

Shoulder impingement syndrome

Wing K. Chang, MD^{a,b,*}

^a*Department of Rehabilitation Medicine, Albert Einstein College of Medicine,
1300 Morris Park Avenue, Bronx, NY 10461, USA*

^b*NYCONN Orthopaedic and Rehabilitation Specialists, 1421 Third Avenue,
Penthouse, New York, NY 10028, USA*

Neer [1] first introduced the concept of rotator cuff impingement in 1972. He described the syndrome as a mechanical impingement of the rotator cuff tendons beneath the anterior-inferior portion of the acromion occurring when the shoulder is placed in the forwardly flexed and internally rotated position. Neer [2] described three stages in the spectrum of rotator cuff impingement. Stage I, which is seen most commonly in patients younger than 25 years of age, has acute inflammation, edema, and hemorrhage in the rotator cuff. This stage usually is reversible with nonoperative treatment. Stage II usually is seen in patients age 25 to 40 and represents a progression from acute edema and hemorrhage to fibrosis and tendinitis of the rotator cuff (Fig. 1). Stage II pathology may not respond to conservative treatment and may require operative intervention [2]. Stage III is typified by mechanical disruption of the rotator cuff tendons and changes in the coracoacromial arch with osteophytosis along the anterior acromion. This stage commonly affects patients older than 40 years of age and commonly requires surgical anterior acromioplasty and rotator cuff repair [2]. In all stages, Neer [2] believed the etiology to be impingement of the rotator cuff tendons under the acromion and the rigid coracoacromial arch, eventually leading to degeneration and tearing of the rotator cuff tendon. Although rotator cuff tears are more common in older populations, impingement and rotator cuff disease are increasingly more common in athletes whose sports involve repetitive overhead motions. The increased forces and repetitive overhead motions can cause attritional changes in the distal part of the supraspinatus tendon, which is most at risk due to its poor blood supply.

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* NYCONN Orthopaedic and Rehabilitation Specialists, 1421 Third Avenue, Penthouse, New York, NY 10028, USA

E-mail address: changwi@yahoo.com

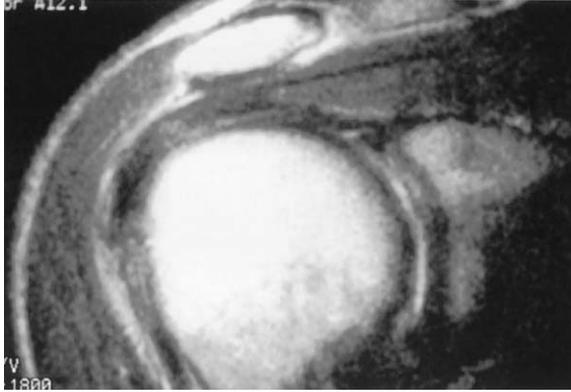


Fig. 1. T2-weighted MRI shows increased signal in the supraspinatus tendon consistent with tendinitis.

Impingement syndrome and rotator cuff disease affect athletes at a younger age than in the general population as described by Neer.

Functional anatomy

The shoulder consists of two bones (humerus and scapula), two joints (glenohumeral and acromioclavicular), and two articulations (scapulothoracic and acromiohumeral), which are joined by several interconnecting ligaments and layers of muscles. Minimal bone stability in the shoulder permits a wide range of motion (ROM) [3]. Soft tissue structures are the major glenohumeral stabilizers [3]. Static stabilizers consist of the articular anatomy, glenoid labrum, joint capsule, glenohumeral ligaments, and inherent negative pressure in the joint [4]. Dynamic stabilizers include the rotator cuff muscles (supraspinatus, infraspinatus, teres minor, and subscapularis), long head of the biceps tendon, scapulothoracic motion, and other shoulder girdle muscles (eg, pectoralis major, latissimus dorsi, and serratus anterior) [5]. The rotator cuff consists of four muscles that control three basic motions: abduction, internal rotation, and external rotation. The supraspinatus muscle is responsible for initiating abduction, the infraspinatus and teres minor control external rotation, and the subscapularis controls internal rotation. The rotator cuff muscles provide dynamic stabilization to the humeral head in the glenoid fossa (fulcrum effect) and form a “force couple” with the deltoid to allow elevation of the arm [6]. The rotator cuff is responsible for 45% of abduction strength and 90% of external rotation strength [7].

The supraspinatus outlet is a space formed by the acromion, coracoclavicular arch, and acromioclavicular joint on the upper rim and the humeral head and glenoid below. It accommodates passage and excursion of the supraspinatus tendon. Abnormalities of the supraspinatus outlet have been

attributed as the cause of impingement syndrome and rotator cuff disease. Other causes since have been discovered.

Impingement implies extrinsic compression of the rotator cuff in the supraspinatus outlet space. Bigliani et al [8,9] discovered and described variations in acromial size and shape that can contribute to impingement. Based on cadaver studies, there seem to be three different variations in the morphology of the acromion: Type I is flat, type II (Fig. 2) is curved, and type III is hooked anteriorly [8,9]. Although the curved configuration was the most common (43% prevalence compared with 17% for flat and 40% for hooked), the hooked configuration was associated most strongly with full-thickness rotator cuff tears [8,9].

Other sites of impingement in the supraspinatus outlet space include the coracoacromial ligament (where thickening can occur) and the undersurface of the acromioclavicular joint (where osteophytes can form). Only rarely is the medial coracoid involved [10]. These various impingement sites in the supraspinatus outlet are compressed further when the humerus is placed in the forward flexed and internally rotated position, forcing the greater tuberosity of the humerus into the undersurface of the acromion and coracoacromial arch.

Nonoutlet impingement also can occur [10]. The causes may be loss of normal humeral head depression from a large rotator cuff tear or weakness of the rotator cuff muscles from a C5-6 neural segmental lesion or a suprascapular mononeuropathy. Another way this impingement may occur is through thickening or hypertrophy of the subacromial bursa and rotator cuff tendons.

Sports-specific biomechanics

Athletes with shoulder pain and rotator cuff disease most often are involved in sports requiring repetitive overhead arm motions, such as

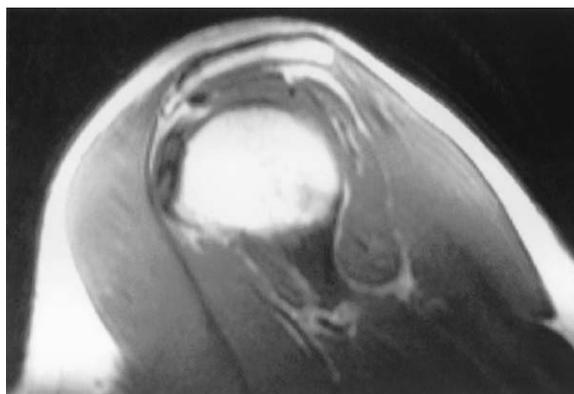


Fig. 2. T1-weighted oblique sagittal MRI shows a type II acromion.

swimming, baseball, and tennis. Rotator cuff disease in this population more likely is related to subtle instability and secondary to factors such as eccentric overload, muscle imbalance, glenohumeral instability, and labral lesions. These instances of rotator cuff disease have led to the concept of secondary impingement, which is defined as rotator cuff impingement that occurs secondary to a functional dynamic decrease in the supraspinatus outlet space due to underlying instability of the glenohumeral joint [10,11]. Young overhead athletes frequently place repetitive large stresses on the static and dynamic glenohumeral stabilizers, resulting in microtrauma and attenuation of the glenohumeral ligamentous structures and generating subclinical glenohumeral instability. This instability places increased stress on the dynamic stabilizers of the glenohumeral joint, including the rotator cuff tendons. These increased demands lead to rotator cuff pathology, such as partial tearing or tendinitis. As the rotator cuff muscles fatigue, the humeral head translates anteriorly and superiorly, impinging on the coracoacromial arch, leading to rotator cuff inflammation. In these patients, treatment should be directed at the underlying instability.

The concept of glenoid impingement has been advanced as an explanation for partial-thickness tears in throwing athletes, particularly tears involving the articular surface of the rotator cuff tendon [10,12]. These tears might occur in the presence of instability due to increased tensile stresses on the rotator cuff tendon either from abnormal motion of the glenohumeral joint or from increased forces on the rotator cuff necessary to stabilize the shoulder. Arthroscopic studies of these patients have noted impingement between the posterior/superior edge of the glenoid and the insertion of the rotator cuff tendon with the arm placed in the throwing position, abducted and externally rotated [12]. Lesions were noted along the area of impingement, at the posterior aspect of the glenoid labrum and articular surface of the rotator cuff [12]. This concept is believed to occur most commonly in throwing athletes and must be considered when assessing for impingement.

History

Age

In patients younger than 40 years of age, history may include the following:

- Usually glenohumeral instability
- Acromioclavicular joint disease/injury

In patients older than 40 years of age, history may include the following:

- Glenohumeral impingement syndrome/rotator cuff disease
- Glenohumeral joint degenerative joint disease

Occupation

Most at risk for impingement are laborers with jobs requiring repetitive overhead activity and athletes (eg, swimming, throwing sports, tennis, volleyball). The following need to be considered in regard to athletic activity:

- Onset of symptoms in relation to specific phases of the athletic event performed
- Duration and frequency of play
- Duration and frequency of practice
- Level of play (little league, pre–high school, high school, college, professional)
- Actual playing time (starter, backup, bench player)
- Position played

Symptoms

The following need to be considered in regard to symptoms:

Onset

- Sudden onset of sharp pain in the shoulder with tearing sensation suggests rotator cuff tear
- Gradual increase in shoulder pain with overhead activities suggests an impingement problem

Chronicity of symptoms

Location of symptoms

- Pain usually lateral, superior, anterior shoulder, occasionally refers to deltoid region
- Posterior shoulder capsule pain usually consistent with anterior instability causing posterior tightness
- Setting during which symptoms come on

Quality of pain (eg, sharp, dull, radiation, throbbing, burning, constant, intermittent, occasional)

Quantity of pain (on a scale of 1–10, with 10 being the worst)

Alleviating factors (eg, change of position, medication, rest)

Aggravating factors (eg, change of position, medication, increase in practice, increase in play, change in athletic gear/footwear, change in position played)

Associated manifestations (eg, chest pain, neck pain, dizziness, abdominal pain, shortness of breath, numbness, weakness)

Provocative positions

- Pain with humerus in forward flexed and internally rotated position suggests rotator cuff impingement
- Pain with humerus in abducted and externally rotated position suggests anterior glenohumeral instability and laxity

Other history

Other questions to ask in regard to the history include questions about previous or recent trauma, stiffness, numbness, paresthesias, clicking, catching, weakness, crepitus, symptoms of instability, and neck symptoms.

Physical examination

Inspection

For the inspection portion of the physical examination, the clinician needs to:

- Visualize the entire shoulder girdle and scapula
- Note muscle mass asymmetry/atrophy and bony asymmetry

Range of motion

Active ROM is tested if possible; if not, passive ROM is tested, as follows:

- Forward flexion (average range 150–180°)
- Abduction (average range 150–180°)
- External rotation (average range with arm in adduction 30–60°; with arm in abduction 70–90°)
- Internal rotation (average range measured by how high the patient can reach the back with the ipsilateral thumb [ie, ipsilateral hip, T12, L5]) (average range above T8)
- Adduction (average range 45°)
- Extension (average range 45°)

The following considerations apply in regard to ROM testing:

Stiffness with external/internal rotation is best tested with the arm in 90° of abduction.

It is best to test external/internal rotation in the supine position with the scapulothoracic articulation stabilized.

Most high-level pitchers have increased external rotation and decreased internal rotation in the pitching arm compared with the nonpitching arm. This may not be pathologic in high-level athletes.

Palpation

Palpation is performed along the joints, noting the biceps tendons, supraspinatus and subscapularis tendons, and anterolateral corner of the acromion. The entire shoulder girdle is palpated (noting tenderness, deformities, and atrophy) from the acromioclavicular joint, clavicle, glenohumeral joint, scapula, scapulothoracic articulation, anterior/posterior shoulder capsule, supraspinous fossa, infraspinous fossa, and humerus, especially proximally.

Manual muscle testing

For manual muscle testing, the clinician should concentrate on the shoulder girdle muscles (especially external/internal rotation, abduction). The supraspinatus may be isolated by having the patient rotate the upper extremity so that the thumbs are pointing to the floor; resistance is applied with the arms in 30° of forward flexion and 90° of abduction (called the *supraspinatus isolation test* or *empty can test* (position imitates the position of emptying of a can).

The following considerations apply in regard to manual muscle testing:

Pain is felt with tendinitis or partial injury to the supraspinatus tendon with the supraspinatus isolation test, but weakness also is found accompanying partial-thickness or full-thickness disruption of the supraspinatus tendon.

Weakness also may be found with tendinitis due to muscle inhibition from the pain stimulus.

Special tests

All tests performed should compare both shoulders either to detect bilateral pathology or to establish a control for comparison with the affected shoulder.

Impingement signs. There are two tests for impingement signs. In the *Neer test*, the examiner forcefully elevates an internally rotated arm in the scapular plane causing the supraspinatus tendon to be impinged against the anterior inferior acromion [2]. In the *Hawkins-Kennedy test*, the examiner forcefully internally rotates a 90° forwardly flexed arm causing the supraspinatus tendon to be impinged against the coracoacromial ligamentous arch [13,14]. Pain and a grimacing facial expression indicate impingement of the supraspinatus tendon—a positive Neer/Hawkins impingement sign.

Impingement test. The examiner injects 10 mL of a 1% lidocaine solution into the subacromial space, then repeats the tests for impingement sign. Elimination or significant reduction of pain constitutes a positive impingement test [2].

Drop arm test. The examiner places the patient's arm in maximum elevation in the scapular plane. The patient is asked to lower it slowly to the side (the test can be repeated after subacromial injection of lidocaine). Sudden dropping of the arm suggests rotator cuff tear [15].

Supraspinatus isolation test. The supraspinatus tendon may be isolated by having the patient rotate the upper extremity so that the thumbs are

pointing to the floor; resistance is applied with the arms in 30° of forward flexion and 90° of abduction (called the *supraspinatus isolation test* or *empty can test*) [16]. The test is positive when weakness is present compared with the unaffected side, suggesting a disruption of the supraspinatus tendon.

Tests for instability

Sulcus sign

For the sulcus sign, the examiner grasps the patient's elbow and applies inferior traction. Dimpling of the skin subjacent to the acromion—the sulcus sign—indicates inferior humeral translation, suggesting multidirectional instability [17].

Apprehension test

The apprehension test is done most effectively with the patient in supine position stabilizing the scapula. The examiner gently brings the affected arm into an abducted and externally rotated position. The patient's apprehension and guarding by not allowing further motion by the examiner denotes a positive test that is consistent with anterior shoulder instability [18].

Relocation test

The relocation test usually is done in conjunction with the apprehension test. After putting the patient in an apprehensive position, posteriorly directed pressure is placed on the anterior proximal humerus, simulating a relocation of the glenohumeral joint that presumably was partially dislocated from the apprehension test. The adept examiner may feel posterior translation of the humeral head on the glenoid. A positive test is reflected by the patient becoming more at ease with the application of pressure on the anterior proximal humerus, suggesting anterior shoulder instability [18].

Other tests

Other tests should be performed to rule out other pathology affecting the biceps tendon, the glenoid labrum, the cervical spine, the sternoclavicular joint, the acromioclavicular joint, and the scapulothoracic joint. A survey of other joint ROM also should be performed to assess for generalized ligamentous laxity.

Neurovascular examination

To complete the shoulder examination, a full neurologic examination must be performed along with assessment of all upper extremity vascular

pulses. The neurologic examination should include all neurologic segments from C5 through T1 myotome, dermatome, and the corresponding stretch reflexes.

Causes

Primary impingement

Primary impingement is indicated by the following:

- Increased subacromial loading
- Acromial morphology (eg, hooked acromion, presence of an os acromiale or osteophyte, calcific deposits in the subacromial space make patients more predisposed for primary impingement)
- Acromioclavicular arthrosis (inferior osteophytes)
- Coracoacromial ligament hypertrophy
- Coracoid impingement
- Subacromial bursal thickening and fibrosis
- Prominent humeral greater tuberosity
- Trauma (direct macrotrauma or repetitive microtrauma)
- Overhead activity (athletic and nonathletic)

Secondary impingement

Secondary impingement [10,11] is indicated by the following:

- Rotator cuff overload/soft tissue imbalance
- Eccentric muscle overload
- Glenohumeral laxity/instability
- Long head of the biceps tendon laxity/weakness
- Glenoid labral lesions
- Muscle imbalance
- Scapular dyskinesia
- Posterior capsular tightness
- Trapezius paralysis

Differential diagnosis

The differential diagnosis includes the following:

- Acromioclavicular joint arthritis
- Adhesive capsulitis
- Glenohumeral joint arthritis
- Cervical herniated disk
- Rotator cuff injury
- Suprascapular nerve entrapment

Diagnostic workup

Standard radiographic studies

The following four standard views are used to rule out glenohumeral/acromioclavicular arthritis:

- Anteroposterior view of the glenohumeral joint
- Internal rotation view of the humerus with a 20° upward angulation to show the acromioclavicular joint [5]
- Axillary view—most useful to rule out subtle signs of instability, such as glenoid avulsion or Hill-Sachs lesion, and to visualize the presence of an os acromiale [5]
- Supraspinatus outlet view—most useful to assess the supraspinatus outlet space (if <7mm, more at risk for impingement syndrome); also assesses the morphology of the acromion (hooked acromion being more at risk for impingement) [5]

Ultrasonography

Ultrasonography [19] is a quick and easy way to diagnose shoulder tendon disruptions (eg, rotator cuff tears, proximal biceps tendon disruption). With the advent of more advanced MRI technology today, ultrasound is reserved more commonly for cases when MRI is not readily available.

Advantages of ultrasound are as follows:

- Noninvasive
- No radiation
- Cost-effective
- Time efficient
- High sensitivity

The disadvantages of ultrasound are that it is operator, interpreter, and technique dependent.

Magnetic resonance imaging

MRI is considered the imaging study of choice for shoulder pathology [19]. Its advantages are as follows:

- Noninvasive
- No radiation
- Able to detect intrasubstance tendon degeneration or partial rotator cuff tears
- Able to detect inflammation, edema, hemorrhage, and scarring
- Able to be used with an intra-articular contrast agent (gadolinium), which improves the MRI ability to detect partial rotator cuff tears

Disadvantages of MRI are as follows:

- Not able to accommodate patients with claustrophobia
- Not able to accommodate larger patients
- Not able to accommodate patients with pacemakers, other metal implants, or particles
- Dependent on quality of the MRI machine
- Dependent on the skill of the technician performing the imaging and the radiologist interpreting the images
- High cost

Arthrography

With arthrography [19], dye is injected into the glenohumeral joint, and postinjection radiographs assess the integrity of the glenohumeral joint. Arthrography frequently is used in evaluating rotator cuff tears. If dye escapes out of the joint and into the subacromial space, it is diagnostic of a full-thickness rotator cuff tear.

Advantages of arthrography include the following:

- Can be used in conjunction with CT to evaluate intra-articular pathology, such as Bankart tears
- Low cost

Disadvantages of arthrography are as follows:

- Size of the tears cannot be quantified
- Patient exposed to radiation
- Contrast dye exposure
- Invasive procedure

Diagnostic arthroscopy

Diagnostic arthroscopy [10] is a minimally invasive visual surgical procedure to assess shoulder pathology. It is able to visualize and assess most shoulder pathology. Diagnostic arthroscopy may give the patient and the physician a chance to diagnose and treat the pathology with one procedure.

Workup for other more systemic processes may be included as clinically indicated.

Treatment

Rehabilitation program

Acute phase

The goals of the acute phase rehabilitation program [21,22] are to relieve pain and inflammation, prevent muscle atrophy, reestablish nonpainful ROM, and normalize arthrokinematics of the shoulder complex. This phase includes a period of active rest, eliminating any activity that may cause an

increase in symptoms. ROM exercises may include pendulum (Codman's) exercises and symptom-limited, active assisted ROM exercises. Joint mobilization may be included with inferior, anterior, and posterior glides in the scapular plane. Strengthening exercises should be isometric in nature, working on the external rotators, internal rotators, biceps, deltoid, and scapular stabilizers (rhomboids, trapezius, serratus anterior, latissimus dorsi, and pectoralis major). Neuromuscular control exercises also may be initiated. Modalities are used as an adjunctive treatment and include cryotherapy, transcutaneous electrical nerve stimulation, high-voltage galvanic stimulation, ultrasound, phonophoresis, or iontophoresis. Patient education is particularly important for this acute phase regarding activity; pathology; and avoiding overhead activity, reaching, and lifting. The general guidelines to progress from this phase are decreased pain or symptoms, increased ROM, painful arc in abduction only, and improved muscular function.

Subacromial shoulder bursa injection. During the acute-to-subacute phase, when pain and inflammation are predominant, a subacromial injection may be diagnostic and therapeutic as an adjunct to the rehabilitation program. Injection of 10 mL of a 1% lidocaine solution without epinephrine into the subacromial space should relieve symptoms if the pain truly is originating from subacromial impingement. The addition of a low-dose, intermediate-acting injectable corticosteroid may provide an additional therapeutic anti-inflammatory effect.

The *technique* of subacromial shoulder bursa injection [20] is as follows:

1. Patient sits with arm hanging by side to distract humerus from acromion.
2. Identify lateral edge of acromion.
3. Insert needle at midpoint of acromion and angle slightly upward under acromion; the depth of needle entry may be confirmed by gently contacting the undersurface of the acromion, which usually places the needle tip right in the supraspinatus (subacromial) outlet space.
4. Slowly withdraw needle, while injecting fluid in a bolus wherever there is no resistance, all the while aspirating before injecting. Sometimes a swelling caused by the fluid is visible around the edge of the acromion. Occasionally, calcification occurs within the bursa, and hard resistance is encountered. In this case, aspiration and infiltration with a large-bore needle and local anesthetic may be helpful. Failing this, surgical evaluation may be necessary.

Aftercare concerns include the following:

Patient is informed that when the effect of the lidocaine wears off, there may be a local reaction to the corticosteroid in the next 24 to 72 hours, in which case the patient is to apply ice (wrapped in a towel) on the affected shoulder 20 minutes on and 20 minutes off, three times in the morning and three times at the end of the day.

Relief of pain after one injection is usual, but the patient must be advised to maintain correct posture with retraction and depression of the shoulder and to avoid the painful arc of elevation for 1 week.

Patient may resume a symptom-limited therapy program in the first week postinjection, then resume the full course thereafter.

Although uncommon with this injection procedure when done correctly, *adverse effects* may occur. The clinician and the patient must be educated about them, and the clinician must know how to manage any related complications.

The following need to be considered in regard to adverse effects of injection:

- Absolute contraindication—documented allergy to any corticosteroid or local anesthetics
- Relative contra-indications—diabetes, hypertension, immunosuppression, cardiac arrhythmias, heart blocks
- Adverse effects of medications—may be minimized when the medication is dosed as recommended

Side effects of injectable corticosteroids are as follows:

- Systemic effects—flushing, menstrual irregularity, impaired glucose tolerance, osteoporosis, psychological disturbance, steroid arthropathy, steroid myopathy, immunosuppression
- Local effects—postinjection flare

Side effects of local anesthetics usually are due to overdose and allergic reactions, which can be minimized by checking the dose before administering and inquiring about and checking records for medication allergies. Effects of overdose and allergic reactions may be catastrophic and may include cardiac, respiratory, and cerebral compromise.

Occasionally a patient may experience a vasovagal reaction (fainting) as an adverse reaction to the injection, owing to pain, apprehension, or needle phobia. The treatment for this situation is to lay the patient down in the supine position, elevate the legs, and reassure the patient strongly that he or she will recover shortly. If the patient loses consciousness briefly, the clinician needs to protect the airway and give oxygen at 35% concentration.

Recovery phase

The initial goals of the recovery phase of rehabilitation [21,22] are to normalize ROM and shoulder arthrokinematics, perform symptom-free activities of daily living, and improve neuromuscular control and muscle strength. ROM exercises should be progressed to active exercises in all planes and self-stretches concentrating on the joint capsule, especially posteriorly. Strengthening should include isotonic dumbbell resistance exercises for the supraspinatus internal rotators, external rotators, prone

extension, horizontal abduction, forward flexion to 90°, upright abduction to 90°, shoulder shrugs, rows, push-ups, and pull-downs to strengthen the scapular stabilizers. Joint mobilization and neuromuscular re-education also should be maintained. Upper extremity ergometry exercises for endurance, trunk exercises, and general cardiovascular conditioning should be maintained. Use of modalities may be continued if necessary. Guidelines to advance are full nonpainful ROM and manual muscle testing results of strength 70% of the contralateral side.

The final goal of this phase is to get the athlete back to throwing. This phase should include improving strength, power and endurance, and sports-specific neuromuscular control. Emphasis is placed on high-speed, high-energy strengthening exercises and eccentric exercises in diagonal patterns. Isotonic strengthening is continued with increased resistance in all planes, and resistance is allowed in the throwing position—90° of abduction and 90° of external and internal rotation. Plyometrics, sports-specific exercises, proprioceptive neuromuscular facilitation, and isokinetic exercises are initiated.

Maintenance phase

The goal of the maintenance phase of rehabilitation [21,22] is to maintain a high level of training and prevent repeat injury. Emphasis is placed on longer, more intense workouts; proper arthrokinematics; and analysis and modification of techniques and mechanics that may re-exacerbate symptoms. Refinements are made in intensity and coordination training. Patient education is re-emphasized. The patient also should exhibit performance of a home exercise program with the proper warm-up, stretching, and strengthening techniques and good understanding of the warning signs of early impingement.

Nonathletic rehabilitation

Although the available literature concentrates on evaluation and treatment of rotator cuff impingement syndrome in athletes, not all patients are athletes or athletic. How do clinicians evaluate and treat rotator cuff impingement syndrome in nonathletes?

Shoulder evaluation of a nonathlete should be as complete and comprehensive as evaluation of the athlete, as delineated earlier in this article. The goals of treatment and rehabilitation are different, however. As clinicians treat athletes, they must view their occupation as the sport they play. As such, clinicians must aim to return athletes to their previous activity level in competitive sports. In the case of nonathletes, the aim also should be returning them to their previous activity level, whether it be activities of daily living or activities specific to their occupations. The acute phase of treatment is unchanged, but the recovery and maintenance phases should include activities more specific to the patient's daily activities (ie, activities related to daily living, household activities, occupational and job-specific activities, and recreational activities). During the acute and recovery phase, the rehabilitation should be

under the supervision of a physical therapist trained specifically in shoulder rehabilitation. As the patient advances into the maintenance phase, he or she usually is educated on techniques to prevent repeat injury, observing proper shoulder biomechanics, activity modification, and continuing a consistent home exercise program of stretching and strengthening.

Surgical intervention

In general, conservative treatments continue for 3 to 6 months. If the patient continues to improve, which is usually the case in 60% to 90% of patients, conservative measures should be continued even beyond the 3- to 6-month time frame [10]. If the patient remains significantly disabled and has no improvement after 3 months of conservative treatment, the clinician must seek further diagnostic workup, reconsider other etiologies, or refer for surgical evaluation. Appropriate surgical referrals are patients with subacromial impingement syndrome that has been refractory to 3 to 6 months of appropriate conservative treatment. Surgery may be particularly beneficial in patients with full unrestricted passive ROM, patients with positive response to injection of lidocaine into the subacromial space, patients with a type III acromion having a large subacromial spur, and patients in whom changes are noted in the rotator cuff tendon on MRI [10].

Discussion of specific surgical techniques and procedures is beyond the scope of this article. Some procedures may include evaluation under anesthesia, arthroscopic evaluation of intra-articular pathology, acromioplasty and subacromial decompression, and rotator cuff tendon debridement or repair.

Postoperative care

Postoperatively an x-ray (supraspinatus outlet view) should be obtained to document the adequacy of the subacromial decompression. The appearance on this radiographic view should be of a type I acromial arch without any residual spurring [10].

After subacromial decompression, the patient is placed in a sling but is encouraged to remove the sling when comfortable and begin active and passive ROM exercises [10]. When pain has decreased significantly and ROM has returned to normal, a program of strengthening similar to the previously mentioned conservative management is instituted. Patients cannot begin sports-specific activities until they have full active ROM in the operated shoulder and normal strength, generally a time course of around 3 to 4 months [10].

Surgical outcome

The following are considerations relevant to surgical outcome:

Subacromial decompression results are generally poor in young, high-performance overhand athletes [10].

Results generally are good for properly selected, middle-aged patients with evidence of impingement on history and physical examination and at the time of arthroscopy [10].

General consensus in the literature is that arthroscopic subacromial decompression results in a good return to the previous level of function in approximately 85% to 90% of patients [10].

Medications

During the acute-to-subacute phases of shoulder impingement syndrome, it is appropriate to use a short course of a nonsteroidal anti-inflammatory drug for its analgesic and anti-inflammatory effects, as an adjunct to the therapy program and other treatment modalities. Every patient's response to a particular nonsteroidal anti-inflammatory drug is different, and a trial of various anti-inflammatory medications is worthwhile to optimize effectiveness.

Follow-up

Return to play

Return to play is restricted until full, painless ROM is restored, rest-related pain and activity-related pain are eliminated, and provocative impingement signs are negative. Strength testing isokinetically must be 90% compared with the contralateral side. Activities are resumed gradually, and the patient must remain symptom-free during the progression to more advanced activities. Flexibility and strengthening exercises must be continued after return to sport to prevent recurrence.

Complications

If shoulder impingement syndrome is not diagnosed and treated promptly and correctly, it can progress to rotator cuff degeneration and eventual tear. Other complications include progression to adhesive capsulitis, cuff tear arthropathy, and reflex sympathetic dystrophy. Other complications may result from surgery, injection, physical therapy, or medication.

Prevention

Primary prevention should be considered an integral part of the treatment of impingement syndrome. Education directed toward patients at risk can do much to circumvent the development of impingement syndrome. Athletes, particularly those involved in throwing and overhead sports, and laborers with repetitive shoulder stress should be instructed in proper warm-up techniques, specific strengthening techniques, and warning signs of early impingement.

Prognosis

In general, the prognosis for patients with prompt, correct diagnosis and treatment of impingement syndrome is good. Of patients, 60% to 90% improve and are symptom-free with conservative treatment. Surgical outcomes also are promising for patients with this syndrome.

Education

Patients should be educated regarding the avoidance of provocative activities and maintenance of proper shoulder arthrokinematics. Education also should stress the performance of an appropriate preventive exercise program, including proper warm-up techniques, specific stretching and strengthening maneuvers, and activity-specific or sport-specific training. Patients also should be instructed to be aware of the potential warning signs of early impingement.

Summary

Shoulder rotator cuff impingement syndrome is treatable—nonoperatively or operatively—if prompt diagnosis and treatment are undertaken. Proper evaluation includes a good history, physical examination, and appropriate diagnostic testing. The main goal in treatment is to return patients to their previous activity level, and the prognosis usually is good. Treatment usually is successful with nonoperative means, but if necessary operative intervention is successful with correct patient selection.

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