitation program. Some exercises are designed to strengthen the internal and external rotators of the shoulder in an attempt to make the rotator cuff a more efficient humeral head depressor and stabilizer (ie, pull the humeral head down and away from the acromion). Next, regaining normal scapulothoracic rhythm is stressed for all types of impingement. Some authors, by contrast, claim that a comprehensive protocol for treatment of secondary shoulder impingement more properly places emphasis on the proper function of the scapulothoracic musculature, including the rotator cuff muscles. Appropriate function of the scapulothoracic muscles fosters synchrony of the scapulothoracic rhythm, which helps ensure minimal abnormal translation of the humeral head and, therefore, should lessen the risk of secondary impingement problems.

Furthermore, for patients with secondary impingement due to instability, stretching should be done carefully and only for muscle groups with obvious tightness, because stretching the anterior muscles and capsule in a patient with anterior instability may cause further laxity and worsening of symptoms. Also, in recent studies, proprioceptive deficits have been noted in patients with impingement syndrome, so restoration of proprioception can have an important role in reestablishing normal functioning. Most importantly, full activity should not be resumed until the patient has regained full shoulder mobility and strength in order to prevent either worsening of their condition or recurrence.

During this rehabilitation period patients should be closely followed by their PCPs and physical therapists for early detection of progression of symptoms in order to prevent worsening of tendinopathies. The majority of patients treated in the early phases of impingement will respond favorably to these conservative measures and return to full activity without further treatment. When patients fail to respond to the first 4- to 6-week course of therapy, they should be carefully reevaluated to rule out other conditions...
that may have been overlooked, such as subtle glenohumeral instability. When symptoms are refractory to the above conservative measures, the pain and inflammation may be relieved with a corticosteroid injection. The use of an injection of corticosteroid with anesthetic at this point can be used to provide both relief of symptoms and to recheck the diagnosis with an impingement injection test. The corticosteroid injection often allows the patient to fully participate in the prescribed rehabilitation program, which can significantly increase chances for recovery with conservative management. This sequence can be repeated up to 3 times over a 6- to 12-month period. Because corticosteroid injections may weaken the tendons and ligaments, strenuous activity should be avoided for at least 2 weeks after each injection. Most physicians will not espouse giving more than 3 injections in any 1-year period.

The patient should maintain overall physical conditioning and cardiovascular fitness through cross-training techniques. Experience with injured workers and athletes of all levels repeatedly demonstrates that by having patients maintain their general levels of conditioning, they return to their preinjury work or athletic levels relatively faster. Such a program can be continued for 6 to 12 months with appropriate supervision by the PCP and therapist before considering any operative approaches. There are obvious exceptions to this treatment strategy, such as patients who sustain full-thickness rotator cuff tears or who have significant anatomic abnormalities that prohibit resolution of symptoms.

Once a successful recovery has been achieved by using an individualized, aggressive, prescribed rehabilitation program, it is imperative to undertake appropriate alterations in a patient’s work or training techniques and form in order to prevent a recurrence of symptoms. There are many ways to adjust a patient’s activity level and style according to the underlying pathoanatomy of that patient’s symptoms.

Conservative treatment is considered to have failed if the patient has not achieved resolution of symptoms and a resumption of previous levels of function and activity within 6 months. Furthermore, surgery is not usually considered unless a patient has undergone a supervised rehabilitation program for at least 3 to 6 months. Thereafter, recent studies report that arthroscopic subacromial decompression generally provides good to excellent results in 70% to 80% of these patients and shows continued improvement at the 5-year postoperative period. Although surgical treatment is often helpful, it can require a long recovery period for throwing athletes and can be associated with residual symptoms.

Further details regarding surgical procedures for this problem are beyond the scope of this article.

In general, referral to a specialist is indicated: if the patient’s symptoms fail to improve with 3 to 6 months of supervised conservative therapy; if the patient’s pain worsens despite appropriate conservative management, as in the case of a significant rotator cuff tear; and when the PCP is unsure of the diagnosis and/or feels uncomfortable treating these conditions. An early and appropriate referral to a colleague with experience treating musculoskeletal injuries may be more cost effective in its avoidance of unnecessary radiologic workups, and may possibly avert irreversible changes that will occur if the condition is allowed to progress. Examples of appropriate specialists include primary care sports medicine specialists who can be located via the American Medical Society for Sports Medicine or orthopedic sports medicine physicians, located via the American Orthopaedic Society for Sports Medicine.

Though coordinating PCP and physical therapist activities is important, ensuring active patient participation is critical. Only through compliance with the prescribed therapeutic course can patients avoid worsening their injuries. With the correct diagnosis and the coordinated rehabilitative efforts of the physician, the physical therapist, and the patient, the majority of patients will return to their prior levels of activity.

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