CHAPTER 5

Shoulder Trauma and Hypomobility

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INTRODUCTION

Optimal functioning of the shoulder and arm can take place only if the delicate balance of healthy anatomy, proper biomechanics, and normal physiology is maintained. Disruption of any one of these three factors may lead to overload and injury, resulting in pain and dysfunction. A vast amount of information regarding the pathoanatomy and biomechanics of the shoulder has contributed to the clinician's ability to understand and manage patients with shoulder complaints more accurately and effectively.[1–11] Surgical techniques grounded in basic scientific principles related to the restoration of normal anatomy, biomechanics, and the physiology of injured or compromised structures have changed to allow for earlier introduction of rehabilitation. Keeping pace with changes in surgical intervention, rehabilitation programs are now based upon principles derived from pathophysiology, functional anatomy, and restoration of the entire kinetic chain from the lower extremity through the trunk, shoulder girdle, and arm.

Proper rehabilitation of the injured shoulder should be based on knowledge of the normal anatomy and function of the entire shoulder girdle complex and the inherent demands and potential mechanisms that may contribute to or cause injury to this region. Goals of simply increasing range of motion (ROM) and/or improving strength at the glenohumeral joint are no longer acceptable; rather, clinicians must be mindful of how the glenohumeral and scapulothoracic complex functions as part of the entire kinetic chain and understand the subtle relationships among all these interrelated components. Rehabilitation programs prescribed for the treatment of shoulder conditions should be centered on **principles of treatment** as well as specific condition exercise protocols, and these principles should be coupled with the clinician's knowledge of the shoulder, particularly how it functions under both normal and abnormal circumstances. Hopefully, advances in management of the injured shoulder are reflected in improved and earlier return to functional status for patients.

An advantage of managing the injured shoulder according to general treatment principles, rather than specific protocols, is the adaptability of these principles to many different situations. In other words, patients with different shoulder pathologies can benefit from the application of some or all of these treatment principles as they share the same goals of "normalizing" shoulder anatomy, biomechanics, and physiology.

Principles of Shoulder Treatment

- Proper and thorough assessment
- Early protected motion after a period of immobilization
- Proper evaluation and treatment of local and distant deficits
- Scapular control
- Pain management
- Sensorimotor control
- Progressions
- Closed chain axial loading exercise
- Functional exercises

Common Principles of Treatment

1. ***Proper and Thorough Assessment:*** As with any musculoskeletal pathology, treatment of the patient with a shoulder injury must begin with a thorough history and physical assessment. Assessment should include an evaluation of all shoulder girdle tissue as well as the cervical and thoracic spine regions. Depending on the patient and his or her shoulder condition, the lumbar spine and lower extremity may be important to also include in the assessment. Patients should be examined statically and dynamically and while they are performing functional activities.
2. ***Protected Motion After a Period of Immobilization:*** The shoulder complex, particularly the glenohumeral joint, is prone to stiffness after trauma or immobilization.[12,13] Early intervention through gentle ROM exercises and joint mobilization techniques may prevent this restriction of ROM, reduce the associated pain, and allow the shoulder to move into positions that can best address normalization of muscle activity and function. Special consideration must always be

given to surgically repaired tissue as well as the adaptive tightening of the glenohumeral posterior capsule, which presents clinically as a medial rotation deficit (i.e., glenohumeral internal rotation deficit [GIRD]), and finally the anterior shoulder muscles (e.g., pectoralis minor), which also tend to shorten.

3. ***Evaluation and Treatment of Local and Distal Deficits:*** Many shoulder injuries are associated with local and/or distal deficits in flexibility, strength, muscle balance, and proper mechanics that affect the whole kinetic chain. For example, hip and lumbar spine inflexibility, altered scapulohumeral rhythm, and reduced medial rotation motion may be part of the cause of the shoulder injury or may occur as a result of the injury. Regardless of the onset, all deficits of the kinetic chain must be noted on assessment and incorporated into a proper treatment plan.
4. ***Scapular Control:*** Scapular dyskinesis, an alteration in the position or motion of the scapula, is caused by weakness or inhibition of the periscapular muscles, especially the lower trapezius and serratus anterior.[14] The resultant loss of scapular control, coupled with an often overactive upper trapezius, can contribute to shoulder impingement and instability syndromes, acromioclavicular (AC) dysfunction, and rotator cuff weakness.[14–17] Proper shoulder rehabilitation must always include a thorough examination of the scapula and correction of any deficits.
5. ***Pain Management:*** Pain is a powerful inhibitor of muscle activation throughout the body. This is especially true at the shoulder because of the degree of muscle activity required to coordinate joint motion, stability, and function. Clinicians must closely monitor and manage pain, making certain that it is not causing inhibition of the weaker, targeted muscles and encouraging perpetuation of an improper muscle recruitment pattern.
6. ***Sensorimotor Control:*** Sensorimotor control is the neuromuscular control basis of sensory input, often known as proprioception. With the increased knowledge about the effect of changes in proprioception and neuromuscular control of joint stabilization in the shoulder, exercises that address retraining of this important factor have been integrated into treatment approaches.[16,18]
7. ***Progressions:*** Indications for progressing a patient to more challenging exercises are based in part on physiological principles of tissue healing and tissue response to injury. Decisions should also be made through the evaluation of the patient's ability to perform key functions at the shoulder girdle (i.e., scapular stabilization) and the entire kinetic chain; these include adequate hip and trunk extension, normal pelvic control, proper scapular control (especially into retraction and depression), normal scapulohumeral rhythm, and glenohumeral joint ROM.
8. ***Closed Chain Axial Loading Exercises:*** Closed chain rehabilitation exercises link the trunk to the scapula and the scapula to the arm; this allows strengthening of the scapular and arm musculature as a functional unit. These exercises should be performed along with early postoperative ROM techniques because they minimize the shear effect and reduce the open kinetic chain loading applied to the glenohumeral joint.[19–22]
9. ***Functional Exercises:*** Rehabilitation of the injured shoulder should always include exercises that are considered functional. However, clinicians must keep in mind that, for an exercise to be considered functional, it must reflect the individual patient's functional activities and goals for work, daily life, and recreation.

These nine shoulder rehabilitation principles form the foundation of treatment for most shoulder pathologies managed conservatively. Greater detail on specific treatments and exercise guidelines are presented throughout the remainder of this chapter and in Chapters 6 and 7.

SHOULDER ANATOMY

When considering the shoulder, the clinician must include the entire shoulder girdle, consisting of three bones (scapula, clavicle, and humerus) linked to each other and to the body by four joints (glenohumeral, AC, sternoclavicular, and scapulothoracic articulation). The combined effect of these four articulations is a high degree of mobility, which not only allows the arm and hand great functional capacity but also makes the shoulder particularly vulnerable to injury because stability is sacrificed for mobility. The glenohumeral joint has an almost global ROM due to the shallow glenoid socket, which is approximately one third to one fourth the size of the humeral head. The glenoid labrum, attached tightly to the bottom half of the glenoid and loosely to the top half, increases the glenoid depth approximately 2 times, adding to glenohumeral stability.[3,4] Normally when the humeral head is moved through its large ranges of motion, only a small amount of translation or excursion occurs between the humeral head and the glenoid. If the dynamic structures controlling this translation (primarily the rotator cuff) are injured, translation may increase, leading to increased wear on the glenoid labrum, failure of the static restraints, and eccentric overload of the dynamic restraints, resulting in instability, impingement, or both.[2,4,6,7,23–25]

The restraints of the glenohumeral joint typically are classified as either active (dynamic) or passive (static). Table 5-1 presents a list of the two groups. It is worth noting that even though the stabilizing components are divided into two groups, the actual mechanism of stability is not achieved through distinctive, separate processes but rather by one that is highly interconnected.

TABLE 5-1

Restraints about the Glenohumeral Joint

Passive (Static)	Active (Dynamic)	
Capsule Labrum Coracohumeral ligament Superior glenohumeral ligament	Supraspinatus Infraspinatus Subscapularis Teres minor	Humeral stabilizers
Middle glenohumeral ligament Inferior glenohumeral ligament Geometry of humeral articular surface Geometry of glenoid articular surface Coracoacromial ligament Articular cartilage compliance	Pectoralis major Latissimus dorsi Long head of the biceps Triceps Deltoid Teres major	Movers of glenohumeral joint
Joint cohesion	Serratus anterior Latissimus dorsi Trapezius Rhomboids Levator scapulae Pectoralis minor	Scapular stabilizers

Modified from Zachazewski JE, Magee DJ, Quillen WS, editors: *Athletic injuries and rehabilitation,* p 510, Philadelphia, 1996, WB Saunders.

SHOULDER FUNCTION

The shoulder is the most proximal link of the upper extremity kinetic chain. It has evolved to allow humans great mobility so that they can position the most distal segment, the hand, for function. This seemingly simple task is achieved through a highly coordinated pattern of movement that begins in the lower extremities and moves sequentially to the trunk, scapulothoracic joint, glenohumeral joint, and elbow and eventually to the wrist and hand, where the function is performed.

Specific shoulder girdle function relies on the combined motion of the sternoclavicular, AC, glenohumeral, and scapulothoracic joints. This collective motion is achieved through the interaction of the muscles that influence the movement of the scapula, clavicle, and humerus relative to the axial skeleton. Research (in vivo and in vitro) has been done to try to elucidate the complex mechanism by which the shoulder moves and muscles act to facilitate these movements.[26,27] Early radiographic studies of arm elevation identified a 2:1 ratio of glenohumeral to scapulothoracic motion when the humerus is elevated in the coronal plane (Figure 5-1).[28] A similar analysis of elevation in the scapular plane found a ratio of 5:4 after the first 30° of elevation.[29,30] Similar studies carried out by other researchers found slight variations of these earlier observations.[31]

Elevation in a 30° to 40° anterior to the coronal plane customarily is referred to as *scapular plane elevation* or *scaption*. However, the scapula is a highly mobile reference point that not only moves with arm elevation but also inclines forward early in motion, obviously in a different direction from that of the intended elevation. Relative motion between the scapulothoracic joint and the glenohumeral joint has been referred to as the *scapulohumeral* or *glenohumeral rhythm*. The ratios of 2:1 and 5:4 are commonly accepted as representing the ratio of glenohumeral to scapulothoracic motion, but these values are only a limited reflection of the scapulothoracic and scapulohumeral combinations possible to achieve a given arm position. These ratios are based on two-dimensional radiographic projections of angular rotations taken at discrete positions of elevation; however, the arm moves in three dimensions, and for positions other than elevation, much of this motion is under voluntary control. These findings and others are not intended to dismiss completely all of our earlier understanding of scapulohumeral motion but rather to broaden awareness of the dynamic, multiplane model of the shoulder.

Muscle function at the shoulder is considered one of the highly intricate systems in the body. The function of a muscle with respect to any joint depends on the position of the skeletal components, distance of the muscle from the joint's center of rotation, and external and

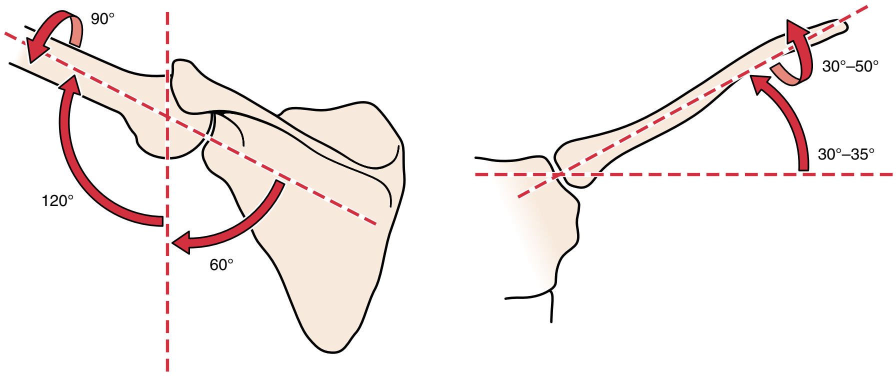

Figure 5-1 Movements of the scapula, humerus, and clavicle during scapulohumeral rhythm. (From Magee DJ: *Orthopedic physical assessment*, ed 6, p 274, St Louis, 2014, Saunders/Elsevier.)

internal forces acting at the time of muscle contraction. Given the three-dimensional, highly mobile nature of the shoulder joint, it is easy to understand why proper functioning of its muscles is essential for a normal, healthy shoulder.

The prime movers of the shoulder are the deltoid, pectoralis major, latissimus dorsi, and teres major muscles. Electromyographic studies[26,32–34] have shown that these muscles function predictably along their line of pull and that they form a drapelike effect over the shoulder, creating the potential for infinite lines of pull that may allow an almost 360° arc of motion.

The rotator cuff contributes to shoulder motion through a variety of means. The muscles of the rotator cuff can function as important movers of the upper extremity if the muscles' line of action is consistent with the intended direction of motion. This group of muscles also serves as a key set of stabilizing muscles for the glenohumeral joint by creating a compressive force that maintains the humeral head on the glenoid surface, and it can function to produce axial rotation of the humerus.

Finally, muscles that attach to the scapula and therefore control scapulothoracic joint motion have been decisively reported as playing a fundamental role in how the shoulder functions or, more appropriately, how *well* the shoulder functions. The work of researchers, such as Kibler[15] and Kibler and Livingstone,[21] has taught clinicians to be mindful of the trapezius, rhomboids, levator scapulae, teres minor, serratus anterior, and latissimus dorsi muscles because their attachments to the scapula allow for control of motion at the scapulothoracic articulation. Dysfunction or inhibition of any of these muscles has been shown to alter the position of the glenoid significantly, which in turn may result in abnormal centering of the humeral head within the glenohumeral joint.[6,15,35] The importance of proper scapulothoracic positioning at rest and during movement cannot be overemphasized, and clinicians must be cognizant of dysfunction at this region of the shoulder girdle.

STERNOCLAVICULAR AND ACROMIOCLAVICULAR JOINT INJURIES

Sternoclavicular (SC) Joint

The sternoclavicular joint is unique because it is the only true articulation between the clavicle of the upper extremity and axial skeleton. It is a diarthrodial type of joint, with the enlarged medial end of the clavicle creating a saddle-type articulation with the clavicular notch of the sternum. The clavicle is the first long bone in the body to ossify. The epiphysis at the medial end of the clavicle, however, is the last long bone to appear and is the last epiphysis to close at approximately 18 to 20 years of age. The epiphysis does not fuse with the shaft of the clavicle until 23 to 25 years of age; therefore the growth plate is the weakest point and more likely to sustain a displaced physeal fracture than a true dislocation.[36] This joint has the distinction of having the least amount of bony stability; therefore it relies heavily on support from the surrounding capsule and ligaments. These ligaments include the intra-articular disc ligament, extraarticular costoclavicular (rhomboid) ligament, interclavicular ligament, and capsular ligament.

The intra-articular disc ligament is a very dense, fibrous tissue that originates from the synchondral junction of the first rib to the sternum and travels through the sternoclavicular joint, dividing it into two distinct joint spaces (Figure 5-2). Anteriorly and posteriorly, the disc blends with the fibers of the capsular ligament. The intra-articular disc ligament functions as a checkrein for medial displacement of the proximal clavicle.

The costoclavicular, or rhomboid, ligament is short and strong and is made up of an anterior fasciculus and a posterior fasciculus. These two different parts give the ligament a slightly twisted appearance. Below, the costoclavicular ligament attaches to the upper surface of the first rib and at the adjacent part of the synchondral junction

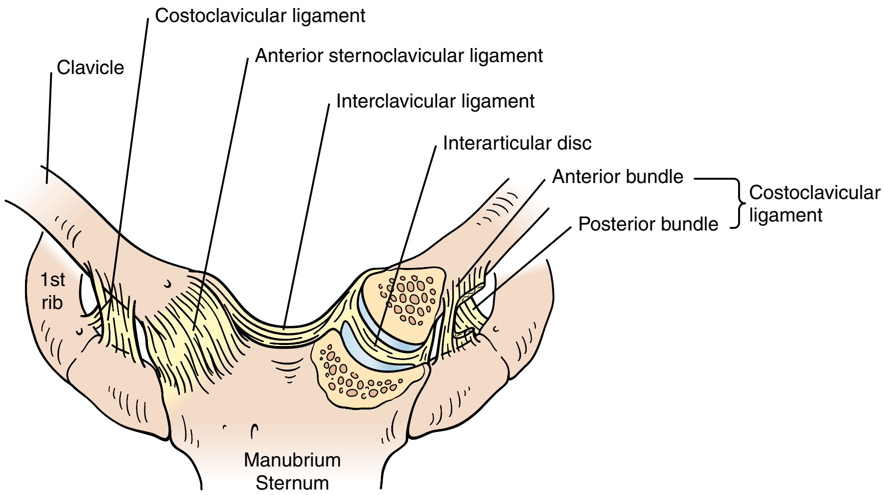

Figure 5-2 Sternoclavicular joint.

with the sternum; above, it attaches to the margins of the impression on the inferior surface of the medial end of the clavicle, sometimes called the *rhomboid fossa*.[37,38] Bearn[39] has shown that the anterior fibers resist excessive upward rotation of the clavicle and the posterior fibers resist excessive downward rotation. Specifically the anterior fibers resist lateral displacement, and the posterior fibers resist medial displacement.

The interclavicular ligament connects the superomedial aspects of each clavicle with the capsular ligaments and the upper aspect of the sternum (see Figure 5-2). Along with the capsular ligaments, the interclavicular ligament helps to hold up the shoulder. To demonstrate this, the clinician can place a finger in the superior sternal notch; the ligament is lax with elevation of the arm but becomes taut when both arms are left to hang at the sides.

The capsular ligament of the sternoclavicular joint, which extends over the anterosuperior and posterior aspects of the joint, represents thickenings of the joint capsule. The posterior capsule is the most important restraint for anterior and posterior translation of the sternoclavicular joint, whereas the anterior capsule acts mostly to prevent anterior translation. These capsular ligaments are considered the most important and strongest ligaments of the sternoclavicular joint and the structures most responsible for preventing upward displacement of the medial clavicle in the presence of a downward force on the distal end of the shoulder.[39]

Function of the Sternoclavicular Joint

The sternoclavicular joint is the most frequently moved, nonaxial joint in the body.[40] It acts similar to a ball and socket joint, allowing for motion in almost all planes, including rotation. In normal shoulder motion, the joint accounts for 30° to 35° of upward elevation, 35° of combined forward and backward movement, and 45° to 50° of rotation about the long axis of the clavicle and sternoclavicular joint.[28,41,42] It is important that clinicians appreciate that almost all motions of the upper extremity impact the sternoclavicular joint and conversely that motion at the sternoclavicular joint influences movement distally, along the upper extremity kinetic chain.

Sternoclavicular Joint Dislocations

Dislocations of the sternoclavicular joint are quite rare, accounting for approximately 3% of all fractures and dislocations affecting the shoulder girdle.[43,44] This is mostly due to the strength and integrity provided by the joint's surrounding capsuloligamentous tissue. Anterior dislocations are 2 to 3 times more common than posterior dislocations, fortunately, because posterior dislocations have a high complication rate with potentially life-threatening consequences (Figure 5-3).[36,38,45] The amount of force required to dislocate the sternoclavicular joint posteriorly is more than 1.5 times the force required for anterior dislocation. In addition, a posterior dislocation may put pressure on the underlying mediastinal structures, including the brachial plexus, vascular structures, trachea, and esophagus (Figure 5-4).[46,47] Complications include brachial plexus lesions, pneumothorax and respiratory distress, vascular injury, dysphagia, and hoarseness.[48,49] Motor vehicle accidents are reported as the most common cause of dislocation of the sternoclavicular joint, followed by direct or indirect trauma during a sporting activity. An example of a direct trauma in a sporting activity might be a hockey player hitting the medial side of the clavicle on the goalpost or on another player's knee. With an indirect injury, the athlete may be lying on his side, and the uppermost shoulder is compressed and rolled backward, resulting in an anterior sternoclavicular dislocation on that side. If the shoulder rolls forward and is compressed, a posterior dislocation is more likely.[45,50]

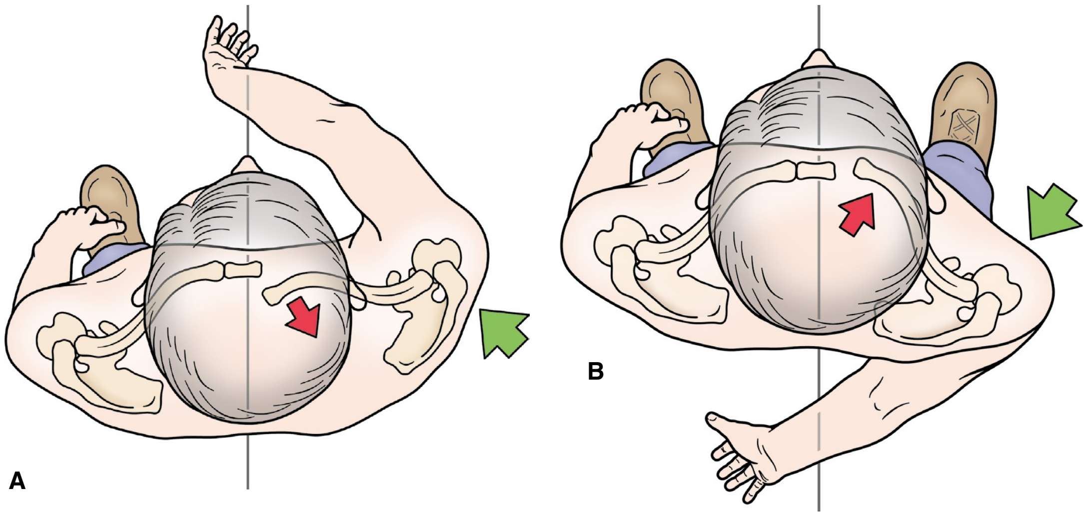

Figure 5-3 Mechanism of injury to the sternoclavicular joint. **A**, Posterior applied force (*green arrow*) directed anteriorly causes proximal end of clavicle to move posteriorly (*red arrow*). **B**, Anterior applied force (*green arrow*) directed posteriorly causes proximal end of clavicle to move anteriorly (*red arrow*). (Redrawn from Emery R: Acromioclavicular and sternoclavicular joints. In Copland SA, editor: *Shoulder surgery,* Philadelphia, 1997, WB Saunders; and Rockwood CA, Green DP, editors: *Fractures,* vol 1, ed 2, Philadelphia, 1984, JB Lippincott.)

Sternoclavicular dislocations are classified according to patient age, severity, and direction of dislocation. Mild sternoclavicular joint injuries are those in which the joint is stable and the ligamentous integrity has been maintained. Moderate injuries have had part of the ligamentous tissue disrupted, and the sternoclavicular joint has partially separated or subluxed. Complete ligamentous disruption, resulting in an unstable sternoclavicular joint, is considered a severe injury. The degree of separation can also be classified as first, second, or third, but this classification is based more on injury to the ligaments supporting the joint rather than injury to the joint itself. A first-degree sternoclavicular dislocation results in minor tearing (first- or second-degree sprain) of the sternoclavicular and costoclavicular ligaments, with no true displacement of the joint. A complete tear (third-degree sprain) in the sternoclavicular ligaments and a second-degree sprain of the costoclavicular ligament constitute a second-degree sternoclavicular dislocation and actually result in subluxation of the joint. A third-degree sternoclavicular dislocation is a true dislocation of the joint, caused by third-degree sprains in the sternoclavicular and costoclavicular ligaments.[51]

Clinical Presentation of Sternoclavicular Joint Dislocations

Sternoclavicular dislocations are characterized by deformity, ecchymosis, crepitus, and local pain or tenderness, especially with arm movements that roll the shoulders forward and/or lying supine. In the acute situation, clinicians should note any tenting of the skin, which could be indicative of an underlying fracture or dislocation of the sternoclavicular joint. A careful neurological and vascular examination should always be performed because of the joint's proximity to these key structures. Observation of an anterior sternoclavicular dislocation will show a prominent medial clavicle that is more anterior compared with the sternum, whereas a posterior dislocation will reveal a medial clavicle that is less visible and often not palpable. With a posterior dislocation or subluxation, some shortness of breath or even venous congestion in the neck may be seen, with decreased circulation sometimes evident in the arm. Reduction of a posterior sternoclavicular dislocation usually is accomplished by extending the shoulders while using some form of roll or sandbag as a fulcrum along the spine. Reduction most often occurs easily, although in some cases supplemental anesthesia may be required. The practitioner also may need to grasp the clavicle with the fingers or, if the anatomy is less defined, with some type of surgical instrument, such as a towel clip.

Treatment of Sternoclavicular Joint Dislocations

Goals of treatment following a sternoclavicular injury include pain control, reduction and/or immobilization as indicated, identification and management of associated injuries, and education regarding protection and prevention of subsequent injury. If the sternoclavicular joint has incurred only a first- or second-degree sprain and the joint is deemed stable, treatment follows a course similar to that for any other injured joint. The patient's shoulder should be immobilized for 3 to 4 days, as pain dictates, and then should make a gradual return to normal use of the shoulder and arm. Local electrical modalities and the use of ice and heat may be helpful for reducing pain and inflammation at the sternoclavicular joint. The clinician should be aware of the impact of a sternoclavicular injury on the entire shoulder complex because the effects of the initial injury may be far-reaching and can lead to compensatory patterns of movement.

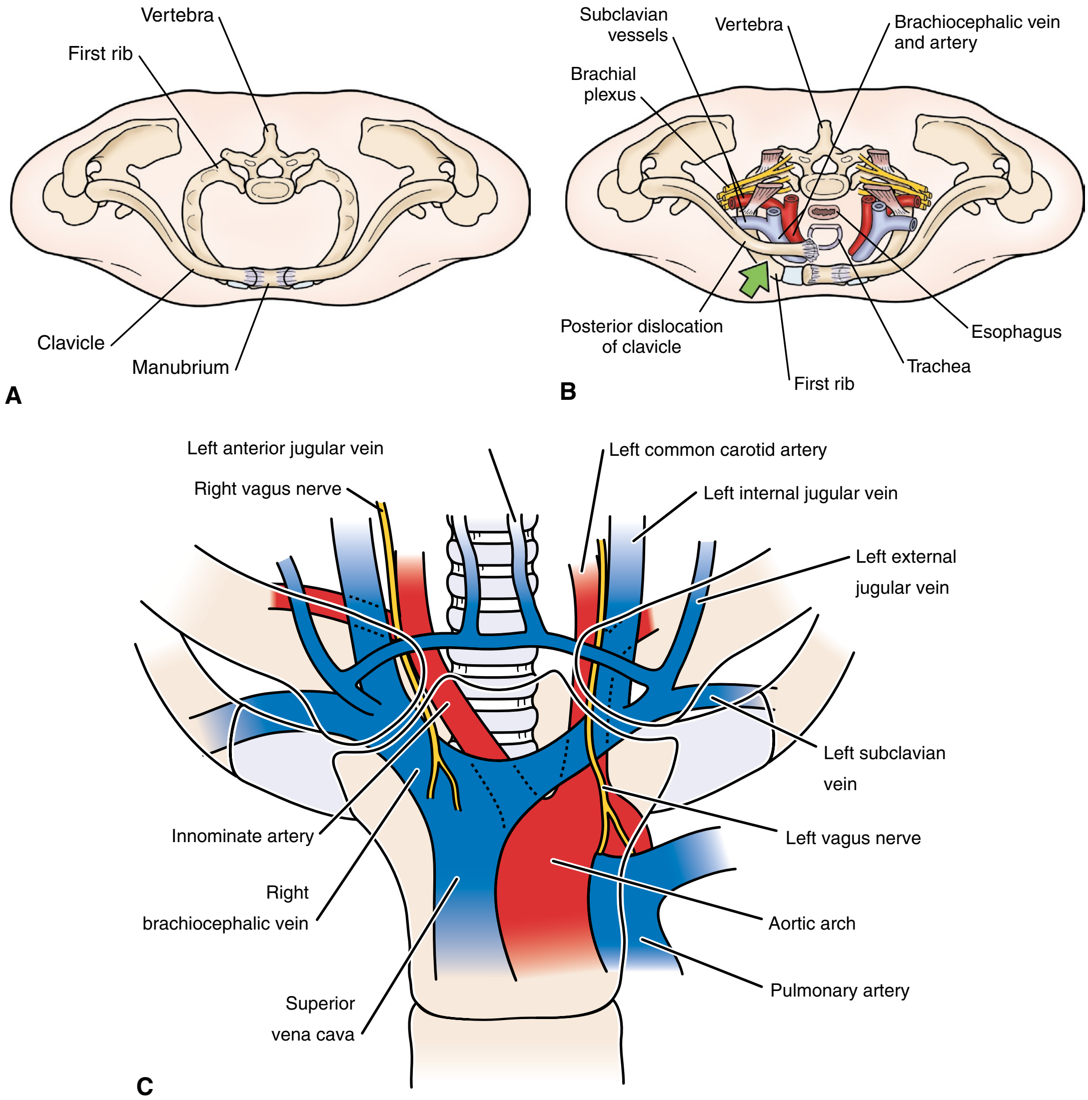

Figure 5-4 Sternoclavicular joint posterior dislocation. **A,** Overhead view of normal anatomical relationships. **B,** Posterior dislocation of the sternoclavicular joint. Note proximity of vessels which could be compressed with dislocation. *Arrow* indicates direction of force. **C**, Anterior view of retrosternal anatomy. Note the proximity of the sternoclavicular joint to the trachea, aortic arch, and brachiocephalic vein. (**C**, From Magee DJ: *Orthopedic physical assessment,* ed 6, p 257, St Louis, 2014, Saunders/Elsevier; redrawn from Higginbotham TO, Kuhn JE: Atraumatic disorders of the sternoclavicular joint, *J Am Acad Orthop Surg* 13:139, 2005.)

In second- or third-degree sprains of the sternoclavicular joint, if the main complaint after reduction is instability, treatment frequently requires sling support for an anterior dislocation or a figure-of-eight bandage for the posterior dislocation. The sling is worn for at least 2 to 3 weeks. Chronic instability may require surgical stabilization of the sternoclavicular joint, which is by no means uniformly successful. Chronic subluxation or damage to the intra-articular disc can produce long-term discomfort with repetitive strong movements of the upper limb.

Complications of anterior sternoclavicular dislocation include cosmetic deformity, recurrent instability, and late osteoarthrosis; complications of posterior dislocation include all of these plus pressure on or rupture of the trachea, pneumothorax, rupture of the esophagus, pressure on the subclavian artery or brachial plexus, voice change, and dysphagia. Although the incidence of one of these complications can be as high as 25%, only about three deaths have been reported in the literature from the more serious complications associated with this injury.[45,51]

Atraumatic anterior subluxation of the sternoclavicular joint during arm elevation is occasionally reported in patients younger than 30 years of age. Many individuals affected by this condition demonstrate signs of generalized ligamentous laxity. Patients report a nontraumatic, spontaneous subluxation of the medial end of the clavicle, occasionally with a pop, that reduces when the arm lowers back down. Most cases are not painful and do not interfere with functional activities; therefore management is conservative with the focus on patient education regarding the anatomical events involved and reassurance that the condition should not alter lifestyle or limit activity (Figure 5-5).[52,53]

Complications of Sternoclavicular Posterior Dislocation

- Cosmetic deformity
- Recurrent instability
- Late osteoarthritis
- Pressure on or rupture of the trachea
- Pneumothorax
- Rupture of the esophagus
- Pressure on the subclavian artery
- Pressure on the brachial plexus
- Voice change
- Dysphagia

Acromioclavicular (AC) Joint

The AC joint is a diarthrodial joint made up of the lateral aspect of the clavicle and the medial margin of the acromion process of the scapula. In conjunction with the sternoclavicular joint, the AC joint provides the upper extremity with a connection to the axial skeleton. The articular surfaces initially are hyaline cartilage until approximately 17 years of age on the acromial side of the joint and approximately 24 years of age on the clavicular side. After this time, the hyaline cartilage acquires the structure of fibrocartilage. The anatomy of the AC joint varies considerably; the articular surface orientation can range from vertical to overriding at an angle of approximately 50°.[45,54–56] The dramatic variation in anatomy may explain why some individuals seem more susceptible to injury at this joint.

Within the AC joint is an intra-articular disc, which may be partial (meniscoid) or complete. This disc can be damaged through injury to the AC joint leading to further degeneration over time. Unstable discs are often implicated as the cause of clicking and recurrent pain at the AC joint.

The AC joint is surrounded by a thin capsule that is reinforced above, below, anteriorly, and posteriorly by the superior, inferior, anterior, and posterior AC ligaments (Figure 5-6). The fibers of the superior AC ligament blend with the fibers of the deltoid and trapezius muscles, which are attached to the superior aspect of the clavicle and the acromion process. These muscle attachments are important as they strengthen the AC ligaments and add stability to the joint.

The coracoclavicular ligament is a very strong, heavy ligament with fibers running from the outer, inferior surface of the clavicle to the base of the coracoid process of the scapula. The ligament consists of two individual ligaments, the conoid and trapezoid, which sometimes have a bursa between them. As the name suggests, the conoid ligament is cone shaped, with the apex of the cone attaching on the posteromedial aspect of the base of the coracoid process and the base attaching to the conoid tubercle on the posterior undersurface of the clavicle. The trapezoid ligament arises from the coracoid process, anterior and lateral to the attachment of the conoid ligament just posterior to the attachment of the pectoralis minor tendon. The ligament travels superiorly to a rough line on the undersurface of the clavicle. The coracoclavicular ligament plays an important role not only in strengthening the AC joint but also in assisting the glenohumeral joint with scapulohumeral motion. As the clavicle rotates upward, it dictates scapulothoracic rotation by virtue of its attachment to the scapula, that is, the conoid and trapezoid ligaments.[55,57]

Function of the Acromioclavicular Joint

Codman[58] described the motion at the AC joint as "swinging a little, rocking a little, twisting a little, sliding a little and acting like a hinge." Subsequent authors[59,60] observed relatively little motion between the clavicle and the acromion during studies involving percutaneous implanted pins in subjects. They described a synchronous, three-dimensional linkage between clavicular rotation and scapular rotation, such that when the clavicle rotates, the scapula

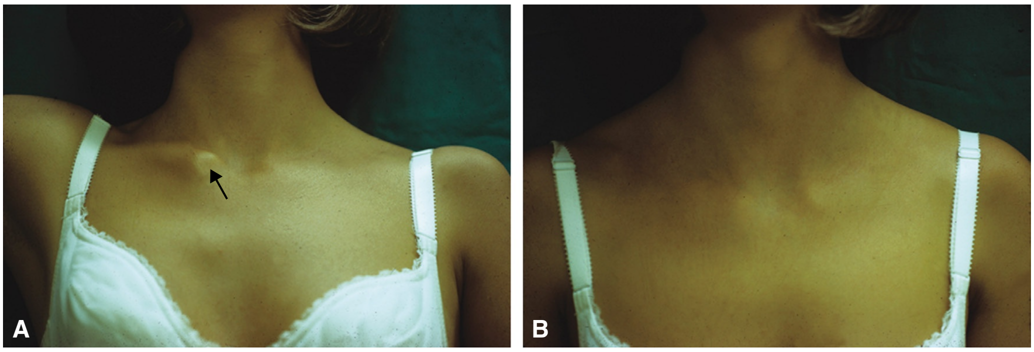

Figure 5-5 Spontaneous subluxation of the sternoclavicular joint. **A,** With the arms in the overhead position, the medial end of the right clavicle spontaneously subluxes out anteriorly (*arrow*) without any trauma. **B,** When the arm is brought back down to the side, the medial end of the clavicle spontaneously reduces. This usually is associated with no significant discomfort. (From Rockwood CA, Matsen FA, Wirth MA, et al, editors: *The shoulder,* ed 4, p 535, Philadelphia, 2009, Saunders.)

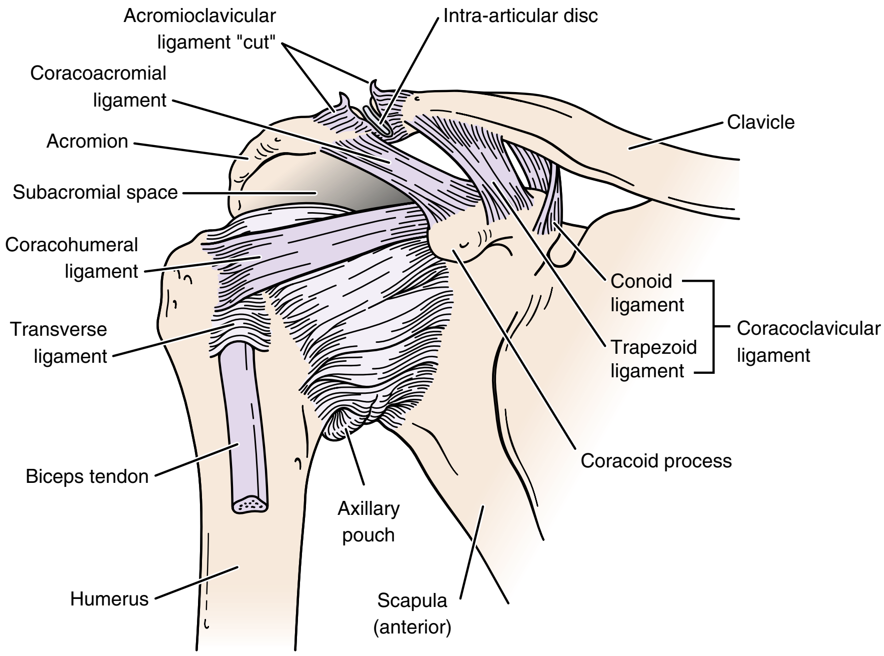

Figure 5-6 Anterior view of the right glenohumeral and acromioclavicular joints. Note the subacromial space or supraspinatus outlet located between the top of the humeral head and the underside of the acromion. The **coracoacromial arch** forming the ceiling of the subacromial space is made up of the undersurfaces of the acromion process, the coracoacromial ligament, and the coracoid process. (From Magee DJ: *Orthopedic physical assessment,* ed 6, p 256, St Louis, 2014, Saunders/Elsevier; modified from Neumann DA: *Kinesiology of the musculoskeletal system: foundations for rehabilitation,* ed 2, St Louis, 2010, Mosby.)

also rotates, concluding that most scapulothoracic motion occurs through the sternoclavicular joint, not the AC joint.

In a classic article published in 1944, Inman[41] suggested that the total ROM of the AC joint was 20° and that clavicular rotation was a fundamental feature of shoulder motion. He noted that motion occurred in the first 30° of abduction and then again after 135° of arm elevation. He also showed that with full elevation, the clavicle rotates upward 40° to 50°. Since these original observations, researchers have disputed the amount of motion that occurs at the AC joint relative to the glenohumeral joint.[54,61,62] Despite the controversy, researchers agree on the important relationship between the two joints and recognize that scapulohumeral rhythm is a delicate balance achieved through a combination of elevation and rotation of the clavicle and proper scapulothoracic and glenohumeral joint motion.

Acromioclavicular Joint Injuries

The AC joint is one of the most frequently injured joints in the body, particularly in sports and activities that require overhead involvement of the shoulder and arm and/or that have a high risk of collision and impact. Injuries to the AC joint result in what is often called a "separated" shoulder, and they account for 9% to 10% or acute injuries to the shoulder girdle in the general population, whereas separations of the AC joint account for 40% of shoulder girdle injuries in athletes.[40,61–64] Common mechanisms of injury include falls on an outstretched hand or elbow (i.e., FOOSH injury), direct blows to the shoulder, or falling onto the point of the shoulder (Figure 5-7).

As with the sternoclavicular joint, AC joint injuries are classified according to ligamentous injury rather than injury to the joint itself. Table 5-2 describes the features and management of the six classifications of AC joint injury (Figures 5-8 and 5-9). Some have suggested that third-degree (type III) disruptions can be further divided according to the magnitude of displacement and the potential accompanying muscle and soft tissue stripping and tearing as well as the direction of displacement of the distal clavicle.[45,59] For instance, displacement of the clavicle of more than 100% is often classified as a fourth-degree (type IV) injury. Type V injuries involve very severe deformity because the distal end of the clavicle is displaced upward a significant degree toward the base of the neck (Figure 5-10). In addition, displacements may occur posteriorly through the trapezius or, in rare cases, inferiorly below the coracoid (type VI). Fourth-degree injuries are associated with considerable disruption of the deltoid and trapezius muscles. A third-degree separation may be treated without surgery, but surgery does have a role in the treatment of fourth-, fifth-, and sixth-degree injuries because of the associated ligamentous disruption and because of cosmetic considerations in leaner individuals (see Figure 5-8).[65–68]

Clinical Presentation of Acromioclavicular Joint Injuries

Patients who have suffered an injury to the AC joint typically present with a history of either a distinctive, traumatic

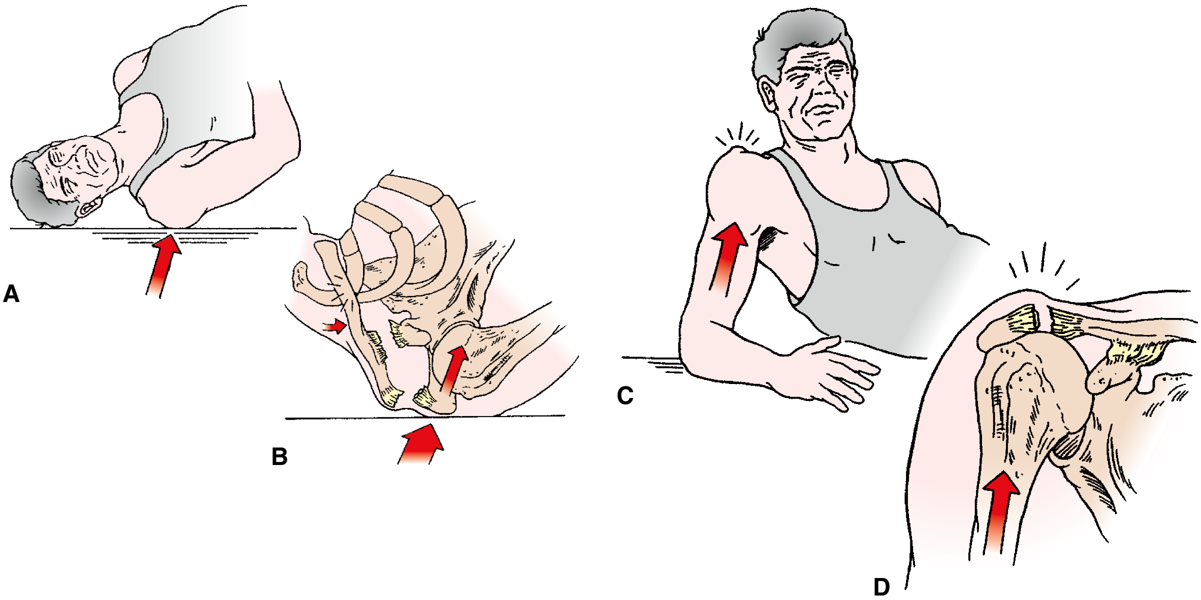

Figure 5-7 Mechanisms of injury to the acromioclavicular joint. **A** and **B,** Direct mechanism of injury. **C** and **D,** Indirect mechanism of injury. (From Field LD, Warren RF: Acromioclavicular joint separations. In Hawkins RJ, Misamore GW, editors: *Shoulder injuries in the athlete: surgical repair and rehabilitation,* p 206, New York, 1996, Churchill Livingstone.)

mechanism of injury or a more insidious type of onset that began with pain and dysfunction. The diagnosis is made in part by assessing the site of local tenderness, the degree of deformity, and whether instability is present. As with the sternoclavicular joint, injury to the AC joint tends to cause localized pain with minimal referral. Stressing the joint with horizontal adduction elicits pain in the injured AC joint, as does loading the joint by applying leverage at the distal arm. A palpable gap (i.e., a step deformity) may be present with the higher degrees of separation (Figure 5-11). With trauma to the AC joint, the clinician must take care to assess for secondary injury to the surrounding soft tissues (e.g., the rotator cuff) and to the other three articulations of the shoulder complex. Long term, most people function well

TABLE 5-2

Classification of Acromioclavicular Joint Trauma

Classification	Salient Features	Management
First degree (type I)	• Minimal structural damage • First-degree sprain of the acromioclavicular ligament • Local tenderness on palpation • Full range of motion (may have pain at extreme) • No loss of structural strength • Normal stress x-ray film	• Re-establish full range of motion and strength
Second degree (type II)	• Subluxation of the acromioclavicular joint • Tearing of the acromioclavicular capsule • Second- or third-degree sprain of the acromioclavicular ligament • First- or second-degree sprain of the coracoclavicular ligament • May affect the deltoid and trapezius muscles • No significant increase in the costoclavicular space on stress x-ray film • Slight widening of the acromioclavicular joint on stress x-ray film • Definite structural weakness • Detectable instability on stress • Palpable gap or step deformity possible • Obvious swelling initially with later ecchymosis	• Requires 6 weeks to heal, although ligaments have good structural strength after approximately 3 weeks • Limb support (sling) • Treatments for pain relief (e.g., ice, transcutaneous electrical nerve stimulation [TENS], interferential therapy) • Therapeutic ultrasound therapy for collagen enhancement • Functional exercises (activities at functional speed control)

TABLE 5-2

Classification of Acromioclavicular Joint Trauma—Cont'd

Classification	Salient Features	Management
Third degree (type III)	• Dislocation of the acromioclavicular joint • Complete disruption of the acromioclavicular joint • Third-degree sprain of the acromioclavicular ligament • Third-degree sprain of the coracoclavicular ligament • Tearing of the deltoid and trapezius muscles from the distal end of the clavicle • Obvious step deformity, often without stress • Increased costoclavicular space on stress x-ray film • Widening of the acromioclavicular joint on stress x-ray film	• May be treated surgically or conservatively • If treated conservatively, deformity remains but athlete will function remarkably well with only slight discomfort or instability at high loads • Conservative treatment as above
Fourth degree (type IV)	• Modifications of type III trauma (rare injuries)	• Require surgical repair
Fifth degree (type V)	• Modifications of type III trauma (rare injuries)	• Require surgical repair
Sixth degree (type VI)	• Modifications of type III trauma (rare injuries)	• Require surgical repair

Modified from Zachazewski JE, Magee DJ, Quillen WS, editors: *Athletic injuries and rehabilitation,* p 519, Philadelphia, 1996, WB Saunders.

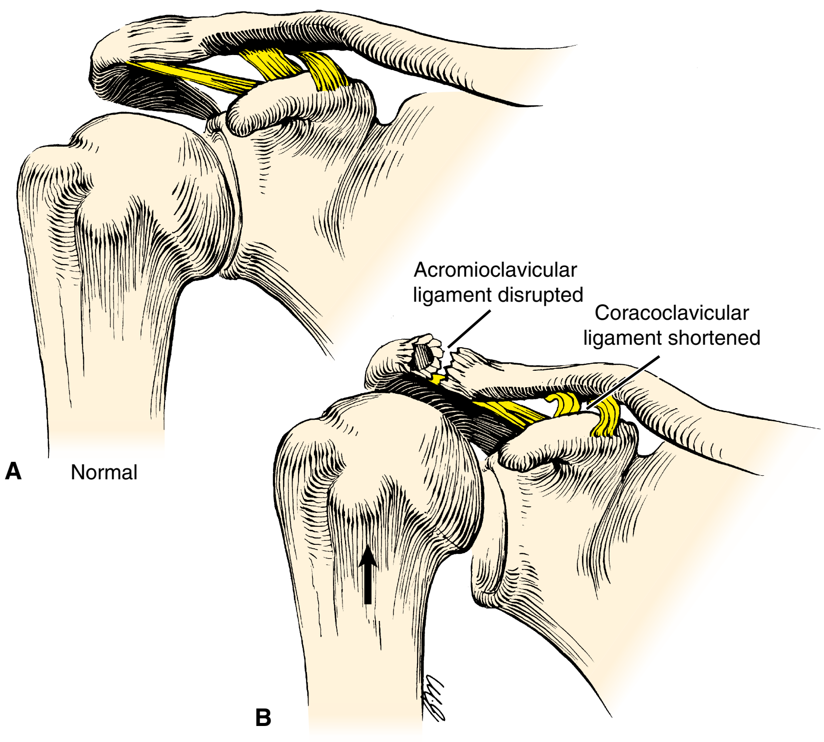

Figure 5-8 Disruption of the acromioclavicular ligament with preservation of the coracoclavicular ligaments. (Redrawn from Rockwood CA, Green DP, editors: *Fractures,* vol 1, ed 2, Philadelphia, 1984, JB Lippincott.)

without surgery, although degenerative changes, including clavicular osteolysis, may be seen in some individuals.

Treatment of Acromioclavicular Joint Injuries

Management of an AC joint injury depends on the type of separation. It is generally agreed upon that higher-grade lesions (i.e., type IV to VI) should be treated surgically, whereas lower-grade lesions (i.e., type I and II) are best managed conservatively. How to best treat type III injuries remains controversial; however, current literature suggests the decision should be made on a case-by-case basis with an emphasis on initial nonoperative treatment.[69,70] The ultimate function of the AC joint depends not so much on the amount of separation as on the amount of pain that persists in the joint. Therefore early conservative treatment for first- and second-degree separations focuses

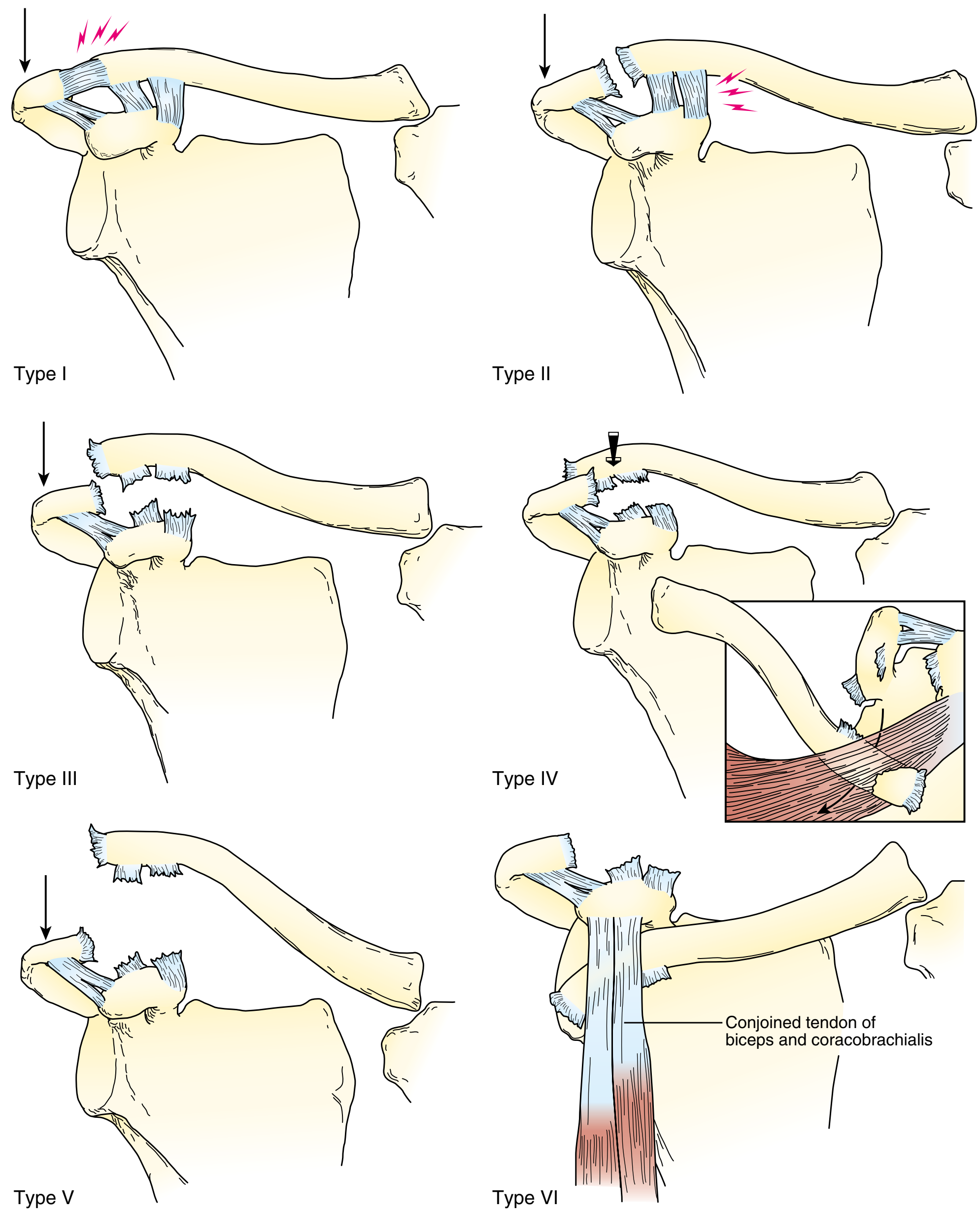

Figure 5-9 Acromioclavicular joint injuries (see Table 5-2). (Rakel RE, Rakel D: *Textbook of family medicine*, ed 9, Philadelphia, 2016, Saunders.)

on pain relief. Initially, the patient's arm may be put in a sling, often accompanied by a swath, to remove the stress of the weight of the limb on the joint and to protect the joint from further damage. This period of immobilization is accompanied by ice and oral analgesic medication, if tolerated. As the pain decreases (within 3 to 7 days), ROM and strengthening exercises, especially for the scapular stabilizers, deltoid, and upper trapezius muscles, are initiated. During this time, heavy lifting and contact sports are avoided to encourage optimal ligament healing. Patients generally return to light-moderate activities 2 to 6 weeks after injury. Return to sport and/or heavy labor is expected following type I and II AC joint separations; however, the timeframe for this is highly dependent upon the individual and his or her specific activity. Associated soft tissue trauma in type II and III injuries typically is more extensive; therefore the time frame is likely to be prolonged for all the suggested treatments.

With third-degree (type III) AC joint injuries, complete reduction is not necessary for satisfactory function; in fact, in terms of strength, endurance, and function, nonsurgical and surgical treatments have been reported to be equally effective.[67–74] Depending on the preference of the surgeon, type III injuries may be treated surgically,

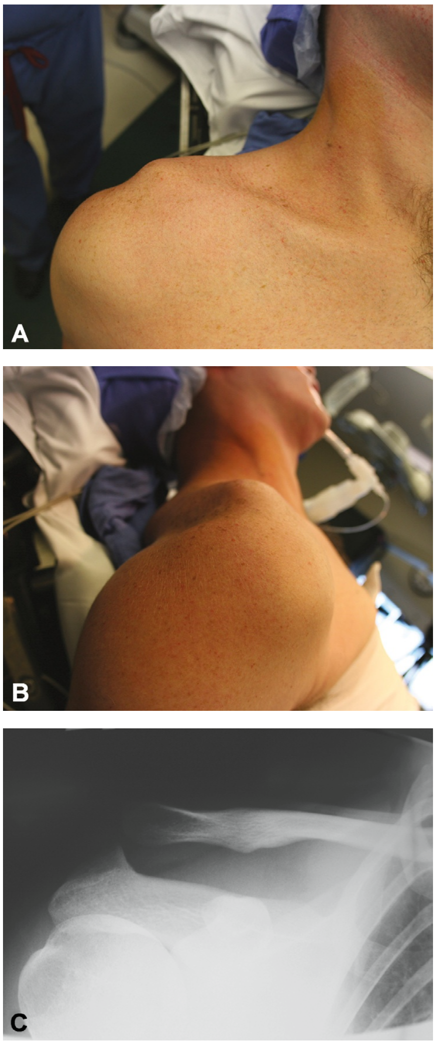

Figure 5-10 Typical findings after a type V AC joint injury, anterior view **(A)** and lateral view **(B)**. There is a twofold to threefold increase in the coracoclavicular distance as well as a stripping of the deltoid and trapezial fascia from the clavicle. This patient was unable to reduce the AC joint with an active shoulder shrug, indicating a true type V injury instead of a type III injury. **C**, Anteroposterior radiographic view demonstrates widening, evidence of a type V injury. (From Miller MD, Thompson SR: *DeLee & Drez's orthopaedic sports medicine: Principles and practices,* ed 3, Philadelphia, 2010, Saunders.)

but the results are seldom better than with conservative treatment, recovery period is longer, and complications of surgery must be considered. For these reasons, most surgeons opt for initial nonoperative treatment, focused on attaining full ROM and scapular stabilization. Surgical consideration is given only to those patients who fail conservative management and present with persistent symptoms and functional limitations.

Types IV, V, and VI require surgical repair to stabilize the joint.

Figure 5-11 Step deformity resulting from acromioclavicular joint trauma (note "step"). (From Magee DJ: *Orthopedic physical assessment,* ed 6, p 265, Philadelphia, 2014, WB Saunders.)

Complications of an AC joint injury include a step deformity produced by the elevated distal end of the clavicle, early skin necrosis if the clavicle is widely displaced in a thin individual, osteoarthrosis, osteolysis, and continuing pain in the long term. For surgical procedures, complications include infection, wound dehiscence, pin migration, and recurrence of the instability, as well as the normal risks of general anesthesia.

FROZEN SHOULDER

The first reported description of frozen shoulder was made by Duplay in the late 1800s.[75] They used the label *scapulohumeral periarthritis* to describe a broad spectrum of pathologies of the shoulder that resulted in pain, stiffness, and dysfunction. This label served as an umbrella term that encompassed disorders such as rotator cuff tendonitis and tears, biceps tendonitis and tears, calcific deposits, AC arthritis, and other painful shoulder syndromes. The term *frozen shoulder* was later coined in 1934 by Codman,[76] who characterized the condition as involving a slow onset, pain near the deltoid insertion, inability to sleep on the affected side, painful and restricted elevation and lateral (external) rotation, and a normal radiological appearance. Even then, Codman described the condition as "difficult to define, difficult to treat and difficult to explain from the point of view of pathology." Subsequent to Codman's observations, Neviaser[77] in 1945 recorded his operative and histological observations from 10 patients with frozen shoulder, noting it was the capsule not the bursa that was thickened and adhered to the humeral head. As a result, he suggested *adhesive capsulitis* as a more descriptive term for shoulders that appeared frozen. More recent investigations have shown that frozen shoulders do not always possess capsular adhesions or, if they do, the adhesions are not always on to the humerus, as first described. Therefore the term *adhesive capsulitis* has been suggested as not appropriate to describe frozen shoulders and should not be used.[78–80]

TABLE 5-3

Types of Secondary Frozen Shoulder

Type	Contributing Causes
Intrinsic	• Calcific tendinosis • Rotator cuff tendinopathy • Biceps tendinopathy • Shoulder surgery
Extrinsic	• Humeral fractures • Clavicular fractures • Cervical radiculopathy • Ipsilateral breast surgery • Chest wall tumor • Cerebrovascular accident
Systemic	• Diabetes • Thyroid abnormalities • Heart disease

Lundberg[81] introduced the terms *primary* and *secondary* to further describe frozen shoulders in 1969. Primary frozen shoulders are those with an idiopathic onset (i.e., no detectable underlying cause), whereas secondary frozen shoulders occur following trauma and/or immobilization. Secondary frozen shoulders have been further classified into intrinsic, extrinsic, and systemic categories by Zuckerman and Rokito[82] and are presented along with contributing causes in Table 5-3.

Despite frozen shoulder being recognized for more than 100 years, there still remains a lack of evidence regarding its etiology, natural history, and best method of management. Clinicians must work closely with their patients with frozen shoulders to tailor treatment plans that best meet the needs of each individual patient, depending on factors such as stage of disease, symptom severity, age, occupation, and expected goals.

Clinical Presentation of Frozen Shoulder

Patients with an idiopathic frozen shoulder tend to progress through three overlapping clinical phases (Table 5-4).[83,84] Phase 1 lasts anywhere from 2 to 9 months and is referred to as the *painful* or *freezing* phase because it is characterized mostly by pain and a progressive loss of glenohumeral joint ROM. Phase 2 is the *stiff* or *frozen* phase and it typically lasts between 4 to 12 months. During this phase considerable stiffness predominates, with a gradual reduction of pain. The final phase (phase 3) is the *resolution* or *thawing* phase, in which there is improvement in ROM and resolution of stiffness. This last phase can range anywhere between 12 and 42 months.[84,85]

Because of the past challenges associated with defining frozen shoulder, authors have begun to adopt definitions of this condition that encompass signs, symptoms, and investigations.[82,86] Table 5-5 presents the symptoms, signs, and investigations that constitute the British Elbow and Shoulder Society's definition of idiopathic frozen shoulder.

TABLE 5-4

Three Phases of Primary (Idiopathic) Frozen Shoulder

Phase	Clinical Presentation
Freezing phase (painful)	Duration: 10 to 36 weeks Pain and stiffness around the shoulder No history of injury Nagging, constant pain that is worse at night Little or no response to NSAIDs
Adhesive/restrictive phase	Duration: 4 to 12 weeks Pain gradually subsides but stiffness remains Pain apparent only at extremes of movement Gross reduction of glenohumeral movements with near total loss of lateral (external) rotation
Resolution phase	Duration: 12 to 42 weeks Spontaneous improvement in ROM Mean duration from onset of frozen shoulder to greatest resolution exceeds 30 weeks

NSAIDs, Nonsteroidal anti-inflammatory drugs; *ROM,* range of motion.
From Dias R, Cutts S, Massoud S: Frozen shoulder, *Br Med J* 331:1453-1456, 2005.

TABLE 5-5

British Elbow and Shoulder Society (BESS) Survey Definition of Frozen Shoulder

Symptoms	Signs	Investigations
• True (deltoid insertion) shoulder pain • Night pain of insidious onset	• Painful restriction of active and passive motion • Passive elevation less than 100° • Passive lateral (external) rotation less than 30° • Passive medial (internal) rotation less than reaching the level of L5 • All other shoulder conditions excluded	• Plain radiographs normal • Arthroscopy shows vascular granulation tissue in the rotator interval

Modified from Guyver PM, Bruce DJ, Rees JL: Frozen shoulder—a stiff problem that requires a flexible approach, *Maturitas* 78:12, 2014.

The two defining symptoms in patients with frozen shoulders are pain and restricted ROM of the glenohumeral joint (Figure 5-12). The pain is characteristically felt around the deltoid insertion but can also be described as diffuse around the entire shoulder. Patients usually describe the pain as severe, affecting sleep and most activities of daily life. As mentioned earlier, pain is often noted first, followed by a decrease in glenohumeral motion.

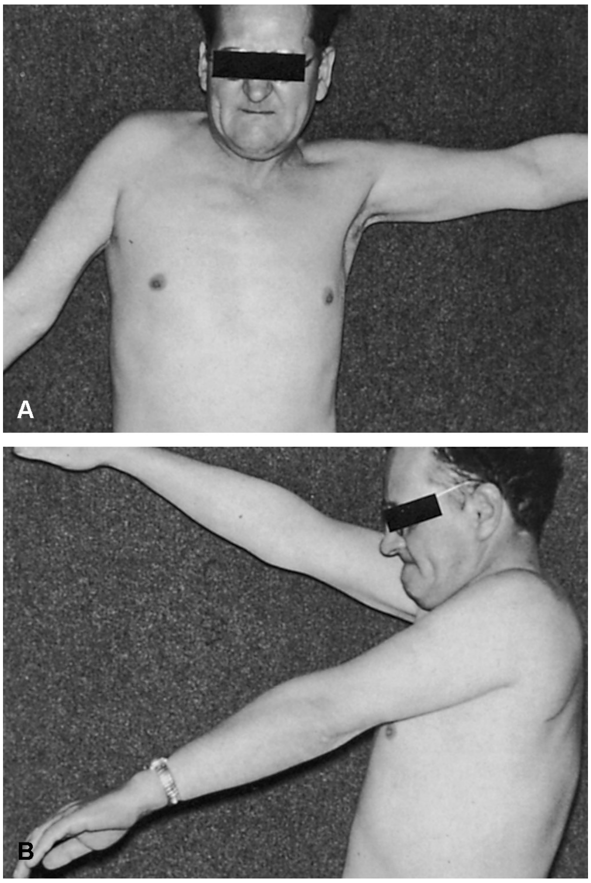

Figure 5-12 **A** and **B,** A 45-year-old man whose right shoulder is in the painful freezing phase of primary frozen shoulder. Note the distress and discouragement in the patient's face. (From Rowe CR, Leffet RD: Frozen shoulder. In Rowe CR, editor: *The shoulder,* p 158, New York, 1988, Churchill Livingstone.)

Patients describe either a slow, progressive loss of movement or occasionally a sudden loss. The reduced ROM follows a *capsular pattern of restriction,* a characteristic pattern of motion loss, secondary to capsular involvement in synovial joints. In the glenohumeral joint, the capsular pattern of restriction is lateral rotation most affected, followed by abduction, and medial rotation. Many clinicians believe that lateral rotation should be reduced more than 50% compared with the unaffected side to consider a diagnosis of frozen shoulder. Plain radiographs are used to rule out bony causes of pain and restricted ROM. To date, there is no evidence to support the use of ultrasound and/or magnetic resonance imaging in diagnosing frozen shoulder.[87]

Clinicians should be mindful of the secondary signs and symptoms that may develop in the patient with a frozen shoulder. Weakness of the glenohumeral and scapular stabilizer muscles is very common as a result of disuse or improper use. This usually occurs in the deep stabilizers of the glenohumeral joint (i.e., the rotator cuff) and in the more superficial movers, such as the three parts of the deltoid muscle. The upper trapezius is often overused in its attempt to compensate for weak and painful shoulder elevation, leading to a reversal of normal scapulohumeral rhythm and further muscle imbalance about the scapula.

Treatment of Frozen Shoulder

Given the uncertainty about the etiology of frozen shoulder syndrome, it is not surprising that the theories of treatment for this condition are equally inconsistent. Systematic reviews regarding the effectiveness of physical therapy[88–91] have concluded limited overall clinical evidence, based upon low-moderate quality evidence, for exercise and manual therapy and low-level laser therapy. The effects of treatment were reported to be greatest during phases 2 and 3 of the condition.

Most patients with a frozen shoulder can be managed successfully with nonoperative treatment, using a multidisciplinary approach of patient education, physical therapy, analgesic medication, and injection therapy.[85,87,88,92] Secondary frozen shoulder requires management of the preceding factor (e.g., shoulder surgery) or contributory factor (e.g., diabetes) as well. Although there is a lack of evidence to support these common treatment interventions, they are noninvasive, inexpensive, and have minimal risk.

Traditional rehabilitation programs focus on exercises and manual therapy techniques that improve glenohumeral ROM and pain-relieving modalities.[87,88,93,94] Joint mobilization techniques, stretching, and passive ROM exercises are done initially because pain usually prevents patients from doing these exercises independently. Some patients may not tolerate any mobility within the first phase of this condition, due to severe pain. In these cases, physical therapy management should focus on patient education and reassurance, pain-relieving strategies and gentle posture, and scapular setting exercises. Patients should also be encouraged to participate in some form of activity that promotes general health and wellness during the time they are dealing with their frozen shoulder. All exercises and techniques should be performed within the limitations of pain to avoid aggravating the affected tissue, especially during the painful, freezing phase.

Clearly no preponderance of evidence points to the best treatment for patients with frozen shoulder syndromes. However, it seems plausible that some, if not all, of the basic principles of shoulder rehabilitation could be applied in dealing with this condition. Patient education about the condition and its expected phases and outcome, postural awareness and treatment (especially of the adjacent cervical and thoracic spine levels), and exercises for the remaining shoulder girdle musculature are all important considerations. In addition, significant care should always be given to reeducate these patients to control movements of the glenohumeral and scapulothoracic joints because these muscles tend to become "detrained" as a result of the disease and the consequent loss of movement.

SHOULDER FRACTURES

Fractures of the shoulder girdle are relatively uncommon, ranging from minor, nondisplaced, hairline fractures to major, comminuted, displaced fractures with life-threatening consequences.[45] Most fractures in this region require some type of immobilization and/or period of protected motion. This immobilization, although essential for bone healing, often results in secondary stiffness of the glenohumeral joint, generalized muscle weakness, and altered scapulothoracic mechanics. Therefore it is important that the objectives of rehabilitation following a shoulder fracture include normalization of the entire shoulder girdle and trunk, not just the joint adjacent to the local fracture site.

Scapular Fractures

Fractures of the scapula account for only 1% of all fractures, 3% of all shoulder injuries, and 5% of fractures involving the entire shoulder.[95–97] They typically occur as a result of a high-energy situation involving a fall or direct blow and often occur in conjunction with other traumatic injuries. With less severe fractures, the muscles effectively stabilize the fracture, and the patient typically can expect a good functional outcome. However, patients may present with associated injuries to the involved arm, shoulder girdle, and thoracic cage; ipsilateral rib fractures are the most common associated injury, occurring in approximately 27% to 50% of patients.[95] Pulmonary trauma (e.g., pneumothorax, hemothorax, or both) is reported in 16% to 40% of patients, and clavicular fractures and injuries to the brachial plexus and subclavian artery reportedly occur in 26% and 12% of patients, respectively.[95–98]

Scapular fractures are classified according to the location of the fracture, specifically the body, neck, glenoid fossa, acromion, spine, or coracoid. The distribution of the different fracture sites has been reported as 35% to 43% at the scapular body, 26% at the neck, 10% at the glenoid fossa, 8% to 12% at the acromion, 6% to 11% at the spine, and 5% to 7% at the coracoid.[95,98–100]

Clinical Evaluation of Scapular Fractures

Most patients with a scapular fracture have experienced a traumatic, well-defined mechanism of injury; therefore clinical evaluation usually is precluded by a thorough radiographic and neurological examination. Local physical findings, such as swelling, ecchymosis, and crepitus with ROM, are common, as are subjective complaints of pain in the scapular region. In most cases, a detailed assessment of the entire shoulder girdle and the cervical and thoracic spine should be performed (see Volume 1 of this series, *Orthopedic Physical Assessment*).

Treatment of Scapular Fractures

Nonoperative treatment is recommended for most scapular fractures, with the exception of those that occur at specific sites and/or according to certain classifications.[101,102] These include (1) acromion or scapular spine fractures with downward tilting of the lateral fragment and resultant subacromial narrowing (Figure 5-13), (2) coracoid fractures that extend into the glenoid fossa (Figure 5-14), and (3) glenoid rim and intra-articular glenoid fractures associated with persistent or recurrent glenohumeral instability (Figures 5-15 and 5-16). Relative indications for the surgical treatment of scapular fractures include displaced acromion or coracoid process fractures (>10 mm), displaced intra-articular glenoid fractures (>5 mm), and those associated with humeral subluxation.[101,103]

Nonsurgical treatment of scapular fractures consists of approximately 7 to 10 days of sling immobilization, followed by a progressing regimen of pendular and gentle passive ROM exercises as comfort and control allow. Once follow-up radiographic findings indicate sufficient healing, the patient is encouraged to discontinue immobilization and proceed with active-assisted and active ROM exercises. Consideration must always be given to restrengthening the muscles that attach to the scapula and those that arise from the scapula (i.e., the rotator cuff and biceps brachii), which may have been affected by disuse and painful inhibition. In a number of cases, patients with a scapular fracture develop stiffness in the adjacent spine, specifically the lower cervical spine to the midthoracic spine. Clinicians must take care to assess these regions and treat accordingly. Failure to address this important

Figure 5-13 Radiograph demonstrating a fracture at the base of the acromion. (From Sanders R: *Core knowledge in orthopaedics: trauma*, Philadelphia, 2008, Saunders.)

Figure 5-14 Axial radiograph of shoulder, demonstrating the coracoid base fracture. (From Alsey KJ, Mahapatra AN, Jessop JH: Coracoid fracture in an adolescent rugby player – case report and review of the literature, *Radiography* 18(4):301-302, 2012.)

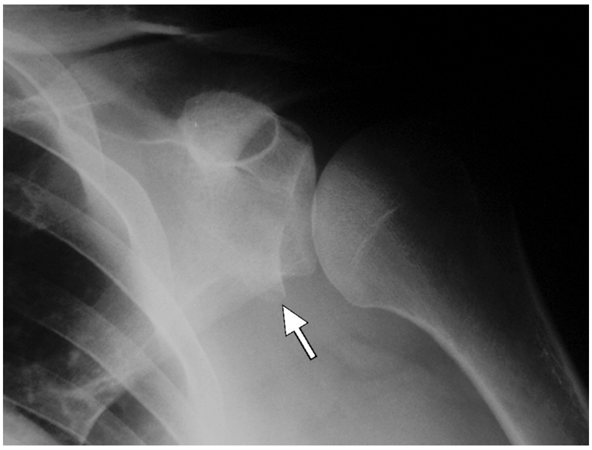

Figure 5-15 True anteroposterior plain radiograph demonstrating an anteroinferior glenoid fracture (*arrow*). (From Browner BD, Levine AM, Jupiter JB, et al: *Skeletal trauma*, ed 4, Philadelphia, 2009, Saunders.)

part of the kinetic chain may lead to further problems in the shoulder region and most certainly will result in a less than optimal outcome. Most minor scapular fractures are sufficiently healed by 6 to 12 weeks and able to withstand the progressive load delivered through ROM and strengthening exercises at this time. Clinicians and patients must keep in mind that maximum functional recovery, which encompasses more than just bone healing, may take as long as 6 to 12 months.[104,105]

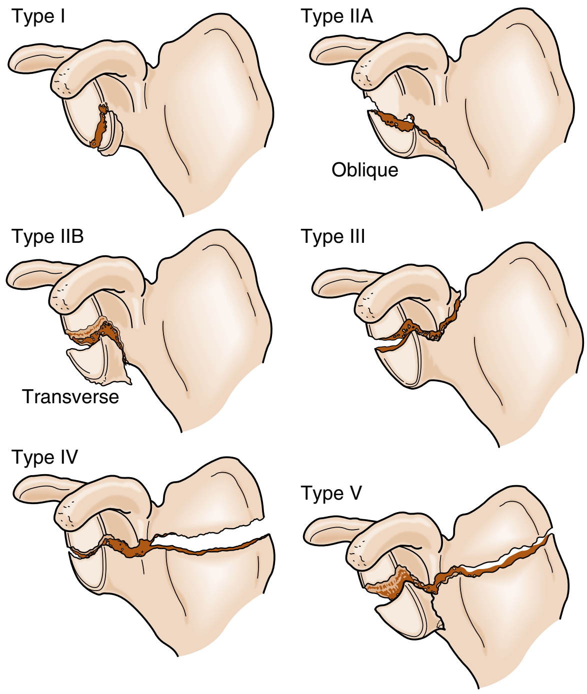

Figure 5-16 Ideberg's classification of glenoid fractures. (From Sanders R: *Core knowledge in orthopaedics: trauma*, Philadelphia, 2008, Saunders.)

"Floating Shoulder"

The term *floating shoulder* was introduced by Herscovici[106] in 1992 to describe an ipsilateral fracture of the clavicular shaft and the scapular neck (Figure 5-17). The combination of these two types of fractures has a significant effect on the stabilizing role of the clavicle. Goss[107] defined *floating shoulder* as "a double disruption of the superior suspensory shoulder complex (SSSC)." He described three struts of this complex: (1) the AC joint–acromial strut, (2) the clavicular–coracoclavicular ligament–coracoid linkage, and (3) the three process–scapular body junction. A potentially unstable anatomical condition exists when the complex is disrupted in at least two places with significant displacement at either or both sites (Figure 5-18). This condition has a high risk of poor bone healing from delayed union or malunion of the clavicle, the scapular neck, or both.

Combined clavicular and ipsilateral scapular neck fractures are usually caused by a high-energy mechanism of injury, most commonly motor vehicle accidents (80% to 100%).[108] Other causes have been cited, such as a direct blow, fall onto the tip of the shoulder, or a FOOSH injury.[106,107] As with most scapular fractures, combined clavicular and ipsilateral scapular neck fractures have a high incidence of associated traumatic lesions, and when severe accompanying injuries are present, the clinical signs and symptoms of this specific fracture type often are overlooked. The definitive diagnosis therefore is made on the basis of radiographic findings.

Very little research to date has addressed the most effective way to treat a floating shoulder. Literature related

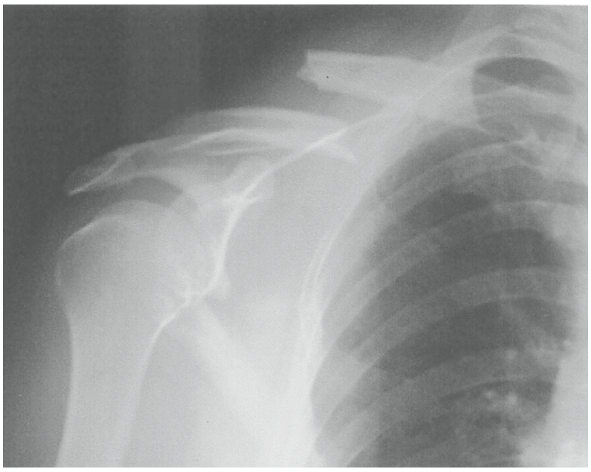

Figure 5-17 Radiograph showing right floating shoulder with scapular neck and ipsilateral midclavicular fractures. (From Hashiguchi H, Ito H: Clinical outcome of the treatment of floating shoulder by osteosynthesis for clavicular fracture alone, *J Shoulder Elbow Surg* 12(6):589-591, 2003.)

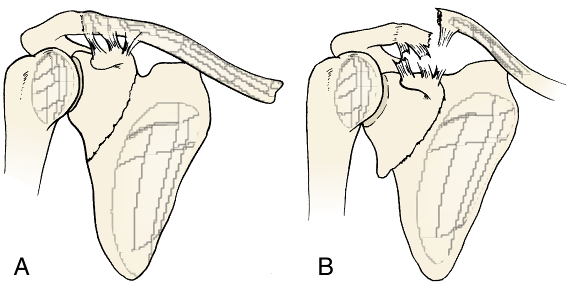

Figure 5-18 Fracture of the scapular neck. **A,** If the coracoclavicular and acromioclavicular ligaments are intact, there is little displacement of the glenoid (stable scapular neck fracture). **B,** Fracture of the scapular neck with disruption of the coracoclavicular and acromioclavicular ligaments creates a floating shoulder (unstable neck fracture). (From Herring JA: *Tachdjian's pediatric orthopaedics: from the Texas Scottish Rite Hospital for Children*, ed 5, Philadelphia, 2014, Saunders.)

to this fracture type is limited to case reports and retrospective studies of small patient series.[106–108] Treatment options mentioned range from conservative management with or without early mobilization to operative treatment through open reduction and internal fixation of the clavicle only or of the clavicle and the scapular neck fracture sites together. Clearly, further research and clinical study are necessary to elucidate this interesting pathology. Any rehabilitation plan should be derived from a careful analysis of the anatomy involved and a thorough examination of the entire shoulder girdle and adjacent spine.

Clavicular Fractures

The most commonly reported fracture of the shoulder girdle is the clavicular fracture (Figure 5-19).[109,110] The incidence is highest among children and adolescents, in whom the mechanism of injury is either a direct blow from a fall onto the affected shoulder or an impact such as a tackle in football. In a direct blow, the compressive force causes buckling of the clavicle, which leads to a fracture once the compressive force exceeds the tensile strength of the clavicle. The clavicle can also sustain a direct impact that results in a fracture. Eighty-seven percent of clavicular fractures occur from falls onto the shoulder, whereas direct impact to the shoulder accounts for 7%. FOOSH injuries account for 6% of clavicle fractu res.[40,110,111] In these instances the shoulder is driven downward, causing the clavicle to forcefully impinge on the underlying first rib, resulting in a fracture (Figure 5-20).

Although a fracture can occur anywhere along the clavicle, the most common site is reported to be between the medial two thirds and lateral one third of the bone (80%) followed by fractures in the distal one third (17%) and those that occur in the proximal one third (2%).[45,109,110,112] Fractures are classified according to their anatomical location: group I are in the midshaft (middle third) of

Figure 5-19 **A,** Clinical photograph of patient with a right clavicle fracture (*arrow*). Note "tenting" of skin. (From Ristevski B, Hall JA, Pearce D, et al: The radiographic quantification of scapular malalignment after malunion of displaced clavicular shaft fractures. *J Shoulder Elbow Surg* 22(2): 240-246, 2013.) **B,** Anteroposterior view of a fractured clavicle showing a comminuted fracture of the midshaft with upward displacement of the proximal fragment. (From Eiff MP, Hatch RL: *Fracture management for primary care,* ed 3, Philadelphia, 2012, Saunders.)

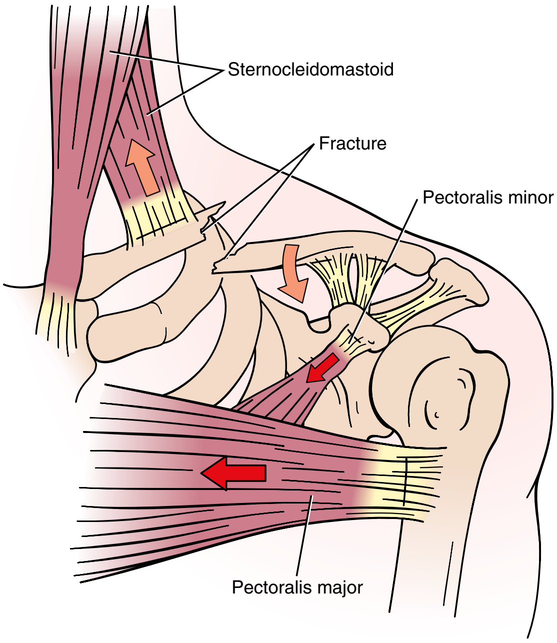

Figure 5-20 Clavicular fracture. With displacement, the proximal fragment is pulled superiorly by the sternocleidomastoid and the distal segment drops forward pulled down by pectoralis minor on coracoid process and pectoralis major on humerus.

TABLE 5-6

Subclassification of Group II Clavicular Fractures

Type	Description
Type I	Fractures lateral to the coracoclavicular ligaments
Type II	Fractures medial to the coracoclavicular ligaments
Type III	Fractures extending into the acromioclavicular joint
Type IV	Fractures in which the proximal fragment is displaced out of its periosteal tube
Type V	Comminuted fractures in which coracoclavicular ligaments remain attached to an inferior bone fragment but are not attached to the proximal or distal fragment

From Craig EV: Fractures of the clavicle. In Rockwood CA, Matsen FA, editors: *The shoulder*, Philadelphia, 1998, WB Saunders.

the clavicle, group II are in the distal (lateral) third of the clavicle, and group III are in the medial (proximal) third of the clavicle. Craig[112] devised a five-part subclassification system of the group II clavicular fractures (Table 5-6).

Clinical Evaluation of Clavicular Fractures

Because of their superficial location, clavicular fractures are easily recognizable through inspection and direct palpation. Patients are able to recall a specific mechanism of injury, describing localized pain and possibly a cracking or popping sound or sensation. If the patient is seen directly following the injury, they may be holding the ipsilateral arm with the contralateral arm or it may be held in adduction at the side. Swelling, ecchymosis, and gross deformity may be visible with possible tenting of the skin. Tenderness to palpation and crepitus may be palpated over the fracture site. If the patient is able to move his or her arm, a grinding sensation may be felt or described.

The degree of deformity depends on the location of the fracture and the amount of displacement (see Figure 5-19, *A*). Associated injuries, although not common, may occur, including scapular fractures, scapulothoracic dissociation, rib fractures, pneumothorax, and neurovascular compromise (Figures 5-21 and 5-22). The brachial plexus is of particular concern because of its proximity to the clavicle, which makes it susceptible to injury.[111,112]

Treatment of Clavicular Fractures

The goal of treatment, relative to clavicular fractures, is to minimize the risk of nonunion and malunion. **Nonunion** is defined as the absence of clinical radiographic healing after 4 to 6 months, whereas **malunion** is associated with angulation, shortening, and poor cosmetic appearance. Generally it was believed that malunion did not affect functional capacity; however, studies have discovered that malunion may lead to weakness and fatigability.[113–116] Most

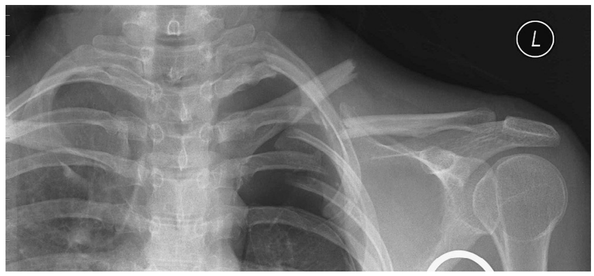

Figure 5-21 Fracture of the left clavicle and ribs with associated pneumothorax. Note the displaced midshaft clavicle fracture. (From Kim W, McKee MD: Management of acute clavicle fractures, *Orthop Clin North Am* 39(4): 491-505, 2008.)

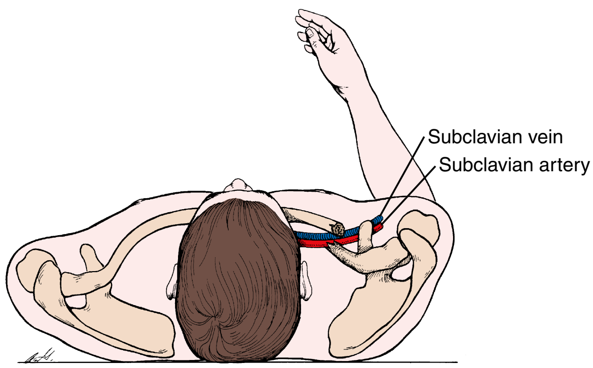

Figure 5-22 Potential injury to the subclavian vessels resulting from a fractured clavicle. (From Rockwood CA, Matsen FA, Wirth MA, et al, editors: *The shoulder,* ed 4, p 400, Philadelphia, 2009, Saunders.)

minimally displaced clavicular fractures do not require surgical intervention and can be treated conservatively with immobilization using either a sling or a figure-of-eight brace for approximately 4 to 6 weeks.[112,114–116] Both immobilizers appear to be effective; however, the sling is reported to provide more comfort with fewer complications, such as skin breakdown because the figure-of-eight brace must be kept in some degree of tension. In addition, the sling allows the patient to more easily perform light ROM exercises.

Clavicular fractures that require surgical consideration include open fractures, concomitant displaced scapular neck fractures that disrupt the superior shoulder suspensory complex, the presence of neurovascular or skin compromise, and a patient with multiple trauma who needs assistance with early mobilization.[116,117] Surgical treatment may be considered in some cases, for certain high-demand patients (such as heavy laborers or athletes), who are exposed to large, repetitive, potentially deforming forces, and individuals who have had a previous nonunion at the same site. However, careful patient selection and preoperative counseling are recommended because of the potential risks (e.g., implant infection, migration of intramedullary wires, neurovascular injuries) and sometimes less than optimal outcomes of the procedure.

Once bone healing is well established (**clinical union** occurs in 2 to 3 weeks), the patient can be guided through active-assisted and then active ROM exercises. Because most clavicular fractures do not involve the adjacent articulations, ROM is quite easily regained. Care must always be taken not to overload the healing fracture site with activities that produce too great a leverage point on this important fulcrum. Restrengthening exercises are prescribed for any muscles that were weakened as a result of painful inhibition and/or the immobilization period. As with any type of shoulder injury, the patient should be checked to ensure that the scapulohumeral rhythm is normal and symmetrical and that the essential stabilizing musculature of the glenohumeral joint is intact. Normal healing times vary slightly depending upon the site of the clavicular fracture but generally range between 6 and 9 weeks in young children and 8 and 12 weeks in adults.[109,111,112] It is important to ensure that both clinical and radiographic healing of the clavicular fracture are obtained before a patient's return to sport or heavy work. Factors to consider include the patient's age, sport or activity, location and severity of the fracture, extent and speed of healing, method of treatment (operative versus nonoperative), and any complications encountered during the recovery period.[114,116] Complications are possible following any fracture of the shoulder girdle, but the most serious consequences occur when neurovascular structures are compromised. Clinicians must be careful to continually assess for changes in sensation related to this and deal with this concern immediately. Less severe clavicular fracture complications include malunion (which results in a bony prominence but rarely a functional deficit), nonunion, and poor cosmesis.

Proximal Humeral Fractures

The proximal humerus is a common site of injury in the young and the elderly. In a skeletally immature athlete, the fracture frequently presents as a fracture at the proximal humeral growth plate or physis (*Little Leaguer's shoulder*); this type of injury most often is associated with young patients involved in throwing sports.[118–121] The injury is caused by a powerful medial rotation and adduction traction force on the proximal humeral epiphysis that occurs during the deceleration and follow-through phases of throwing or pitching. The rotational forces that occur during the arm-cocking and arm-acceleration phases can compound the problem. The result of these forces is a stress fracture, usually a Salter-Harris type I or type II. Radiologic signs, which may take up to 4 to 6 weeks to become evident, include widening of the growth plate, demineralization and rarefaction on the metaphyseal side of the physis, and metaphyseal bone separation.[119–121]

Radiographic Signs of Little Leaguer's Shoulder

- Widening of the epiphyseal plate
- Demineralization and rarefaction of the metaphyseal side of the physis
- Metaphyseal bone separation

The athlete typically complains of acute shoulder pain when attempting to throw hard. If these complaints are ignored, it can result in acute displacement of the weakened physis. Rest is the primary treatment, along with patient education regarding why absolute cessation from activity, at least initially, is essential. The bone may require up to 8 to 12 months to reossify and remodel.

Following a period of rest, rehabilitation should include activities to improve strength, coordination, proprioception, endurance, and ROM. The exact timing for beginning active exercises, as well as when and how much to progress these exercises, varies, depending on the stage of bone healing evident, the patient's tolerance of pain, and the condition of the surrounding soft tissue and adjacent joints. The most prudent decisions related to exercise prescription for these young patients is guided by the radiologic evidence and a thorough assessment and monitoring of the patient's clinical signs and symptoms. Clinicians must proceed cautiously, applying gentle stress to the area to encourage healing but never exceeding what the tissue can sustain.

In the elderly, proximal humeral fractures usually occur in patients with osteoporosis, and women are affected twice as often as men are. Predictions of incidence are estimated to triple during the next three decades as the population ages.[122] Most fractures are caused by minimal trauma, such as a FOOSH injury from a standing height or lower, or a direct blow to the lateral aspect of the shoulder (Figure 5-23).[123,124] Occasionally, they occur in association with a dislocation

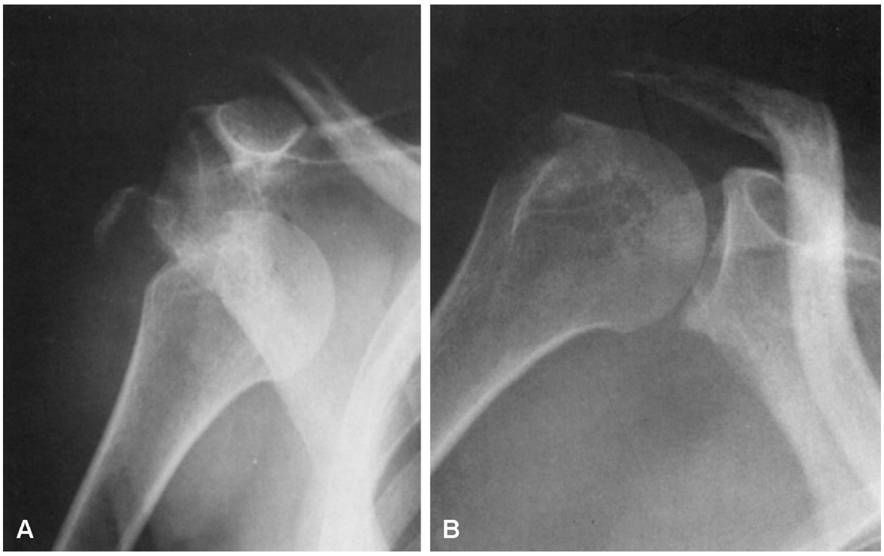

Figure 5-23 **A,** Anteroposterior x-ray film of a two-part anterior fracture-dislocation with a displaced greater tuberosity fracture. **B,** After a closed reduction, the greater tuberosity fracture reduced and healed without further displacement. (From Rockwood CA, Green DP, Bucholz RW, Heckman JD: *Fractures in adults,* ed 4, p 1069, Philadelphia, 1996, Lippincott-Raven.)

or with excessive rotation of the arm, especially while in the abducted position.[58] In this situation, the humerus locks against the acromion in a pivotal position, resulting in a fracture.

Most proximal humeral fractures are nondisplaced or minimally displaced; these should be differentiated from the more serious displaced fractures, which are managed quite differently. The most commonly used classification system is the four-part fracture classification developed by Neer in 1970, in which he divided fractures into displaced and nondisplaced (Figure 5-24).[125,126] Displaced fractures, according to this classification, have more than 1 cm of displacement or more than 45° of angulation. Nondisplaced fractures may be called *one-part fractures,* regardless of how many fracture lines are seen. A *two-part fracture* involves displacement of the anatomical neck, surgical neck, lesser tuberosity, or greater tuberosity in relation to the remaining intact proximal humerus. Similarly *three-* and *four-part fractures* involve displacement of two or three fragments in relation to the main humeral fragment.

According to Neer's classification, the most commonly reported fracture of the proximal humerus is a displaced or nondisplaced (one-part or two-part) fracture involving the surgical neck (Figure 5-25). Researchers have investigated the clinical impact of greater tuberosity displacements less than 1 cm and found that the shoulder has very little tolerance for greater tuberosity displacement. Displacements of 5 mm to 1 cm were found to result in substantial dysfunction from either impingement or rotator cuff tearing.[122,126,127]

Clinical Presentation of Proximal Humeral Fractures

Because most proximal humeral fractures present acutely, common symptoms are pain, swelling, and tenderness about the shoulder, particularly in the region of the greater tuberosity. Crepitus may be evident with movement of the shoulder, but in most cases the patient is reluctant to initiate any active movement and holds the arm closely against the chest wall. The definitive diagnosis is made through radiographic evaluation.

A thorough assessment should be performed, including evaluation of the integrity of the bony tissue and careful examination of the neurovascular and musculotendinous structures intimately situated in this region. A fracture-dislocation or a fracture resulting from a high-energy trauma is much more likely to be associated with neurovascular injuries. The axillary nerve can be injured during a shoulder fracture–dislocation; in most patients the injury results in axonotmesis and a full recovery is expected. Of particular concern is the rotator cuff, which attaches to the different tuberosities of the humerus and consequently may have a deforming effect, depending on where the proximal humerus has been injured. For example, with a fracture of the greater tuberosity, the fracture fragment is pulled superiorly and posteriorly due to the muscle action of the supraspinatus, infraspinatus, and teres minor muscles. Conversely, in a fracture of the lesser tuberosity, the fragment is pulled anteriorly and medially by the subscapularis muscle (Figure 5-26).[127,128]

The deltoid and pectoralis major muscles must also be evaluated in patients with a proximal humeral fracture. Because the deltoid muscle attaches to the lateral shaft of the humerus, it can cause displacement of fractures of the proximal humeral shaft. Similarly, the pectoralis major muscle's insertion onto the lower portion of the bicipital groove can displace the proximal shaft of the humerus medially, as is typically seen in surgical neck fractures.

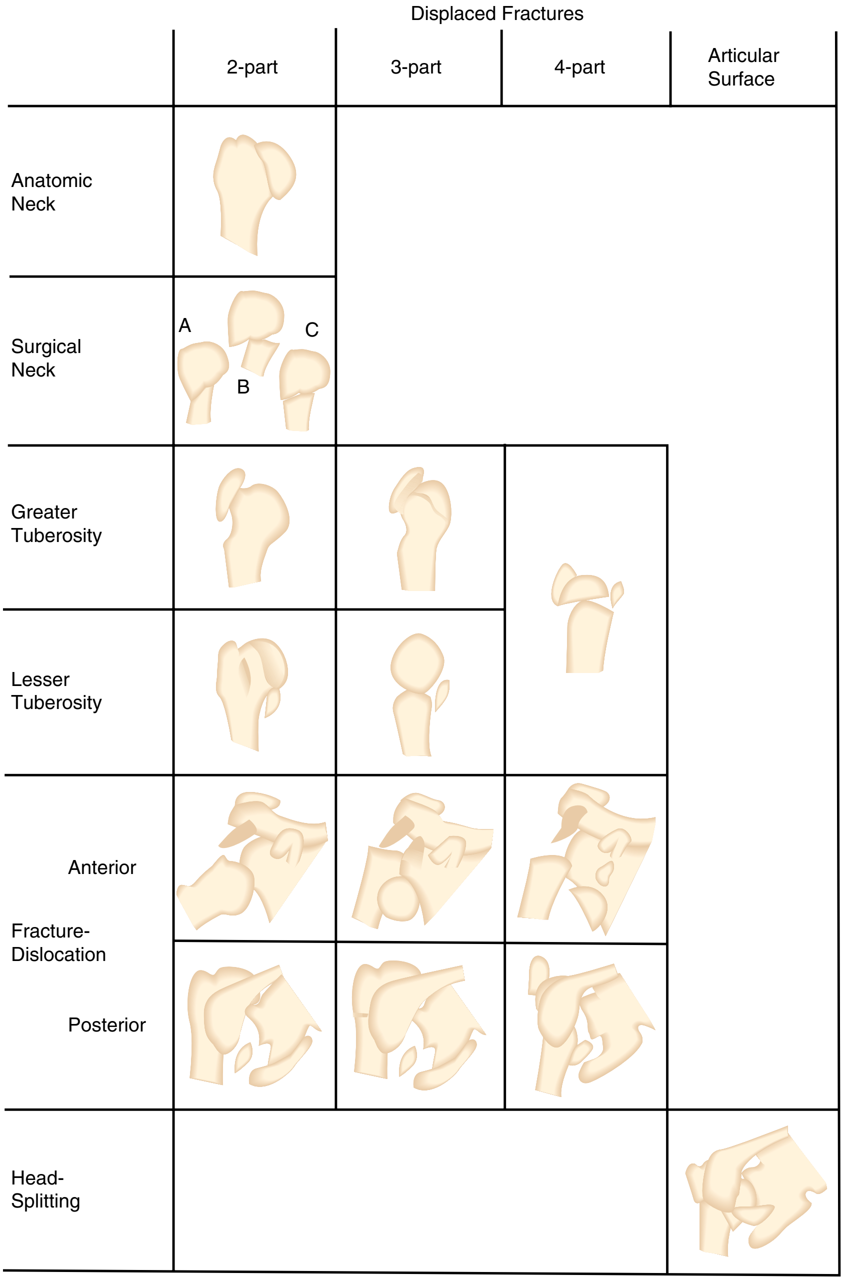

Figure 5-24 Neer four-segment classification of displaced proximal humeral fractures. A fragment is considered displaced when greater than 1 cm of displacement or 45 degrees of angulation is present. A fracture-dislocation is present if the articular segment is no longer in contact with the glenoid. (Redrawn from Neer CS: Displaced proximal humeral fractures. Part I. Classification and evaluation, *J Bone Joint Surg Am* 52:1077-1089, 1970.)

Treatment of Proximal Humeral Fractures

Most proximal humeral fractures are minimally displaced and are successfully treated without surgery.[121,127,129] If the fracture is classified as nondisplaced and is impacted or stable, an initial period of immobilization is indicated, using either a conventional sling or a collar and cuff sling that allows the weight of the arm to apply slight traction to the fracture. Early ROM exercises follow, at approximately 2 weeks after injury. If the fracture is classified as nondisplaced but is considered unstable, the immobilization period is more strictly adhered to with ROM exercises beginning after approximately 4 weeks when clinical union has occurred.

Diminished pain and an easing of the patient's apprehension are reasonable indicators for beginning gentle exercises. Obtaining intermittent radiographs before

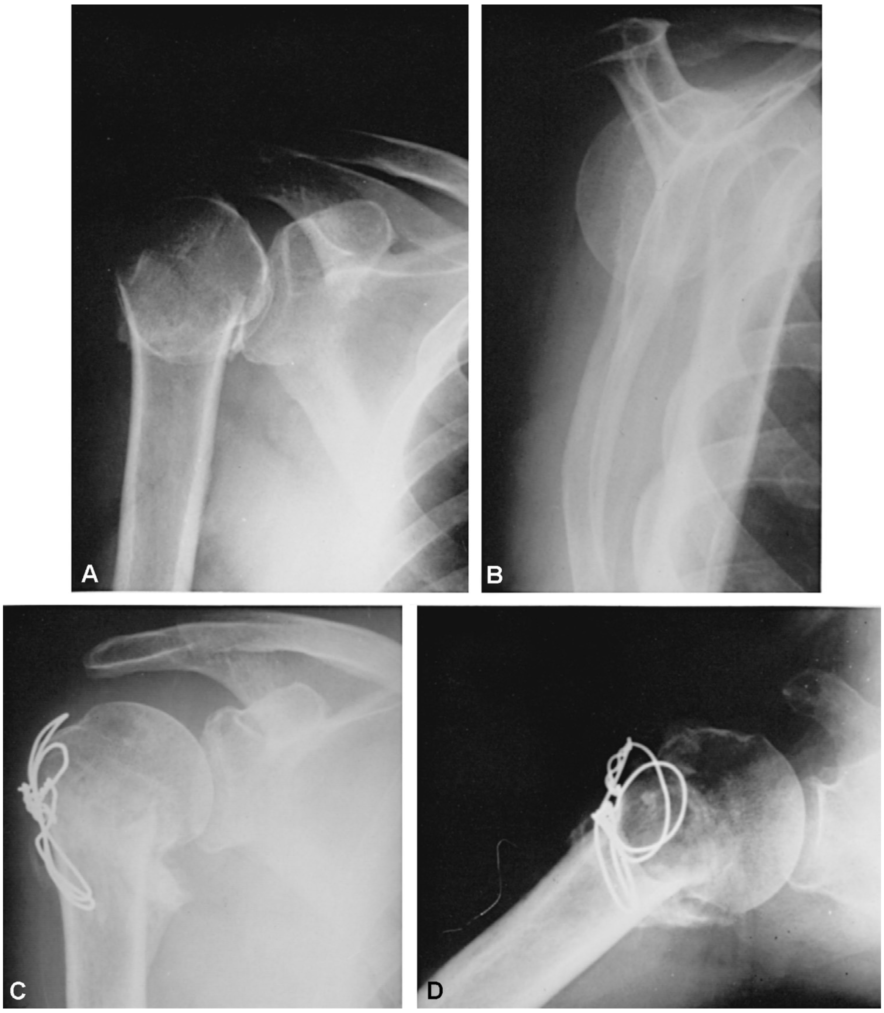

Figure 5-25 Displaced fracture of the surgical neck. **A** and **B,** Anteroposterior and lateral x-ray films of a displaced surgical neck fracture. The fracture was treated for 3 weeks as an undisplaced fracture on the basis of an anteroposterior x-ray film only **(A)**. The follow-up lateral x-ray film **(B)** in the scapular plane revealed a significant anterior shaft displacement. **C** and **D,** Anteroposterior and lateral x-ray films after open reduction and internal fixation with two figure-of-eight wires. (From Rockwood CA, Matsen FA, editors: *The shoulder,* p 353, Philadelphia, 1998, WB Saunders.)

beginning rehabilitation is important to establish the fracture is clinically stable and the bone moves as a unit. Rehabilitation programs should follow general shoulder treatment principles of patient education, mobility of the entire shoulder girdle and relevant kinetic chain segments, optimal neuromuscular strength, and return to functional activities. Bertoft et al.[129] reported that the greatest amount of improvement in ROM occurs between 3 and 8 weeks after injury.

Management of the young athlete with an epiphyseal fracture of his or her proximal humeral growth plate is nonoperative with a 3-month period of rest followed by a program of shoulder ROM exercises, scapular and rotator cuff strength, and endurance exercise and functional exercises incorporating the entire kinetic chain. A progressive throwing program and subsequent return to play are considered when the athlete demonstrates resolution of symptoms as well as optimal shoulder ROM and neuromuscular control.

Complications following nonoperative management of a proximal humeral fracture are primarily related to shoulder stiffness and limited motion or to the consequences of displacement of the greater tuberosity. Early initiation of passive and active-assisted ROM exercises compared with delaying exercises until 3 weeks post injury results in less pain and faster recovery in people with stable fractures.[130] Additional complications that may affect outcomes in patients with proximal humeral fractures are those related to associated soft tissue damage (e.g., rotator cuff) and neurovascular injury. Clinicians must be diligent in evaluating for and treating any associated injuries that may have been injured, along with

Figure 5-26 Greater tuberosity malunion resulting from unopposed pull of the supraspinatus and infraspinatus muscles. (From Antuña SA, Sperling JW, Sánchez-Sotelo J, et al: Shoulder arthroplasty for proximal humeral malunions: long-term results, *J Shoulder Elbow Surg* 11(2):122-129, 2002.)

the humerus, in the original trauma or at the very least may have been strained or traumatized as a result of the fracture.

Surgical indications for a fracture of the proximal humerus are not straightforward. Humeral head vascularity, fracture pattern, bone quality, and overall geometry are important considerations in determining appropriate treatment. Osteoporosis influences the degree of comminution, magnitude of cancellous defects secondary to impaction, and likelihood of fixation failure or loss of reduction.[131] Surgery is reserved primarily for fractures that are significantly displaced; it involves mainly closed reduction with or without percutaneous pinning or open reduction and internal fixation.[131,132] An osteoporotic fracture is often more appropriately treated with arthroplasty than with reduction and internal fixation. Treatment of proximal humeral fractures with surgery is considered difficult, not only because of the complex anatomy of the shoulder girdle but also because of the difficulty achieving and maintaining anatomical reduction and stable fixation. Rehabilitation following these types of surgical procedures is rigorous and involved, and practitioners should ensure patients are well informed and educated about the procedure and are suited to handle the important postoperative period. In some cases, because of the co-morbidity status and the patient's age, nonsurgical treatment is the most appropriate choice despite substantial fracture displacement.

ROTATOR CUFF INJURIES

Injuries affecting the rotator cuff comprise the largest proportion of all injuries reported at the shoulder.[133–136] They range from minor tendon or muscle injuries to full-thickness tears and have been reported to cause disability in individuals that has been compared with health issues such as congestive heart failure, diabetes, myocardial infarction, and depression.[137] More information on rotator cuff injuries and their management can be found in Chapter 7.

BICEPS TENDON AND SUPERIOR LABRAL LESIONS

The long head of biceps is a tendinous tissue, susceptible to trauma and injury by the nature of its anatomical site and its role in shoulder mechanics. Some debate exists regarding the etiology of biceps injuries, namely, whether they have a primary cause directly associated with the biceps tendon complex or whether they occur secondary to dysfunction elsewhere in the shoulder girdle. Some authors suggest that greater than 90% of patients with a diagnosis of biceps tendonitis actually have a primary diagnosis of impingement syndrome or scapular instability that is causing secondary involvement of the biceps tendon.[138,139] Studies based on the results of magnetic resonance imaging and arthroscopic procedures have shed new light on the pathophysiology of the long head of the biceps tendon.[138–142]

One of the most significant contributions to understanding biceps injuries has come as a result of studies that have investigated the superior glenoid labrum. Andrews et al.[143] in 1985 described tears in the superior labrum of the glenohumeral joint noting the placement of these tears at the attachment of the biceps tendon (Figure 5-27). In 1990, Snyder et al.[144] first described the superolabral anterior to posterior (SLAP) lesion, a lesion of the superior labrum and adjoining biceps anchor. They categorized SLAP lesions into four types. Type I lesions show degenerative fraying with no detachment of the biceps insertion; type II lesions show detachment of the biceps insertion; type III lesions show a bucket handle tear of the superior aspect of the labrum with an intact biceps tendon insertion to bone; and type IV lesions show an intrasubstance tear of the biceps tendon with a bucket handle tear of the superior aspect of the labrum.

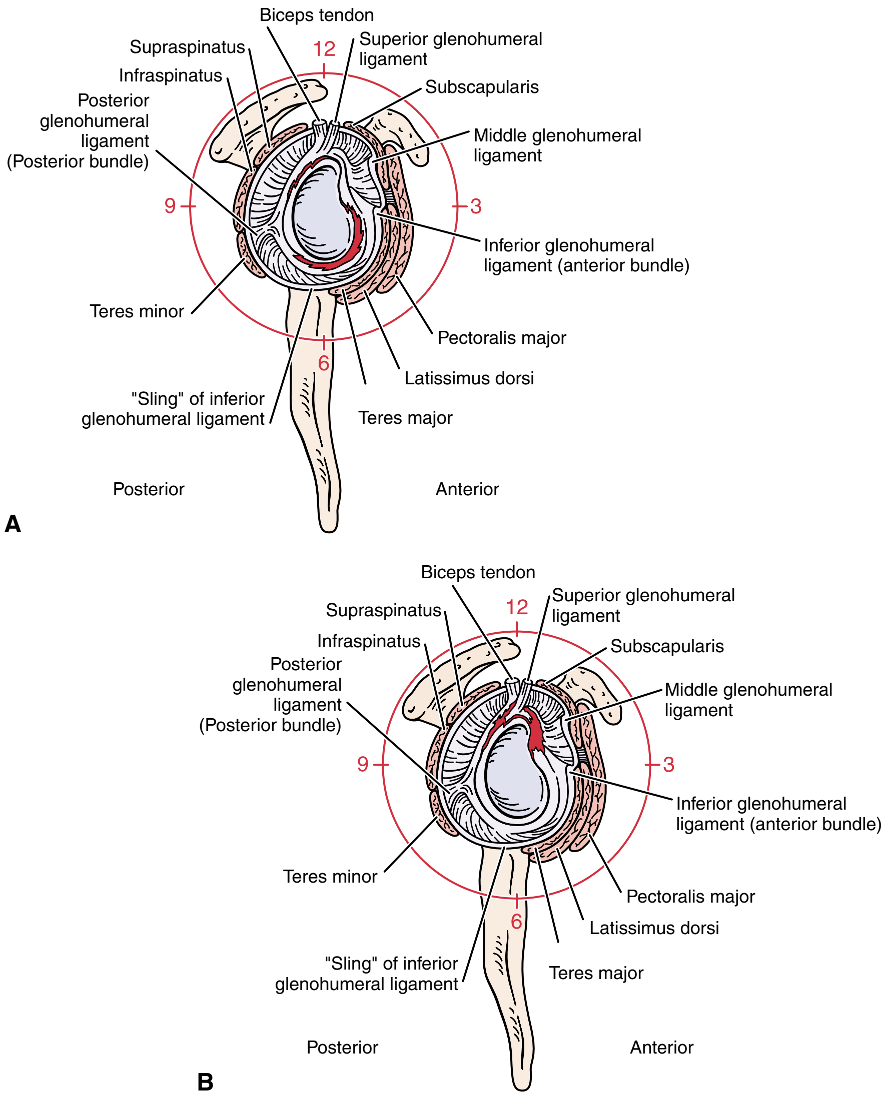

Figure 5-27 Labral lesions of the right shoulder. **A,** Bankart lesion. **B,** SLAP lesion. (From Magee DJ: *Orthopedic physical assessment*, ed 6, p 320, Philadelphia, 2014, WB Saunders.)

Morgan[145] further divided type II SLAP lesions into three subtypes, depending on whether the detachment of the labrum involved the anterior aspect of the labrum alone, the posterior aspect, or both.

In their three-part series, *"The Disabled Throwing Shoulder,"* Burkhart et al.[5,14,146] described the type II SLAP lesion (i.e., involving detachment of the biceps tendon) as the most common pathological entity associated with a "dead arm" syndrome, which they defined as any pathological shoulder condition that deems an athlete unable to throw due to pain and subjective unease in the shoulder. In an arthroscopic study done earlier, these same authors observed what they called a dynamic "peel-back phenomenon" in throwers with posterior and combined anteroposterior SLAP lesions.[147] They found that with the arm in the cocked position of abduction and lateral rotation the peel-back occurred as a result of the effect of the biceps tendon as its vector shifted to a more posterior position in late cocking. The change in angle and the twist of the biceps tendon produced a torsional force to the posterosuperior labrum, causing detachment if the superior labrum was not well anchored to the glenoid. These findings suggested a mechanism of injury for SLAP lesions in throwers that was different from the mechanism postulated earlier by Andrews et al.[143] These researchers had described a *deceleration* mechanism of labral injuries in throwers, which occurred as the biceps contracted to slow the rapidly extending elbow in the follow-through phase. They believed that this created a high tensile load in the biceps that acted to pull the biceps and superior labrum complex from the bone. Burkhart et al.[146] essentially described the opposite, an *acceleration* mechanism of injury; specifically, in late cocking, as the arm began to accelerate forward from an abducted and laterally rotated position, the long head of the biceps and the superior labrum were peeled back rather than pulled from the bone.

Also of interest in studies of type II SLAP lesions are the clinical factors associated with the primary pathology. Burkhart et al.[146] called these factors the "ultimate culprits" in dead arm syndrome: (1) a tight posteroinferior capsule, which causes a GIRD and a shift in the glenohumeral rotation point, (2) the peel-back mechanism produced by the biceps tendon, which leads to a SLAP lesion, (3) hyperexternal (lateral) rotation of the humerus, and (4) scapular protraction. Of the throwers with SLAP lesions whom they examined, 31% were also found to have rotator cuff tears (38% were full-thickness tears, and 62% were partial-thickness tears).

As can be seen, the biceps tendon plays an important role in the overall anatomy and function of the shoulder joint. Most importantly it has a strong relationship to its adjacent tissue, namely the glenoid, glenoid labrum, and rotator cuff. Injury to any one of these structures undoubtedly affects the integrity of the biceps tendon and the well-being of the entire shoulder girdle.

Etiology of Biceps Lesions

Two main etiologic categories have been proposed relative to biceps lesions.[148–151] One includes young patients with anomalies of the bicipital groove in whom repetitive trauma is the most important causal factor; the other includes patients in an older age group, where associated degenerative changes in the tendon tend to be the predominant causal factor. In most cases, the cause of biceps pathology is multifactorial and is related, in some manner, to the anatomical location of the tendon. As mentioned, the potential for impingement of the biceps tendon under the coracoacromial arch is great, especially if dysfunction of the rotator cuff and/or scapular misalignment are present. The blood supply of the biceps has been shown to be diminished in the long tendon region, with a "critical zone" similar to that seen in the supraspinatus tendon.[152] In abduction, a zone of avascularity exists in the intracapsular portion of the biceps tendon, which is felt to be caused by pressure from the head of the humerus, a phenomenon referred to as *wringing-out*. Considering these positions, it is easy to see why patients involved in overhead lifting and throwing are more susceptible to ruptures, elongations, and dislocations of the biceps tendon. The past several years have produced a proliferation of information about the superior labrum and its relationship to the biceps tendon.[138,140,142–146] As noted earlier, Andrews et al.[143] and Snyder et al.[144] originally described lesions of the biceps and superior labrum, known as *SLAP lesions*. Two cited causes of SLAP lesions are (1) a FOOSH, which drives the humeral head up onto the labrum and the biceps tendon, and (2) excessive and forceful contraction of the biceps in throwing athletes (e.g., baseball and football players).

Relevant Anatomy

The long head of the biceps tendon originates from the superior glenoid labrum and the supraglenoid tubercle and travels obliquely within the shoulder joint, turning sharply inferiorly to exit the joint underneath the transverse humeral ligament within the bicipital groove. Interestingly, the biceps tendon does not slide in the groove; rather, the humerus moves on a fixed, passive biceps tendon during shoulder motions. The groove can move along the tendon as much as 4 cm (1.5 inches). The origin of the biceps tendon reportedly varies, not only in the type of insertion (single, bifurcated, or trifurcated) but also in the specific anatomical location where it inserts.[148,153,154] Vangsness et al.[148] dissected 105 cadaveric shoulders to study the biceps tendon origin and its relationship to the labrum and the supratubercular groove. They classified their findings into four insertion types. Type I insertions occurred in 22% of the subjects and showed a labral attachment that was entirely posterior with no contribution to the anterior labrum. In type II insertions the labral contribution was primarily posterior with a small anterior band present. Type III insertions had equal contributions to the anterior and posterior labrum and were found in 37% of the cadavers. Type IV insertions occurred in 8% of the cadavers and consisted mainly of an anterior labral attachment. A different study[138] noted the biceps tendon originating from the supraglenoid tubercle 20% to 30% of the time, from the posterosuperior portion of the glenoid labrum 48% to 70% of the time, and from both the supraglenoid tubercle and the glenoid labrum 25% to 28% of the time. It is clear to see from these studies why the biceps tendon and superior labrum are commonly injured together.

The biceps tendon is surrounded by a synovial sheath, which ends at the distal end of the bicipital groove, making the tendon an intra-articular but extrasynovial structure. As the long head of the biceps tendon moves from its origin on the superior glenoid labrum and supraglenoid tubercle to its muscle insertion, it is stabilized in position by the supraspinatus and subscapularis tendons and the capsuloligamentous tissues. The triangular-shaped area between the supraspinatus and the subscapularis tendons is referred to as the *rotator interval*. It contains the coracohumeral and the superior glenohumeral ligaments and has on its medial side the coracoid process. Histoanatomical studies have found that both the superior glenohumeral ligament and the coracohumeral ligament are important structures in the stabilization of the biceps tendon in its groove, and lesions associated with any of the components of the rotator interval leave the biceps tendon and the rotator cuff vulnerable to injury.[141,152,155]

Function of the Biceps

The biceps extends from the scapula to the forearm and as a result plays an important role in function of the shoulder and elbow. Basmajian and Latif[150] characterized the action of biceps as flexion of the elbow when the forearm is in the neutral or supinated position. The muscle is also described as having an important role in decelerating the rapidly moving arm during activities such as forceful overhand throwing. The exact function of the biceps at the shoulder is not well understood. Most references regard it as a weak flexor,[140,153,155] but its proposed role as a humeral head depressor remains controversial.[152,154,155] One study, which used simulated muscle forces on a biomechanical cadaver model, showed that the biceps tendon force restrained superior humeral translation. A key factor in this study was that this occurred in the presence of a large rotator cuff tear.[141] A similar study demonstrated that the biceps assisted glenohumeral anterior stability through increased resistance to torsional forces. Other studies have disputed biceps role as a shoulder stabilizer, finding only a small amount of muscle activity during shoulder motion.[140,155–157] Despite the debate, the relationship between the long head of biceps and the function and stability of the glenohumeral joint is an important clinical feature, which must always be considered in the management of patients with a shoulder dysfunction.

Classification of Biceps Lesions

Historically, lesions involving the biceps tendon have been categorized as being either a *tendonitis* or *instability*. Tendonitis can be further divided into *primary tendonitis,* which occurs as a result of pathology of the tendon sheath, or *secondary tendonitis,* which occurs secondary to an underlying injury that causes subsequent biceps irritation and injury. Many believe that the pathology seen in the biceps is directly related to its intimate relationship with the rotator cuff because they both pass under the coracoacromial arch and are therefore susceptible to impingement. "Isolated" ruptures of the long head of the biceps tendon are reportedly uncommon. Neer believed that most ruptures of the long head of the biceps tendon were associated with supraspinatus tears.[158,159] One study documented isolated biceps tendon ruptures in as many as 25% of patients,[145] but this incidence has not been reproduced in subsequent studies.

Slatis and Aalto[160] developed a three-part classification for biceps lesions. A type A lesion is referred to as *impingement tendonitis* because it occurs secondary to an impingement syndrome and rotator cuff disease. The defective cuff exposes the biceps to a rigid coracoacromial arch, resulting in tendonitis. This is the most common cause of biceps tendonitis. Type B lesions describe a *subluxation of the biceps tendon*. All subluxations and dislocations of the biceps tendon are included in this category. Lesions of the coracohumeral ligament allow the biceps tendon gradually to displace medially; the slipping of the tendon into and out of its groove leads to inflammation and fraying. A type C lesion is called *attrition tendonitis.* These are primary lesions of the biceps tendon that occur inside the canal, resulting in inflammation and eventual degeneration of the biceps tendon. Often spurring and fraying are evident with this type of tendonitis.

Biceps lesions have also been classified according to their anatomical location, specifically, at their origin, in the rotator interval, or in association with rotator cuff tears.[138] As noted earlier, the third category has spurred considerable study regarding the intimate relationship between the rotator cuff and the biceps tendon complex.

Clinical Presentation of Biceps Lesions

Patients with biceps tendon injuries typically present with pain in the proximal anterior area of the shoulder directly over the biceps tendon, with occasional radiation of pain down into the muscle belly. Similar to rotator cuff impingement syndromes, the pain can also be described at the deltoid insertion. It is unusual for biceps-related pain to radiate into the neck or distally beyond the elbow joint. Night pain is sometimes reported with biceps lesions. However, this likely can be explained by overall problems associated with trying to sleep with a painful shoulder. Most shoulder conditions are worse at night for a number of reasons. A compressive load may be present (e.g., lying on the affected shoulder) or in some instances traction may be applied to the shoulder (e.g., arm hanging over the bed); either of these results in pain. Positioning to avoid pain is difficult; however, most patients find that, with the use of many pillows, lying on the contralateral side with the affected arm in neutral, resting on the pillows, is among the most comfortable positions. Many patients with a painful shoulder report that their best rest is achieved in a semireclined position, such as in a recliner, with the arm resting on a pillow in the shoulder resting position.

Patients with a biceps injury usually describe an insidious onset of their symptoms; however, the pattern of pain can be linked to repetitive types of activities. Acute trauma may be part of the original injury that predisposed the biceps tendon to subsequent rupture or dislocation. Patients with bicipital tendonitis tend to be young or middle aged, with a pain pattern that is less at rest and more intense with activity. This is especially true with overhead activities in daily living, work, and/or sports. Rotation of the humerus at or above the horizontal level brings the tuberosities, bicipital groove, biceps tendon, and rotator cuff in direct contact with the anterior acromion and the coracoacromial ligament. Biceps instability is most commonly associated with the throwing athlete, where in certain positions of overhead rotation, the motion is accompanied by a palpable snap and pain. Pain is felt locally at the anterior aspect of the shoulder and is aggravated by elevating the arm to around 90°. If the biceps tendon ruptures, there is associated acute pain and sometimes an audible pop in the shoulder, followed in a few days by a notable change in the contour of the arm, with ecchymosis that often tracks downward to the distal muscle belly.

Numerous special tests have been described that can assist in the diagnosis of biceps conditions; however, their findings should be interpreted in combination with other clinical tests because the sensitivity and specificity of these tests have been reported to be only moderate (see Volume 1 of this series, *Orthopedic Physical Assessment*, Chapter 5). Most biceps lesions present with tenderness on palpation in the bicipital groove, which often disappears as the lesser tuberosity and groove rotate medially under the short head of the biceps and coracoid process. This is different from the tenderness noted with subdeltoid bursitis and impingement syndromes, which has a more diffuse pattern that does not change with arm rotation. According to Burkhead et al.,[153] this "tenderness in motion" sign is among the most specific for differentiating biceps lesions. ROM may be slightly restricted and painful into full abduction and medial and lateral rotation, usually as a result of pain rather than capsular constriction. Resisted isometric testing reveals a painful weakness with forward flexion of the shoulder.

Treatment of Biceps Injuries

As is almost universally true of all injuries, the most effective form of treatment of a biceps injury is prevention. As noted, the biceps is often part of the domino effect that occurs as a result of other primary shoulder pathologies. Therefore prevention of injury and overuse in these regions of the shoulder (e.g., rotator cuff) conceivably could prevent injuries to the biceps tendon. Individuals involved in manual labor with either repetitive overhead work or heavy lifting, as well as individuals participating in sports that demand overhand motions, are particularly at risk for biceps injury and should be educated on how to monitor and prevent injuries in this region. Developing a balanced shoulder musculature that includes a strong, healthy rotator cuff and scapular muscle region can help prevent the vicious cycle of impingement tendonitis, irritation, and muscle weakness that leads to altered kinematics, instability, and further impingement of tissue, such as the long head of the biceps.

Once the biceps tendon has been injured, the first step in proper management is a thorough examination of the anterior shoulder, structures of the shoulder girdle, and, if appropriate, kinetic chain. Failure either to identify associated lesions (e.g., those affecting the glenoid labrum and rotator cuff) or to note contributing factors (e.g., poorly stabilized scapula, hypomobile cervical and/or thoracic spine, or altered muscle recruitment patterning) may lead the clinician to direct the sole treatment to the affected biceps tendon rather than addressing the true cause of the shoulder injury. Depending on the specific nature and severity of the biceps injury, treatment can range from managing an early, acute inflammatory situation locally at the tendon to treating the biceps tendon by retraining the scapular stabilizing muscles and rotator cuff to properly position the glenohumeral joint and therefore reduce impingement on the long head of the biceps anteriorly. Whichever is the case, the message is always the same: the clinician must make sure that treatment encompasses not only the affected tissue but also the causal tissue.

NERVE INJURIES OF THE SHOULDER

Nerve injuries of the shoulder represent a relatively small percentage of overall problems reported.[43,161–165] They are most commonly caused by direct trauma but also may occur as a result of repetitive microtrauma. In some instances, nerve injuries affecting the shoulder occur secondary to the predominant injury (e.g., axillary nerve damage from an anterior glenohumeral dislocation) or they may be the consequence of an underlying shoulder condition (e.g., poor posture and/or scapular instability that leads to thoracic outlet syndrome). Nerve injuries affecting the shoulder are being recognized with increasing frequency in sports, such as football and rugby, that comprise higher risk of injury from direct contact.[162–164,166] Nerve injuries as a result of compression and/or stretching from muscle hypertrophy have been reported in weight lifters, body builders, and/or volleyball players as well as individuals who perform repetitive motions in their occupations, such as computer analysts and assembly line workers.[161,163]

It is important to note that a fair number of neurological-like symptoms that patients experience may originate from sites well beyond the actual shoulder. These could include the spinal cord, cervical disc, cervical or thoracic nerve roots, nerve branches, and peripheral nerves, as well as unsuspected sites (e.g., the pleural or abdominal cavity) that can refer pain to the shoulder. Patients with nerve injuries often describe inconsistent, vague symptoms that may involve some combination of pain, paresthesia, or motor weakness in the limb; however, this is highly variable and depends on the patient's particular injury and the component of the nerve affected.[161,163,165] To make a diagnosis, the clinician must recognize the muscles innervated, sensory distribution, muscle stretch reflex, and compressive sites associated with a particular nerve. Commonly affected nerves in the shoulder region include the dorsal scapular, long thoracic, suprascapular, axillary, and musculocutaneous nerves. Table 5-7 presents the clinical features associated with injury to these nerves. Conservative management of most nerve injuries includes avoidance of precipitating factors to allow

TABLE 5-7

Clinical Findings of Nerve Injuries About the Shoulder

Nerve	Injury Site	Motor Findings	Sensory Findings	Other Findings
Dorsal scapular nerve	Within scalenus medius muscle	Weakness in pressing the elbow backwards against resistance while hand is on hip or in pushing the palm backward against resistance with arm folded behind the back	None	Scapular winging with lateral displacement; shoulder pain
Long thoracic nerve	Supraclavicular region	Weakness in raising arm above the head	None	Scapular winging when patient pushes arm against the wall while elbow is extended
Suprascapular nerve	Suprascapular notch Spinoglenoid notch	Weakness in both abduction and lateral (external) rotation of arm Weakness of only lateral (external) rotation of arm	None	Deep scapular pain
Axillary nerve	Lateral aspect of the humerus and quadrilateral space	Weakness of abduction of shoulder between 15° and 90°	Lateral shoulder	Deltoid atrophy
Musculocutaneous nerve	At the level of the coracobrachialis	Elbow flexor and forearm supination weakness	Lateral forearm	Proximal forearm pain aggravated by elbow extension; reduced biceps reflex

recovery, physical therapy, and anti-inflammatory medications. Most injuries resolve spontaneously within 6 to 24 months.[161–165] For nerve injuries that have failed conservative treatment, surgical techniques involving various combinations of fascial graft or transfer of adjacent muscles may be effective.[167] A more detailed description of nervous tissue and the pathology of nerve injuries about the shoulder can be found in Chapter 25 and in Volume 2 of this series, *Scientific Foundations and Principles of Practice in Musculoskeletal Rehabilitation*, Chapter 8.

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CHAPTER 6

Shoulder Instability

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INTRODUCTION

The impression gained from a cursory glance at the shoulder belies its complexity. This proximal part of the upper kinetic chain starts at the trunk and includes the scapula, glenohumeral joint, acromioclavicular joint, and sternoclavicular joint.[1] As part of the kinetic chain, the shoulder acts as a funnel, transferring the forces generated by the lower extremity and trunk to the arm.[2–5] The human shoulder has evolved to allow an incredible range of motion (ROM) for reaching and placing the human hand through a large functional arc, largely at the expense of bony stability. Elevation, depression, protraction, retraction, and rotation of the scapula, along with elevation and rotation of the clavicle, enhance or facilitate the motions of forward flexion, extension, abduction, adduction, medial and lateral rotation, and circumduction of the glenohumeral joint, which allow the arm and hand to be placed in many positions. Unfortunately, every anatomical adaptation toward mobility creates the potential for problems, which can be compounded by the large ROMs needed, frequent repetitions, and high stress loads seen in sports and occupations involving the upper limbs.

It is imperative that clinicians consider the whole kinetic chain as well as the shoulder girdle, rather than any one articulation when assessing the shoulder. With less bony support than other articulations, the shoulder girdle must be supplemented by strong ligaments and careful arrangement of all muscle groups to ensure stability.[7,8] With such a large ROM available at the shoulder, glenohumeral stabilization comes from the osseous anatomy, specifically the "pear-shaped" glenoid (Figure 6-1), as well as joint compression. This "concavity" compression is the result of precise sequencing of all the shoulder muscle groups involved along with the labrum, glenohumeral ligaments, and joint capsule.[9–12]

FUNCTIONAL ANATOMY

Although bony containment is lacking in the scapulohumeral articulation, the articular surfaces are positioned to contribute to stability, especially with the arm in elevated positions. For example, the scapula faces 30° anteriorly to the chest wall and is tilted upward 3° to augment functional reaching motions above shoulder height.[3,13,14] The "pear-shaped" glenoid is almost a perfect circle, being much wider in the lower half than in the superior half. It is also tilted upward 5° to help to control inferior instability and to further augment movements above shoulder height (see Figure 6-1).[15]

At the glenohumeral joint, a large humeral head articulates with the shallow glenoid cavity, which is deepened by the glenoid labrum. The surface area of the humeral head is significantly larger than the glenoid.[13,16,17] However, the articular surfaces of the humeral head and glenoid are functionally congruent, having radii within 3 mm. During movement, the area in contact between the two surfaces constantly changes, with the greatest contact in mid-elevation rather than at the extremes of glenohumeral motion, where the potential for instability is greatest.[18] Only approximately 25% to 30% of the large, spherical humeral head is in contact with the glenoid surface at any one time.[13,19] Stability in the glenohumeral joint is achieved through several factors: the bony glenoid, labrum, glenohumeral ligaments, rotator cuff, negative pressure within the joint capsule, and coefficient of friction of the synovial fluid.

During 180° of elevation of the arm, there is 120° of glenohumeral motion, along with 60° of scapulothoracic motion. This large ROM (i.e., scapulohumeral rhythm) is brought about by precise muscle sequencing, and the resultant force couples generate rotary torques on the scapula and humerus, creating complex arm movement patterns. The term *force couple* refers to muscles working together by applying different forces to create a common motion—some act concentrically to initiate and provide movement or control movement and others act isometrically, concentrically, or eccentrically to control or decelerate movement (Figure 6-2).[20] For example, abduction by the deltoid, which is part of a force couple, is counterbalanced by the supraspinatus, infraspinatus, and subscapularis, the other part of the force couple, to allow abduction by compressing the humeral head in the glenoid and preventing its

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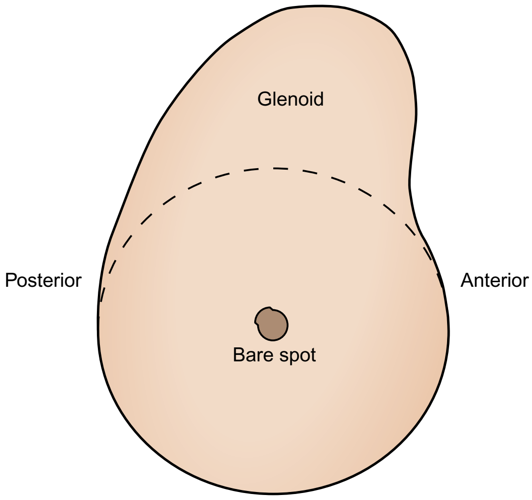

Figure 6-1 "Pear shape" of the right glenoid. (Redrawn from Lo IKY, Parten PM, Burkhart SS: The inverted pear glenoid: an indicator of significant glenoid bone loss, *Arthroscopy* 20(2):171, 2004.)

elevation under the acromion (see Figure 6-2, *A*). Similarly the serratus anterior is counterbalanced by the three parts of trapezius, levator scapulae, and rhomboids to control the scapula (see Figure 6-2, *B*).

Any structural injury, scapular dyskinesia (i.e., abnormal positions or patterns of movement of the scapula), or dysplasia (i.e., abnormal development) of the humerus or the glenoid or its labrum can affect the stability of the glenohumeral joint.[21] Normally, translation of the humeral head on the glenoid is limited to a few millimeters in every direction during movement.[20,22–24] If the dynamic structures controlling this translation (i.e., primarily the rotator cuff) or the passive structures (e.g., the labrum or ligaments) are injured, this translation increases. The increased translation leads to increased stress on the glenoid labrum, failure of the static restraints (i.e., ligaments) (see Table 5-1), and eccentric overload of the dynamic restraints (i.e., rotator cuff and biceps), which work individually and in unison to provide stability in the joints of the shoulder, where mobility is paramount. The result is instability, impingement because of the translation and/or abnormal movement, or both.[25,26]

Any alteration in biomechanics or trauma may lead to a tear in the glenoid labrum. The glenoid labrum increases the glenoid depth approximately twofold, which contributes to glenohumeral stability.[13,16,17,19] However, labral anatomy can be quite variable, even being deficient in certain locations. Such variations often occur in the anterosuperior location and are referred to as a sublabral foramen or **Buford complex**.[27–29] A **Bankart lesion** is a traumatic labral tear that usually occurs with an anterior dislocation. However, the labrum can tear anywhere along its attachment on the glenoid. A Bankart lesion can also be a bony lesion. This typically occurs in the anteroinferior quadrant of the glenoid

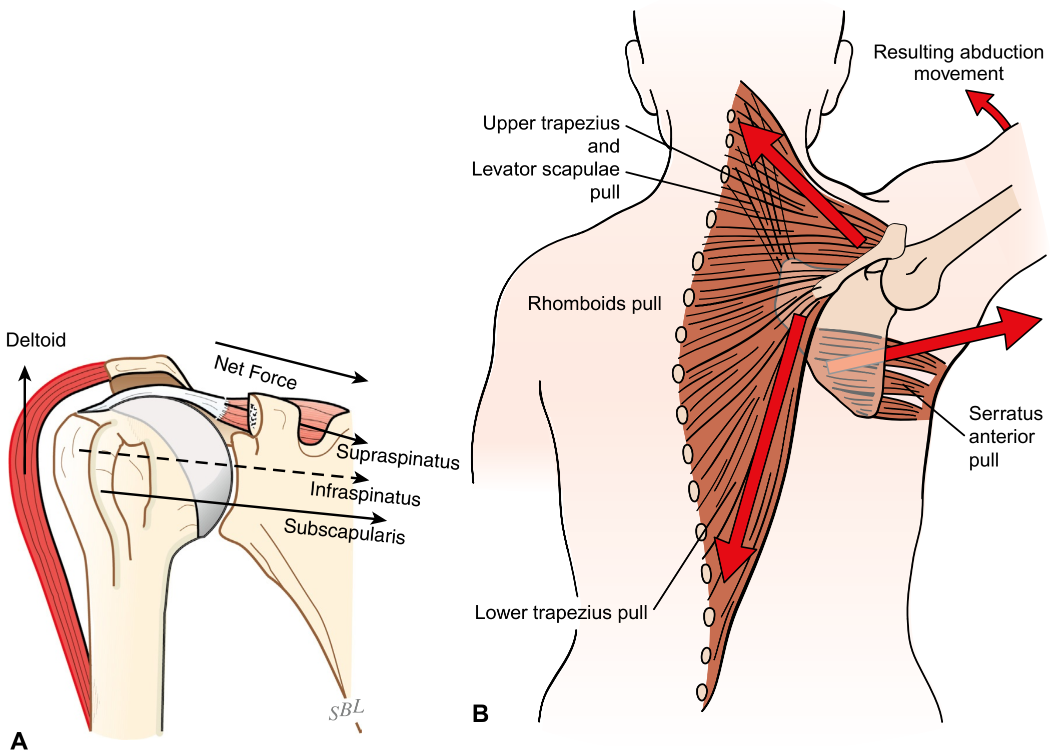

Figure 6-2 Force-couple examples about the shoulder. **A**, Glenohumeral joint. **B**, Scapula. (**A** Modified from Matsen FA, Lippett SB, Sidles JA, Harryman DT, editors: *Practical evaluation and management of the shoulder*, p 98, Philadelphia, 1994, WB Saunders.)

after traumatic anterior glenohumeral dislocation. As the size of the bone fragment increases, so does the risk of recurrent dislocation, especially in young, contact athletes. A **Hill-Sachs lesion** is the corresponding lesion or impaction fracture that usually occurs in the posterolateral aspect of the humeral head as the soft cancellous bone of the humeral head rests on the hard cortical glenoid bone during a traumatic glenohumeral dislocation. A condition that occurs in the superior labrum anterior and posterior (SLAP) to the biceps, **SLAP lesion**, is a lesion around the long head of the biceps where it crosses over the glenohumeral joint. Single or repetitive trauma can cause the labrum to fray, tear, or peel back from the bone, resulting in a SLAP lesion.[2–4]

The surrounding soft tissue envelope (i.e., the capsule) is the primary contributor to stability in the normal glenohumeral joint, with a normal lax inferior capsule allowing movement into full elevation. The capsule is reinforced by the coracohumeral and glenohumeral ligaments, which act as checkreins at the extreme ROM, assisting the compression caused by the dynamic rotator cuff.[21,29] These two components augment normal scapulohumeral rhythm to create functional stability. The concavity formed by the glenoid labrum, along with the muscles that provide compression and the unique tightening mechanism of the inferior glenohumeral ligament, provides a "concavity-compression" mechanism that stabilizes the glenohumeral joint.[13,21]

The glenohumeral ligaments, which are thickenings in the capsule, show a high degree of variability, especially the superior and middle glenohumeral ligaments. Of the three, the inferior glenohumeral ligament is the most important and shows the least variability. It acts like a hammock for the humeral head, preventing some inferior translation, with the anterior and posterior bands tightening on rotation (torsion), which, in turn, limits anterior and posterior translation.[24] This ligament takes on even greater importance with glenohumeral elevation. At 0° abduction, the subscapularis muscle, labrum, and superior glenohumeral ligament are the primary restraints to anterior translation. At 45° abduction, the subscapularis muscle and middle and inferior glenohumeral ligaments, along with the labrum, prevent anterior translation. When the arm is abducted more than 90°, which is the usual position of anterior dislocation, the anterior fibers of the inferior glenohumeral ligament are the primary restraints to anterior movement.[13,16,17,19,30] The long head of the biceps tendon can sometimes act as a secondary dynamic support to reduce stress anteriorly in the abducted and laterally rotated position.[31] Similarly, the posterior band of the inferior glenohumeral ligament prevents posterior translation above 90°.[16]

If the ligaments are hypomobile, they can restrict translation of the humeral head in the glenoid,[32] and restricted translation can adversely affect shoulder motion, especially when the shoulder is under load. If the labrum, capsule, or ligaments are injured or stretched through repetitive microtrauma, such as occurs with repetitive throwing or swimming activities, the resulting hypermobility can lead to functional instability even without major trauma.

The tendons of the rotator cuff have important dynamic roles, but they can perform these roles only when acting from a stable scapular base from which they all originate. The muscles serve in a complementary function to adjust the tension in the capsuloligamentous system.[33] It is possible that stretch receptors within the capsular ligaments are activated by tension to induce selective contraction of the surrounding musculature, protecting these structures at the extremes of motion.[34] As stated previously, contraction of these muscles compresses the humeral head into the glenoid cavity and increases the force needed to translate the humeral head.[16,35,36] Cadaveric studies have shown that simulated maximal contraction of the posterior rotator cuff muscles reduces anterior ligamentous strain and posterior rotator cuff contraction reduces anterior ligamentous strain.[37]

A second and equally important group of muscles that affects glenohumeral stability is the scapular control muscles (see Table 5-1). These muscles control (stabilize) the scapula, which is the dynamic base (i.e., the origin) for the rotator cuff, biceps, and triceps (long head) muscles. Through their action on the scapula, these muscles position the glenoid beneath the humeral head, adjusting for the changing position of the arm. With scapular dyskinesia, failure to maintain the stable glenoid platform (i.e., the scapula) may occur, causing an alteration in scapulohumeral rhythm and mild-to-severe winging of the scapula.

These concepts form the basis for rehabilitation of the unstable shoulder. Four key components must be addressed in rehabilitation: (1) normal scapulohumeral rhythm, (2) normal arthrokinematics at the joint, (3) balancing of the ligamentous structures, and (4) normal function of the rotator cuff muscles, biceps and scapular stabilizers (normal osteokinematics).[1]

PRINCIPLES OF SHOULDER ASSESSMENT

The essential ingredients for assessing the spectrum of shoulder injuries are a detailed history, identification of the precipitating or perpetuating factors (i.e., the mechanism of injury), and a careful physical examination, including special tests for differential diagnosis. Palpation of the specific structures generating the pain or dysfunction concludes the physical examination. This process should lead the examiner to choose the specific investigations (e.g., diagnostic imaging) needed to confirm the diagnosis, and it also provides a framework that is essential to successful treatment.

Key Elements for a Clinical Diagnosis

- Recognition of the salient points in the history
- Identification of precipitating or perplexing factors
- Careful examination including individual muscle testing
- Palpation of pain-generating structures

In the shoulder, dysfunction and pain may arise from a variety of sources, including the scapulohumeral complex, acromioclavicular or sternoclavicular articulations, rotator cuff and other shoulder muscles, associated subacromial (subdeltoid) bursa, biceps tendon, or labrum. Pain may even be referred from the cervical and thoracic spine and ribs. Shoulder pathology shows a common pattern of pain along the anterior glenohumeral joint line and biceps tendons, at the origin of the levator scapulae, and at the teres minor on the posterior joint line. Pain also may be present at the deltoid insertion and upper trapezius, along the medial border of the scapula and, in some cases, over the whole scapula.

When evaluating pain, clinicians should keep in mind the patient's age, occupation, recreational goals, and side dominance. The examiner must make sure that the patient is not confusing pain with weakness, instability, or apprehension. The mode of onset is important and may present as a spectrum of injury mechanisms, from a single traumatic episode to repetitive microtrauma.

Detailed examination of the shoulder can be found elsewhere (see Volume 1 of this series, *Orthopedic Physical Assessment,* Chapter 5). When assessing the shoulder, the examiner must keep in mind several important concepts:

1. *Shoulder pain must always be distinguished from referred pain arising from the cervical spine* (Table 6-1). The embryological derivation of the shoulder girdle from the cervical myotomes results in a close relationship for specific innervation, as well as for referred pain. With any complaint of shoulder girdle discomfort, cervical pathology must be ruled out. Neck pain and dysesthesia (i.e., numbness, tingling, burning) that radiate past the elbow in a dermatomal distribution normally implicate cervical disc disease, although nerve entrapment at the elbow or wrist or a local pathological lesion in the hand must be considered.[1] True shoulder pain rarely extends below the elbow. In any patient over age 50, a coexisting cervical degenerative abnormality must also be considered.[38]
2. *Generally shoulder pathology is characterized by pain on use, weakness, stiffness, and guarded apprehension.* (In many cases, the patient reports that the shoulder "just doesn't feel right.") These findings help distinguish shoulder pathology from cervical pain, which often is present even at rest and generally is aggravated by chronic compensatory postures, such as sitting, reading, or studying at a desk. Neck and shoulder pain cannot be totally isolated. The clinical points that are most suggestive of pain of spinal origin include absence of shoulder tenderness, guarded cervical spine motion, decreased biceps strength and reflex, and a positive cervical compression test (see volume I of this series, *Orthopedic Physical Assessment,* Chapter 3).[1] The most common pattern of cervical disc disease (i.e., spondylosis) refers into the C5-C6 dermatome with pain and/or paresthesia over the shoulder, the lateral arm, and even into the forearm and the thumb side of the hand.
3. *The patient's ability to maintain a static and dynamic stable core is important in shoulder rehabilitation.* A stable core (i.e., the ability to maintain control of the pelvis and spine) is the transfer zone between the lower extremity and the upper extremity, and as such, it plays a significant role in shoulder rehabilitation. If the practitioner tries to correct shoulder and/or cervical posture without addressing the unstable or incorrectly positioned core, treatment will be unsuccessful.
4. Neer[39] thought that mechanical "*impingement" could sometimes occur against the anterior edge of the acromion, coracoacromial ligament, or inferior acromioclavicular joint.* Others think that the anterior pain is due more to a complex interplay of rotator cuff dysfunction and degeneration ("rotator cuff disease") rather than impingement.[40] Structural narrowing of the subacromial space may occur secondary to inferior acromial tilting (i.e., anti-tilt), the so-called hooked acromion (i.e., type III), an unfused acromial ossification center called an **os acromiale**, or subacromial spurring. In addition, acromioclavicular joint hypertrophy and marginal osteophytes may irritate the rotator cuff.[9] Postural changes, particularly slouching with rounded shoulders and a protruding, chin-forward head posture, contribute to impingement. Magnetic resonance imaging (MRI) has demonstrated narrowing of the subacromial space as the shoulder girdle moves from a retracted to a protracted position.[41]
5. *Stability of the shoulder complex depends on an intimate relationship between the muscles controlling the scapula and those controlling the humerus.* The examiner must ensure correct functioning of the periscapular

TABLE **6-1**

Factors Suggesting the Site of Origin of Shoulder Pain

Neck Pathology	Shoulder Pathology
• Pain at rest • Pain with neck motion • Pain with overpressure on the neck • Pain aggravated by postural positions • Pain past the shoulder • Reflex changes • Altered peripheral sensation • Guarded cervical spine motion	• Pain with use • Pain when working overhead • Feeling of instability • Local palpable tenderness • Painful arc of motion • Pain into the deltoid area • Pain mainly on the dominant side • Pain relief with local injections

From Zachazewski JE, Magee DJ, Quillen WS, editors: *Athletic injuries and rehabilitation,* p 511, Philadelphia, 1996, WB Saunders.

stabilizers and the rotator cuff (i.e., inner cone muscles). Commonly, the power (i.e., outer cone) muscles (i.e., the pectoralis major, deltoid, and latissimus dorsi) are not the problem, although they may contribute to the problem by being tight or substituting for the inner core muscles. Weakness or injury in any of the inner cone muscles leads to an imbalance of the synergistic inner cone (i.e., stabilizer) and outer cone (i.e., mobilizer) muscles and the prime mover muscles, resulting in abnormal shear forces which, in turn, can lead to instability (i.e., functional subluxation), labral injury, impingement, muscle imbalance, abnormal joint mechanics, and/or rotator cuff tears.[6,7,42] A thorough, detailed assessment of the strength and flexibility of each individual muscle of the shoulder complex is essential to identify weak and tight links in the kinetic chain and to ensure proper return to correct function. (For muscle testing, the reader is referred to the appropriate muscle testing books.[43–45])

6. *The examiner should watch for scapular dyskinesia, especially if the patient is under 35 and has anterior shoulder pain.*[2–4,46–48] If the scapula cannot be controlled by its stabilizer muscles, extra load is transferred to the humeral control muscles and they become overused, which leads to tendon and labral problems. Scapular instability is a common cause of rotator cuff and bicipital tendonitis or tendinosis and secondary impingement. A functional scapular base is essential for correct shoulder movement, especially in sports.[49]
7. *Hypomobility can be a major issue in shoulder instability and impingement.* The clinician must determine the mobility of the glenohumeral joint capsule (especially posteroinferiorly), ribs, scapula, and acromioclavicular and sternoclavicular joints, as well as assessing for tight muscles (e.g., pectoralis major and minor). Addressing tight structures helps to ensure normal shoulder arthrokinematics and function.[2–4] For example, glenohumeral arthrokinematics are markedly altered by a **glenohumeral internal (medial) rotation deficit (GIRD)** or by a tight posterior capsule when medial (internal) rotation is limited and lateral (external) rotation is excessive (also called **glenohumeral external (lateral) rotation gain [GERG]**). One theory proposed by Burkhart and coworkers[2–4,29] states that GIRD of 10° to 20° has a beneficial effect of offsetting the center of rotation of the glenohumeral joint posterosuperiorly. The advantage is that the greater tuberosity can "clear" the posterosuperior glenoid and labrum. Unfortunately, this process can get worse; with medial (internal) rotation deficits greater than 20° of the contralateral shoulder, combined with loss of the total motion arc, there continues to be stress on the superior labrum, and a SLAP lesion eventually results. Thus, a tight posterior capsule results in a pathological "decentering" of the humeral head.
8. *Immobility of the thoracic spine and ribs can significantly influence the movement patterns of the shoulder complex and can affect the dimensions of the subacromial space.* A careful assessment of the cervical and thoracic spine and ribs is an important part of any shoulder assessment.[50]
9. *Any shoulder rehabilitation program must involve assessment of the whole kinetic chain.*[3] The clinician cannot assess the upper quadrant in isolation. Power movements and accelerating motions begin in the lower limb and trunk musculature. In patients with shoulder problems, the clinician should look for inflexibility in the hips (especially on the nondominant side) and in trunk rotation and the anterior shoulder muscles, along with weakness of the hip abductors and the pelvic core.[2]
10. *Exertional left shoulder pain in individuals over age 45 with coronary risk factors should be considered as cardiac pain until proven otherwise.* Coronary risk factors include smoking, obesity, hypertension, and a family history of cardiac disease.
11. *Pain from the lungs and abdominal organs can be referred to the shoulder, and injury to these structures should be considered in conditions involving trauma.*
12. *The initial observation should include the thoracic spine, ribs, and shoulder girdle and requires adequate exposure of the patient.* Viewing the disrobed patient from behind allows the clinician to note any wasting of the shoulder girdle muscles, especially the supraspinatus, infraspinatus, serratus anterior, and posterior deltoid. Such wasting may indicate a nerve injury (e.g., suprascapular, long thoracic, or axillary nerve)[51] or apparent hypertrophy of the upper trapezius as a result of superior migration of the scapula (i.e., type III dyskinesia).[2–4,46–48] The presence and size of stretch marks or scars often reflect poor quality collagen and may be a clue to the possibility of generalized ligamentous laxity or steroid abuse in athletes. Excessive lordotic curvature, a kyphotic thoracic spine, and/or associated poking chin with rounded shoulders should be noted. The clinician must assess for lack of pelvic or core control with these postural problems, which often contributes to shoulder problems. In addition, these anterior and posterior postural imbalances can lead to a tight pectoralis major and minor, resulting in increased stress on the anterior capsule, tightness of the posterior capsule, and overuse of scapular control muscles.[51]
13. *The examiner must always be aware of the possibility of referred pain.* The response to palpation must be put in the context of the patient's presentation and the implications of this presentation (e.g., secondary gain, pain threshold) must be judged correctly.

14. *Clinicians must know their patients.* The patient may have other issues and agendas that affect the clinical presentation.

ROTATOR CUFF DEGENERATION AND DYSFUNCTION (SHOULDER "IMPINGEMENT")

Shoulder impingement or "pinching" is associated with various degrees of inflammation or rotator cuff degeneration. It frequently occurs as a result of microtrauma, macrotrauma, or functional instability. The suspicion of impingement is raised by the presence of a painful arc during attempted active abduction or forward flexion or in patients with a complaint of anterosuperior or posterior shoulder pain with no history of trauma. There has been a tendency to move away from calling pain in the anterior region "impingement" because this implies that this is largely a mechanical phenomenon alone, when, in fact, it may be a more complex interplay of rotator cuff dysfunction and degeneration and less to do with attritional irritation from rotator cuff impingement.[40]

The subacromial space can be identified as a source of impingement by taking the arm passively into full forward elevation with gentle overpressure (i.e., Neer impingement test).[51] This test can be supplemented with the Hawkins-Kennedy impingement test, which involves taking the patient's arm to 90° of abduction and then moving it into horizontal adduction, followed by forced medial rotation.[51] The Hawkins-Kennedy impingement test can be enhanced by having the patient resist a force applied downward to the dorsum of the hand. A positive Hawkins-Kennedy test result implies impingement of the rotator cuff under the acromion. Historically a positive Neer impingement test result is thought to represent impingement of the rotator cuff on the anterosuperior glenoid rim or coracoacromial ligament.[52] Unfortunately, the sensitivity and specificity of these tests are quite poor and localizing the pain to one area is quite difficult.[53] Many sources of pain exist in this anterior region, including the bursa, rotator cuff, long head of biceps, anterosuperior labrum, and even the acromioclavicular joint.

Shoulder pain caused by impingement would seem to be easily distinguishable from that caused by posttraumatic instability. However, over time, clinicians have come to recognize that these conditions comprise a spectrum of both injury and pathology (Figure 6-3). At one end of the spectrum is an unstable painful shoulder along with rotator cuff degeneration and dysfunction, and at the other end is frank dislocation.[54,55] Rotator cuff degeneration is most often the result of a gradual attrition and irritation of the tendons that is associated with pain and dysfunction and that often is the result of repetitive overuse. Frank dislocation frequently goes on to become a recurrent dislocating joint. Similarly the spectrum of natural collagenous laxity ranges from extreme tightness to abnormal looseness; therefore the degree of

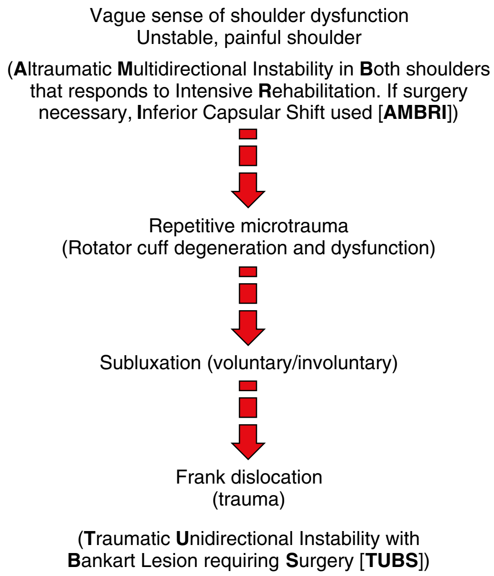

Figure 6-3 Shoulder instability spectrum. (Modified From Zachazewski JE, Magee DJ, Quillen WS, editors: *Athletic injuries and rehabilitation*, p 520, Philadelphia, 1996, WB Saunders.)

trauma or overuse required to generate a dislocation or subluxation varies.

Between these extremes lies the mildly unstable shoulder that predisposes the patient to impingement and pain; this condition is often seen in athletes, such as swimmers, and assembly line workers. The throwing shoulder in which the pitcher alters the mechanics to increase range of lateral rotation to get into the "slot" is another example.[2]

The aging process also fits into this spectrum, and its effects are manifested more in the soft tissue rotator cuff and in the joint surface itself.[52] Furthermore, internal derangements in the form of labral tears, attrition of the rotator cuff muscles, and degeneration of or partial tears in the biceps tendon have begun to be diagnosed with more prevalent use of the arthroscope and the increasing availability of MRI and computed tomography (CT) scanning. As a result, the previously simple, distinct diagnostic categories have become blurred into a spectrum of injuries.

Rotator cuff degeneration and dysfunction is often the result of cyclical repetitive abduction, forward flexion, and medial rotation motion at the glenohumeral joint.[56,57] Such movement is seen in swimmers who commonly swim approximately 10,000 to 14,000 m (6.2 to 8.7 miles) or more per day.[52] Impingement may also occur when the arm is forward flexed, medially rotated, and abducted within the coracohumeral space. This **coracoid impingement syndrome** occurs when the lesser tuberosity of the humerus encroaches on the coracoid process and has been associated with subscapularis tears.[58,59] The condition has been reported in swimmers,

Figure 6-4 Posterior internal impingement. **A**, Posterior contact between the glenoid lip and the insertion of the cuff to the tuberosity occurs in the apprehension, or fulcrum, position, especially if the anteroinferior capsule has been stretched, allowing the humerus to extend to an unusually posterior scapular plane. This contact can challenge the integrity of the posterior cuff insertion and the tuberosity. **B** and **C**, Activity positions causing potential posterior impingement. (**A**, From Matsen FA: Stability. In Matsen FA, Lippett SB, Sidles JA, Harryman DT, editors: *Practical evaluation and management of the shoulder*, p 98, Philadelphia, 1994, WB Saunders.)

tennis players, weight lifters, and brick layers. With force overload, the humeral and scapular control muscles (see Figure 6-2) become fatigued, resulting in muscle weakness and muscle imbalance in the scapulohumeral force couples, which leads to incorrect movement patterns.[56,57] The resultant abnormal shear stresses can lead to secondary impingement.

Another type of impingement occurs in individuals over age 40, arising from alterations in the soft tissues (e.g., rotator cuff, labrum) and possibly bony changes. This has been called **primary anterior (external) impingement**. Although it has been classically thought of as a pure mechanical process of irritation and attrition of the rotator cuff secondary to physical contact between the anterosuperior rotator cuff and under the surface of the acromion, it is now recognized that this is a largely oversimplified point of view.[40] Although some mechanical irritation certainly occurs, it is unlikely that this is the source of the problem and is more likely the result of an aging, fatigued, and degenerating rotator cuff. This patient seldom has instability.[5,60,61]

In younger patients (ages 15 to 35), muscle dynamics is the primary problem (i.e., weakness of the scapular and humeral stabilizers). This has been called **secondary anterior (external) impingement**, which primarily is due to scapular dyskinesia and posterior rotator cuff fatigue.[5,60,61]

The fourth type of impingement is **posterior internal impingement** (Figure 6-4), which involves contact of the undersurface of the rotator cuff with the posterior superior glenoid labrum.[62–64] Burkhart et al.[3] reported that posterior internal impingement is a normal phenomenon that sometimes can result in pathology of the rotator cuff and labrum.

If significant inflammation is present, impingement tests are less specific because all testing positions tend to be uncomfortable. Very inflamed tissues are uncomfortable even on passive stretching or when put under tension by contraction of the associated muscle. By testing for pain in the nonimpingement position for each specific structure, the clinician can distinguish and isolate the inflammatory response of the tendon from a simple impingement area. For example, a partial rupture of the biceps tendon within the joint may give positive impingement signs and may also be maximally painful with resisted forward flexion. The clinician can further confirm the diagnosis by injecting approximately 3 mL of 0.25% to 1% lidocaine (Xylocaine) directly around the most painful areas (or 5 mL into the subacromial space) and then waiting a suitable interval (usually a few minutes) before repeating the impingement test.[62] Relief of at least 50% of the patient's pain through the painful arc or the impingement positions helps to confirm the source of the pain. However, it is important to keep in mind that although the source of the pain has been found, the source of the real problem may be more elusive. Most nontraumatic shoulder pain is the result of posterior muscle problems involving the muscles stabilizing the scapula resulting in loss of scapular stabilization or control, overuse of the rotator cuff, loss of dynamic thoracic and pelvic control, and muscle imbalance in all four areas. In addition, when a rotator cuff tear is suspected, elimination of most of the pain allows a more specific examination of strength. The classic findings with a rotator cuff tear are pain even at rest (and especially at night), wasting of the supraspinatus and infraspinatus muscles, weakness of scaption (i.e., abduction in the scapular plane), and loss of lateral rotation strength. Small tears or partial-thickness tears may generate impingement signs without significant loss of rotator cuff strength. Diagnostic tests, such as ultrasound, MRI, or CT arthrograms can help to identify the true problem.

Functional Assessment of the Unstable or Dysfunctional Rotator Cuff and "Impinging" Shoulder

As the clinician goes through the assessment of the shoulder, he or she should carefully watch how patients position themselves and their shoulders to do the tasks they are asked to do. Proper alignment and a "streamlined" posture (i.e., major joints in line, ears squeezed by the arms, pelvis in neutral, feet and hands together, head in neutral) both in standing and sitting are key both during assessment and when doing exercises (Figure 6-5). Patients must be able to place the pelvis "in neutral" (anterior superior iliac spine [ASIS] one to two finger widths lower than posterior superior iliac spine [PSIS]) with the lower spine in proper postural alignment. Any alteration resulting in poor posture or altered scapular stabilization can lead to alterations in the position of the glenoid fossa, which can, in turn, lead to several different sequelae. For

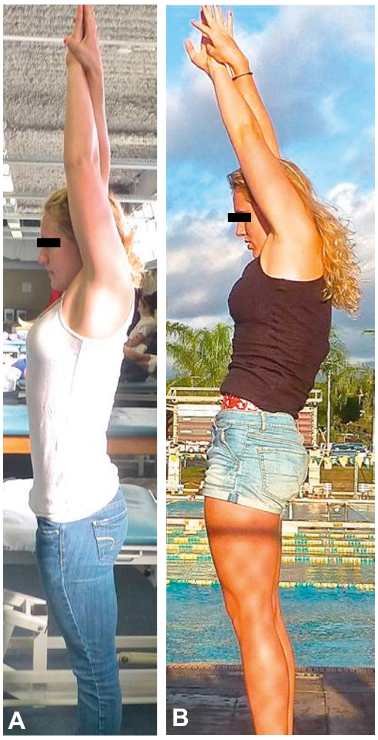

Figure 6-5 Postural alignment. **A**, Correct postural alignment. **B**, Compensatory postures due to hypomobility and weakness.

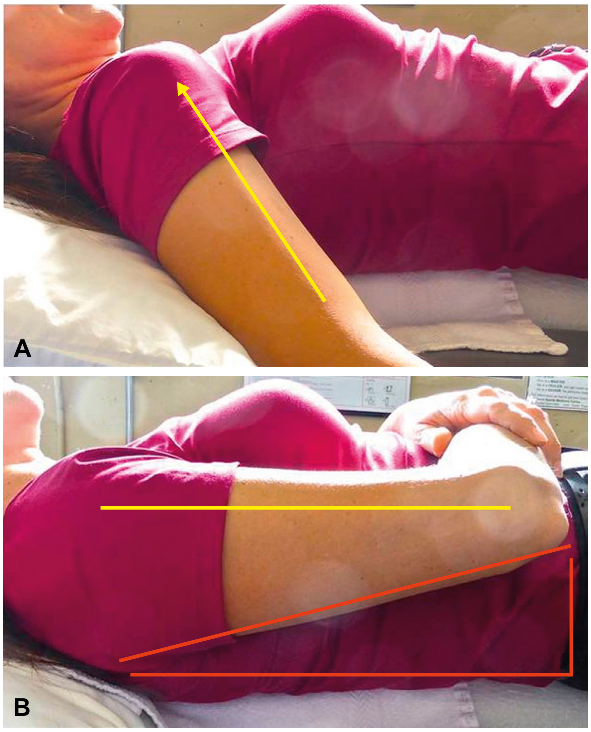

Figure 6-6 Compensatory "shoulder poke" position. **A**, Note how humeral head is translated anteriorly ("pokes" forward) in glenoid as arm extends. **B**, Correct resting position with head of humerus placed "neutrally" in glenoid.

example, when the patient is lying, supine or prone, the humeral head should align with the cervical spine and not project anteriorly (Figure 6-6). Similarly there should be no "shoulder bump" (i.e., shoulder hiking) (Figure 6-7). If the patient is slouching, the head of the humerus shifts anteriorly against the anterior capsule and labrum, resulting in undue pressure on these anterior structures. Shoulder ROM and rib motion both must be observed with motion on both sides of the body compared during the assessment.

Scapular posture should show the medial border of the scapula parallel to the normally straight thoracic spine and approximately 5 to 8 cm (2 to 3 inches) apart (Figure 6-8). There should be no scapular tilt (i.e., the inferior angle tilted away from the ribs), and the inferior angles of both scapula should be horizontally level and equidistant from the thoracic spine. The upper trapezius and levator scapulae should not be tight on palpation. To observe scapular functional movement, the examiner can have the patient hold the core and do a squat posture (Figure 6-9, *A*), with the shoulders in the power square position (Figure 6-9, *B*), open or closed shutters (Figure 6-9, *C*), and fly shutters. Lateral plank exercises may be performed from the knees or ankles as long as the patient can hold proper alignment (Figure 6-10).

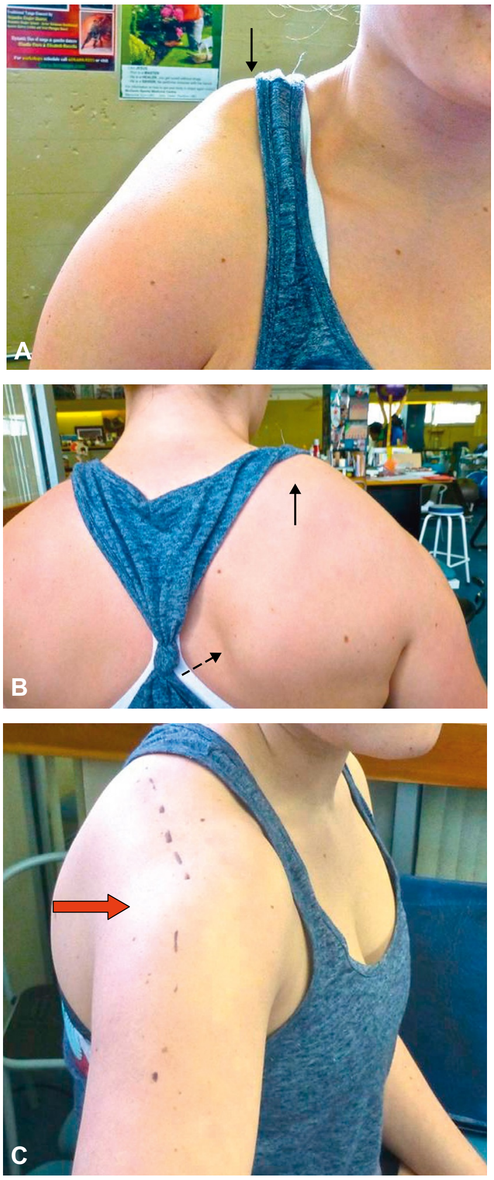

Figure 6-7 Compensatory "shoulder bump." **A**, Tight upper trapezius (*solid arrow*) causing bump. **B**, Tilting of inferior angle of scapula (*dotted arrow*) and tight trapezius (*solid arrow*). **C**, Forward position of humeral head in glenoid (*red arrow*).

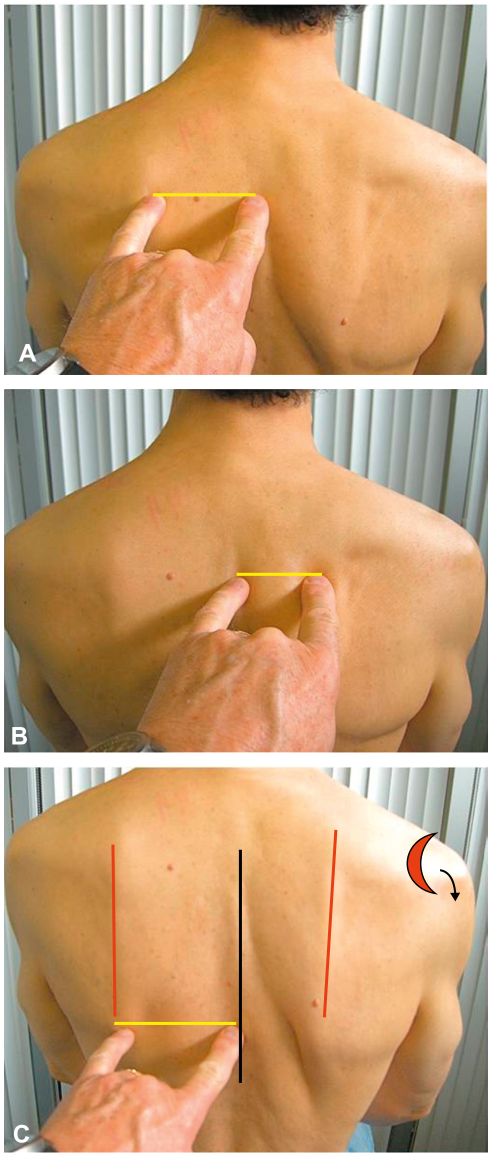

Figure 6-8 Position of scapula. **A**, Normal at level of base of spine of scapula. **B**, Abnormal retraction at level of base of spine of scapula. **C**, Normal position of left inferior angle of scapula. Note angle of scapula on right, resulting in anti-tilt of glenoid.

Treatment of the Unstable Shoulder and Impingement

In the early clinical stages, the patient may complain of aching only after activity, and ROM is preserved. The clinician seldom sees the patient at this point, which is unfortunate, because postural correction involving the scapula, spinal postural line, and pelvis (see Figure 6-5, *A*) along with patient education could play a significant role. If the patient detects the early onset of symptoms, preventive measures can be instituted, such as modification of an activity. For example, during their workouts, swimmers could use a different stroke (e.g., the breaststroke), do only kicking drills, use speed fins, use a center snorkel, or alter their stroke pattern (e.g., different hand entry, altered body roll). The use of resistance devices should be kept to a minimum or, if used, monitored closely to ensure incorrect movement patterns do not develop and the arm is not being positioned in compromising positions. For example, the use of hand paddles or swimming with elastic cords, use of water barrels, or pulling foam can increase the stress on a swimmer's shoulder. In addition, using a flutter board held in front of the head while

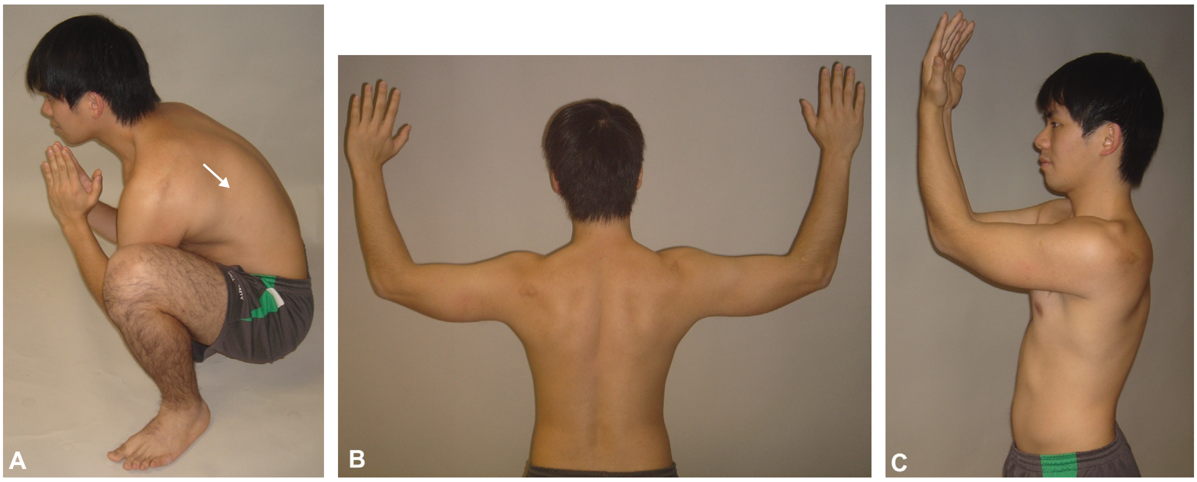

Figure 6-9 Scapular functional movement exercises. **A**, Core-hold squat posture (push against knees, scapula pulled down and back). **B**, Power square position. Also "open" start position for open or closed shutter. **C**, Closed shutters end position.

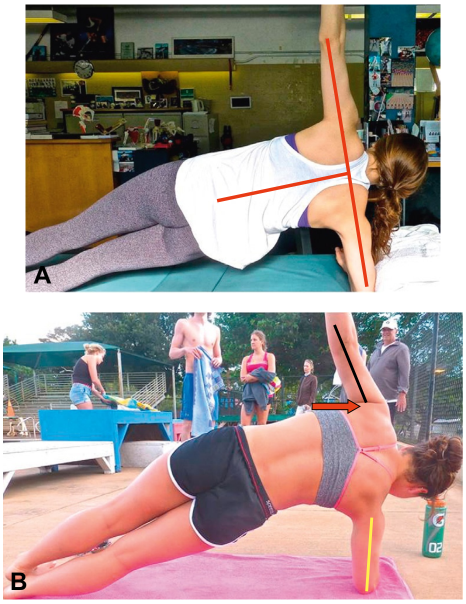

Figure 6-10 Side plank exercises. **A**, Proper alignment–note how humeral head is compressed into glenoid, scapular muscles are engaged, shoulders are square, and spine is straight. **B**, Incorrect alignment with compensatory postures. *Red arrow* shows potential posterior impingement.

Figure 6-11 Protecting the shoulder. The shoulder should NOT be allowed to poke forward. **A**, In supine. Pillow used to put shoulder in its resting position (40°-55° abduction, 30° horizontal adduction [scapular plane]). **B**, Sitting support (note shoulder is not elevated). **C**, Standing support with bolster.

doing kicking strokes in prone position may position the arm in a vulnerable impingement posture.

The patient should be instructed in healthy shoulder care (e.g., positioning or supporting the shoulder in resting positions) (Figure 6-11), the application of ice, especially after a workout, and how to take the shoulders slowly through a full ROM in a controlled fashion while ensuring a dynamically stabilized scapula and normal shoulder activation patterns. The patient should avoid compensatory shoulder impingement positions in activities of daily living and dry land training programs. Ideally, muscle strength should be monitored regularly for weakness of the scapular stabilizers, compensatory movement patterns, loss of ROM (which is demonstrated by unequal movement or range when comparing both sides), pain, and compensatory postures. *Correct posture is an important concept that clinicians must keep in mind and continuously monitor when asking patients to do shoulder exercises.* The practitioner should not allow poor posture with protracted scapulae, which leads to **anti-tilt,** or the position in which the glenoid fossa is no longer superiorly and anteriorly tilted. This anti-tilt position allows the humeral head to "fall" out of the glenoid (Figure 6-12). With instability and impingement, any combination of poor posture or altered scapular stabilization and control can lead to compensation with the glenoid fossa facing inferiorly (anti-tilt). Matsen's "scapular dumping" seen in dislocations[32] is another, more traumatic, example of this glenoid anti-tilt.

As the pathology progresses, the patient experiences discomfort and pain during and after the activity. A positive impingement sign is elicited on examination (i.e., positive Hawkins and Neer tests, restriction in abduction and scaption). Palpation may demonstrate tenderness along the upper corner and medial border of the scapula, over the origin of the levator scapulae, around the insertion of deltoid, and long head of biceps, anterior capsule, and off the lateral edge of the acromion. These patients may also present with tenderness over the infraspinatus and teres minor muscles. Pathological changes may consist of thickening and fibrosis of the tendon, involvement of the subacromial bursa, residual adhesions, and possibly fraying of the labrum.

In the final stages, the patient complains of continuous pain over the supraspinatus and biceps tendons and tenderness over the coracoacromial ligament. A painful arc, especially in the empty can (Jobe's) position, is commonly demonstrated, and ROM can be restricted into medial and lateral rotation motions, which are not equal bilaterally, and impingement signs can be grossly positive. X-ray films may demonstrate infra-acromial and infraclavicular spurs, bony sclerosis near the supraspinatus insertion, and subacromial erosion.

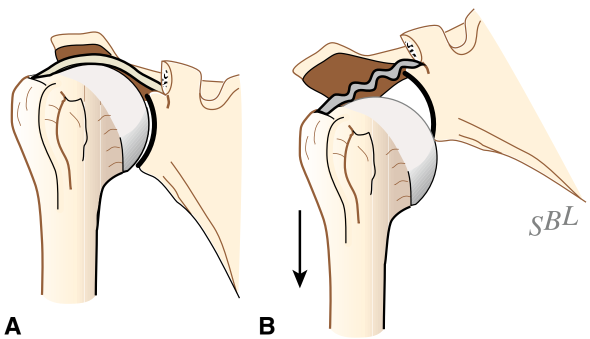

Figure 6-12 Scapular anti-tilt or dumping. With the scapula in a normal position (**A**), the superior capsular mechanism is tight, supporting the head in the glenoid concavity. Drooping of the lateral scapula (**B**) relaxes the superior capsular structures and rotates (anti-tilts) the glenoid concavity so that it does not support the head of the humerus. Conversely, stability is enhanced by elevating the lateral aspect of the scapula. (From Matsen FA, Lippett SB, Sidles JA et al: *Practical evaluation and management of the shoulder*, p 81, Philadelphia, 1994, WB Saunders.)

Surgical Treatment of Rotator Cuff Dysfunction

Surgery is considered a treatment of last resort for impingement-type syndromes. The indications for surgery include unremitting pain and failure to respond to nonoperative, conservative treatment. The timing of surgery depends on the functional disability and pain. There has been a strong trend away from surgical management of this condition, with aggressive physical therapy programs having equal or greater success to surgery.[65–67]

The surgical procedures used historically have been performed arthroscopically and were designed to increase, or decompress, the subacromial space. In the case of anterior impingement, this is achieved by some combination of shaving or excising the undersurface of the anterior edge of the acromion process with release or resection of the distal part of the coracoacromial ligament, thus decompressing the acromioclavicular space. The decision to remove inferior osteophytes or resect the acromioclavicular joint, combining the procedure with diagnostic arthroscopy of the glenohumeral joint and varying degrees of correction of labral lesions, and bicipital tendon pathology usually is a preoperative determination refined by intraoperative findings. It should be noted that several randomized controlled trials all have failed to show the benefit of subacromial decompression in the setting of anterosuperior shoulder pain.[65,66,68–70] Even in the setting of rotator cuff repair, routine use of subacromial decompression appears to not be warranted.[65,66,69,70] Select cases do still exist in which the degree of bony pathology is severe, such as those with huge subacromial spurs encroaching into the subacromial space, and may still warrant a subacromial decompression.[71] Surgical management of coracoid impingement is directed toward subscapularis repair and debridement of the undersurface of the coracoid if it is determined that the space between the coracoid and the humerus is narrowed.[72] Internal impingement is best identified early because surgical management is not often successful in returning the individual back to the same level of activity.[2–4] Once superior labral and rotator cuff damage occurs, the result of surgical repair becomes quite unpredictable. Significant, recalcitrant posterior capsular tightness that has failed to respond to an aggressive stretching program may be addressed by posterior capsular release, which is aimed at reducing GIRD. Associated labral and rotator cuff tearing may warrant debridement and/or repair.[3,73]

As might be expected with the array of surgical decisions to be made, the progress of postoperative rehabilitation varies. The clinician should have accurate and detailed information before starting the planning of the treatment regimen. In the simplest scenarios of surgical debridements and arthroscopic subacromial decompression, gentle ROM can be commenced within the tolerance of discomfort. The initial emphasis is on isometric muscle contractions, with progression to ROM exercises and strengthening. This progression is dictated by the ability to restore appropriate scapulohumeral rhythm and functional scapular control. Impingement positions must be avoided, and the exercises should focus on medial and lateral rotation in neutral, along with forward flexion motions, before abduction motions are initiated. It is better to go slow early, until most of the discomfort has subsided. The clinician should follow the principles of rehabilitation outlined later in this chapter, including those for nonoperative treatment of impingement.

SHOULDER INSTABILITY, SUBLUXATION, AND DISLOCATIONS

Shoulder instability, due either to significant trauma or to inherent ligamentous laxity and overuse with associated functional instability, is a common problem, especially in those under 35 years of age and in individuals who engage in a lot of overhead activity. The condition may be compounded by pseudo-laxity, which is an apparent increase in anterior laxity that results from a decreased cam effect, along with a tear of the superior labrum (i.e., SLAP lesion), congenital joint laxity (i.e., hypermobility), congenital muscle weakness, congenital anomalies, and/or muscle weakness or atrophy after injury.[2]

The glenohumeral joint is the most frequently dislocated major joint in the body. In one case series, glenohumeral dislocations are more common than all other joint dislocations combined.[63] Anterior dislocations of the shoulder account for 80% to 95% of all initial shoulder girdle dislocations, and recurrence is common.[64,74,75]

Classification of these injuries is necessary to develop a logical treatment plan. As previously mentioned, instabilities can range from subtle subluxation to a frank dislocation in which the articular surfaces are no longer in contact (i.e., true dislocation). Recurrent transient subluxation results in a feeling of instability or loss of control due to positioning of the arm. An example of this is forced hyperextension of the arm in elevation and/or lateral rotation, which may result in the "dead arm" syndrome seen in throwing athletes or in tennis players during a serve. In these cases, altered mechanics or traction, or pressure on the nerves generates the symptoms.[2,76,77]

Instabilities are basically classified as traumatic or atraumatic instabilities; Neer added a third type, acquired instabilities.[78] A traumatic dislocation is caused by a single force that applies excessive overload to the joint. This often damages the bony glenoid and/or the labrum and may even disrupt the capsule. This type of dislocation is seen in patients who present with a condition known as a **TUBS lesion** (i.e., *T*raumatic *U*nidirectional instability with a *B*ankart lesion that responds well to *S*urgery), as described by Matsen et al.[32] An atraumatic dislocation usually occurs in patients with multiple joint laxities who frequently have experienced episodes of subluxation before a relatively minor injury results in a dislocation. These individuals often show functional instability (i.e., lack of muscular control) as a result of congenital hypermobility or congenital muscle weakness. This classification includes patients who present with an **AMBRI lesion** (i.e., *A*traumatic *M*ultidirectional instability that is *B*ilateral and the primary treatment is *R*ehabilitation, not surgery;[32] if surgery is required, an *I*nferior capsular shift is often recommended). Capsular laxity is common in these patients, and Bankart lesions are not seen, although the labrum may be frayed.[48]

However, the clinician must keep in mind that overlapping gradations of instability cover the entire spectrum, from TUBS to AMBRI lesions (see Figure 6-3).[25] Individuals with acquired instability who become symptomatic usually are engaged in overhead occupational or recreational activities, such as plastering ceilings, installing ceiling lighting, swimming, gymnastics, and baseball (i.e., pitching), in which the repetitive microtrauma of ill-conceived stretching or rapid, large range motion gradually contributes to capsular stretching. Although the shoulder becomes hypermobile, other joints may test within the normal range. A traumatic episode may push the joint over the edge and produce the first dislocation, but this major episode is only a small component of the problem.[63]

Many clinicians recognize functional instability as a pathological entity, particularly when the joint is put under high stress loads. Scapular control is lacking in these patients, which is one of the clinician's primary concerns.[3] This instability manifests itself from muscle fatigue or scapular dyskinesia after excessive load or repetitions. The mechanical etiology of **functional instability** is uncontrolled translation (i.e., loss of the arthrokinematic control) of the humeral head within the glenoid cavity.[19,56,79–83] Strong, coordinated muscle activity and proper neuromuscular balance contribute significantly to a reduction in functional instability and the often accompanying impingement, and these elements form the basis of treatment.

The glenohumeral instability, which is commonly due to asynchronous firing and fatigue of the scapular and humeral control muscles, can lead to subluxation, rotator cuff strains, and tendonitis or tendinosis injuries. Therefore movement control becomes the primary focus of the clinician.[3,21,84–87] Movement control focuses on maximizing coordinated and correct contraction of muscles through strength, endurance, proprioception, and coordination. Burkhead and Rockwood[8] reported that 80% of patients with atraumatic subluxations had good or excellent results with conservative treatment when appropriate rehabilitation was used. Other researchers are more conservative regarding the efficacy of the outcomes.[88–90] An accurate diagnosis and an appropriate, conservative treatment program can lead to a successful outcome in most patients and are essential for successful rehabilitation.[88] Functional instabilities occur primarily in one direction. However, they can present with global laxity, commonly referred to as *multidirectional instability.*[91]

Voluntary instability, another classification of instability, is usually due to congenital hypermobility or laxity combined with the patient using the shoulder muscles to purposely and spontaneously subluxate the joint.[63,92] For example, these patients commonly say, "Look what happens when I do this," as they voluntarily sublux the shoulder. This condition may provide an element of secondary gain for these individuals, and treatment is difficult. *Involuntary instability* is a recurrent dislocation in a person whose shoulder is so unstable that it dislocates spontaneously.[9]

A dislocation may be acute or chronic. An *acute, frank dislocation* is commonly traumatic and is usually reducible with a variety of manipulation techniques described elsewhere.[93,94] Reduction of a traumatic dislocation by manipulation should be attempted only by an experienced clinician. Occasionally the dislocation is irreducible, and open reduction may be required to extricate the structures preventing reduction. This condition is called a *complex dislocation,* in which interpositioning soft tissue blocks reduction.

With a *chronic dislocation*, the humeral head has been out of contact with the glenoid cavity for a protracted period (i.e., days to years). Unreduced posterior dislocations are the most common chronic dislocations because of the incidence of missed diagnosis. If the chronic nature of the dislocation is not recognized, attempts at closed reduction will be unsuccessful or may even result in a fracture.[75] Chronic dislocations invariably require open reduction. In studies of posterior instability, damage occurs to both the anterior and posterior aspects of the capsule.[94,95]

The principles of diagnosis of a dislocated or unstable shoulder were outlined earlier in the chapter. Usually the diagnosis is self-evident from the history, signs, and symptoms. The two most common mechanisms of traumatic injury are the arm being forced into 90° or more abduction and 90° lateral rotation and falling backward with the arm in extension.

The signs and symptoms of any dislocation or instability vary, depending on the severity. Patient complaints range from a vague sense of shoulder dysfunction to marked apprehension and pain, especially if the injury has been traumatic and the dislocation is unreduced. Reduction of the dislocation or subluxation usually provides immediate relief of pain, which is followed by a residual dull ache. Muscle spasm may make reduction difficult without the use of muscle relaxants, especially for the glenohumeral joint. A step deformity and loss of ROM are evident with a dislocation. Several days later, bruising and ecchymosis may be seen. If the patient complains of paresthesia, numbness, or muscle weakness, the clinician must be alert to the possibility of nerve injury, especially to the axillary nerve.

For dislocations, at least two radiographic views of the shoulder from two different angles are desirable to prevent a missed diagnosis, particularly with a posterior dislocation. X-ray films for an anterior dislocation should include a true anteroposterior (AP) view of the shoulder and a true lateral view of the scapula (Table 6-2).[51] An axillary view is difficult to obtain in an acutely injured shoulder but is extremely useful in ruling out a dislocation and thus should be obtained whenever possible. Specialized x-ray views, including Stryker Notch and a West Point Axillary, are required to further elucidate the size of Hill-Sachs lesion and Bankart fractures, respectively, but are not typically obtained in the acute setting.

TABLE **6-2**

Radiographic Imaging for Different Shoulder Pathologies

Pathology	Radiographic Imaging
Impingement	Anteroposterior (AP) x-ray film in the plane of the scapula Supraspinatus outlet view Axillary AP x-ray film, 30° caudal view (anterior acromial view)
Instability	AP x-ray film in the plane of the scapula Stryker Notch view West Point axillary view Axillary
Arthritis	AP x-ray film in the plane of the scapula Full humerus AP x-ray film in the plane of the scapula with laterally rotated 30° Axillary
Trauma	AP x-ray film in the plane of the scapula Axillary True scapular lateral view (Y view)

Anterior Dislocations

Anterior dislocations account for approximately 95% of acute traumatic glenohumeral injuries.[95] They occur most frequently in young adults. With an anterior glenohumeral dislocation, the head of the humerus is driven or levered anteroinferiorly. Typically, the glenoid labrum is stripped from the anteroinferior aspect of the glenoid (i.e., Bankart lesion). As previously noted, it frequently involves the anteroinferior glenoid bone (Bankart fracture) (Figure 6-13) and an impaction fracture in the posterolateral aspect of the humeral head (Hill-Sachs lesion) (Figure 6-14). The bony Bankart lesion can be an acute fracture or an erosive process in which the glenoid slowly becomes deficient anteroinferiorly with multiple recurrent disclocations[96] (see Figure 6-13). If no Bankart lesion is found, the anterior or lateral capsule may be disrupted at the humeral insertion site. This is called **humeral avulsion of the glenohumeral ligament**, or **HAGL lesion** (Figure 6-15).[94,96] With significant labrum damage, especially with increasing bone loss, the chances for recurrent dislocation are greater because the potential for healing and spontaneous reattachment of the fibrocartilaginous labrum and/or Bankart fracture fragment back to its anatomical location is poor. The disrupted osseus or soft tissue Bankart lesions do heal back to the glenoid but often in a more medial position along the glenoid neck,

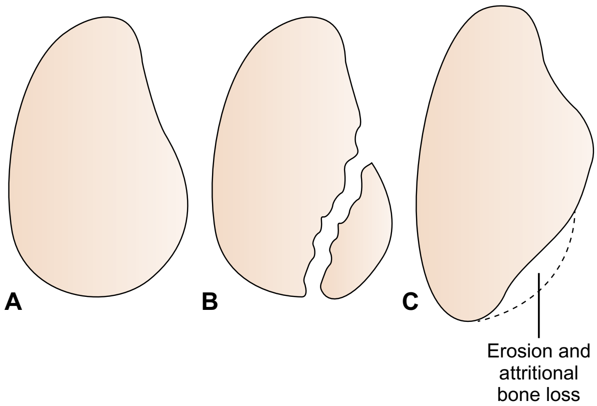

Figure 6-13 Bankart lesion. **A**, Pear-shaped normal glenoid. **B**, Bankart fracture. **C**, Erosive or attritional bone loss. (Redrawn from Burkhart SS, De Beer JF: Traumatic glenohumeral bone defects and their relationship to failure of arthroscopic Bankart repairs: significance of the inverted-pear glenoid and the humeral engaging Hill-Sachs lesion, *Arthroscopy* 16(7):684, 2000.)

resulting in an inadequate anterior buttress to humeral head translation. This is further exacerbated if there is a large Hill-Sachs lesion.

The intact labrum is important for maintaining a suction-like vacuum effect to hold the humeral head in the glenoid cavity and for its buttress effect. Although it is most important as a static stabilizer, it also enhances dynamic support of the joint.[16,17] The glenohumeral joint usually is bathed in less than 1 mL of synovial fluid,[97] which helps hold the articular surfaces together and enhances the normal negative intra-articular pressure.[18,33] However, excessive fluid (usually blood) in the joint can negate this effect.

On examination, an acutely dislocated shoulder demonstrates a deformity (i.e., step deformity), or a space usually is detected under the tip of the acromion, resulting in prominence of the acromion, flattening of the deltoid muscle, pain, and loss of motion. The patient tends to support the arm in 30° of abduction with the other arm while listing or leaning to the affected side. Attempts at movement in any direction are painful, and the humeral head may be palpable in the axilla.

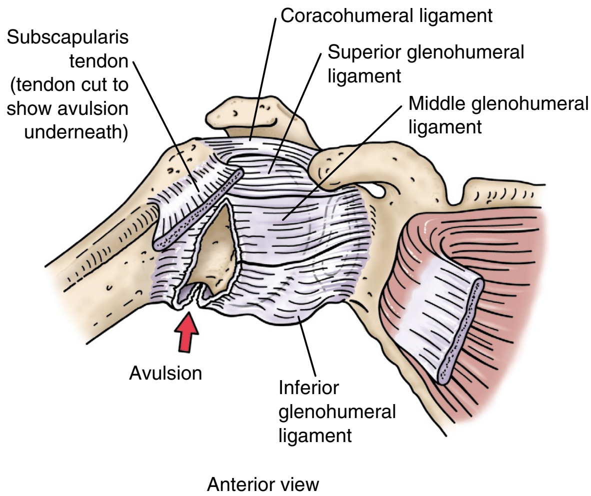

Figure 6-15 Schematic of humeral avulsion (*arrow*) of the glenohumeral ligament (HSGL Lesion) showing avulsion of middle and inferior glenohumeral ligaments from the humerus (subscapularis tendon cut for clarity). (Redrawn from Wolf EM, Cheng JC, Dickson K: Humeral avulsion of the glenohumeral ligaments as a cause of anterior shoulder instability, *Arthroscopy* 11(5):602, 1995.)

Anterior glenohumeral dislocations have several associated complications (of which recurrence is the most common). These may include injury to the axillary, musculocutaneous, or median nerves or more rarely the brachial plexus.[20] Only approximately 20% of these dislocations show minimal clinical signs, and 5% show significant evidence of neurological involvement; however, Rockwood demonstrated that up to 80% of individuals show electromyographic evidence of nerve injury.[63] This damage initially may be subclinical, with the patient showing no detectable weakness. Consequently *it is essential that the clinician repeatedly check and monitor for paresthesia and changes in the strength of the deltoid muscle*. Other complications include rotator cuff tears

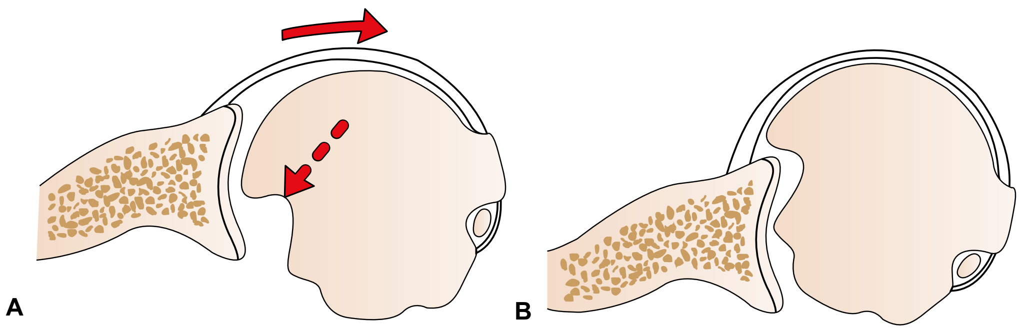

Figure 6-14 **A**, Hill-Sachs lesion (*dashed arrow*). **B**, Hill-Sachs lesion causes engagement of the humeral head in the anterior glenoid with lateral rotation. (Redrawn from Burkhart SS, De Beer JF: Traumatic glenohumeral bone defects and their relationship to failure of arthroscopic Bankart repairs: significance of the inverted-pear glenoid and the humeral engaging Hill-Sachs lesion, *Arthroscopy* 16(7):681, 2000.)

(the likelihood of these tears increases with advancing age, typically greater than 40 years of age), damage to the biceps tendon, and, on occasion, fractures of the greater tuberosity or the neck of the humerus. Vascular injury must also be considered, especially if distal pulses are diminished, and can be potentially limb threatening.[98]

Recurrent dislocations typically occur in active male patients under age 20, with recurrence rates being possibly as high as 90% after a traumatic dislocation.[95] Other factors that increase the risk of occurrence include contact, overhead activities, and significant glenoid and humeral head bone loss. The recurrence rate declines with age, with fewer recurrent dislocations occurring after age 40.[22,24,99,100] Most of these dislocations (75%) occur within 2 years of the initial traumatic event.[99]

Examination of a patient with recurrent instability is confirmed by a positive apprehension and relocation test. Signs of hyperlaxity, including lateral (external) rotation beyond 85° or hyperabduction of greater than 105°, should be watched for because these patients have a higher incidence of failure of a surgical repair.[101,102] Intact rotator cuff function should be confirmed. If there is concern for rotator cuff integrity, ancillary imaging, such as an ultrasound or an MRI, should be performed.

Nonoperative treatment may be successful in some patients with recurrent dislocations because restoration of kinetic chain function, shoulder strength, and proprioception may suffice in rendering the joint stable. Unfortunately, it is ineffective for most individuals with recurrent episodes; therefore, surgical stabilization is appropriate for those with significant functional instability, and excellent success rates usually are achieved.[24] The choice of surgical technique and the timing and extent of surgery depend on the degree and direction of instability, the requirements for ROM, the associated soft tissue and bone damage, and the surgeon's preference and skills.

Posterior Dislocations

Posterior dislocations result from the arm being forced or thrust backward when it is forward flexed and medially (internally) rotated; these injuries account for approximately 2% of all glenohumeral dislocations. Although falling on an outstretched, adducted arm can cause the injury, the typical mechanism is the result of a grand mal seizure. On examination, posterior prominence of the humeral head may not be evident, and rounding of the shoulder or deltoid is maintained; therefore these dislocations are sometimes missed or misdiagnosed, leading to prolonged periods of dislocation, especially in more heavily built individuals. Some flattening of the anterior shoulder may be seen, and the coracoid process may be more prominent than on the uninjured side. The patient will have limited lateral (external) rotation (less than 0°) and limited elevation (less than 90°), which are signs that further investigation is needed. In addition, medial rotation and cross-flexion (horizontal adduction) cause pain and apprehension. Posterior dislocations are recognized by the "empty glenoid" sign on the x-ray film (Figure 6-16). The true glenohumeral AP view of the posteriorly dislocated shoulder shows abnormal overlapping of the humeral head and glenoid shadows, which are normally on this view. Again, an axillary view will clearly show this dislocation but is often difficult to obtain in cases of an acutely posterior shoulder dislocation. If this injury is missed, it results in considerable morbidity because the relatively soft humeral head becomes severely damaged as it rests on the edge of the hard glenoid. Reduction becomes very difficult and it can often only be achieved through operative intervention.

In recurrent posterior dislocations, the patients complain of apprehension with shoulder elevation, especially with the arm adducted. Physical examination confirms excessive posterior translation of the humerus and subluxation, or even frank dislocation, with a posteriorly directed force on the humerus. The complex pathophysiology

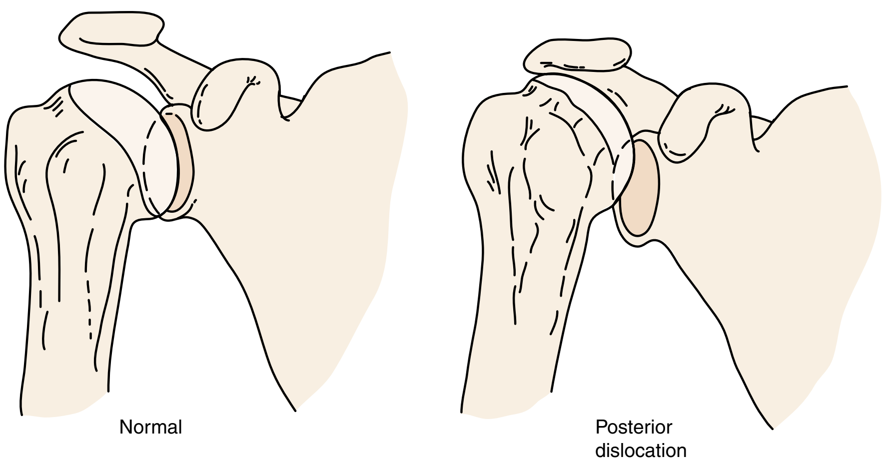

Figure 6-16 "Empty glenoid" sign of posterior dislocation on an anteroposterior x-ray film. Note that the head of the humerus fills the glenoid in the normal x-ray film (*left*). With a posterior dislocation, the glenoid is "empty," especially in its anterior portion (*right*). (From Zachazewski JE, Magee DJ, Quillen WS: *Athletic injuries and rehabilitation*, p 523, Philadelphia, 1996, WB Saunders.)

involved in posterior shoulder dislocations has led to the development of numerous surgical techniques to address this problem.[100] Soft tissue techniques range from a simple arthroscopic posterior labral and capsular plication to a posteroinferior capsular shift.[103] Additional bony techniques include wedge osteotomies with or without a bone block, and humeral rotation osteotomies can be performed in only the most severe and rare cases.[104] With advanced arthroscopic techniques, surgeons have been able to perform arthroscopic stabilization with fairly good functional outcomes.[105] Postoperative care varies widely, but if the practitioner has an appreciation of the techniques used and follows the treatment principles outlined in this chapter, a customized treatment program can be developed to suit each specific situation. However, in all cases of posterior stabilization, the patient is immobilized in neutral to slight lateral rotation to protect the repaired posterior structures.

TREATMENT PRINCIPLES FOR A DISLOCATED SHOULDER

In the management of acute dislocations of the shoulder, an attempt must be made to characterize the injury. A major difficulty in developing a treatment plan from the older literature is the failure of the literature to separate groups adequately according to the underlying degree of injury and the direction of laxity.[106] A glenohumeral dislocation usually is reduced using closed techniques and adequate anesthesia. Post reduction, it is important to recheck the patient's neurovascular status both before and after reduction and to obtain second AP and lateral x-ray films to ensure proper positioning.

With dislocations, subluxations, or instability, a concerted, focused effort at nonoperative treatment with a well-planned, well-coordinated therapy program must be attempted. This principle is more important in cases in which the shoulder is more lax, the collagen hyperelasticity is greater, and/or less trauma is involved in the initial dislocation. If a larger Bankart fracture (i.e., greater than 25% of glenoid area) or other structural deficiencies, such as an acute rotator cuff tear, are found, they usually are dealt with surgically as soon as possible.

For acute anterior dislocations, treatment begins with early controlled immobilization. After closed reduction, the shoulder may be immobilized with a swath and sling or just a sling for up to 6 weeks (in young adults) to prevent extension, abduction, and lateral rotation, which helps to promote capsular healing. Commonly, after 3 weeks, the sling is used primarily to protect the glenohumeral joint from potential injurious outside forces (e.g., a fall, bumping into people) Some researchers have reported that the period or position of immobilization bears little relation to the recurrence of the dislocation,[100] and some authors advocate a shorter period of immobilization (1 to 3 weeks).[36,99] The duration of immobilization depends on the philosophy of the physician or surgeon and the type of reconstruction, if surgical repair is performed.

In the first 2 weeks following reduction, a program of strong isometric deltoid, shoulder abductor, adductor, and biceps work is instituted within the limits of pain tolerance. Scapular control exercises should be initiated right away within a pain-free ROM in standing or prone positions (Figure 6-17). This minimizes muscle wasting, keeping the muscles active following injury and decreases pain as the edema and hemorrhage resolve and the shoulder becomes more comfortable. The sling can be removed for exercises, controlled ROM, and unresisted eccentric exercises (to biceps and triceps) "in the cone" several times a day. When doing biceps exercises, the clinician should use a "block brace" or towel (Figure 6-18) to ensure the

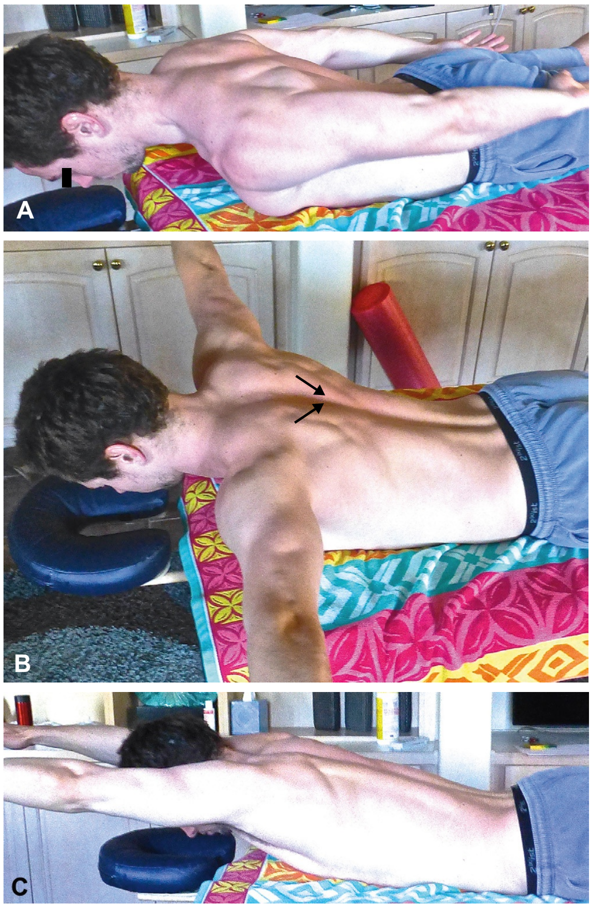

Figure 6-17 "Prone series of 3" scapular exercises used to engage scapula in streamlined position. **A**, Patient lies in prone with hands on gluteals, palms up. Patient then reaches for feet, lifting head and chest off bed, maintaining a straight spine with scapula engaged, core set, and neck in neutral (i.e., not looking up). **B**, Shoulders brought to 90° ("T" position) with thumbs facing forward. Patient then squeezes scapulae together and down (*arrows*) while maintaining a straight spine, core set, and neck in neutral. **C**, Patient brings arms over head and then pulls scapulae down and toward opposite hips while maintaining a straight spine, core set, and neck in neutral.

Figure 6-18 "Block brace" (towel or wedge) to prevent arm adduction in early rehabilitation. Note: The "block" (*arrow*) should be used with caution for glenohumeral rotator cuff exercises especially if the patient does not have good control of scapula. When using a towel at the elbow, the clinician must ensure that the scapula is able to stabilize. If the patient does not have good control of the scapula, stabilization of the shoulder occurs through the elbow with the rotator cuff working through reverse origin insertion to stabilize the scapula.

arm does not adduct or stays in alignment with the body line during elbow movement. Adduction may result in the humeral head being laterally levered against the lateral structures of the glenohumeral joint.

"In the cone" implies movement at the shoulder of 30° abduction (i.e., the range in which scapula becomes engaged or the scapular muscles begin to contract to stabilize the scapula and allow it to move in coordination with the moving arm), 30° to 60° forward flexion, and lateral (external) rotation to neutral (the hand does not move outside the plane of the body), with the elbow never extending through the frontal plane (behind) of the body (to prevent the humeral head from translating too far forward) (Figure 6-19). Pendular exercises may also be initiated at this stage (Figure 6-20), starting with momentum pendular exercises (i.e., leaving the arm hanging while rotating the hips) and progressing to controlled pendular exercises (i.e., moving relaxed arm in circles, forward and backward and side to side), and finally to modified pendular exercises (e.g., placing hand on ball and moving ball). Small range, gentle, closed kinetic chain exercises can be initiated, provided the movement is pain free.

The emphasis in the early stages is still on isometric activities, with neutral (i.e., not outside the body plane) shoulder medial and lateral rotator isometric exercises taking precedence using "closed stick" and "power square" drills in the supine position (Figure 6-21). The clinician must ensure that the patient is able to maintain and hold starting postures when doing the exercises. The patient should always start in supine position (to help to stabilize the scapula), performing the exercises in three positions—at the level of the diaphragm, shoulder, and forehead. Exercises should be colinear and coupled so the Hawkins position (which stabilizes the glenoid under the humerus) (Figure 6-22), and the "power square" position (which is the basis of many shoulder movements) may be used as foundations for a number of different exercises.

In addition, concentric exercises through a limited range ("in the cone") are permitted, with the clinician bearing in mind that capsular healing is well under way at 3 weeks. The limits of range are determined by pain and whether the patient can comfortably control the movement. When stability is obtained "within the cone," the patient can carefully begin to increase the ROM outside the cone.

Controlled movements are essential during proper rehabilitation. The controlled active movement applies small amounts of stress to the joint structures to reduce the adverse effects of immobilization on the glenohumeral joint.[36,42,107,108] During this time, active abduction beyond 20° to 30°, forward flexion beyond 90°, or lateral rotation past neutral (outside the cone) should be performed carefully. In older individuals, in whom the danger of stiffness leading to adhesive capsulitis and frozen shoulder is greater than the risks of redislocation, the movement program is initiated at the beginning. Once immobilization has ended and dynamic control achieved, the treatment protocol follows the same principles as for treatment of patients with instability (see later discussion).

SURGICAL TREATMENT OF SHOULDER INSTABILITY

Indications for surgery and open reduction include the inability to reduce by closed reduction, presence of a large flake of avulsed bone on the inferior glenoid margin (which may contribute to future instability), a fracture through a significant portion of the articular surface, vascular impairment, and an associated displaced tuberosity fracture or a fracture of the neck of the humerus. On occasion, very athletic individuals may want a guarantee of stability in the shoulder by having early surgery. Postoperative care depends on the type and degree of associated problems that led to open reduction and repair.

Surgical procedures may be arthroscopic or open and involve either a soft tissue or bony reconstruction. The spectrum of surgical approaches depends largely on the pathology involved. Patients with largely soft tissue lesions, such as simple labral tears, are ideal candidates for a simple arthroscopic Bankart repair. If the inferior capsule is avulsed from the humeral side (i.e., an HAGL lesion), an open or arthroscopic capsular repair is indicated. A capsular plication is indicated for the rare occasion when surgery is indicated for the patient with multidirectional instability. Bony procedures also may be performed,

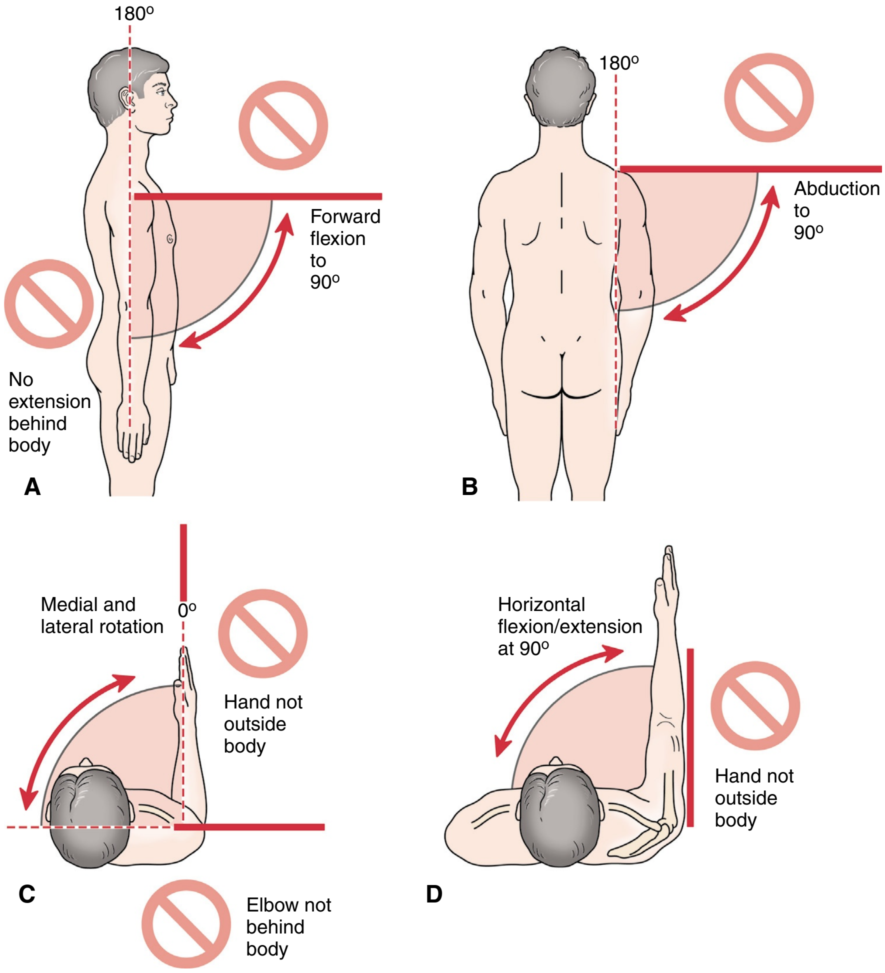

Figure 6-19 Low-risk postsurgical movement allowed "in the cone." **A**, Forward flexion to 90°. **B**, Abduction to 90°. **C**, Lateral rotation—hand not outside the body, elbow not behind body. **D**, Horizontal flexion—hand not outside body. Note: Horizontal flexion/extension is also called horizontal adduction/abduction. (Modified from Magee DJ: *Orthopedic physical assessment*, ed 6, p 273, Philadelphia, 2014, WB Saunders.)

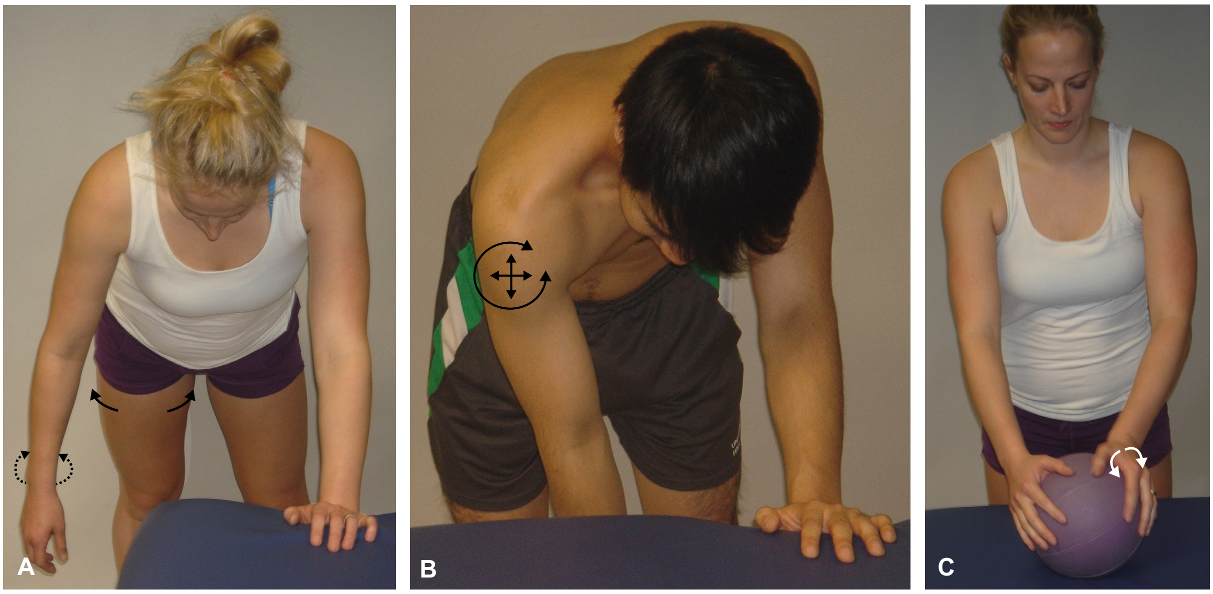

Figure 6-20 Pendular exercises. **A**, Injured arm hangs down free. "Momentum" pendular movement achieved by swinging hips to initiate movement in different directions. **B**, Active controlled pendular movement—muscles control movement in different directions. **C**, Ball pendular movement. Both hands press down on a ball to compress humeral head into glenoid. The unaffected left arm moves the ball in circles, back and forth, or side to side while injured right arm "goes along for a ride."

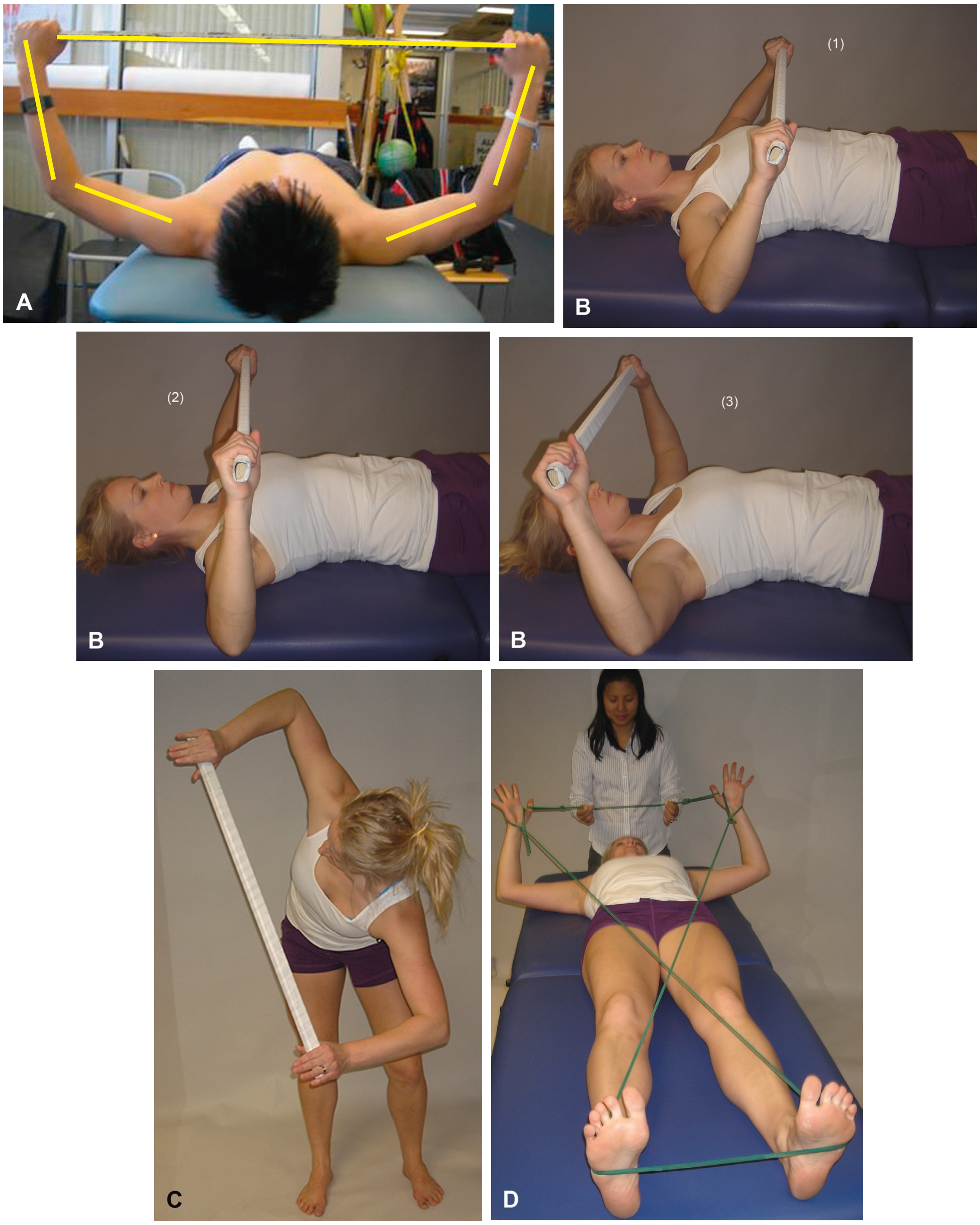

Figure 6-21 Power square position. **A**, Basic start position using stick (tubing could also be used). **B**, Three basic positions of power square—at diaphragm-nipple-xiphysternum line (*1*), at shoulder height (*2*), and at forehead/nose height (*3*)—to do co-contractions. **C**, Functional movement out of power square position with stick. **D**, "Spiderman" exercise out of power square position. Clinician is providing perturbations on "thumb-tube."

mainly focusing on restoring anteroinferior glenoid bone loss with local bone graft from a transferred coracoid and the attached conjoined tendon. Historically the Bristow procedure involves transferring the tip of the coracoid, whereas the more recently popularized Latarjet procedure involves transferring a larger fragment of the coracoid, allowing for a more robust bony reconstruction of the anteroinferior glenoid bone stock (Figure 6-23).[29] This procedure has the added stabilizing effect of lowering the inferior half of the subscapularis muscle belly, as well as adding the dynamic sling effect of the conjoined tendon with the shoulder in positions of apprehension (i.e., abduction and lateral rotation). This procedure is usually reserved for severe cases of instability in which there is substantial glenohumeral bone loss and patients who require a robust repair, such as athletes involved

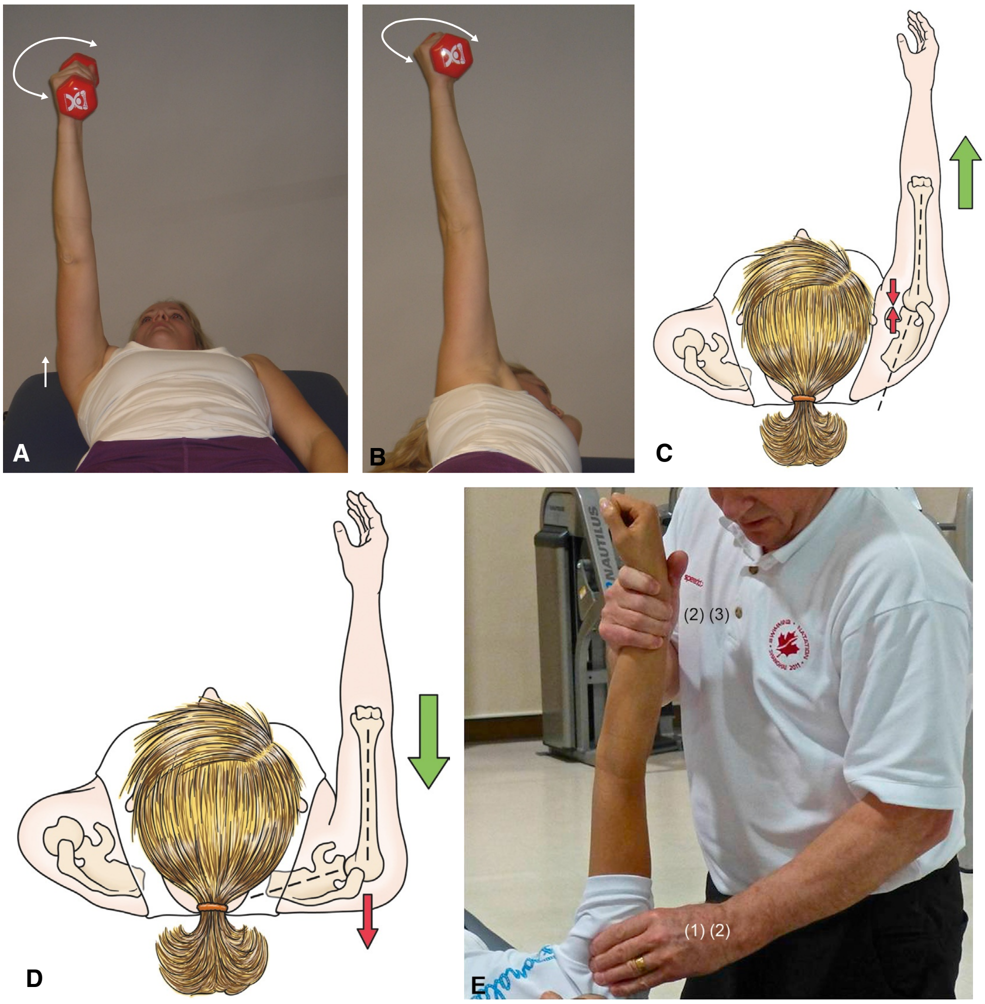

Figure 6-22 Hawkins ("punch out") position ensuring glenoid is sitting under humeral head for added stability. **A,** In forward flexion doing circles, horizontal, or up and down movement. **B,** In abduction. **C,** Line drawing shows how the glenohumeral joint is more stable with the glenoid lying under the head of the humerus in the Hawkins position than in **D**. **E,** To gain arthrokinematic control, the clinician applies resistance in all directions at the glenohumeral joint (*1*). To ensure arthrokinematic control at the glenohumeral joint while doing osteokinematic movement, the examiner applies resistance at the glenohumeral joint (arthrokinematic stabilization) and the wrist (osteokinematic movement) (*2*). To strengthen osteokinematic movement, the clinician applies resistance at the wrist (*3*) while palpating the glenohumeral joint to ensure there is minimal translation and to ensure arthrokinematic control during the perturbations. (**C** and **D,** modified from Matsen FA: Stability. In Matsen FA, Lippett SB, Sidles JA et al: *Practical evaluation and management of the shoulder,* p 64, Philadelphia, 1994, WB Saunders.)

in contact sports. The inferior glenoid can also be augmented by other bone graft options, including iliac crest and other cadaveric sources.[109] Large Hill-Sachs lesions have been addressed using allograft bone implantation to the humeral head defect or suturing the posterior cuff into the Hill-Sachs lesion. The latter procedure is called a *Remplissage*, which is the French word meaning "to fill" (Figure 6-24).[110,111] Other procedures have been described and may now be only of historical interest. Such procedures as the Putti-Platt and Magnuson-Stack involved excessive tightening of the anterior structures of the shoulder, specifically the subscapularis and anterior capsule, thus restricting lateral rotation and thereby preventing pathological translation and engagement of the Hill-Sachs lesion. Unfortunately, there is a report of these procedures directly resulting in early osteoarthritis of the glenohumeral joint.[112] Excessive anterior capsular tightness results in posterior subluxation of the humeral head on the glenoid, and this eccentric loading of the glenohumeral joint can lead to the rapid development of arthritis. In addition, any procedure that involves restriction of lateral rotation is very undesirable to an athlete who requires full shoulder motion.[36,113–128]

Complication rates of instability surgery vary depending on the procedure performed. Arthroscopic procedures carry a higher risk of recurrent dislocation, especially in young athletes involved in contact sports with significant bone loss.[29,129] In the past, these arthroscopic approaches have been associated with worrisome redislocation rates.[130,131] With strict technical criteria,

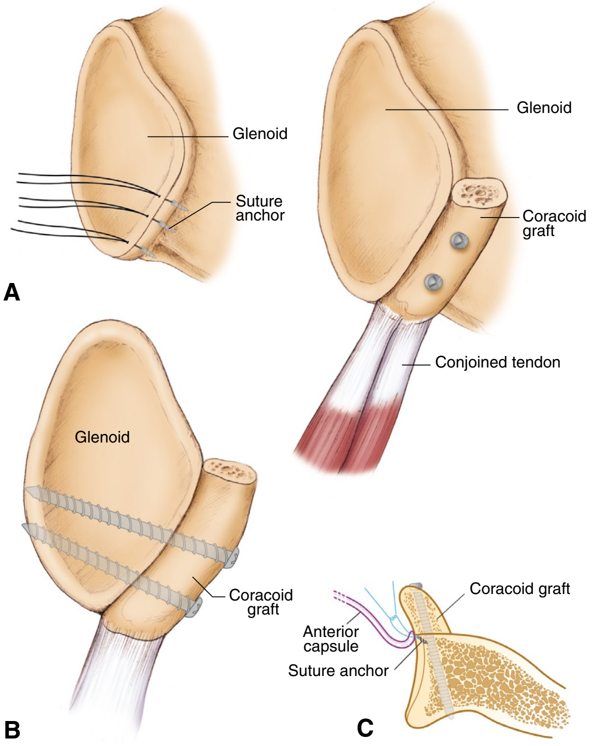

Figure 6-23 Latarjet procedure. **A**, Conjoined tendon of biceps sutured to glenoid. **B**, Screws hold conjoint tendon. Note how graft restores pear shape of glenoid. **C**, Graft is placed so that it acts as an extension of the articular arc of the glenoid. (Redrawn from Burkhart SS, De Beer JF: Traumatic glenohumeral bone defects and their relationship to failure of arthroscopic Bankart repairs: significance of the inverted-pear glenoid and the humeral engaging Hill-Sachs lesion, *Arthroscopy* 16(7):677-694, 2000.)

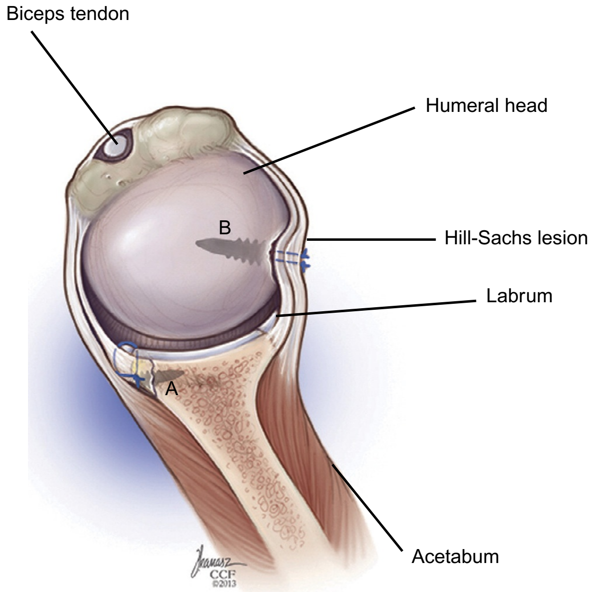

Figure 6-24 **A**, Bankart repair–reattachment of the anterior labrum. **B**, Remplissage (to fill in): Hill-Sachs lesion with the posterior capsule and infraspinatus muscle anchored into the lesion. (Courtesy of Cleveland Clinic Foundation, Cleveland, OH.)

including restoration of the proper resting length of the anterior-inferior glenohumeral ligament and recreation of the labral "bumper" or "chalk-block" effect, current arthroscopic techniques offer success rates comparable to open techniques. Strict clinical guidelines for suitable patient selection also are important. Open bone grafting procedures, such as the Latarjet procedure, have lower recurrence rates documented in the literature but unfortunately have higher rates of infection, hematoma formation, iatrogenic nerve injury, and degenerative changes related to poor bone graft positioning.[132,133]

With soft tissue surgical techniques, it is important to allow sufficient time for capsular healing with minimal stress to these tissues. With bony techniques, such as the Latarjet procedure, a period of approximately 4 to 6 weeks is required to allow for union of the bone block before stress is applied. After operative treatment, the traditional Putti-Platt procedure has a 1% to 5% incidence of recurrent dislocation; the Magnuson-Stack operation, 1% to 9%; and the Bristow repair, 1% to 6%.[125] These results should be compared with the increasingly successful outcomes of arthroscopically assisted techniques, which, as surgeons have become more experienced, have reached and surpassed the open techniques. Return to a high level of function is the rule with an appropriately selected and executed technique performed with due regard for the pathological lesion, follow-up rehabilitation, and the activity goals of the patient.

Although postoperative therapy is based on tissue healing principles and proper stress to the tissues, each surgeon has certain criteria that he or she wants to be followed in a rehabilitation program. The clinician must be aware of these criteria, including the reasons for them, along with any restrictions or modifications to treatment, to prevent any misunderstanding with the surgeon and patient's desired outcome.

POSTSURGICAL TREATMENT OF THE SHOULDER

The basic rehabilitation plan may be modified, depending on the type of surgical procedure performed, precautions to treatment noted by the surgeon, the individual surgeon's preferences, and whether pain, restriction, or both are factors. For the most part, the treatment of patients who have undergone arthroscopic procedures parallels that for patients who have had open techniques, although the former have less tissue disruption and muscle wasting. The patient's willingness to move and the quality of the patient's movements also are of concern to the clinician. Only broad guidelines are given here for progression. The treatment principles remain constant: to relieve pain, observe for complications, regain muscle control, strengthen, regain ROM safely, improve endurance, and retrain proprioceptive control. These guidelines form the foundation of an activity-based protocol. Specificity plays a major role with each program being individualized to the patient and his or her activities.

Principles of Specificity Related to Sports and Rehabilitation

Every activity makes specific demands in terms of load (stress), rate, repetitions, duration, and neurophysiological adjustment. When a new or modified task with a different demand in intensity, load, rate, repetitions, duration, or neurophysiological adjustment is instituted, an entirely new pattern of adjustment must be acquired. Therefore training and rehabilitation must be specific to the sport or activity in which the patient hopes to take part or return to. This principle applies to all of the following:

1. Cardiovascular fitness (aerobic and anaerobic activities)
2. Strength training (isometric, isotonic [concentric and eccentric], econcentric, isokinetic)
3. Open-chain and closed-chain activities
4. Motor skills and motor learning
5. Flexibility (static, dynamic)
6. Coordination
7. Proprioceptive or kinesthetic control (stimulation of different mechanoreceptors)
8. Timing, reaction time, and movement time
9. Progressions
10. Biomechanical demands
11. Tissue healing and stress on tissues

To achieve progression in these areas, the clinician proceeds from:

1. General to specific
2. Simple to complex
3. Easy to difficult
4. Lesser to greater volume
5. Lesser to greater intensity
6. Lesser to greater frequency
7. Smaller to greater ROM

Modified from Zachazewski JE, Magee DJ, Quillen WS, editors: *Athletic injuries and rehabilitation,* p 527, Philadelphia, 1996, WB Saunders.

Postsurgical treatment initially concentrates primarily on the scapula. Scapular control exercises focus primarily on the lower and middle trapezius and serratus anterior. These exercises include scapular retraction, depression, protraction, and elevation with the arm at the side. Careful observation should detect compensatory trunk, pelvic, and especially shoulder postures that indicate incorrect firing (i.e., contraction) sequences and lack of proper stabilization. As the patient improves, the glenohumeral joint can be taken out of "the cone" into abduction, scaption (i.e., plane of the scapula), forward flexion, and other elevation movements, provided the patient can maintain control of the scapula and can stabilize the humerus in the glenoid. Lateral rotation should be performed only with extreme care and never beyond neutral in the early stages of rehabilitation (i.e., the hand should never go outside the sagittal plane of the body, and the elbow should never go behind the frontal plane of the body). These are important early rules, regardless of the surgical procedure used. Keeping inside "the cone" reduces the stress on the anterior labrum and the repaired soft tissues to a minimum. During rotation exercises, the clinician should watch for excessive scapular winging or motion because the scapula may be compensating for tight medial or lateral rotators. If present, this can be treated with joint play techniques or muscle energy or proprioceptive neuromuscular facilitation (PNF) techniques, depending on whether the restriction is caused by inert (i.e., collagen) or contractile (i.e., muscle) tissues.

As control of the scapula is being achieved, isometric exercises to the muscles that control the humeral head can be initiated, starting with light contractions (importantly, not maximum voluntary contraction [MVC]) and progressing to stronger isometric contractions for the deltoid and rotator cuff muscles. Initially the clinician should start with exercises that cause the muscles controlling movement at the glenohumeral joint to compress the humeral head into the glenoid for stability (see Figure 6-22). If the patient has good pelvic and scapular control, plank exercises may be performed as long as correct posture can be maintained while doing the exercise (see Figure 6-10).

Postoperative exercises for the shoulder should not cause sharp or severe pain. It is acceptable for the patient to be slightly uncomfortable when doing exercises, but the exercises must be performed with confidence and a sense of control. The discomfort should disappear when the exercise stops. The clinician must ensure that the exercises are performed correctly. Correct movement patterns are more important than load or speed. Once proper scapular control is achieved up to 30° of abduction, the clinician can move to 30° to 90°, and when proper dynamic control has been gained in that range, the patient can be progressed to 180°.[134]

Once control of the scapula is achieved, isometrics to the muscles of the glenohumeral joint are instituted at several positions in the ROM below shoulder level, with no lateral rotation beyond 0°. The positioning and resistance depend on the patient's response to pain and discomfort. The clinician must ensure that compensatory pelvic, trunk, and cervical postures are addressed and that the scapular and humeral control (i.e., stabilization) muscles receive attention.[35] The patient is instructed to perform proper functional isometric position exercises in abduction, medial rotation, lateral rotation (to 0°), extension, and a "full can" (neutral with thumb up) scaption (below horizontal) for the supraspinatus.

Rhythmic stabilization exercises and co-contraction at the joint (e.g., supine or lateral Hawkins positions [see Figure 6-22, *C*]) can be performed to the affected shoulder. The progression of stabilization-co-contraction sequence follows three steps in each selected isometric position. Ensuring the patient has assumed a correct posture and has the appropriate scapular muscles engaged and, if possible, the glenoid under the head of the humerus for greater stability (e.g., supine Hawkins with punch out [see Figure 6-22, *C*], the clinician places one hand around the glenohumeral joint and one hand distally near the wrist. For the first step, the clinician applies perturbations to the humeral head while asking the patient to hold the head of the humerus stabilized (i.e., not moving or translating) in the glenoid. This is called **establishing arthrokinematic control** at the joint. The hand around the wrist offers no resistance but is there to keep the patient's arm extended. Once arthrokinematic control at the glenohumeral joint is gained, the clinician can progress to the second step, in which the clinician applies perturbations not only to the humeral head but also at the wrist. It is important that the clinician ensures that minimal translation occurs at the glenohumeral joint during the double perturbations and the hand applying the perturbations to the humeral head applies only enough counterforce that translation does not occur. Thus this hand is applying perturbations while palpating to ensure no unwanted translation occurs at the glenohumeral joint. If translation does occur, the wrist perturbations being applied at the wrist are too strong. The hand at the wrist can also apply "eccentric breaks" to the whole arm. This second phase establishes **osteokinematic control of the arm** along with arthrokinematic control at the glenohumeral joint. The final or third stage is resistance at the wrist doing eccentric breaks into functional movements. The position of the clinician's hands is the same (around the humeral head and at the wrist), and perturbations are applied only at the wrist into functional positions. The hand around the glenohumeral joint only palpates to ensure minimal translation is occurring at the glenohumeral joint. The result should be eccentric movement of the arm with minimal translation at the glenohumeral joint, which mimics normal arthrokinematic control at the glenohumeral joint and osteokinematic control of the arm. Once co-contraction and coupling are obtained, functional isometrics can be started. If the patient is having difficulty engaging different muscles (e.g., different parts of the deltoid—anterior deltoid often does not engage or contract following injury to the anterior shoulder), electrical stimulation may be used (Figure 6-25).

If the patient complains of pain in the shoulder, ice can be applied to give some relief. In addition, active exercise to the other joints of the arm, cervical spine, trunk and pelvis, and the rest of the body may be instituted to ensure involvement of the whole kinetic chain.

As the patient improves (as evidenced by decreased pain and discomfort), isotonic exercises, especially flexion and extension, can be initiated (usually by the third or fourth day), again below shoulder level and with no lateral rotation beyond 0°, within the pain-free and controllable range. By the fourth day, medial and lateral rotation to 0° with the arm in the adducted position may be performed very carefully. For the lateral rotators, only very careful isometrics should be performed, to prevent excessive stress to the healing tissues. By the fifth day, the patient may begin assisted abduction. To achieve proper scapular and humeral control, exercises are used when appropriate and when strength and pain allow.[134] Once the pain has diminished and as the patient is weaned from the sling, the clinician can follow the treatment concepts for rehabilitation of shoulder instability described later in the chapter.

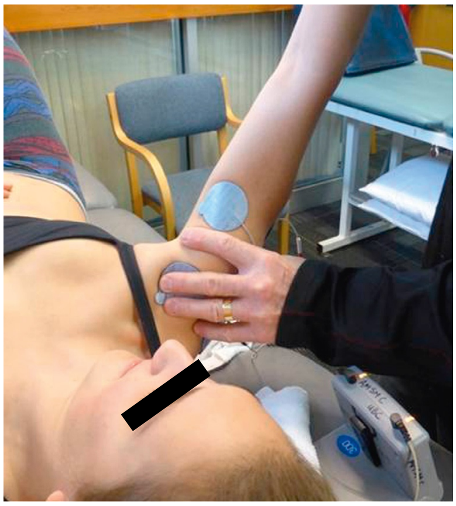

Figure 6-25 Electrical stimulation of anterior deltoid.

PITFALLS OF TREATMENT FOR SHOULDER INSTABILITY AND IMPINGEMENT MANAGEMENT

As with any treatment, the clinician must be aware of common pitfalls. If the following mistakes are kept in mind, the rehabilitation program will have every chance of success.

1. *Failure to perform a complete and thorough assessment.* The assessment must involve the shoulder complex, trunk, pelvic core, and lower kinetic extremity to determine which structures are weak and which are tight and to check for incorrect movement patterns.[85,135]
2. *Failure to assess and address scapulohumeral dysfunction.* The clinician must remember that scapular motion is dynamic for the most part and that the scapula constantly moves, even as it functions as a stable base from which most of the glenohumeral muscles originate. Proximal stability (i.e., scapular control) must be achieved early in the rehabilitation process, before distal mobility (i.e., glenohumeral movement) is encouraged.[47,85] Coordinated scapulohumeral movement is affected by muscle length, muscle strength (especially eccentric muscle strength), capsular tightness, the type of muscle contraction (e.g., isometric, concentric, eccentric, econcentric), timing, and pain. Scapular movement itself is affected by gravity, altered posture, tight muscles, whether the activity is an open or closed kinetic chain, the amount of load the arm lifts, and the speed of arm movement. The clinician must keep all these issues in mind when progressing activity.
3. *Failure to understand the mechanics and demands of the activities to which the patient is returning.* The clinician must have a clear picture of these activities, whether they involve sports or are part of the

individual's everyday life, such as work or recreational activites.[85] Driving is obviously not the same as throwing. Although patients may present with similar types of painful movement, the clinician must be able to assess the problem with a clear understanding of the demands of the activities the patient wants to accomplish. The practitioner must understand that an injury in one part of the body may cause an alteration in another part (i.e., alteration in shoulder mechanics, movement patterns, and how they are accomplished). The shoulder is a link in a kinetic chain of movement.[2–4,85] This kinetic chain allows the generation, summation, transfer, and regulation of forces from the foot to the hand during everyday activity.[2–4,47,81,85] Although individual muscles are the early emphasis in rehabilitation, eventually, as the patient progresses, the whole body must be integrated with shoulder movement patterns (i.e., so that the lower extremity and "core" drive the shoulder).

4. *Failure to deal with any hypomobility early in the treatment to ensure that the joint can regain normal arthrokinematic movement.*[85] Particular attention should be paid to tightness of the posterior glenohumeral capsule, restricted glenohumeral medial rotation (GIRD) or imbalance between the medial (internal) and lateral rotation ROM,[3] tightness of the pectoralis minor and pectoralis major muscles, tightness of the inferior glenohumeral ligament (caudal glide of the glenohumeral joint), and hypomobility in the thoracic spine and ribs.[3] Treatment of structures must ensure proper movement patterns. Testing for tight anterior structures may be accomplished by doing the Mattison 20 cm test and the horizontal extension test. For the **Mattison 20 cm test**, the patient lies supine while the examiner places one hand over the humeral head to palpate humeral head anterior movement with the patient's arm in 90° abduction. The clinician, using the other hand, medially rotates the patient's arm over the clinician's arm (Figure 6-26). Normally, the patient's hand should be within 20 cm (horizontally) of the ASIS on the same side without the humeral head projecting anteriorly. For the **horizontal extension test**, the patient again lies supine with the arm abducted to 90°. The examiner is positioned such that the elbow of one hand lies over the humeral head to prevent it translating forward. While holding the humeral head down, the examiner horizontally extends the patient's arm (Figure 6-27). The examiner should normally be able to horizontally extend the arm at least 20° to 30° without the humeral head translating anteriorly.
5. *Failure to make sure that stretches are performed properly.* Although stretching may be necessary to restore normal arthrokinematics in a joint, it should not compromise other structures (e.g., scapular stabilizers)

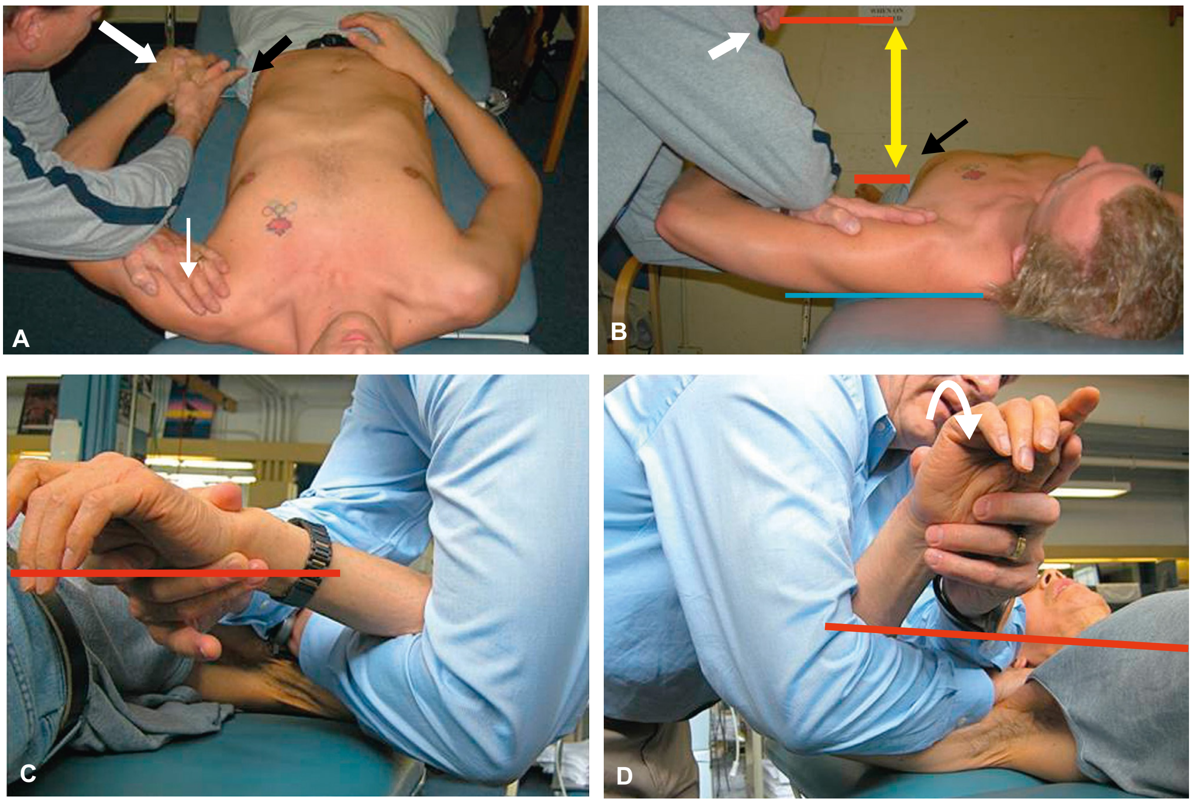

Figure 6-26 Mattison 20 cm test. **A**, Medial rotation of the patient's arm over the clinician's arm. Note how the clinician is holding the humeral head down with his hand. **B**, The distance from the patient's wrist to the ASIS is measured. **C**, Negative test. **D**, Positive test.

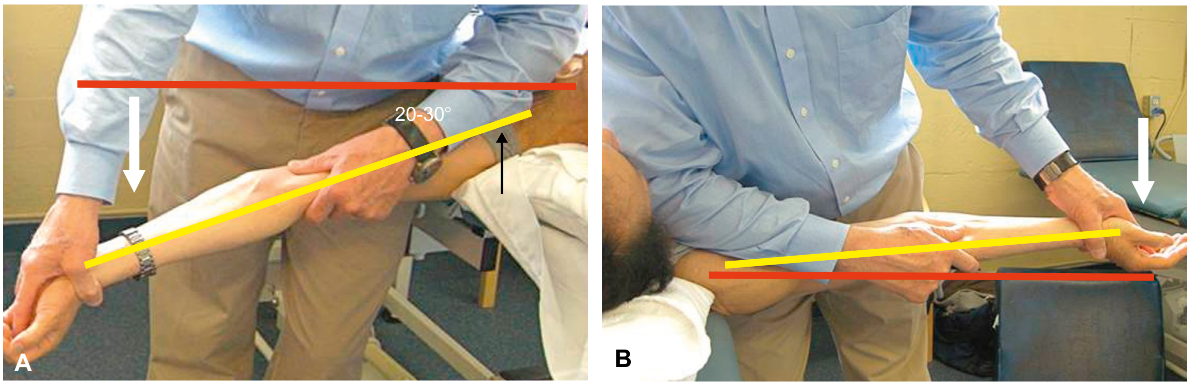

Figure 6-27 Horizontal extension test. Note how the examiner is using his elbow to prevent anterior movement of the humeral head in the glenoid. **A**, Normal. **B**, Restricted movement.

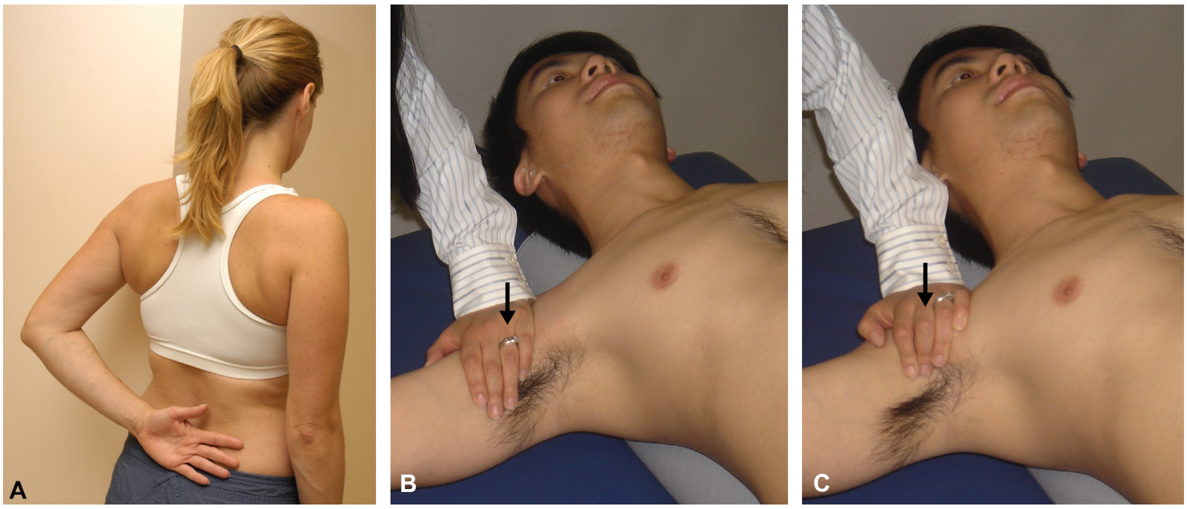

Figure 6-28 Stretching pectoral muscles. **A**, Doorway stretch (shoulder should not hike up) for pectoralis major. **B**, Clinician stretching pectoralis major (push down on head of humerus). **C**, Clinician stretching pectoralis minor (push down on coracoid process).

or reinforce faulty movement patterns. The clinician should teach the patient how to do stretches correctly so that normal tissues are not compromised and surgically repaired structures are not abused.[3,85] Anterior stretching involving a tight pectoralis major may be done by doing a "doorway" stretch (Figure 6-28, *A*). Pectoralis major and minor may be stretched by the clinician ensuring that the humeral head and scapula do not tilt forward and by giving resistance over the coracoid process for pectoralis minor and over the humeral head for pectoralis major (Figures 6-28, *B* and *C*, respectively). Posterior scapular stretching should be performed in three positions—at nipple-diaphragm line, shoulder-acromion line, and chin-mouth (Figure 6-29). Traction stretching (Figure 6-30) can be used to stretch the whole upper kinetic chain, but the clinician must ensure stress is applied to (i.e., is felt by the patient in) the tight structures. The sleeper stretch and modified sleeper stretch (Figure 6-31) are two examples of stretches for a tight posterior glenohumeral joint capsule, provided the clinician is sure the stretching is occurring posteriorly and is not the result of impingement.

6. *Failure to control pain, fatigue, and painful movement.* Exercises must be virtually pain free (pain is less than 4 on a 0 to 10 pain scale [0 being no pain; 10 being the worst pain the patient can imagine]). Otherwise, rehabilitation will be compromised. Ideally, exercises should be performed in a pain-free ROM. The clinician must design exercises that enable the patient to do the activity without pain. It is important to watch for signs of fatigue and to terminate the exercise before excessive fatigue leads to incorrect movement patterns. For example, early activity or overactivity of the upper trapezius is an early sign of fatigue in shoulder exercises.
7. *Failure to correct poor posture.* A normal posture (i.e., no rounded shoulders) is important for reducing anterior pressure on the anterior labrum and anterior structures of the shoulder both statically and dynamically, so the clinician must watch the patient closely to ensure correct

Figure 6-29 Stretching the posterior capsule of the glenohumeral joint. The clinician forward flexes the arm to 90° and then pushes the humeral head backward by pushing down on the elbow. While pushing down, the clinician carefully adducts the arm, increasing the tension on the posterior capsule. The stretch is performed at the nipple-diaphragm line, shoulder acromion line, and chin-mouth line, and the patient should feel the stretch posteriorly.

Figure 6-30 Self-traction stretch. **A**, In elevated forward flexion. **B**, In abduction. Patient should relax muscles around glenohumeral joint as much as possible while doing the stretch.

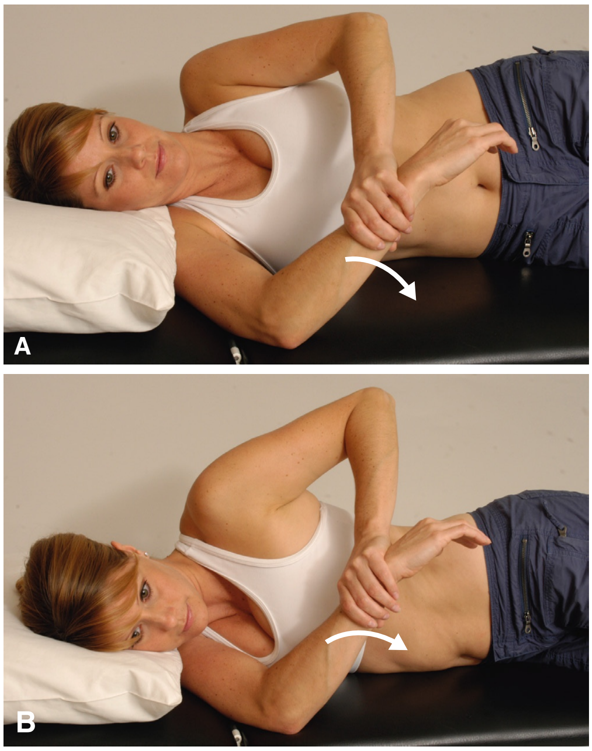

Figure 6-31 **A,** Sleeper stretch. **B,** Modified sleeper stretch.

posture and muscle action during movement. The clinician must make sure the patient can maintain a neutral pelvis position (i.e., patient can hold the neutral pelvis statically and dynamically by ensuring correct alignment of the pelvis and spine), proper trunk alignment, rib mobility, and scapular positioning. The clinician should also teach the patient to maintain the correct shoulder position by discouraging exercises that cause rounding of the shoulders (e.g., scapular dumping).

8. *Failure to control for fatigue and compensating postures.* The number of repetitions a patient is asked to do should be based on the number the patient can do correctly with control. The clinician should be specific in the instructions regarding the number of repetitions or sets the clinician wants the patient to complete. Normally, a specific number of repetitions should not be given; rather, the clinician should watch for and

Indicators of Fatigue or Loss of Control or that Load Being Used is Too Great

- Erratic movement
- Incorrect movement patterns
- Breath holding
- Inability to "hit" target
- Phasic movements
- Instability jog or catching movement
- Trick movements, loss of control of the scapular base
- Facial signs of fatigue

teach the patient to be aware of evidence of fatigue. The patient should recognize fatigue and its compensating postures and stop when they occur. Motor control, muscle memory, the firing (contraction) sequences of muscles (stabilizers, then mobilizers), fatigue patterns, and muscle and cardiovascular endurance all play major roles in the rehabilitation process.

9. *Failure to note habitual exercises performed in compensatory postures.* Many clinicians tend to progress exercise routines too quickly, before achieving isolated muscle control, especially of the scapular control muscles. Scapular control implies that the patient is able to control the scapula through contraction of the scapular force couple (lower and upper trapezius, serratus anterior) (see Figure 6-2, *A*). This is essential because it enables the patient to establish proper scapular rhythm while moving the arm (i.e., the humerus) in a controlled fashion. Many activities start with little or no weight, often with gravity eliminated or only very small amounts of weight (0.5 to 0.8 kg [1 to 2 lb]). Progression in resistance should be small. No advantage is gained from lifting heavy weights.
10. *Failure to select appropriate (low risk) exercises.* When asking the patient to do exercises, the clinician must remember to correct the patient's posture, ensure scapular control or stabilization, ensure functional isometric exercises are performed in uncompromising positions, and retrain co-contraction or arthrokinematic control at the glenohumeral joint. Speed of movement should only come toward the end of treatment and should not be a major focus until the functional phase. Initially the goal should be to use low-risk exercises that use the rotator cuff to compress the head of the humerus into the glenoid, such as two-hand stick drills (narrow and wide handle), medial and lateral rotation, and wiper drills (Figure 6-32). Such exercises may include rhythmic stabilization and co-contraction using the supine or lateral Hawkins position (i.e., placing the glenoid under the humerus) (see Figure 6-22, *A* and *B*).[2-4,85] Hawkins drills can include, in the Hawkins positions, small circles, bilateral open and closed gate, scapular movements on a stable arm, one and two arm punch outs, and table and floor push ups with a plus to flys and arm scissors and, as the patient progresses, core exercises—scapular push ups, wall or floor planks (hold core while allowing scapula to move in and out of winging), lateral plank, and V-plank (Figure 6-33).

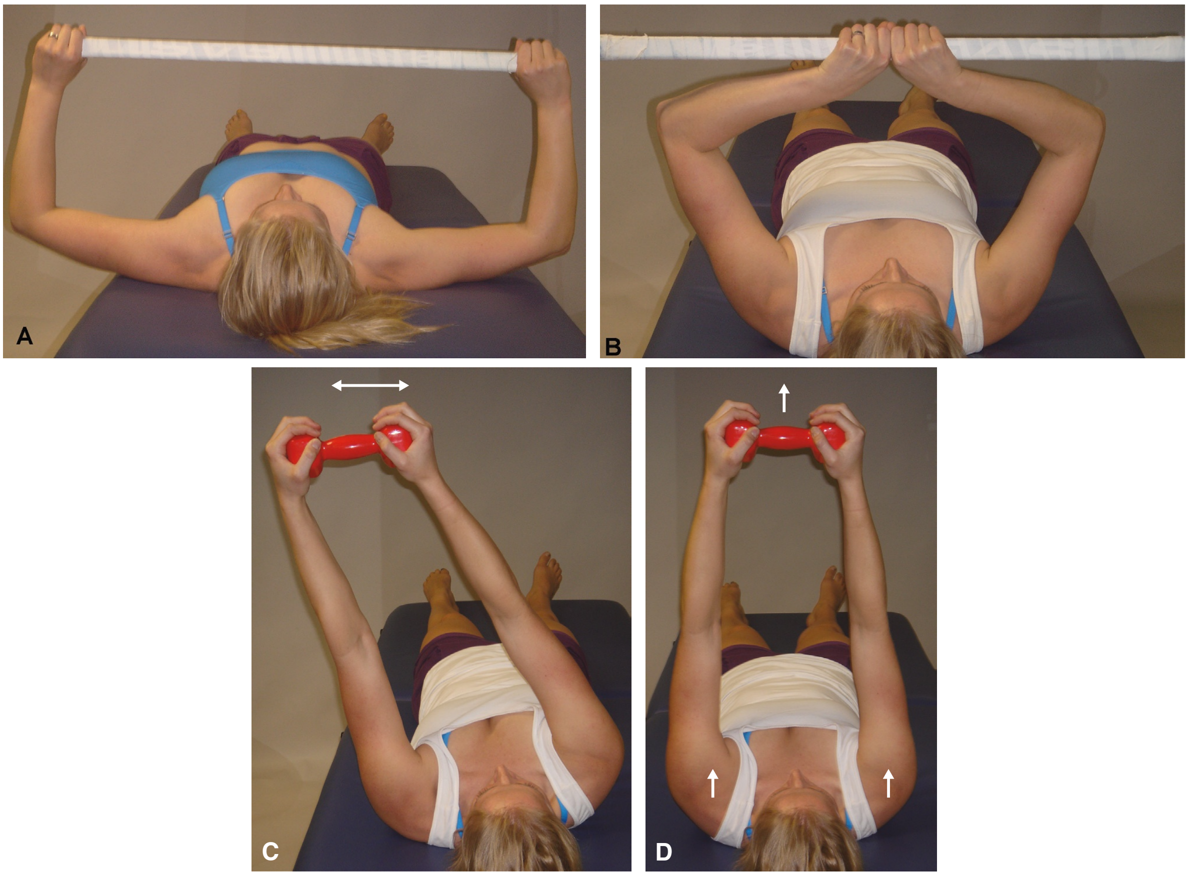

Figure 6-32 Low-risk exercises. **A**, Stick drill–wide hands (power square position). **B**, Stick drill–narrow hands. **C**, Supine wiper drill (can go side to side and up and down to 90°). **D**, "Reach for ceiling" then do circles while maintaining reach. (All exercises can be done in the three zones - i.e., level of diaphragm, shoulder, and forehead.)

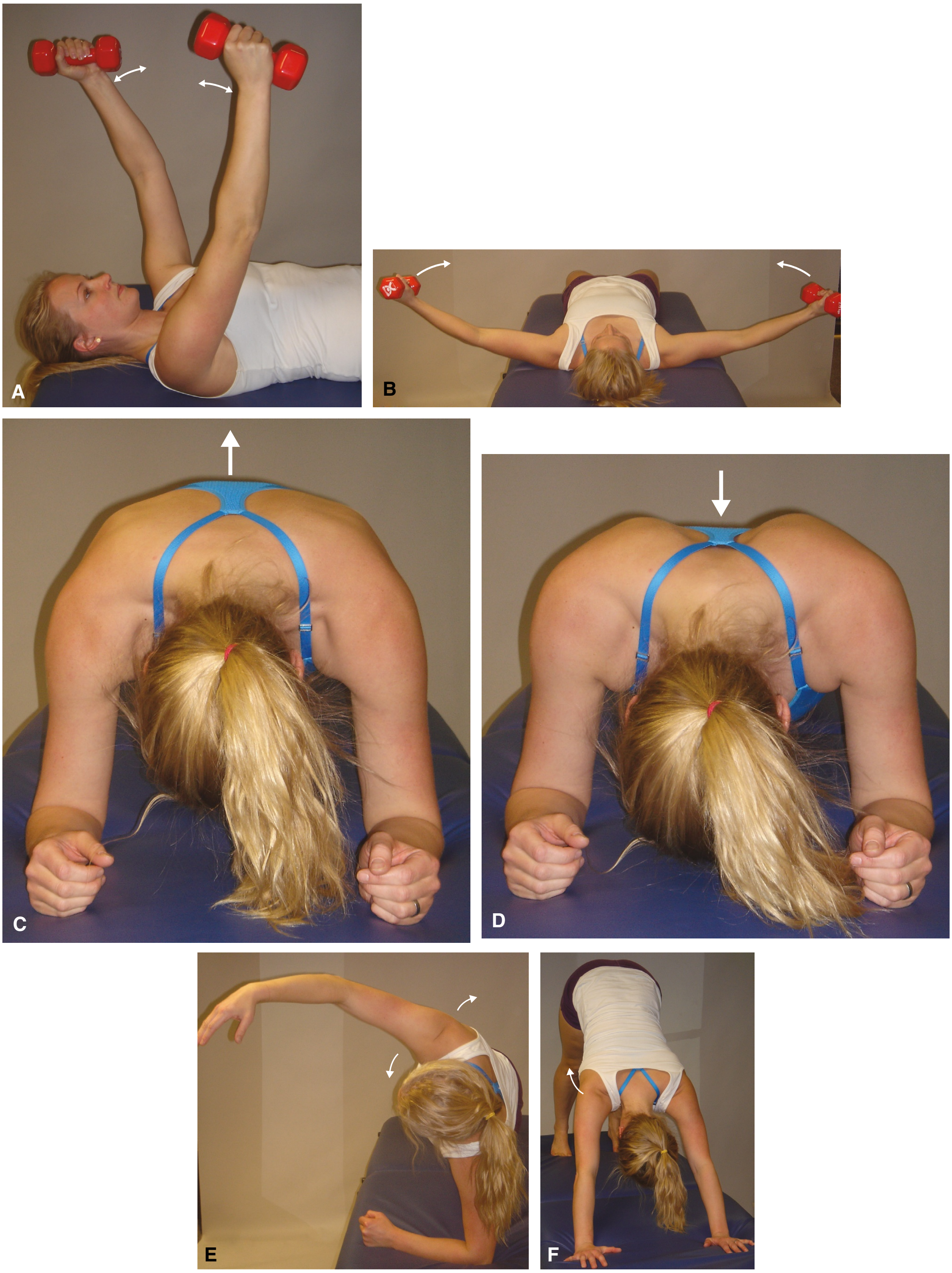

Figure 6-33 Advanced low-risk exercises. **A**, Scissors (punch to ceiling). **B**, Pectoral flies. **C**, Scapular push up (stabilize core, straight spine, scapula should be flat against ribs). **D**, Scapular push up down (relax). Note how scapula wing away from ribs. **E**, Lateral plank with arm motion. Note how left humerus is compressed into glenoid. Motion of right arm and trunk allows arthrokinematic slide at left glenohumeral joint. **F**, "V" plank. Patient can roll back and forth over shoulder again allowing arthrokinematic sliding at the glenohumeral joint.

Low-risk exercises that help to stabilize the humeral head and keep the shoulder away from compromising pinching or impingement postures are preferable. While the patient does the stabilization exercises, the clinician can palpate the humeral head to make sure that it is properly aligned, compressing into the glenoid, and not translating excessively which would indicate loss of arthrokinematic control. The clinician also can make sure that the anterior deltoid contracts and is part of the exercise routine. Shoulder depression against tubing resistance while doing circles or figures of eight (Figure 6-34) is another example of a low-risk exercise. Punch out exercises (Figure 6-35, *A*), dynamic hug (Figure 6-35, *B*), and push ups with a plus (Figure 6-35, *C*) along with PNF exercises (D2 patterns) (Figure 6-36) work the serratus anterior and help to ensure minimal upper trapezius activity. Forward flexion wall exercises with the hands on a ball in a closed kinetic chain (Figure 6-37) also are low-risk exercises. The clinician's repertoire of rotator cuff exercises must include more than simple medial and lateral rotation in neutral.

The empty can position puts the glenohumeral joint in a compromising impingement posture and in the anterior tipped (i.e., scapular anti-tilt) position. The classic empty can strengthening exercise actually is an impingement posture and is not functional for most activities.[136] This exercise does not help to re-establish scapulohumeral rhythm and often is painful early in the rehabilitation process, leading to counter-productive exercises.

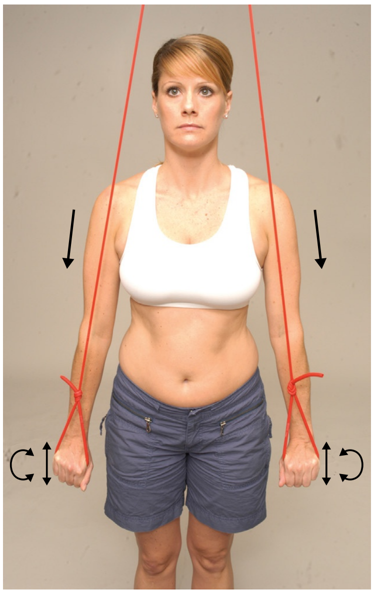

Figure 6-34 Shoulder depression (i.e., dynamic caudal glide) using tubing. Example exercises include having a patient do circles or figures of eight or write his or her name. Clinician should always watch for scapular control.

The clinician should always be aware that traditional weight room exercises used to strengthen the shoulder musculature and surrounding structures can often compound an injury and should be discouraged until the person is in the functional stage of recovery. Traditional gym exercises, such as the bench press and military press, may compound or precipitate shoulder problems. A common mistake of clinicians is to introduce these exercises too early in the rehabilitation program. Early rehabilitation concentrates on training the individual stabilizers of the scapula and humerus. Once this has been achieved, more complex functional motor patterns can be initiated.

11. *Failure to create an exercise program or to make use of functional patterns in later stages of rehabilitation that integrate lower extremity and trunk movements.* These exercises incorporate the whole kinetic chain and are complementary to traditional rotator cuff and scapulohumeral exercises.[2-4,48,82] Examples include the lunge walk with and without trunk rotation (Figure 6-38) and using "lift and chop" patterns.
12. *Failure to rehabilitate the deceleration (eccentric) component of shoulder activity.* Controlled eccentric lengthening of muscles, especially the posterior shoulder muscles, is essential for proper functional return to activity. The patient needs to control both the concentric and, even more important, the eccentric components (i.e., eccentric braking and shock absorption, especially for the posterior muscles). The patient must be able to control movement regardless of its direction. This is especially important when tubing is used. If the patient has to eccentrically control the shortening of the tubing as well as its lengthening (concentric movement), the exercises become more difficult, the patient will be able to do fewer repetitions, and control will improve more quickly.
13. *Failure to address proprioception (static) and kinesthesia (dynamic).* Proprioception is an important and integral part of the rehabilitation process for shoulder instability and any exercise performed by the patient has a proprioceptive or kinesthetic component. The patient must develop an understanding of the sensation of joint movement as well as joint position. Lephart and Kocker[137] recommended a progression of activities to restore proprioception and neuromuscular control, including starting with joint position sense, moving on to dynamic joint stabilization (i.e., kinesthesia), then reactive neuromuscular control, and finally function-specific exercises. For joint position sense, the Hawkins position can be used to position the glenohumeral joint with the patient's eyes open or closed, for rhythmic stabilization and eccentric break exercises (see Figure 6-22, *C*, and text for description). To do these exercises, the clinician positions the patient

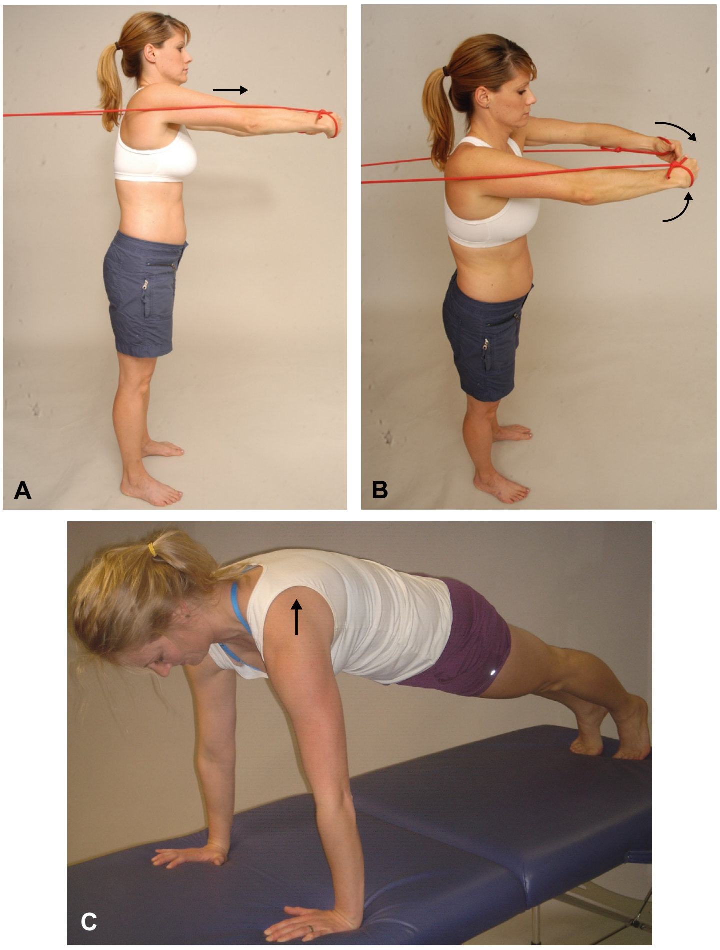

Figure 6-35 Exercises for serratus anterior. **A**, Punch out. **B**, Dynamic hug. **C**, Push up with a plus.

Figure 6-36 Exercise for proprioceptive neuromuscular facilitation (e.g., D2 pattern) using tubing in a lunge position. Note patient aiming for a target, which will help to give an indication of onset of fatigue. If the patient cannot consistently hit the target, he or she is fatigued or the resistance is too great.

and then says, "Don't let me move you," while applying co-contraction forces at the joint, along with eccentric breaking forces on the arm. PNF exercises can be used for dynamic joint stabilization and joint loading with closed kinetic chain exercise (Figure 6-39). To train reactive neuromuscular control, plyometrics, throwing and catching, balance drills, and "falls" (Figure 6-40) all play a role later in the rehabilitation process. For functional motor patterns, PNF (acceleration and deceleration), Body Blade® and Boing® exercises (Figure 6-41), and actual controlled functional activities can be used.

14. *Failure to take the time to work one on one with the patient.* Clinicians must make sure the patient performs the proper movement patterns and controls posture, and they must assist with the timing and sequencing with compensatory motions. This is performed by using individualized, accommodating resistance through all parts of the ROM.
15. *Failure to integrate the patient into the treatment program.* The clinician must convince the patients that their efforts play a crucial role in the rehabilitation process. Treatment for shoulder instability

Figure 6-37 Forward flexion closed kinetic chain exercises. **A**, Without a ball against wall (stable). **B**, With a ball against wall (unstable). Patient can be told to retract, elevate, depress, and protract the scapula while pushing against wall or ball.

Figure 6-38 Lunge walk with "lift and chop" patterned movement. Patient does lunge as arm goes through PNF pattern, stopping at ASIS bilaterally.

Figure 6-39 Joint stabilization in closed kinetic chain. Resistance for arthrokinematic control (*1*) or osteokinematic control (*2*).

The Patient as "Quarterback"

- Follow clinician instructions
- Ensure correct posture before beginning
- Stabilize the "core," then the scapula before beginning
- Start with "power square" position in supine
- Watch for compensating postures and stop when they occur
- Be sure movement pattern is performed correctly
- Stop when fatigued
- Ask clinician questions if you do not understand

will not succeed unless the patients are actively involved—"they are the quarterback!" who must follow the instability treatment guidelines. The patients must feel confident and in control of the rehabilitation exercise program. They also must have a thorough understanding of the exercises they need to do and why they need to do them.

16. *Failure to use caution when using exercise machines.*[48] Generally machines use long lever arms; this makes controlling the motion difficult and increases the risk of a joint shear, especially if the patient has not

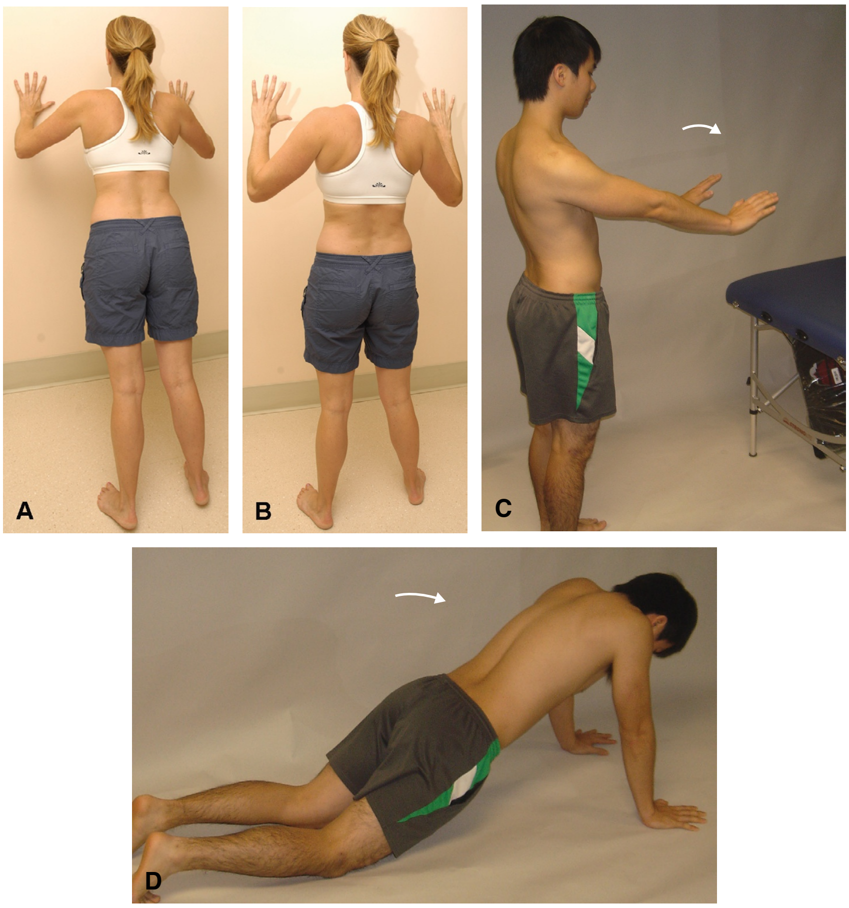

Figure 6-40 Proprioceptive exercises. **A,** Wall push up. **B,** Wall fall: the patient falls forward and catches himself/herself. This can be performed with the eyes closed. **C**, Table fall. **D**, Floor fall.

gained arthrokinematic control. Exercises performed on these machines often are not functional and only work isolated parts of the kinetic chain. In addition, they often emphasize anterior muscle groups rather than posterior muscle groups, which are often more important, especially with anterior instability.

17. *Failure to maintain or restore an appropriate fitness level.* Patients must be encouraged to maintain or do activity during their recovery from injury. Depending on the fitness level of the individual, this may involve low-level activities or higher-level activities within the confines of what the injury will allow them to do. Activities such as walking ("taking the shoulder for a walk"), water activities (walking in water with breast stroke action), and using an upper body exerciser (UBE) (Figure 6-42) to "bicycle the shoulder" are all activities that can help to maintain some aerobic activity and contribute to the feeling of well-being. Improving fitness will also enable the patient to recover from an exercise regime more quickly.

CONSERVATIVE TREATMENT OF INSTABILITY

For proper conservative treatment of impingement or instability syndromes, patients must understand the problem and must recognize that the outcome depends on their compliance with the clinician's instructions about what they should and should not do. The physician will prescribe rest, anti-inflammatory medication, pain-relieving medication, and physical therapy in the early stages. However, in most cases, sound rehabilitation is the key to resolution of these problems.

If the problem is one of atraumatic instability, the rehabilitation process follows a course that ensures that the exercises activate the appropriate muscle or muscle groups in the proper sequence and that functional control is achieved (see Volume 2 of this series, *Scientific Foundations and Principles of Practice in Musculoskeletal Rehabilitation*, Chapter 19).

Although many modalities may be used to treat instability and impingement, the clinician's primary concern is to restore normal shoulder mechanics and function by

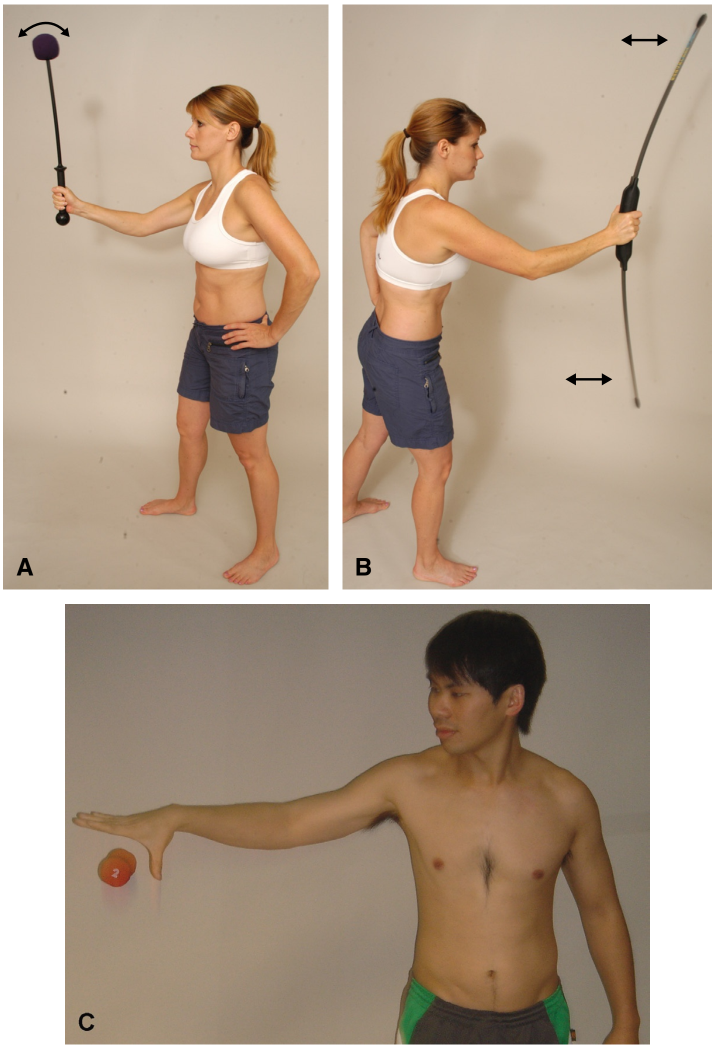

Figure 6-41 Proprioceptive drills using (**A**) Boing, (**B**) Body Blade, and (**C**) Dumbbell drop and catch.

Stabilization Training Sequence

Stages	Steps	Purpose
Stage 1	1.1	Reduce pain
	1.2	Allow freedom of movement (normal arthrokinematic movement)
Stage 2	2.1	Ensure proper muscle function
	2.2	Ensure proper muscle recruitment and motor patterns
	2.3	Correct muscle imbalances
Stage 3	3.1	Correct endurance discrepancies
	3.2	Correct strength discrepancies
Stage 4	4.1	Retrain proprioception and kinesthesia
Stage 5	5.1	Re-educate the muscle stabilizers statically
	5.2	Teach advanced static stabilization exercises
	5.3	Re-educate the muscle stabilizers dynamically
	5.4	Teach advanced dynamic stabilization exercises
	5.5	Teach functional stabilization
Stage 6	6.1	Restore or maintain fitness throughout program

restoring the muscles and improving their coordinated action, contraction sequence, strength, endurance, and proprioceptive or kinesthetic control.[2–4,47,85]

In the early stages of rehabilitation, the emphasis is on scapular control, humeral head stabilization, and restoring the coordinated action of the force couples of the scapulothoracic and glenohumeral joints.[35,85,134,138] This should include integration of the lower limb and trunk as early as possible. Examples of glenohumeral force couples include the subscapularis muscle (a primary stabilizer following anterior dislocation), counterbalanced by the infraspinatus and teres minor muscles, and the deltoid moving the humerus while the supraspinatus, infraspinatus, teres minor, and subscapularis muscles compress the humeral head into the glenoid.[30] By establishing strength and neuromuscular control of these force couples, the patient will regain control of humeral head translation (i.e., arthrokinematic control) during dynamic movement.[79]

Figure 6-42 Upper body exerciser.

Proper rehabilitation of instability of the shoulder involves controlled aggressiveness in terms of pushing the patient and pushing the envelope of progression for the patient as much as possible, while making sure the patient can control the movement and do it correctly. Because corrective movement depends on force generation and coordinated action of the muscles, as well as freedom of joint movement, the clinician must ensure that patients work only in the ROM in which they are able to control the movement and to do the movement correctly (i.e., using correct movement patterns). This is demonstrated by patients working at a speed at which they demonstrate movement control through precise, smooth (nonjerky) movement patterns, although part of the later rehabilitation continuum involves ensuring that patients progress to functional speeds and loads. With instability, the clinician is concerned with dysfunction that is primarily a motor control problem. The stabilization regimen is a multifaceted program that corrects impairment by improving neuromuscular control and coordination of specific muscles and movements of the shoulder. At the same time, the clinician must work to correct the mechanical factors that predispose or contribute to the abnormal movement patterns. This requires active participation and "buy in" by the patient and an accurate diagnosis by the clinician to determine what restrictions are present; that is, what muscles are at fault and which movements are being done incorrectly. This education and exercise program involves maximum psychological concentration on the part of the patient and very specific movement patterns at minimal speed in a range in which the patient has control. Throughout the process, the principles of specificity apply (see the Principles of Specificity Related to Sports and Rehabilitation box on p. 209). Stabilization is best achieved by isometric (co-contraction) exercises for static stabilization and concentric-eccentric exercises for dynamic stabilization.

The following steps are not necessarily sequential, but the clinician must consider each one to obtain optimum treatment outcomes. For example, with anterior instability, the clinician may treat the anterior glenohumeral joint for pain (step 1.1) while at the same time, treating the scapula by ensuring isolated contraction of the lower trapezius (step 2.1).

Step 1.1: Reduce Pain (Aim: Pain Control)

The first step is to relieve pain or at least diminish it because the aim is to establish pain control.[85] Instability training may be fatiguing, but it should not be painful. Generally, pain should be kept below a 4 on a 0 to 10 visual analogue scale or at a level that ensures control throughout the exercise (Figure 6-43). This can be accomplished through the use of pain-relieving modalities (e.g., ice, transcutaneous electronic nerve stimulation [TENS], interferential therapy) or by positioning the shoulder in the resting position (see Figure 6-11, *A*). Other approaches that have a role in pain management include modified traction (e.g., shaking out the arm and "throwing" the arm [Figure 6-44]), glenohumeral compression activities, using a sling, nonsteroidal anti-inflammatory drugs (NSAIDs), and gentle mobilizations.[2-4] Exercises should be low risk (i.e., isometric or slow speed) starting from a stabilized or the resting position and ensuring humeral head compression in the glenoid.

Rest from activity is relative and implies decreased length, intensity, and/or frequency of activity. It seldom means total rest. It is important to "take the shoulder for a walk" for 30 minutes each day, which implies moving the shoulder (e.g., swinging the arms while walking).

Step 1.2: Allow Freedom of Movement (Aim: To Restore Normal Arthrokinematic and Osteokinematic Movement)

The clinician should address any movement restriction in any of the joints, muscles, or inert tissues of the shoulder complex, including the spine and ribs. This often is necessary to restore normal arthrokinematic movement in the glenohumeral joint (i.e., allowing the humeral head to centralize in the glenoid cavity), acromioclavicular joint, sternoclavicular joint, scapulothoracic "joint," and thoracic

Figure 6-43 Pain-monitoring system (visual analogue scale).

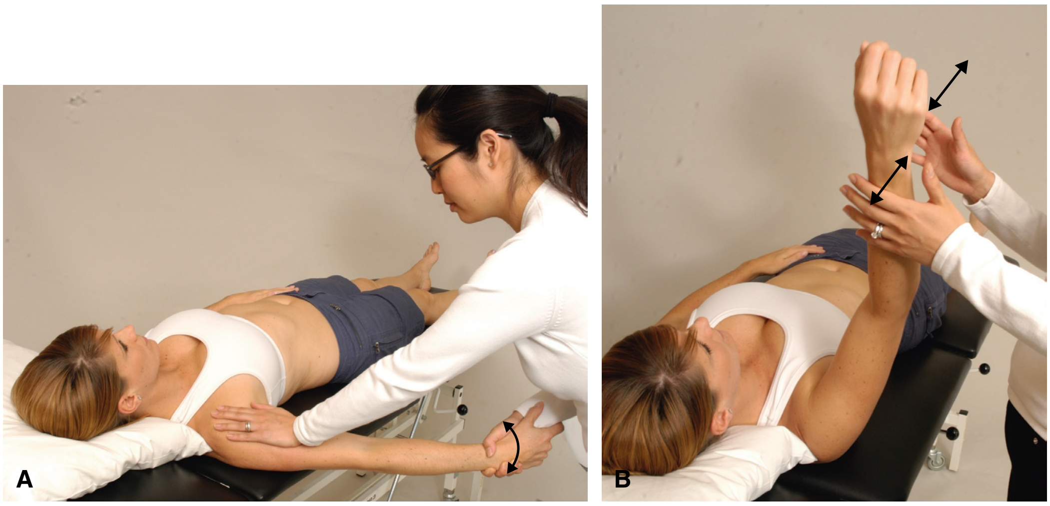

Figure 6-44 **A**, Shaking the arm. **B**, Throwing the arm.

spine and upper ribs.[21,85] The clinician should check for loss of medial (internal) rotation (Mattison 20 cm test) (see Figure 6-26), restriction of the inferior glenohumeral ligament (decreased inferior glide at the glenohumeral joint), a tight posteroinferior capsule (causes the humeral head to migrate anteriorly and superiorly[2]), and any loss of range due to tightness of the pectoralis major and/or pectoralis minor. Restricted rib motion can limit end ranges of shoulder motion. All have the potential of being tight.[2–4]

A tight posterior capsule, which is often seen with instability, can be suspected if, in supine, there is a loss of medial rotation at the glenohumeral joint, tight anterior chest muscles, restricted shoulder extension performed with the elbow at the nipple-diaphragm line, clavicle-shoulder, and/or chin-forehead (three positions), decreased inferior glide, and restriction at arm 90°-90° (arm forward flexed to 90° and elbow bent to 90°), followed by medial rotation.

Tight Structures Seen in Shoulder Instability and Impingement

- Pectoralis major and/or minor
- Subscapularis
- Posteroinferior capsule
- Ribs
- Thoracic spine

The clinician must always keep in mind the possibility of hypermobility at least in some directions, which tends to be common in individuals with an impingement or instability disorder. Altered glenohumeral arthrokinematics must be assessed and treated as necessary. The clinician must assess carefully to determine which movements are restricted and should treat only these movements, so that the joint play mobilization techniques to stretch the capsule are effective.[138–142] As the circle concept of movement implies, if the joint is hypermobile in one direction, it may be hypomobile in the opposite direction. With anterior instability, the posteroinferior capsule tends to be tight and therefore requires mobilization, whereas the anterior capsule is hypermobile and requires protection. The clinician should always check the ribs (Figure 6-45) and upper thoracic spine because these are commonly tight with associated chronic shoulder problems.

Active-assisted exercises using a rigid bar (i.e., a T bar, L bar, wand, or stick) may be used to increase ROM.[8,93,138] Stretching of the posterior structures can be accomplished using a stick (Figure 6-46), with stretches performed in different ROMs of shoulder abduction. In the United States, it is currently popular to use a sleeper, modified sleeper, or rollover sleeper stretch or doorway stretch (see Figures 6-28 and 6-31). If the sleeper stretch is to be used, the clinician must ensure the patient feels the stress or stretch posteriorly. Many patients feel pain anteriorly when doing the sleeper stretch, indicating a compromising position with the tissues being impinged anteriorly. Traction stretch (see Figure 6-30) in several positions of abduction will also gain medial rotation.

Posterior stretching may also be accomplished by forward flexing the arm and then pushing the humerus posteriorly (see Figure 6-29). The technique may also be performed actively by having the patient resist as the clinician pushes the humerus posteriorly. Humeral head depression (Figure 6-47) is an effective technique for ensuring inferior glide of the humeral head if excursion of the humeral head under the acromion is restricted.[143] It also poses less risk of compromising the medial scapular stabilizers. Other posterior stretch techniques include anterior chest eccentric breaks and anterior–posterior traction scoop.

Figure 6-45 Checking the mobility of the ribs. **A,** First rib with patient in the supine position. **B,** First rib with patient in the prone position. **C,** Other ribs. **D,** Gentle "pounding" ribs feeling resistance to movement. **E,** Extension and rotation of thoracic spine and ribs.

Following anterior surgery, the clinician should not stretch or stress the anterior capsule in the early stages of recovery. In most postoperative programs, stretching of vulnerable tissues is contraindicated for the first 6 weeks. In these cases, it is important to follow the surgeon's protocols. In nonsurgical cases, stretching should be performed only in hypomobile directions using precise positions and complete control. Restriction may be the result of inert tissue tightness or muscle tightness, and different techniques are used to address these two problems. If the patient has signs of muscle tightness (most commonly, the pectoralis minor and subscapularis),[86] the clinician may use muscle energy techniques, hold-relax, or active release techniques or passive (anterior chest) stretching (Figure 6-48). Trigger point therapy, another muscle technique, can be used effectively to increase

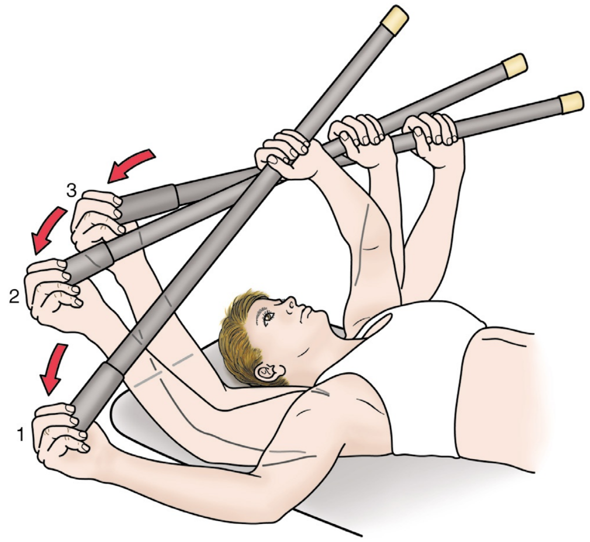

Figure 6-46 A stick can be used to increase the ROM in the glenohumeral joint by going through different parts of the ROM.

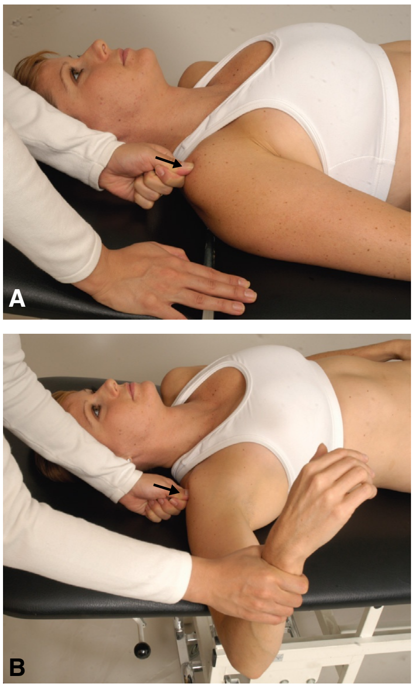

Figure 6-47 Humeral head depression. **A**, Arm by the side. **B**, Arm in 90° abduction.

Figure 6-48 Stretching the anterior structures of the shoulder and trunk on a foam roll ("open book" stretch).

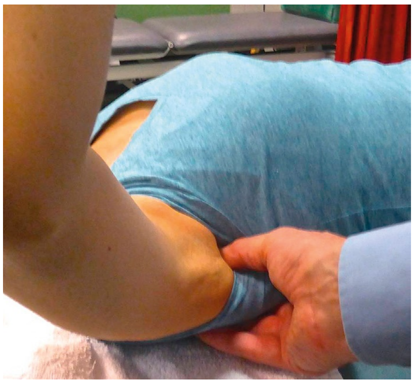

Figure 6-49 Trigger point therapy to "strip" subscapularis. The clinician holds the arm in slight abduction and forward flexion. Using the thumb (or middle and ring fingers) of the other hand, the clinician palpates for the trigger points on the striations of subscapularis. The clinician then applies trigger point pressure with the thumb while with the other hand the clinician resists the patient isometrically holding the arm in various postures. The clinician then eccentrically breaks the postural holds. The clinician does 3 to 5 hold and break positions while applying different trigger point pressure for each of the hold positions

range with isolated muscle tightness (e.g., the subscapularis) (Figure 6-49). Restriction or loss of abduction, inferior humeral glide, or medial or lateral rotation of the shoulder may also need to be addressed.[85]

Clinicians must also consider the effect of crossed syndromes (Figure 6-50) on the mobility and strength of muscles about the shoulder. **Janda's "upper crossed" syndrome** showed that muscles (primarily postural) on one diagonal at a joint could be tight and hypertonic, whereas muscles on the other diagonal were weak and long.[144] Because similar cross syndromes can affect flexibility in the whole kinetic chain, inflexibility in the upper and lower kinetic chains may need to be addressed.[85] The clinician should assess for and treat any tight lower kinetic chain structures, including the Achilles-calf musculature, hamstrings, hip flexors, abductors, and extensors. Tight trunk flexors and abnormal gait patterns must also be considered.[85]

Pendular exercises (e.g., the Codman pendular arm swing exercises) may be used initially (see Figure 6-20), provided the exercises are pain free and the patient can control the movement (start with small pendular exercises progressing to scapular and humeral muscle control

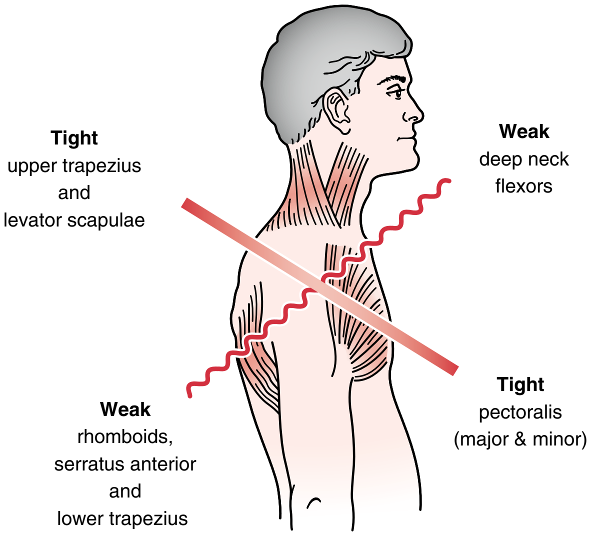

Figure 6-50 Upper crossed syndrome. (From Magee DJ: *Orthopedic physical assessment*, ed 6, p 165, Philadelphia, 2014, Saunders/Elsevier.)

with circles). Caution is required with pendular exercises, especially after arthroscopic surgery, because they apply gravity traction to the repaired vulnerable structures. The clinician may also try modified Codman exercises, which eliminate gravity traction by having the patient roll a ball on a low chair or table with the elbow extended (see Figure 6-20, *C*). This prevents inferior translation and caudal traction on the glenohumeral joint. Anterior translation of the humeral head (common in active lateral rotation) must be avoided, especially in patients with anterior instability.[145] The clinician must not allow scapular hitching (i.e., upward movement) or dumping (i.e., downward tilting of the glenoid) with any assisted exercises. The arm may be swung horizontally in a variety of planes. These exercises are useful in the early stages to maintain ROM, but compensated shoulder postures must be avoided because some reports have indicated that in pathological shoulder conditions, the upper trapezius and supraspinatus are less likely to relax.[146] Shoulder depression exercises using tubing (e.g., dynamic caudal glide) help to improve range, enhance scapular control, and promote coordinated scapulohumeral and glenohumeral activity.

Step 2.1: Ensure Proper Muscle Function (Aim: To Ensure All Muscles Contract Individually)

Ensuring Proper Muscle Function

- Start by positioning the muscle in the "muscle test" position
- Use low (10% to 30% MVC) isometric contraction
- STOP as soon as fatigue is evident
- Work muscle in inner range of movement

This stage involves restoring individual muscle function to ensure proper muscle recruitment or proper activation of the muscle. Proper contraction of the scapular force couple, including the lower trapezius with the serratus anterior, requires special attention. If the patient does appropriate exercises and compensatory postures are not allowed, these muscles will contract or "turn on." The upper trapezius and latissimus dorsi can dominate in compensatory postures, causing an incorrect movement pattern. For example, with a Kibler type III dyskinesia of the scapula, the upper trapezius, levator scapulae, and posterior deltoid do not function in concert most of the time, resulting in the postural deformity.[2–4,86] Biofeedback may be used to teach the patient to contract the desired muscle and to "turn off" the unwanted compensatory muscle contraction.

To encourage isolated muscle contractions (i.e., specificity), the patient is positioned in the "muscle test" position for that muscle.[43–45] The clinician positions the patient so as to isolate the muscle and then asks the patient to hold the position against minimal clinician resistance to ensure that the desired isometric contraction is achieved. The patient contracts the muscle only to 10% to 30% of the MVC to ensure an isolated contraction of the muscle while the clinician watches for no compensatory contraction of other muscles. Functional activation may be tested by doing "open shutter" and supine and standing "streamline" exercises, "four corner" drills, and PNF diagonals (Figure 6-51).

During these early stages of treatment, it is important that the clinician watch for the first sign of fatigue and stop the patient when it appears. Signs of fatigue are shaking when doing movement, not being consistently able to hit a target, loss of or change in segmental colinear posture, inability to hold proper functional posture, or contraction

Figure 6-51 Functional activation exercise. PNF D2 diagonal patterns.

of compensatory muscles. Early on, patients commonly find contracting the isolated muscles difficult and in some cases, impossible—these people need electrical stimulation to the muscles to "reboot" them, and they frequently show psychological fatigue, which mirrors physiological fatigue.

If inferior instability is present, the scapula must be able to stabilize both statically and dynamically, the humeral head must be balanced in terms of ROM (e.g., medial and lateral rotation should be close to equal), compression of the humeral head by the rotator cuff must be evident, and the patient must be able to contract the anterior and posterior deltoid muscles simultaneously.

Postural correction is an essential component of this step. The clinician should determine whether the patient can achieve the neutral pelvis position (i.e., stabilize the core) and hold the position during both static and dynamic activities. As the patient begins to assume and hold the neutral pelvis position (i.e., demonstrating a stable core), the posture will begin to correct. If the connection is slow or the patient appears to be having problems, the clinician must look for potential restrictive and weak cross syndromes that are limiting the return to normal posture and stretch (e.g., pectoralis minor, hip flexors) and strengthen these components (e.g., abdominal flexors, scapular control muscles).

Step 2.2: Ensure Proper Muscle Recruitment and Motor Patterns (Aim: To Ensure All Muscles Contract When They Should)

Ensuring Proper Muscle Recruitment and Motor Patterns

- When muscle can be recruited, the aim is then to ensure the muscle contracts when it is supposed to
- Make sure the patterns of movement are correct
- Watch for any compensatory or incorrect movement patterns
- Start with low-load isotonics and progress to eccentric (for muscle shock absorption and braking functions) and concentric (for correct movement) loaded exercises
- Can initiate proprioceptive exercises at this point keeping in mind the different proprioceptive receptors to be stimulated

At this stage, treatment is directed primarily at the stabilizer muscles of the scapula, followed by the stabilizers of the glenohumeral joint, because these muscles maintain the normal relationship between the glenohumeral and scapulothoracic joints. The scapular stabilizers provide a stable base for the arm, preserve deltoid fiber length so that acromiohumeral length is maintained, and help control impingement of the rotator cuff. Proper activation and functioning of the scapular stabilizers is essential (see Table 5-1).[49,85,147] The clinician must ensure that scapular substitution patterns are not occurring and that the stabilizer muscles contract before the mobilizer muscles.[148] For example, when the patient abducts the arm to 30°, the scapula should stabilize and "lock in" in this phase of scapulohumeral rhythm. In the first phase of arm abduction (0° to 30°), no or minimal scapular movement should occur. If the scapula keeps moving during the first 30° of abduction, the scapular stabilizers are not functioning properly and the mobilizers are dominating or the exercise is too advanced (i.e., too hard) for the patient. In the second phase (30° to 90°), the scapula should rotate but show minimal protraction or elevation. In the third phase (90° to 180°), lateral rotation to the humerus is necessary along with some elevation.

In scaption (i.e., the plane of the scapula), scapulohumeral rhythm is slightly different, with more individual variation in the movement. More scapular rotation and protraction often occur. In scaption, less lateral rotation of the humerus occurs and less stress is placed on the tissues of the shoulder. Therefore scaption is a good position for working the patient initially because there tends to be less pain with movement and scaption is a more common functional movement pattern.

If the patient is having difficulty determining how to contract individual muscles, electrical stimulation or biofeedback may be used.[149] Useful scapular control exercises include retraction and depression of the scapula, as well as elevation and protraction. These involve individual exercises for the three parts of the trapezius muscle, serratus anterior, rhomboids, and levator scapulae. Exercises for the scapular control muscles include scapular squeezes or pinch, thumb tubes in the three positions starting in the "power square" (90°-90°) position for the middle and lower trapezius, dynamic hug and punch outs for the serratus anterior, shoulder depression with tubing and press down exercises (Figure 6-52),[85] and V-plank exercises (see Figure 6-33, *F*) for compression of the humeral head into the glenoid.

Figure 6-52 Press down exercise for shoulder depression using bars.

At this step, force-couple training also plays a predominant role. Force-couple training involves recruitment of the muscles, along with synchronized action of the muscles using loaded and unloaded activities. Co-contraction (i.e., arthrokinematic stabilization) with rhythmic stabilizations has a stabilizing and stiffening effect, adds compression for joint position sense, and tends to be done isometrically.[85] Coordinated coactivation results in less torque or sheer loads on the glenohumeral joint on the shoulder and increased joint control. Co-contraction primarily works the small (inner cone) muscles (i.e., the rotator cuff) of the joint. An example is rhythmic stabilization to the humeral head in the Hawkins punch out position (see Figure 6-22, *A*) and scapular movements on a stable arm (e.g., closed kinetic chain scapular "clock" exercises—the patient moves the scapula through the different hours of a clock while stabilizing the hand against the wall). These exercises also help emphasize the rotator cuff, stabilize the glenohumeral joint by compressing the humeral head in the glenoid, and reduce deltoid activation.[48,85]

Closed kinetic chain activities in the shoulder (e.g., rhythmic stabilization) are more stable, involve less translation, provide static stability, and are often used with coactivation.[85] Closed kinetic chain activities provide motion control, sequencing, and improved proprioception and often are more functional.[48] Exercises include weight shifts with the hand on a stable or unstable surface, rotator cuff clock exercises, and wall washes ("wax on, wax off") exercises (Figure 6-53), and "wiper" and "Spiderman" (see Figure 6-21, *D*) exercises.[48]

Once scapular control has been achieved, humeral control exercises can be instituted for the rotator cuff and deltoid. These help to prevent humeral head migration and to restore voluntary arthrokinematic control of the humeral head through rotator cuff coactivation (especially of the supraspinatus and subscapularis) and stabilization, as well as activation of the biceps and triceps (Figure 6-54) and pectoralis major.[134–137,149] These exercises also can be used later in the rehabilitation program, as long as the patient has no impingement signs and good scapular control.

Figure 6-53 Exercises for proprioception, motion-controlled sequencing, and increasing ROM. **A,** "Clock" exercise for the scapula with the arm in abduction. **B,** Wall washes ("wax on, wax off"). **C,** Rhythmic stabilization with a stick in power square position. Note how clinician is palpating for arthrokinematic control of patient's right shoulder. **D,** Rhythmic stabilization with thumb tube in power square position. Clinician is applying perturbations to tube.

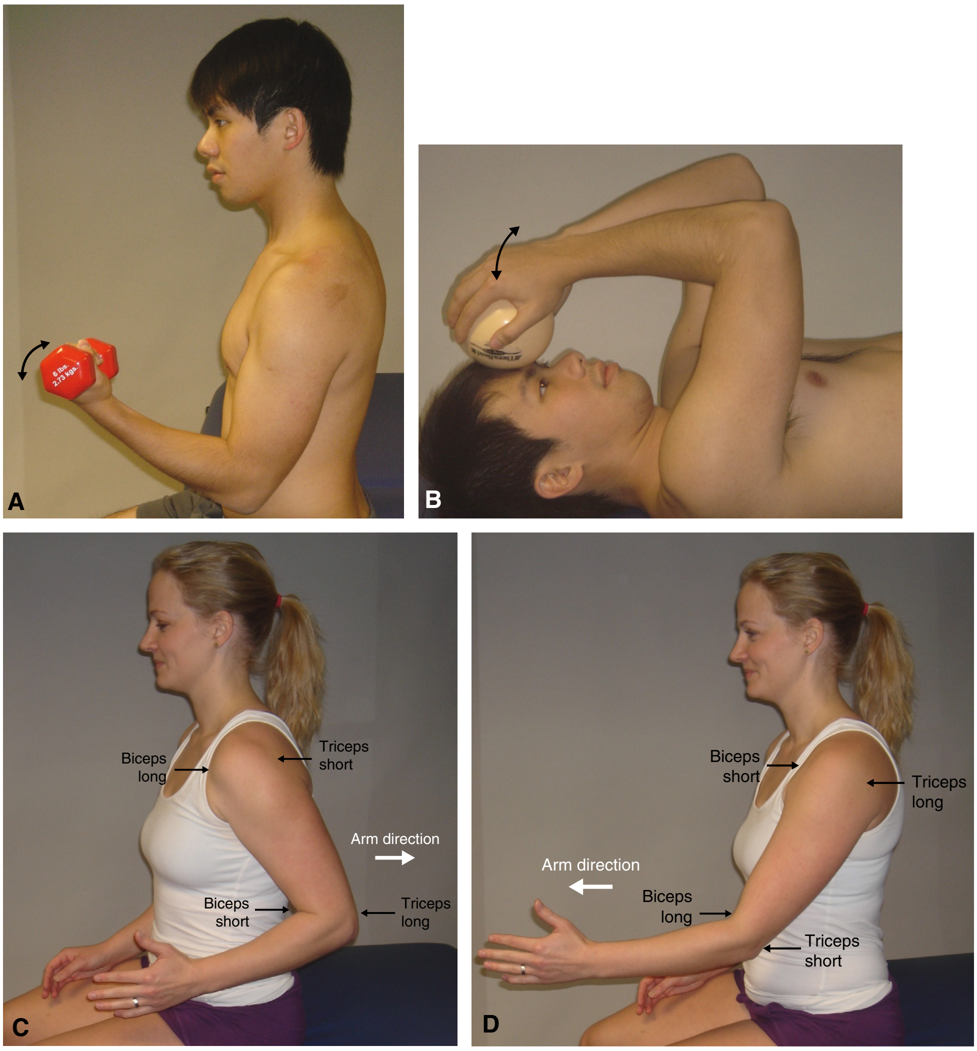

Figure 6-54 Biceps and triceps exercises. **A**, Biceps curls. **B**, Skull crushers (triceps). **C**, Econcentric biceps and triceps in shoulder extension. **D**, Econcentric biceps and triceps into forward flexion. Doing C and D together would result in a functional arm pump.

The patient must concentrate to make sure that the movements are done correctly. If necessary, each movement should be broken down into its component parts so that the patient can do each correctly. At this stage, maximum strength contractions are contraindicated. A contraction of 10% to 30% of MVC at controlled speeds is the aim to ensure correct movement patterns and to keep fatigue of the stabilizers to a minimum. Fatigue is most evidenced by muscle substitution (e.g., contraction of the upper trapezius and posterior deltoid).

Short arc exercises (in this case, in a pain-free range) into scaption, abduction, and forward flexion to 30° initially and then to 90° can be prescribed, especially in the initial treatment phase, once scapular control has been achieved. If scapular control has not been achieved, these exercises should be limited to 30°. Once the patient is able to control motion up to the horizontal plane, movement to full elevation can begin. Exercises above 90° should be performed in lateral rotation to facilitate clearance of the greater tuberosity under the coracoacromial ligament.[52] Tubing exercises, if performed properly, are effective. In most cases, when and how tubing is used should be controlled unless the patient has learned to do the tubing exercises correctly. To be performed correctly, tubing exercises should be performed only at the speed at which the patient has complete functional control of the activity. If functional control is lost, dysfunctional compensatory movement patterns result (usually seen in winging or abnormal movement of the scapula or excessive contraction of the upper trapezius or posterior deltoid).

Once control in most of the ROM has been achieved, diagonal PNF exercises may be used to teach control and stabilization throughout the ROM. The shoulder D2 pattern (flexion, abduction, and lateral rotation) is especially useful for instability. Full range should not be attempted in patients with signs of impingement or those showing apprehension.[93,134,150,151] Techniques such as slow reversals, contract-relax, rhythmic stabilization, and timing for emphasis are useful for treating instability by increasing awareness of movement, stimulating appropriate weak muscles, and strengthening weak patterns.[93,95,145] Tubing exercises using these patterns have also been found to be effective, provided they are performed with control.

Once the muscles can contract in the correct sequence, the primary shoulder accelerators or movers of the glenohumeral joint (i.e., pectoralis major, anterior deltoid, latissimus dorsi, teres major, biceps, and pectoralis minor) are trained concentrically, whereas the primary decelerators (i.e., infraspinatus, teres minor, supraspinatus, posterior deltoid, and triceps) and scapular stabilizers are trained eccentrically. Concentric and econcentric exercises of the biceps and triceps should begin as the patient progresses toward more functional movement. *Econcentric* or *pseudo-isometric* exercise is so named because commonly during function these two-joint muscles are lengthening over one joint while they shorten over the other joint. For example, when swinging the arms into extension with bent elbows during running, the biceps is lengthening over the shoulder but is shortening at the elbow as the elbow flexes (see Figure 6-54, *C* and *D*). Thus, to ensure correct movement patterns, two-joint muscles need to be trained to function econcentrically.

Step 2.3: Correct Muscle Imbalances (Aim: To Ensure Correct Force-Couple Action)

Correcting Muscle Imbalances

- Ensure joint can move through full ROM by stretching tight structures and allowing lengthened structures to tighten
- Correct any unbalanced cross syndromes
- Ensure near-equal right and left body strength and endurance
- Correct any kinetic chain movement imbalances that involve injured structures
- Proprioceptive (static) and kinesthetic (dynamic) exercises keeping in mind the different proprioceptive receptors to be stimulated and whether they function statically or dynamically

To correct muscle imbalances, any lengthened muscles must be exercised in the inner range to shorten them, and short muscles must be stretched to lengthen them. The clinician must make sure that strength and endurance are equal between the left and right sides for what the patient wants to do and that any strength and/or endurance discrepancies are corrected.[152] For example, with the humeral control muscles, a good relationship should exist between the medial and lateral rotators, between the flexors and extensors, and between the abductors and adductors, which will lead to many exercises being performed bilaterally.[152] Cross syndromes with their strong and tight muscles on one diagonal and weak and long muscles on the other diagonal must also be addressed.[144] People with anterior shoulder instability commonly demonstrate overactivity of the upper trapezius and underactivity of the serratus anterior. A standard push up "with a plus" (this implies scapula is fully protracted) has been reported to be a good exercise to accentuate the serratus anterior while minimizing the upper trapezius, thus reversing this abnormal movement pattern.[152] Isokinetic tests have shown that medial-to-lateral rotation strength ratios should be approximately 3:2, extension-to-flexion strength ratios should be 5:4, and adduction-to-abduction strength ratios should be 2:1.[113,137]

Muscle balance does not necessarily imply maximum strength of the muscles. Rather, it implies that the force couples are able to work in a coordinated fashion and with sufficient strength and endurance to do the correct motor patterns during activity. The "mover" muscles must have the strength and endurance to move an object concentrically one or more times, and the "stabilizer" muscles must have the strength and endurance to stabilize isometrically (statically) and eccentrically (during dynamic movement or as a shock absorber) in the correct movement pattern.[85,153] Eccentric control in throwing activities is more important than concentric control, and if eccentric fatigue occurs, injury is more likely.[154] Tubing may be used provided the resistance and speed of contraction are carefully controlled. The shoulder should not be overloaded, and the patient must understand that the exercises can be done only at the speed at which control and correct movement (i.e., smooth, coordinated movement) are demonstrated. If ROM is a problem, pulleys or T bar routines may be used as active-assisted exercises to help to increase the ROM and control large lever forces.[155,156]

Because synchronization of shoulder movement is important during normal activities, the patient must develop neuromuscular functional control. Therefore any treatment program must ensure good muscle balance between the agonist and antagonist and enable the patient to work at properly controlled speeds. This control eventually must be demonstrated at functional speeds. Too often, the clinician rehabilitates the patient only to have the patient return, complaining of problems when performing the activity. The problem commonly is lack of control at functional speeds.

Step 3.1: Correct Endurance Deficiencies (Aim: To Ensure Sufficient Muscle Endurance to Do Activity and Minimize the Likelihood of Injury)

The clinician must take extreme care in choosing the type of strengthening program to which the patient is exposed, especially if the rotator cuff or biceps muscles are

Correcting Endurance Discrepancies

- Use low load, high repetitions
- Patient must be able to control the pelvis (neutral pelvis), thorax (alignment), and scapula (dynamic stabilization)
- Need to build up endurance to delay fatigue with "built in protection" factor
- Ensure correct movement patterns
- Watch for compensatory postures
- Work at only speeds at which the patient can control the movement (i.e., can do the movement correctly)

involved. Because instability often has an overuse component, unsuitable exercises or too many exercises may aggravate the condition. The quality of the exercises is more important than the quantity. That being said, endurance plays a significant role in instability training because incorrect movement patterns are more likely to be seen with fatigue.

Once scapular control is achieved and the patient can control active exercises, progression to free weights (2.3 kg [5 lb] or less for up to 30 repetitions) and tubing exercises may be initiated, in the range in which the patient is able to control the movement.[157] Pappas et al.[157] found that weights heavier than 2.3 kg (5 lb) tended to precipitate loss of muscle balance in the shoulder. Initially, this movement may occur over 10° to 30° (i.e., a short arc). Once control is demonstrated by smooth, coordinated movement with minimal or no evidence of pain and discomfort, the exercises can be progressed into greater ROMs. The clinician must monitor the exercises to make sure the patient can do them correctly without fatigue, to provide a baseline for improving endurance. For example, if a patient can do four repetitions correctly, this is the starting point for the exercises. The aim is to increase the number of repetitions to a level that meets the patient's eventual functional needs plus a built-in protection (BIP) factor. If a patient needs to do 30 repetitions in a 15-minute period, the aim is to get the patient to be able to do the 30 repetitions in 15 minutes with a 10% to 15% cushion (*the BIP factor*) (i.e., 33 to 35 repetitions), so that there is less chance of injury due to fatigue. It is essential that the patient demonstrate control in terms of scapular stabilization, movement pattern, strength, endurance, and movement direction when progressing into greater ROMs. Exercises such as controlled tubing routines and wall dribbles (Figure 6-55) can enhance endurance.[149]

Step 3.2: Correct Strength Deficiencies (Aim: To Ensure Sufficient Muscle Strength to Do Activity and Minimize the Likelihood of Injury or Reinjury)

To improve strength, the patient initially should use low repetitions with high-load contractions. Active and resisted exercises may include progressive resisted exercises with minimal weight at the beginning, working up to heavier weights or using various commercial exercise machines, keeping in mind the limitations of these devices (Table 6-3).[149,158]

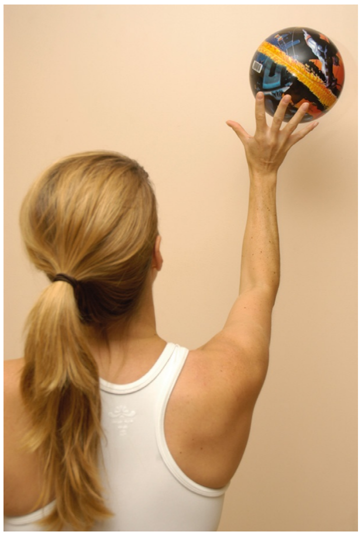

Figure 6-55 Wall dribbles used for proprioception and endurance.

Correcting Strength Discrepancies

- Start with isometric exercises moving on to concentric, eccentric, and econcentric exercises
- Use concentric exercises to ensure correct movement patterns
- Watch for compensatory postures
- Use eccentric breaks to retrain muscle shock absorption and braking action
- Use econcentric exercises to strengthen and increase endurance of two-joint muscles

Once the scapular stabilizers are functioning correctly, the glenohumeral stabilizers may be strengthened. Common exercises for strengthening the glenohumeral stabilizers include those involving the supraspinatus and deltoid ("open can" exercises or prone horizontal abduction at 100° with full lateral rotation),[136] subscapularis (medial rotation, side lying, eccentric breaks), infraspinatus and teres minor (side lying lateral rotation at 0° to 45° abduction, eccentric breaks) (Figure 6-56), biceps (flexion), and triceps (extension).[159,160] Strengthening of the scapular control muscles, along with the supraspinatus, infraspinatus, teres minor, biceps, and all three parts of the deltoid should be stressed.[143,145,146]

Eccentric exercises for the scapular and humeral control muscles help improve their strength and endurance for decelerating the arm and for shock absorption.[30,162] Closed-chain shoulder activities should be encouraged when the patient has attained suitable strength and control. Open-chain activities may begin

TABLE 6-3

Correcting Strength Deficiencies

Muscle	Exercise
Supraspinatus	Side lying, 45° abduction[159] Full can at 90° elevation in scaption[126]
Subscapularis	Side lying, 45° abduction[159] Lift off (Gerber push)[126]
Infraspinatus	Side lying, 0° to 45° lateral rotation[159] Sitting, lateral rotation of the arm from –45° to 0° (multiple angles)[126]
Teres minor	Side lying, 0° to 45° and lateral rotation
Biceps	Yergason's drill (Yergason's test used as an exercise) Speed's drill (Speed's test used as an exercise) Arm pumping
Upper trapezius	Military press[159]
Middle trapezius	T exercise: Scapular retraction with the arm at 90° abduction in the prone position
Lower trapezius	Y exercise: Scapular retraction with the arm abducted to 120° in the prone position, Spiderman exercise
Serratus anterior	Wall walks, dynamic hug, V-plank
Deltoid	Side lying, 45° abduction[159]
Rhomboids	With shoulder abduction at 90°, the patient resists forward flexion of the arm[126] (also exercises the posterior deltoid)

Figure 6-56 Lateral rotation eccentric break. Patient laterally rotates the arm and clinician eccentrically "breaks" the contraction into medial rotation.

early with high repetitions using low weights, as long as the patient can control the movement. High-risk activities (e.g., dead lifts, military presses, hands behind head "lats" for latissimus dorsi and shrugs with weights in the hands) should be avoided if scapular protraction occurs because they can cause harmful sheer forces and lead to lack of control.[150] Similarly, daily activities such as sleeping in a supine position with the hand under the pillow or over the head, fixing the hair, reaching into the back of a car from the front seat, leaning back on the arms while sitting for long periods, and putting on a backpack should be discouraged because of the stress they place on the anterior capsule.

In terms of strength about the shoulder, adduction should be the strongest, followed by extension, flexion, abduction, medial rotation, and lateral rotation. It is especially important to strengthen the medial humeral rotators (i.e., subscapularis, pectoralis major, and latissimus dorsi) and the scapular control muscles, especially the serratus anterior and lower and middle trapezius muscles, all of which show marked weakness when impingement or instability is demonstrated.[35]

Controlled pulleys or tubing may be used in the pain-free ROM for abduction, scaption (thumbs up), and forward flexion with the palm supinated. Supinating the palm causes lateral rotation of the humerus and moves the shoulder out of the impingement position. Such movement in standing may provide gravity assistance to humeral depression.[162] Strengthening exercises should progress from isometric pain-free activity to isotonic pain-free activity.

As the patient progresses through the strengthening regime, special attention must be paid to two-joint muscles because these are the muscles most commonly injured during activity. Functionally, these muscles are moving two joints at the same time and should be exercised this way (i.e., econcentrically).

Step 4.1: Retrain Proprioception and Kinesthesia (Aim: To Ensure Voluntary Control and Awareness of Movement)

Retraining Proprioception and Kinesthesia

- Remember that proprioceptive receptors are stimulated only with specific actions (e.g., muscle spindle–muscle stretch; Golgi tendon organs–muscle contraction; Golgi ligament endings–end range stretch)
- Start with slow, precise movements
- Stop when fatigue evident
- Ensure correct movement patterns
- Watch for incorrect compensatory postures
- Break complex movement patterns down into simple components

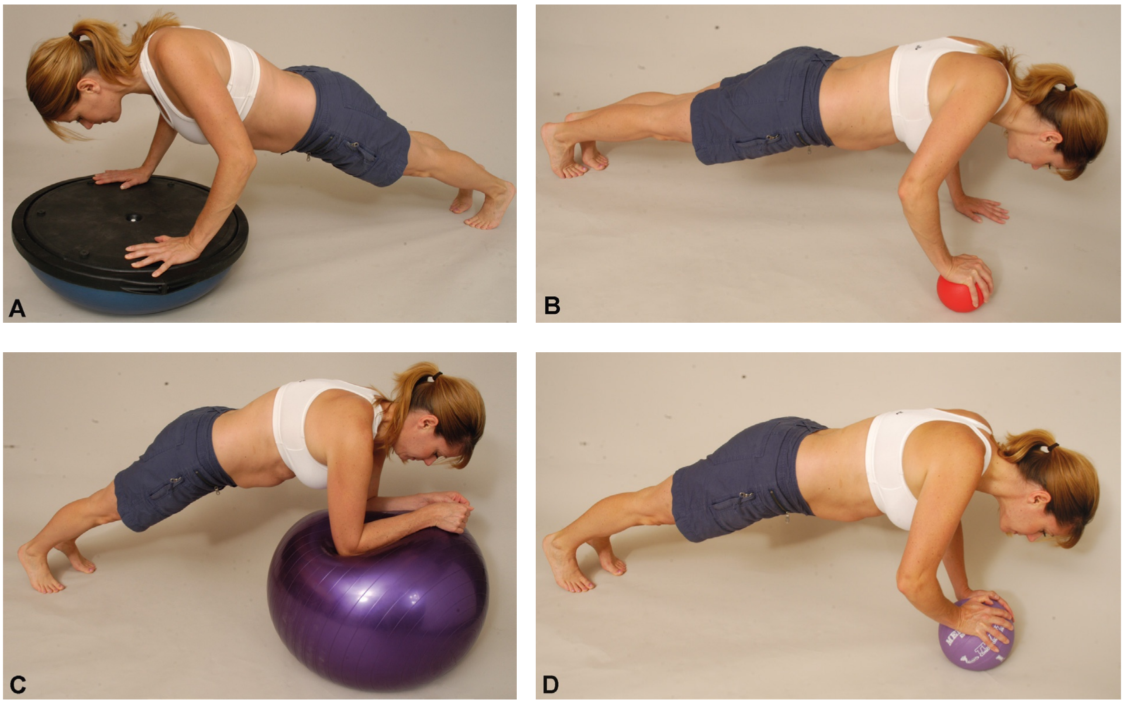

Figure 6-57 Push up on **(A)** a Bosu Ball®, **(B)** small ball using one hand, **(C)** big ball, and **(D)** small ball using two hands for proprioception.

In reality, any exercise can be used for proprioceptive or kinesthetic training because they all have a proprioceptive or kinesthetic effect. However, to specifically target individual proprioceptive receptors, exercises have to be individualized. As soon as the patient has gained scapular control, proprioceptive or kinesthetic training can be instituted to restore proprioceptive deficits.[163] Proprioceptive and kinesthetic training involves gaining voluntary control of different movement patterns, increasing the patient's awareness of correct movement patterns, and establishing automatic control of movements. Proprioceptive and kinesthetic exercises may involve closed kinetic chain weight shift, recovery phase closed kinetic chain exercises, and concentric as well as eccentric motion, movement in different parts of the ROM, and activities at different speeds.

In closed-chain activities, the distal segment is fixed or the patient holds on to an immovable object so that fixation feedback is possible, thus providing better proprioception, co-contraction, and stabilization.[164] Examples include arm wall slides into abduction, flexion diagonal patterns, and "wax on, wax off" routines (i.e., wall washes).[86,87] Dips and many proprioceptive control activities (e.g., balance board, Bosu Ball®, or using different size balls) are considered closed-chain activities (Figure 6-57). Overuse injuries are less likely to occur during closed-chain activities. Closed-chain activities for the shoulder should be primarily eccentric to reduce the effects of gravity and inertia. These activities tend to compress the joint, increasing stability, and may be used in the reverse origin insertion mode. For example, although the latissimus dorsi is viewed as an extensor and medial rotator of the shoulder, it may also be used as a trunk extensor. The clinician should look at each exercise to determine which proprioceptive or kinesthetic receptors he or she wants to stimulate (e.g., different parts of the ROM, different speeds, acceleration or deceleration) and design the exercises to meet that goal.

As the patient progresses and the scapula can be stabilized, closed-chain proprioceptive or kinesthetic activities can be performed. These can include push ups (progressing from rhythmic stabilization) and co-contraction at the joint; wall push ups progressing to wall falls (see Figure 6-40), then table push ups progressing to floor push ups (with or without a ball) (Figure 6-57); weight shifting from the push up position, shifting from a quadruped to a tripod position; ball rolling; target drills; wall washes; balancing on balls; using a Boing® or Body Blade®, dumbbell and ball drops (see Figure 6-41) to cause sudden alterations in joint position; sitting push ups; Profitter® exercises using the hands; use of an UBE (see Figure 6-42) or similar cycling device for the upper limb; hand balancing or using a balance board; hand stair climbing; and hand treadmill walking.* All of these activities are excellent for improving strength, endurance, and proprioception, but the clinician must always watch for faulty compensatory postures, pain, discomfort, and

*Refs. 30, 85, 87, 138, 158, 160, 161, 165, 166.

fatigue resulting in loss of scapular and/or humeral control, leading to compensatory patterns.

Open kinetic chain activities may be used, but they are not as effective, and they do not provide the same stability. However, they are often more functional. They must also be done with good posture. Shoulder dumps and punch outs with weight (e.g., 3 sets of 20) are some open kinetic chain activities that simulate a shoulder function.[85]

Step 5.1: Reeducate the Stability Muscles Statically (Aim: To Ensure Isometric Positioning for Form and Skill Execution)

Stage 5, consisting of five parts, is where true integrated stabilization training begins. The previous parts are individual parts of a stabilization training program that must be addressed before functional integrated stabilization training can begin.

Reeducating the Muscle Stabilizers Statically

- To begin, clinician must ensure the patient has control of the pelvis, thorax, and scapula
- Clinician places the patient in the correct position and asks the patient to hold the contraction isometrically (statically)
- Basic movements can be broken into segments and isometric hold exercises performed in each segment to establish isometric control in different positions
- Initially the contraction is submaximal
- Resistance to holding the correct position is started close to the joint (proximal) and moved distally as the patient demonstrates isometric stabilization of the joint
- Clinician watches for correct force-couple activation
- Co-activation or co-contraction used to ensure arthrokinematic control of the joint–*resistance is given at the joint*
- Closed kinetic chain activities are used to assist joint stabilization (aids compression at the joint and stress distribution)
- Positions close to the close-packed position provide more stability
- Clinician must stop exercise if fatigue or compensatory postures are evident

Once the patient has gained control of the shoulder segments (e.g., the scapulothoracic and glenohumeral joints) through isolated contraction of muscles, ensuring proper muscle function and recruitment in isolation, true integrated stabilization training can be initiated. First, the clinician must ensure that the stabilizer muscles function statically. Part of this training will have been accomplished during training of the individual stabilizer muscles. This stage also involves maintaining a stable scapula, along with correct posture (i.e., stable pelvis, or core), while doing controlled movements of the arm below 30° abduction initially (above 30°, the scapula will rotate) and then progressing to full ROM. To reeducate the stabilization muscles statically, the clinician must be concerned primarily with positioning the arm in different positions. The exercises must be performed isometrically in specific positions with control. The focus of muscle reeducation moves from proximal (i.e., ensuring a stable scapula proximally with correct posture) to distal (while allowing distal movement). Static force-couple action ensures that the muscles work in a coordinated fashion to produce a smooth, coordinated isometric hold both when the upper extremity is loaded (in close pack, or closed kinetic chain) and unloaded (in open pack, or open kinetic chain). Rhythmic stabilization and closed kinetic chain isometric scapular stabilization on a balance board or ball are examples of static stabilization exercises.

Step 5.2: Advanced Static Stabilization Exercises (Aim: Isometric Positioning for Activity-Specific Skills)

Teaching Advanced Static Stabilization Exercises

- Clinician places the patient in the desired position and asks the patient to hold the contraction isometrically (statically) (e.g., start position, follow-through position, mechanism of injury)
- Movements can be broken into segments and isometric hold exercises performed in each segment to establish isometric control in different positions
- Resistance to holding the correct position is given distally to gain static osteokinematic control while maintaining arthrokinematic control at the joint
- Clinician watches for correct force-couple activation
- Move from closed kinetic chain to open kinetic chain activities
- Stronger contractions used to stimulate isometric activation of whole kinetic chain
- Clinician must stop exercise if fatigue or compensatory postures are evident

In advanced static stabilization exercises, the patient works to stabilize the scapula statically in positions above 30°, most commonly in open kinetic chain.[85] The patient may be taken into the position of the mechanism of injury to do rhythmic stabilization exercises or taken into positions related to function, working from submaximal to maximal isometric effort to ensure control of different positions. The clinician can have the patient do isometric hold or eccentric break activities of the medial and lateral rotators in various positions of glenohumeral abduction, scaption, and forward flexion. Rhythmic stabilization with compression with the glenoid under the humeral head in the Hawkins position (see Figure 6-22), "throwing the arm," resisted isometric PNF D1 and D2 patterns, and rhythmic stabilization of the medial and lateral rotators in various postures from low risk to high risk should be attempted. To accomplish this, the patient's arm is taken into the desired position and the patient is asked to hold the position while the clinician applies perturbations to the arm and affected joint. For example, the clinician can place the patient's shoulder in the position close to the mechanism of injury and then ask the patient to statically stabilize the shoulder while doing rhythmic stabilizations. Wall falls, table falls, and floor falls from the knees are progressions, and balancing on different balance devices are closed kinetic chain examples of this.

Step 5.3: Dynamic Stabilization Exercises (Aim: Dynamic Form and Execution with Voluntary Control)

Re-educating the Muscle Stabilizers Dynamically

- Training correct (specific) movement patterns with voluntary concentric and eccentric control
- Need to ensure proximal stability with distal mobility involving the whole kinetic chain
- Want to establish voluntary control of functional movement patterns
- Clinician watches for correct force-couple activation
- Resistance movement is given distally to gain dynamic osteokinematic control while maintaining dynamic arthrokinematic control
- Clinician watches for correct force-couple activation
- Open kinetic chain activities
- Clinician must stop exercise if fatigue or compensatory postures are evident

Dynamic stabilization exercises involve movement of the joints of the shoulder girdle performed slowly and in control above 30° abduction. This involves controlled eccentric movement of the scapula while the arm moves. When the clinician teaches dynamic stabilization exercises, the focus is on correct movement patterns. The clinician must make sure that the patient has voluntary control and that agonist and antagonist activities are performed involving both concentric and eccentric exercises. Exercises such as bent-over rowing, "lawn mower" pulls using tubing, dumbbell or tubing punch outs, and lunges with dumbbell or tubing reaches are examples of open kinetic chain dynamic stabilization exercises (Figure 6-58).[87,153]

Step 5.4: Advanced Dynamic Stabilization (Aim: Dynamic Form and Execution of Activity-Specific Skills)

Advanced dynamic stabilization involves faster movements and the whole kinetic chain, including multidimensional activities. These exercises should stress functional diagonal patterns that the patient will use when he or she returns to previous activities. They may include exercises against gravity, exercises in water, the use of free weights or tubing, isokinetic exercises, PNF

Teaching Advanced Dynamic Stabilization Exercises

- Training control in activity-specific movement (diagonal) patterns
- Multidirectional stability activities
- Progressive eccentric exercises at functional speeds
- Need to ensure proximal stability with distal mobility involving the whole kinetic chain
- Clinician watches for correct force-couple activation
- Resistance movement is given distally to gain dynamic osteokinematic control while maintaining dynamic arthrokinematic control in diagonal patterns
- Clinician watches for correct force-couple activation
- Open kinetic chain activities
- Clinician must stop exercise if fatigue or compensatory postures are evident

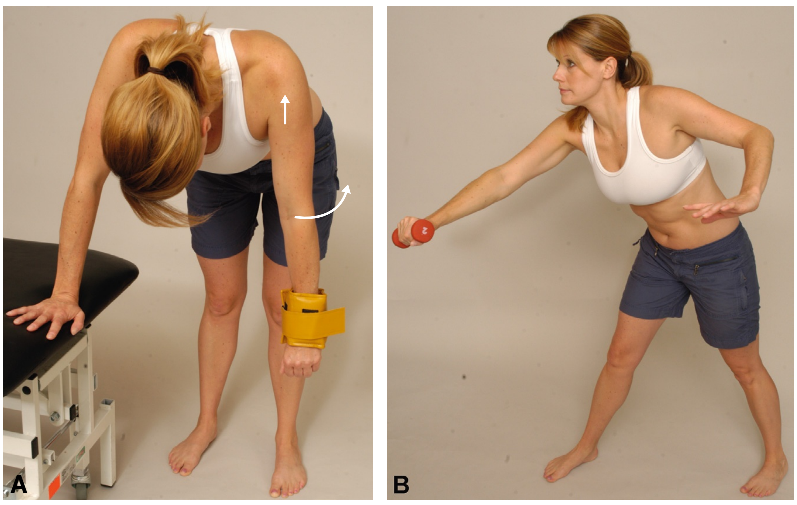

Figure 6-58 Examples of open kinetic chain dynamic stabilization exercises. **A,** Bent-over rowing. **B,** Lunges with dumbbell reaches.

patterns, and medicine ball and plyometric activities. Clock drills and reach-and-stretch (target) drills are examples of these exercises. Oblique or torsional movement, along with plyometrics and eccentric deceleration or braking, plays a major role.

The clinician should keep in mind the rules of specificity and the specific requirements of the patient in the rehabilitation program. Open-chain activity is a concentric movement against gravity with no fixation feedback. Injury to the shoulder from overuse is most likely to occur during open-chain activities and is often the result of loss of functional control of the scapula, the humerus, or both. Consequently, the clinician must make sure the patient has good scapular and humeral control before progressing to open kinetic chain exercises.[164] Open-chain activities include free weight exercises, which often are combined with proprioceptive activities (i.e., eyes open or closed, positioning or mirroring) to teach "free hand" feedback.

Step 5.5: Functional Stabilization (Aim: Functional Form and Skill Execution)

In the final stage of the stabilization program, the clinician teaches the patient functional stabilization, in which functional activities are broken down into their component parts so that the patient can develop particular motor skills that he or she will need in everyday life.[98,167,168] Patients are asked to perform specific, synchronized, multidirectional skilled pattern sequences (Figure 6-59). Eccentric patterns and power development are emphasized to develop the braking action and shock absorption of the muscles to protect the glenohumeral joint and the ability to control movement at functional speeds. All activities should involve most if not all of the whole kinetic chain.

Teaching Functional Stabilization

- Training synchronized skilled movement sequences and activity-specific skills
- Clinician must watch for ***patient carriage*** (e.g., weight shift, weight acceptance, movement symmetry, appropriate limb braking), ***control*** (e.g., smooth, automatic, and unrestricted execution and movement with proper form and skill), and ***confidence***
- Clinician watches for correct force-couple activation
- Clinician must stop exercise if fatigue, apprehension, or compensatory postures occur
- Teach injury or reinjury prevention strategies

Open-chain activities using devices, such as the Body Blade® and Boing® activities, plyometrics, and the use of a medicine ball are helpful in later stages of rehabilitation to teach functional stabilization and control.

Plyometric programs for the upper limb (Figure 6-60) are designed to increase the excitability of the neurological receptors and improve the reaction of the neuromuscular system.[85,126] The patient must be taught to accept greater loads (the clinician must keep in mind that with plyometrics, the rate of stretch rather than the length of stretch

Criteria for Return to Activity[85,167,168]

- Little or no pain
- No or minimal apprehension
- Functional ROM with involvement of the whole kinetic chain
- Near-normal strength (80% to 90%)
- Normal movement patterns
- Normal functional ability
- Able to perform the skills the patient wants to resume
- Appropriate level of fitness
- Appropriate patient expectations

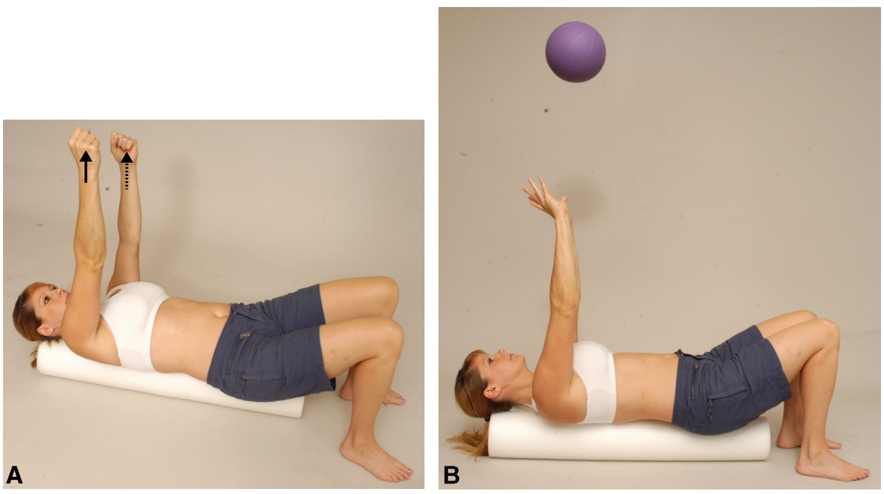

Figure 6-59 Multidirectional instability exercises. **A**, Punch out on roll. **B**, Ball toss on a roll.

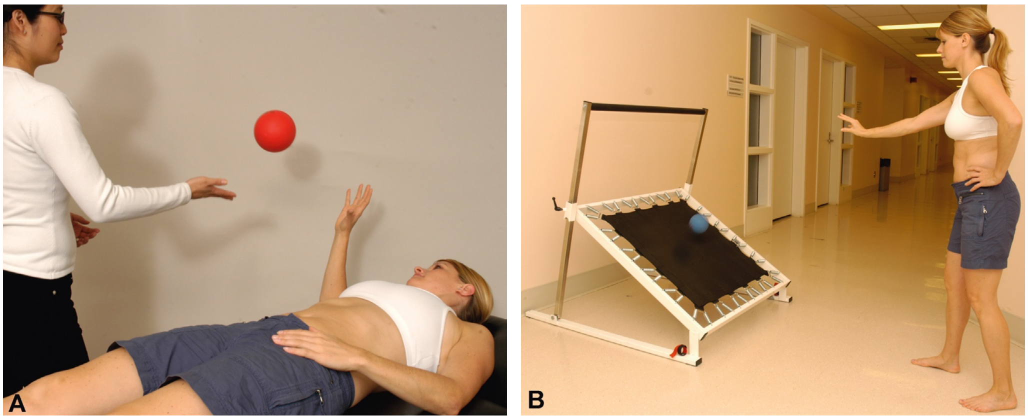

Figure 6-60 Plyometric exercises. **A**, Ball throw. **B**, Plyoback exercises using a weighted ball.

is important).[169,170] Activities using a 0.9- to 1.8-kg (2 to 4 lb) ball (e.g., medicine ball or Plyoball), a bounce-back device (Plyoback™) or partner, or tubing (progressing from slow to fast sets) can be effective.

Step 6.1: Fitness Aim: To Restore or Maintain Fitness throughout Program

While the patient's shoulder is being rehabilitated, the clinician must keep in mind that the patient at some point will be returning to activity. A program to maintain or restore cardiovascular fitness should be instituted and should be as specific as possible to the patient's occupation or activity. Little good is achieved if the shoulder is rehabilitated and the patient is cleared medically to participate in activities or return to work, if patients do not have the cardiovascular fitness or activity-specific fitness to compete or to do their job. This process is sometimes referred to as *work hardening*.

To keep the patients involved and to enhance their recovery, it is imperative to have them work to maintain their fitness level if they have been active or to work to improve their fitness level if they have not been active. In both cases, fitness training will help their recovery physiologically and psychologically. Activity may be as simple as asking them to "go for a walk around the block" for someone who has done little activity to someone doing a vigorous workout on a stationary bike or elliptical apparatus. This stage can, in reality, start presurgery or postsurgery using lower limb activities that will not stress the patient's shoulder. Most commonly, patients are started with activity at about 60% of their maximum heart rate working up over time to 75% to 85%, but this will depend on their level of fitness to begin with and the activity they want to return to. There are several ways patients' target heart rate can be calculated, but probably the easiest is $(220 - \text{age}) \times$ percentage of maximum heart rate. This will give a target heart rate for training. If the activity is going to take place in water (e.g., running in water), then the target heart rate should be 10 to 13 beats less than the target heart rate calculated above because of the increased stress placed on the heart due to water resistance.

Fitness training may be modified based on what the clinician is trying to accomplish and based on the activity the individual wants to return to. For example, initially the desire may simply be to establish a good aerobic base, which will enhance the patient's recovery from exercise. Depending on the activity the individual wants to return to, the clinician may initiate different exercise regimes which may tax the alactic anaerobic system (ATP-PC system), lactic anaerobic system (glycolysis), and/or the aerobic system, each of which have their own special characteristics.

SUMMARY

Rehabilitation of the unstable shoulder can be both a challenging and rewarding experience. Clinicians must always be aware of possible pitfalls and special concerns in the treatment of the unstable shoulder. The process of treating an unstable shoulder takes time, and the patient may require anywhere from 6 months to 2 years to fully rehabilitate and regain the preinjury or preoperative status. The clinician may devise a sterling treatment plan, but ultimately the patient's dedication to and involvement in the rehabilitation program are the main predicators of a successful outcome. Therefore the patient must have enough information to "buy into" the program, understand what to expect as the program progresses, and things to watch for that may indicate he or she is pushing too hard or not hard enough, as well as having realistic expectations as to the outcomes.

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CHAPTER 7

Rotator Cuff Pathology

JUDY C. CHEPEHA, MARTIN J. BOULIANE, DAVID M. SHEPS

INTRODUCTION

Shoulder pain is well established as one of the most common musculoskeletal complaints managed by physicians and physical therapists.[1–3] Up to half the population experiences at least one episode of shoulder pain per year,[3] and nearly 50% of patients have some type of rotator cuff disease.[1] Conditions affecting the rotator cuff include tendinopathy of one or more of the muscles of the rotator cuff, partial-thickness tears, and full-thickness tears.[4] Rotator cuff injuries affect individuals across the life span—they are common in young, overhead athletes, as well as in individuals 65 years and older.[5–7] The prevalence of rotator cuff tendinopathy is reported at approximately 30% of all shoulder-related disorders.[8] Studies specific to rotator cuff tears have reported incidence rates between 13% and 32%, with a significant increase in occurrence as an individual ages.[9,10] Kibler et al.[11] suggested that more than 50% of individuals older than 60 years of age have at least a partial-thickness tear of their rotator cuff and that this estimate may be low because a large number of individuals are asymptomatic.[12,13] Rotator cuff disease tends to progress over time, becoming more painful and/or involving more muscles.[14] Because of the current aging population, and that many older individuals are maintaining an active lifestyle, the incidence of symptomatic rotator cuff pathology is expected to grow.

Rotator cuff disease manifests itself clinically in a variety of ways; however, most patients complain of pain worsened with overhead activities, and exhibit strength deficits and functional impairment(s).[9,15,16] These symptoms can cause significant disruption of an individual's physical functioning within activities of daily life (ADL), work, and recreational activities, as well as negatively affect the individual's mental health and social participation.[17] The effect that rotator cuff pathology has on a patient's quality of life and resulting impairment has been compared with that of congestive heart failure, diabetes, myocardial infarction, and depression.[18]

Physical therapy, consisting of cryotherapy, therapeutic exercise, manual therapy, and electrical modalities, is the usual first course of treatment for rotator cuff-related pathology. Surgical intervention is generally considered only after a failed course of appropriate, conservative treatment lasting at least 3 months.

Clinical Note

Rotator cuff surgery should only be considered after at least 3 months of appropriate conservative treatment.

ROTATOR CUFF ANATOMY

Successful treatment of the patient with rotator cuff disease begins with a thorough understanding of the anatomy of the rotator cuff and its adjacent structures. Advances in basic science and surgical technology over the past decade have improved knowledge regarding the osseous and soft-tissue structures of the shoulder and have led to a better understanding of the role these tissues play in normal and abnormal shoulder function.

Rotator Cuff Muscles

- Supraspinatus
- Infraspinatus
- Subscapularis
- Teres minor

The four muscles that constitute the rotator cuff (supraspinatus, infraspinatus, teres minor, and subscapular muscles) arise from the scapula, a bone stabilized by different muscles, and converge with the articular capsule, coracohumeral ligament, and glenohumeral ligaments, to attach on the tuberosities of the humerus (Figure 7-1).[19–21] The supraspinatus muscle arises from the supraspinatus fossa on the upper one third of the posterior scapula. It passes beneath the acromion and the acromioclavicular joint and attaches to the superior aspect of the greater tuberosity. It is innervated by the suprascapular nerve after

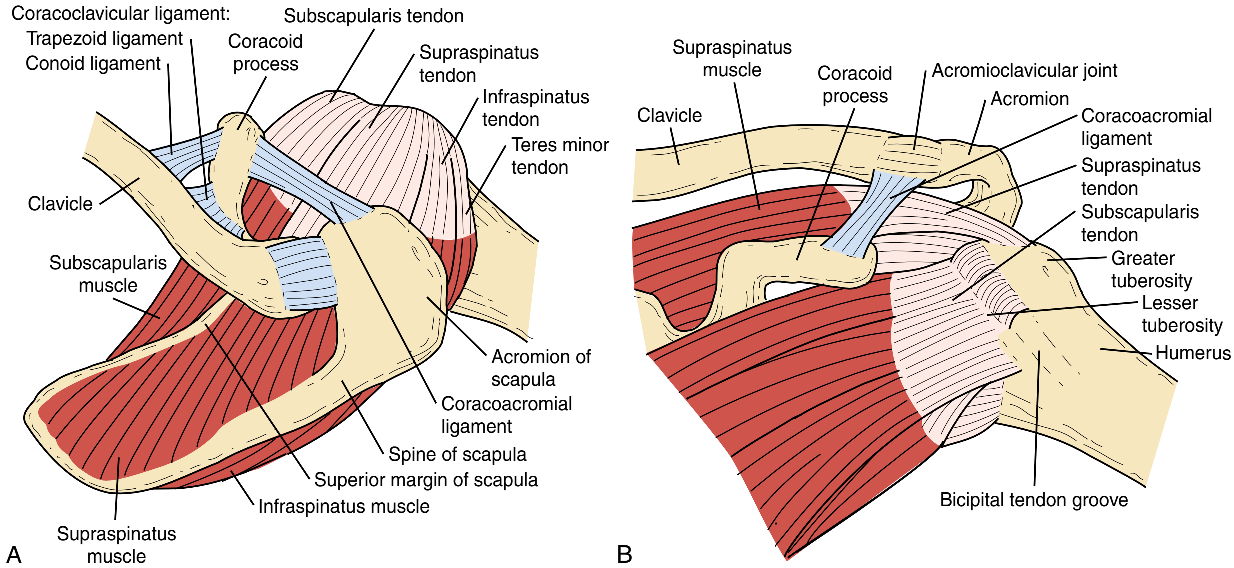

Figure 7-1 **A,** Superior view of the rotator cuff musculature as it courses anteriorly underneath the coracoacromial arch to insert on the greater tuberosity. **B,** Anterior view of the shoulder reveals the subscapularis, which is the only anterior rotator cuff muscle inserting on the lesser tuberosity. It rotates the humerus medially (internally) and provides dynamic anterior stability to the shoulder. (From Firestein GS, Budd RC, Harris ED et al: *Kelley's textbook of rheumatology*, ed 8, Philadelphia, 2008, Saunders/Elsevier.)

it passes through the suprascapular notch. The infraspinatus muscle arises from the infraspinatus fossa on the lower two thirds of the posterior scapula and attaches to the posterolateral aspect of the greater tuberosity. It is innervated by the suprascapular nerve after it passes through the spinoglenoid notch. Despite their distinct attachment sites, the tendons of supraspinatus and infraspinatus join together to form a single tendon, approximately 15 mm proximal to the insertion points on the greater tuberosity.[22] The teres minor muscle, innervated by a branch of the axillary nerve, arises from the lower lateral aspect of the scapula and attaches to the lower aspect of the greater tuberosity. The subscapularis muscle arises from the anterior aspect of the scapula and attaches over much of the lesser tuberosity. It is innervated by the upper and lower subscapular nerves. The tendons of supraspinatus and subscapularis, along with the superior glenohumeral and coracohumeral ligaments, form a sheath that encapsulates and stabilizes the biceps tendon as it enters the bicipital groove. Part of the supraspinatus tendon forms the roof, whereas the subscapularis tendon forms the sheath floor. Tearing or rupture of the anterior supraspinatus or superior subscapularis muscle can cause injury to the biceps "sling" and lead to instability of the long head of biceps.[23–25] The rotator cuff insertions form a horseshoe pattern that tapers away from the anatomic neck inferiorly. The superior insertion is tendinous, becoming more muscular inferiorly.[26] This continuous cuff around the humeral head allows the muscles to provide an infinite variety of moments to rotate the humerus and to oppose unwanted components of the deltoid and pectoralis muscle forces. Research related to the insertional anatomy of the rotator cuff tendons, known as the **rotator cuff footprint**, has helped to improve diagnosis of rotator cuff tears and assist surgeons in surgical repair.[26,27]

The long head of the biceps tendon is an important part of the rotator cuff complex; so much so, it is sometimes referred to as the fifth rotator cuff tendon (Figure 7-2).[28] It attaches to the supraglenoid tubercle of the scapula and travels between the subscapularis and supraspinatus muscles, exiting the shoulder through the bicipital groove under the transverse humeral ligament, finally attaching to its muscle in the proximal arm. Tension in the long head of the biceps tendon assists the rotator cuff in compression of the humeral head into the glenoid and guides the head of the humerus as it is elevated.[24,25,29]

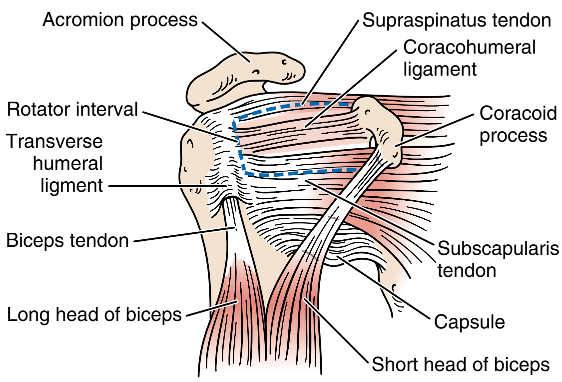

Figure 7-2 Rotator interval (*dashed lines*) showing the relationship between the supraspinatus tendon, long head of biceps, subscapularis tendon, and the coracohumeral ligament. (From Magee DJ: *Orthopedic physical assessment*, ed 6, p 255, St Louis, 2014, Saunders/Elsevier.)

The coracoacromial arch refers to the inferiorly concave surface consisting of the anterior undersurface of the acromion, the coracoid process, and the coracoacromial ligament (see Figure 5-6). It provides a strong ceiling for the shoulder joint, along which the rotator cuff tendons must glide during all shoulder movements. The coracoacromial arch has received considerable attention over the years because of its anatomic relationship to the rotator cuff tendons and the role its structures may play in causing rotator cuff injury.[30–33] Investigations[34,35] have highlighted the importance of contact and load transfer between the rotator cuff and the coracoacromial arch in the function of normal shoulders. Because there is normally no gap between the superior rotator cuff and the coracoacromial arch, the slightest amount of superior translation of the humerus compresses the rotator cuff tendons between the humeral head and the arch, resulting in impingement. Bigliani et al.[32] studied 140 shoulders in 71 cadavers with an average age of 74.4 years. They identified three acromial shapes: type I (flat) in 17%, type II (curved) in 43%, and type III (hooked) in 40%. Of the shoulders, 33% had full-thickness tears, 73% of which were seen in association with a type III acromion; 24% with type II; and 3% with type I (Figure 7-3). The clinical significance of this relatively small difference is unknown and controversial because other authors have discovered no significant association between acromial morphology and rotator cuff pathology.[36–38] A fourth type has been added to the classification as convex or upturned, but has no correlation to impingement. However, a significant correlation between age and rotator cuff tears was noted, with older patients more likely to have type II and III acromion.[36,38] These findings suggest that correlations between acromial morphology and rotator cuff tears may be exaggerated because of the confounding variable of age.

The coracoid process acts as the medial attachment site for both the coracohumeral and coracoacromial ligaments. The neighboring supraspinatus and subscapularis tendons must be able to glide past the coracoid process with their full excursion during shoulder motion. Scarring of one or both of these tendons to the coracoid process can inhibit passive and active shoulder motion. Although the coracoid process does not normally contact the anterior subscapularis tendon, forced medial (internal) rotation of the arm, particularly in the presence of a tight posterior capsule, can produce such contact because of the obligate translation, and lead to impingement of the rotator cuff tendons.[34,39]

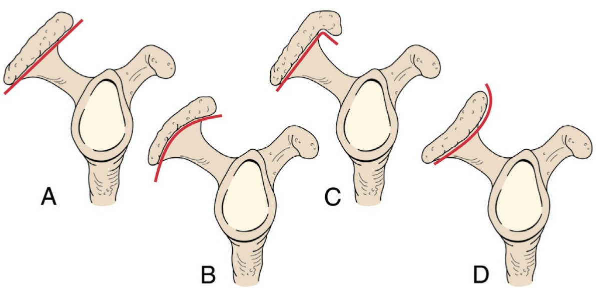

Figure 7-3 Acromion morphology. **A,** Type I (*flat*). **B,** Type II (*curved*). **C,** Type III (*hooked*). **D,** Convex (upturned). (From Magee DJ: *Orthopedic physical assessment*, ed 6, p 257, St Louis, 2014, Saunders/Elsevier.)

The coracoacromial ligament (see Figure 5-6) originates along the distal two thirds of the lateral aspect of the coracoid process as a broad ligament. It passes posteriorly to insert onto the anteromedial and anteroinferior surfaces of the acromion. Spur formation can occur preferentially on the anterolateral band and contribute to impingement syndrome, although it has been suggested that substantial alterations in morphological and biomechanical properties occur in the coracoacromial ligament during aging.[40] Studies are needed to determine whether variations in the coracoacromial ligament morphology cause impingement or are a function of the problem.

There are three bursae relevant to the development of shoulder pain and rotator cuff disease: subacromial, subdeltoid, and subcoracoid (Figure 7-4). The subacromial bursa occupies a space above the rotator cuff and under the acromion. It is a synovium-lined cavity that acts as a gliding surface in two locations: (1) between the rotator cuff tendons and the coracoacromial arch and (2) between the deltoid muscle and the rotator cuff tendon. The subdeltoid bursa is an independent structure located on the deep surface of the deltoid muscle. The subacromial and subdeltoid bursae act together and extend as far medially as the coracoid process. The subcoracoid bursa is located inferior to the coracoid process between the subscapularis tendon and the conjoined tendon of the coracobrachialis muscle and short head of the biceps muscle. Subacromial "disease" encompasses a spectrum of pathologies ranging from bursitis to adhesion formation. Rotator cuff tears are often associated with subacromial bursitis, and this bursitis can lead to the formation of adhesions, which then contribute to rotator cuff impingement. Machida et al.[41] studied 18 patients with shoulder pain and found that subacromial bursa adhesions increased impingement between the acromion process and the rotator cuff insertion.

Anatomical consideration should also be given to the proximal portion of the rotator cuff muscles on the scapula and the relationship between the rotator cuff origins and the scapulothoracic articulation. The scapula, which is stabilized by muscles, provides a stable base for the controlled movements of the humeral head in the glenoid fossa. The muscles that attach to and stabilize the scapula (i.e., trapezius, rhomboids, latissimus dorsi, serratus anterior, levator scapulae, and pectoralis minor) aid in the positioning of the glenoid fossa to accommodate the head of the humerus (Figure 7-5). Scapular dysfunction, from altered muscle balance and/or instability, is one of the contributing factors of rotator cuff injury, especially in the young, active patient population.

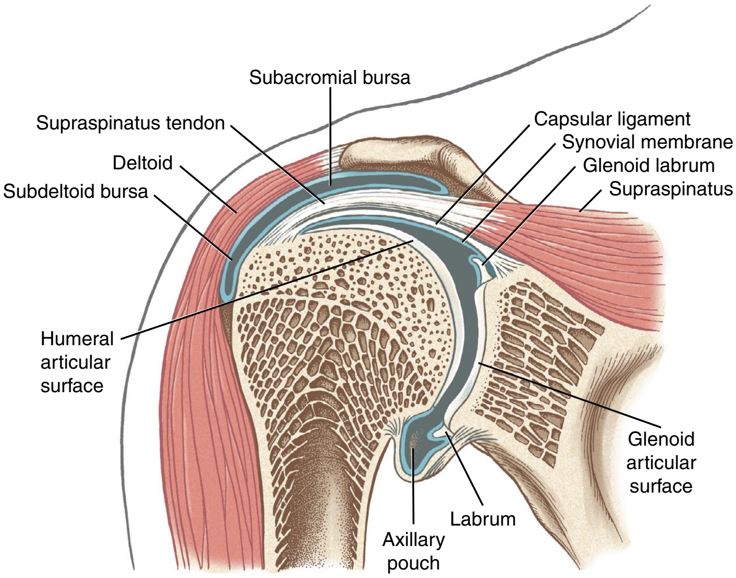

Figure 7-4 Anterior view of a frontal plane cross section of the right glenohumeral joint. Note the subacromial and subdeltoid bursa within the subacromial space. Bursa and synovial lining are depicted in blue. The deltoid and supraspinatus muscles are also shown. (From Neuman DA: *Kinesiology of the musculoskeletal system: foundations for rehabilitation*, ed 2, p 143, St. Louis, 2010, Mosby/Elsevier.)

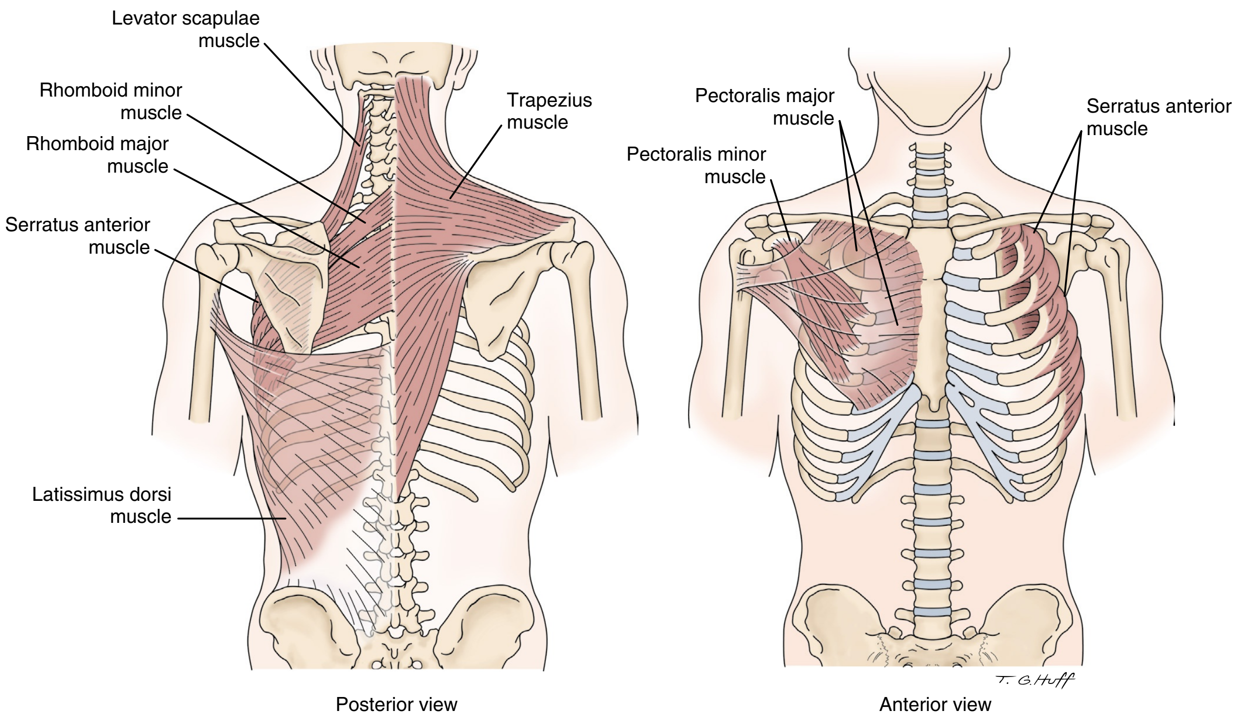

Figure 7-5 The muscles that attach to and stabilize the scapula aid in the positioning of the glenoid fossa to accommodate the head of the humerus. Note that if the elbow is braced, the rotator cuff muscles (supraspinatus, subscapularis, infraspinatus, and teres minor), teres major, deltoid, and pectoralis major can also move the scapula by "reverse origin-insertion."

Muscles Controlling the Movement of the Scapula

- Trapezius (three parts)
- Rhomboids
- Serratus anterior
- Levator scapula
- Pectoralis minor
- Latissimus dorsi (inferior angle)

ROTATOR CUFF FUNCTION

The term *rotator cuff* may be a misnomer because its role relative to shoulder function involves not only joint rotation and muscle force but also joint compression and stability. In the past, the rotator cuff has been referred to as a humeral head depressor; however, evidence now indicates that the inferiorly directed force of the rotator cuff is small compared with the humeral head compressive force it exerts to stabilize the glenohumeral joint (Figure 7-6).[42,43] The four tendons of the rotator cuff perform as a unit, precisely centering the humeral head through compression (called concavity compression) into the glenoid cavity to improve stability, resist translation, and provide rotation about the three major axes of motion.[42–44] Joint stability, through the rotator cuff's concavity-compression mechanism, is provided mostly within the shoulder's midrange positions, where passive capsuloligamentous restraints are lax; however, it has been shown to also be important at the end of shoulder range of motion (ROM), where forces acting on the joint are increased.[45] Rotator cuff compression acts to protect the capsuloligamentous structures in extreme positions by limiting the shoulder's ROM and reducing strain on these structures.[43] The humeral head maintains a relatively constant position in relation to the glenoid during shoulder rotation. The rotator cuff muscles are closer than any other muscles to the axes of rotation and therefore have shorter lever arms to affect rotation.

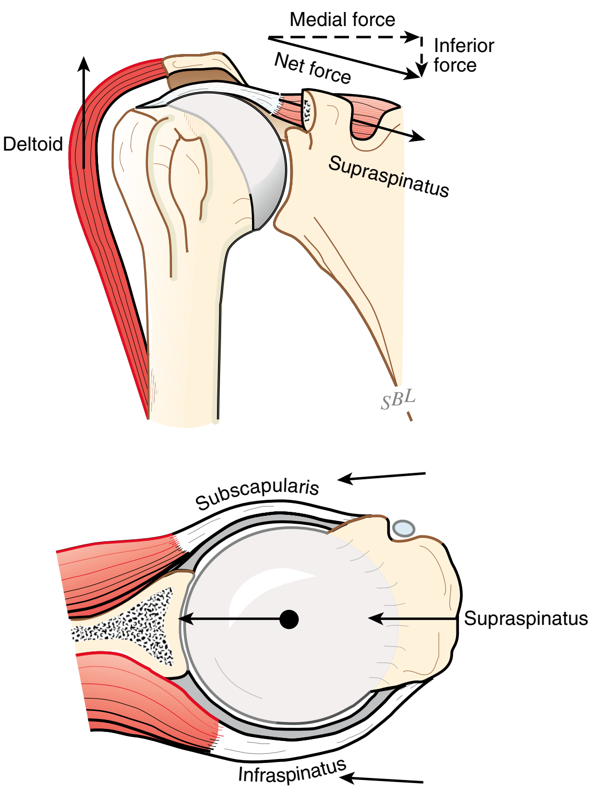

Figure 7-6 Concavity compression of the rotator cuff. (Modified from Matsen FA III, Lippitt SB: *Shoulder surgery: principles and procedures*, p. 85, Philadelphia, 2004, WB Saunders.)

Clinical Note

The rotator cuff muscles work as a unit to center the humeral head in the glenoid cavity through compression, to improve stability at the joint, resist translation at the glenohumeral interface, and provide rotation.

The amount of force that can be generated by a rotator cuff muscle is determined by its size, health or condition, and joint position. The individual rotator cuff muscles' contribution to shoulder strength has been well studied.[44–46] The supraspinatus functions mainly to aid the deltoid muscle in shoulder abduction. The dynamic interplay of supraspinatus and deltoid is well established as essential for proper glenohumeral function.[22,42,44–46] With the arm in adduction at the initiation of elevation, the pull of the deltoid muscle is nearly parallel to the glenoid surface, producing a well-documented upward shear component. The stability of the humeral head provided by supraspinatus compressing and stabilizing the humeral head inferiorly opposes the upward pull of the deltoid, avoiding impingement of the rotator cuff between the humeral head and the undersurface of the acromion. The infraspinatus muscle assists the supraspinatus in its role as a humeral head depressor, especially with the shoulder at 90° of abduction and neutral rotation. Subscapularis functions primarily as a stabilizer during lateral (external) rotation, controlling excessive anteroposterior translation as the shoulder abducts and laterally (externally) rotates. This may explain why subscapularis' contraction has been shown to be so strong in overhead athletes at the initiation of the acceleration phase of throwing. Several powerful muscles contribute to medial (internal) rotation, but tears of the subscapularis may be shown by testing function at the extreme of medial (internal) rotation, indicating the importance of this muscle at this end ROM. Patients who have sustained subscapularis tears feel the loss of joint compression and stabilization normally provided by this portion of the rotator cuff. The infraspinatus and teres minor are the only two muscles that produce lateral (external)

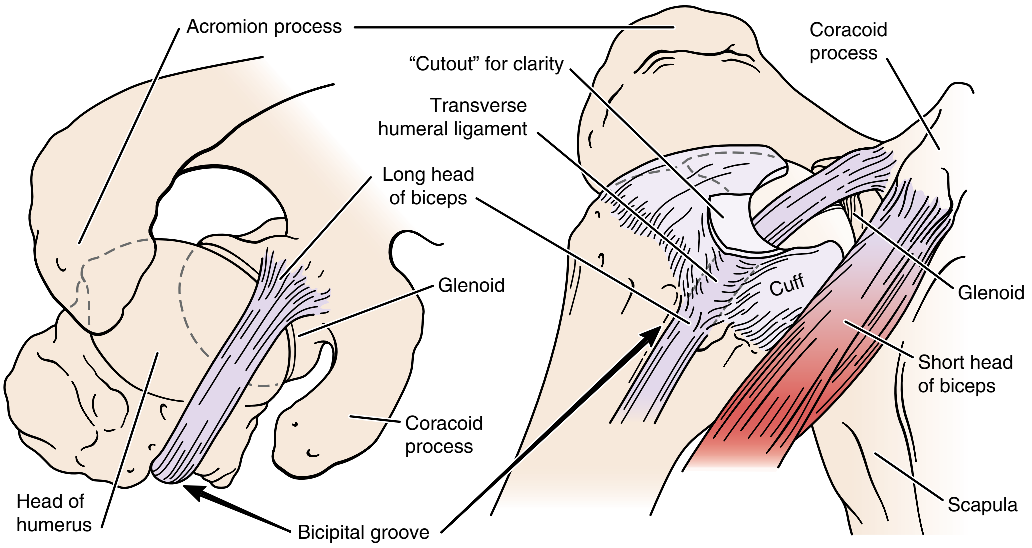

Figure 7-7 The biceps apparatus. (From Magee DJ: *Orthopedic physical assessment*, ed 6, p 254, St Louis, 2014, Saunders/Elsevier.)

rotation, and large tears in this region of the rotator cuff have profound effects on a patient's motion and strength.

The long head of biceps is often considered a functional part of the rotator cuff complex due, in part, to its intimate anatomical relationship with the rotator cuff tendons at the glenohumeral joint (Figure 7-7).[23,29] Its superior-anterior location assists the superior-posterior rotator cuff tendons in stabilizing the glenohumeral joint through humeral head compression, especially into forward elevation. The importance of the relationship between the rotator cuff and long head of biceps is evidenced through injury to one or both of these tissues. Greater than 90% of patients with a diagnosis of biceps tendinitis were found to have underlying shoulder impingement and/or instability believed to be the cause of irritation of the biceps tendon.[47] Lesions of the superior labrum, anterior to posterior (i.e., a SLAP lesion), and adjoining biceps anchor have received considerable attention over the past two decades, including work that has demonstrated a high incidence of rotator cuff tears in overhead athletes with superior labral defects (see Figure 5-27).[48–50] Rotator cuff tears were discovered in 31% of throwers with type II SLAP lesions: 38% of these were full thickness, and 62% were partial thickness. Finally, biceps were found to have an increased tendon-tension force in the presence of a large rotator cuff tear, suggesting the increase of its role in restraining superior humeral head translation to compensate for the rotator cuff failure.[25]

It is important to remember that the shoulder joint differs from single axis joints, such as the knee, in that it functions in the absence of a fixed axis. In a specified position, activation of a muscle creates a unique set of rotational moments. For example, the anterior deltoid can produce moments in forward flexion, medial (internal) rotation, and horizontal adduction. If forward elevation is to occur without rotation, the cross-body and medial (internal) rotation moments of the anterior deltoid must be neutralized by other muscles, such as the posterior deltoid and infraspinatus. As another example, use of the latissimus dorsi in a movement of pure medial (internal) rotation requires that its adduction moment be neutralized by the superior rotator cuff and deltoid. Conversely, use of the latissimus dorsi in a movement of pure adduction requires that its medial (internal) rotation moment be neutralized by the posterior rotator cuff and posterior deltoid muscles.[51]

The timing and magnitude of these balancing muscle effects must be precisely coordinated to avoid unwanted directions of humeral motion. Therefore, the simplified view of muscles functioning in isolation or as part of "force couples" must be replaced with the understanding that all shoulder girdle muscles function together in a precisely coordinated way: opposing muscles canceling out undesired elements and leaving only the net torque that is needed to produce the desired action. This degree of coordination requires a fine-tuned strategy of muscle activation or motor engram that must be established before the motion is carried out. The rotator cuff muscles are critical elements of this shoulder balance equation.

Functions of the Rotator Cuff

- Rotation of the humerus relative to the scapula
- Compression of the humeral head into the glenoid fossa, providing a critical stabilizing mechanism known as concavity compression
- Provision of muscle balance and force

ETIOLOGY

Injury to the rotator cuff occurs via several different mechanisms. Often, more than one mechanism of injury and multiple contributing factors occur simultaneously in the same patient. Injuries that are traumatic tend to be acute, with a well-defined mechanism, and occur in individuals younger than 40 years of age. Concurrent clinical findings such as glenohumeral and/or scapulothoracic instability, secondary impingement, and muscle imbalances are common in this population. More frequently, rotator cuff injuries occur atraumatically as a result of multiple, repetitive, overuse mechanisms, in individuals over the age of 40 years of age. Clinical findings in this population often include postural alterations of the shoulder girdle and/or adjacent spine, subacromial impingement, and rotator cuff degeneration.

Although the exact cause of rotator cuff disease is unknown, it is generally accepted that injury occurs because of multiple intrinsic and extrinsic factors.[52–54] Intrinsic factors include traumatic, reactive, or degenerative changes that originate within the tendon from inferior tissue mechanical properties, direct tendon overload, intrinsic degeneration, or other insult. Extrinsic mechanisms are those that damage the tendon through external forces such as compression against surrounding tissue.[55]

The proposed causes of intrinsic degeneration are primarily the vascular supply of the rotator cuff tendon, aging, and tensile overload. Several researchers have suggested that hypovascularity within the area of greatest impingement (i.e., Codman's critical zone) is the main reason why rotator cuff tears occur in this region (Figure 7-8).[56,57]

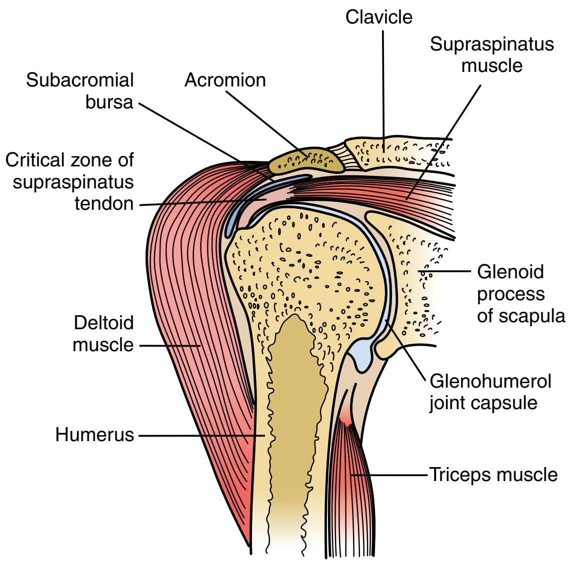

Figure 7-8 The "critical zone." Note the close relationship of the supraspinatus tendon and subacromial bursa to the humeral head and acromion, making an exact clinical diagnosis very difficult. (From Foley BA, Christopher TQ: Injection therapy of bursitis and tendinitis. In Roberts JR, Hedges JR, editors: *Clinical procedures in emergency medicine*, ed 5, Philadelphia, 2010, Saunders/Elsevier.)

Evidence has emerged that both supports and refutes this claim.[58–61] Cadaveric studies confirmed this hypovascular area within the supraspinatus tendon's critical zone, whereas microvasculature studies of the rotator cuff revealed the critical zone as being hypervascular in patients with impingement syndrome.

The effect that age has on rotator cuff tendons is much less controversial. Aging is believed to have a negative effect on the strength and resiliency of the rotator cuff, as evidenced through histological studies that reveal calcification, fibrovascular proliferation, and microtears, suggestive of degenerative changes, in individuals greater than 40 years old.[40,53,60] These same changes are rarely noted in the nonathletic population of those under 40 years. Moreover, it is generally accepted that a correlation exists between a patient's age and the healing abilities of a tendon, where the healing property of tendons of elderly individuals is diminished, as compared with that of younger tendons.[61]

Tension overload is another intrinsic factor found to contribute to rotator cuff injury. Studies have attempted to distinguish specific mechanisms that may contribute to tension overload injuries. One study reported that if the joint and bursal sides of the supraspinatus tendon are subjected to similar loads, the joint side will be more susceptible to failure.[62] Methods to measure intratendinous strain using magnetic resonance imaging have discovered that intratendinous strain fields increased with increasing glenohumeral joint angle, but the strain did not change between the articular and bursal sides.[63] These findings suggest that individuals, such as overhead throwing athletes or those working overhead, may be at risk of articular-sided rotator cuff tears from tendon overload because the joint side of the tendon may be closer to its failure strain during these activities.

Extrinsic factors that contribute to rotator cuff pathology include anatomical findings such as narrowing within the coracoacromial arch or other osseous or soft-tissue changes that result in anterior, external mechanical impingement of the rotator cuff tendons (Figure 7-9). Numerous studies have confirmed that abnormal soft-tissue and bony anatomy surrounding the rotator cuff affect the incidence of pathology; however, the degree to which anatomical variations *cause* rotator cuff injury is less clear.[30,33,34,39] Osteoarthritis of the acromioclavicular joint as well as changes to the coracoacromial ligament have also been implicated as extrinsic factors that could cause anterior impingement and mechanical irritation of the rotator cuff tendons.[38] Internal impingement can occur posteriorly, between the posterior-superior labrum and the posterior rotator cuff tendons (Figure 7-10).[5,50] This is a common problem in overhead throwing athletes because of the tremendous amount of stress placed on the rotator cuff as the athlete accelerates and decelerates the arm during the throwing motion. The rotator cuff muscles must counteract these forces, especially during the

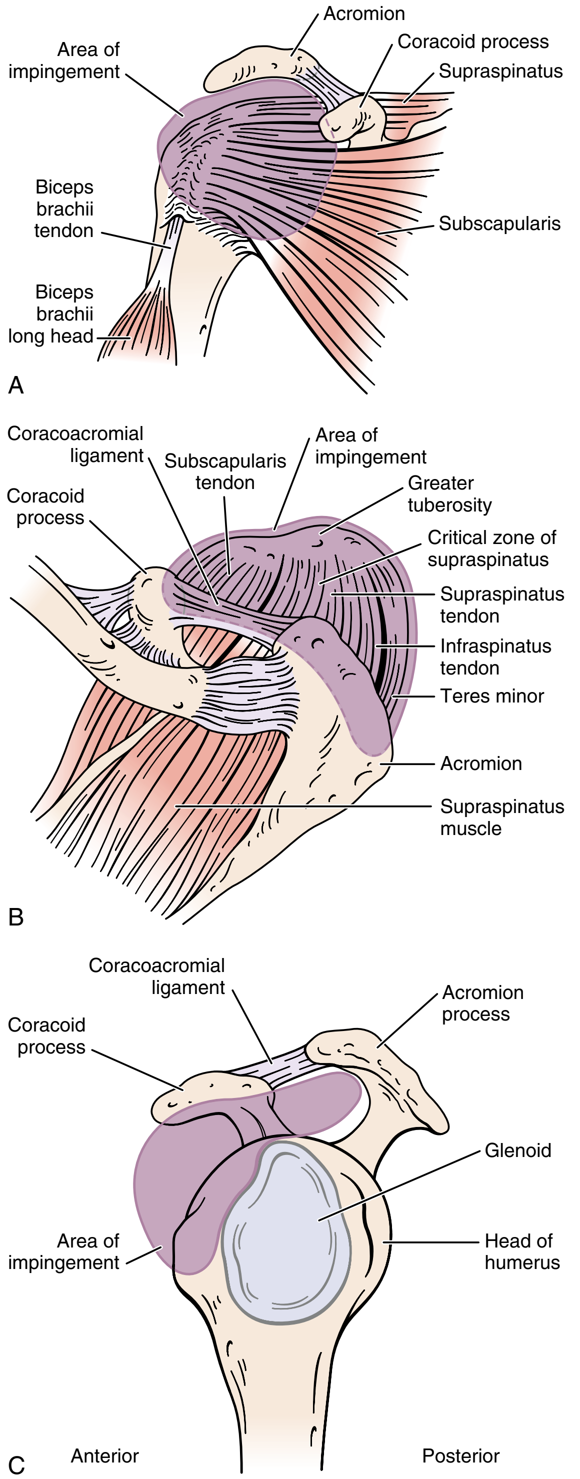

Figure 7-9 Impingement zone. **A,** Anterior view. **B,** Superior view. **C,** Lateral view. (From Magee DJ: *Orthopedic physical assessment*, ed 6, p 316, St Louis, 2014, Saunders/Elsevier.)

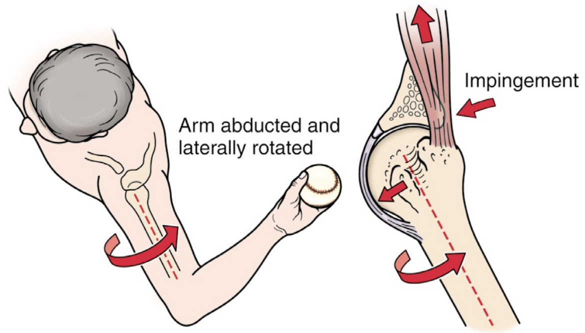

Figure 7-10 Internal impingement of the undersurface of the rotator cuff against the posterior aspect of the labrum in maximum lateral rotation and abduction. (From Magee DJ: *Orthopedic physical assessment*, ed 6, p 318, St Louis, 2014, Saunders/Elsevier.)

deceleration phase, to keep the humeral head centered in the glenoid. Thus, repetitive throwing motion can lead to repetitive microtrauma or impingement on the posterior glenoid margin, usually involving the undersurface of the posterior half of the supraspinatus and the superior half of the infraspinatus. With repeated injury, articular-sided partial-thickness tears can develop.[5,6,50]

Finally, glenohumeral instability and altered scapulothoracic motion must also be considered as possible extrinsic factors leading to rotator cuff injury. Whether instability is due to a structural defect, such as a torn labrum or from muscle imbalance and altered kinematics, the resultant increased translation at the glenohumeral joint affects the integrity of the rotator cuff through either direct compression and/or increased demand on the rotator cuff to stabilize the humeral head on an unstable glenoid.

Pain in the shoulder exists as a vicious cycle of instability, impingement, weakness, and inflammation: all signs and symptoms associated with rotator cuff pathology. Clinicians must have a clear understanding of the multiple etiological factors that can contribute to rotator cuff injuries, and identify clinical findings associated with the cause of the injury, in addition to those resulting from the injury process. Failure to recognize and address *both* of these etiological factors will affect patient outcomes and possibly lead to recurrent rotator cuff injury.

ROTATOR CUFF INJURIES

Optimal functioning of the rotator cuff occurs if all of its muscles are healthy, intact, and well conditioned, and if there is a normal amount of capsular laxity present at the glenohumeral joint, a smooth contour of the undersurface of the coracoacromial arch, a thin, lubricating bursa, and concentricity of the glenohumeral joint and rotator cuff and coracoacromial spheres of rotation.[19–21,34] Given its intricate anatomy and complex role, it is easy to understand why injuries are so common in the rotator cuff.

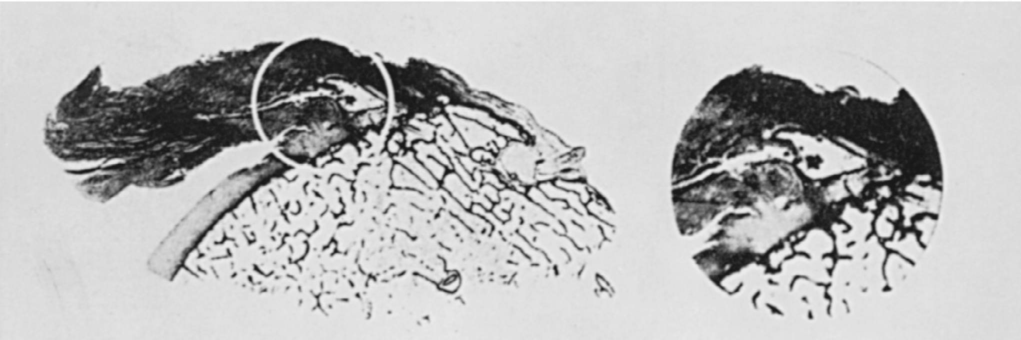

Figure 7-11 Codman's illustration of "a rim rent where all the tendon is torn away from the sulcus except the superficial portion which extends into the periosteum." These photomicrographs provide a convincing argument that cuff tears frequently begin on the deep surface and extend outward until they become full-thickness defects. (From Codman EA: *The shoulder: rupture of the supraspinatus tendon and other lesions in or about the subacromial bursa*, Malabar, 1984, Robert E. Krieger.)

Pathology of the rotator cuff includes a spectrum of injuries, ranging from minor tendinitis to various degrees of partial- and full-thickness tearing. Authors dating back to the 1930s have proposed terms and classification systems to help describe the different types of rotator cuff disease. Codman, considered a pioneer in the realm of rotator cuff injuries, described four different lesions of the supraspinatus tendon, including what he described as a "condition of great clinical importance"—partial tears or "rim rents" (Figure 7-11).[64] The Neer three-stage classification of subacromial impingement lesions has been used extensively to describe injury in this area.[30] Stage I refers to reversible edema and hemorrhage within the rotator cuff and is typically seen in patients under 25 years of age. Stage II occurs with repeated inflammation and results in fibrosis and tendinosis of the rotator cuff, typically in patients 25 to 40 years of age. Stage III occurs with further degeneration of the rotator cuff and adjacent osseous tissue(s) and results in partial or complete tears in individuals older than 40 years of age. Newer classification systems have focused on describing tears of the rotator cuff. Ellman[65] graded partial-thickness tears into three types according to depth. Grade 1 is noted as less than 3 mm deep; grade 2, 3 to 6 mm deep; and grade 3, greater than 6 mm deep. He also included information on the location of the tear, identifying bursal surface, articular surface, or interstitial tears. Fukuda[7] labeled rotator cuff injuries as grade 1, a subacromial bursitis or tendinitis (a "pretear"); grade 2, a partial-thickness tear (involving at least one-quarter thickness of the supraspinatus tendon); and grade 3, a full-thickness tear. Fukuda also classified the location of partial-thickness tears as bursal-side tears, intratendinous tears, and joint-sided tears. Snyder[66] described a classification based on tear location and severity: articular and bursal for partial-thickness tears, named "A" and "B," and "C" for complete tears. Degree of damage is labeled from zero (normal tendon) to four (very severe partial-thickness tear).

Tendinopathy

Tendinopathy of the rotator cuff encompasses a fairly broad category of pathology involving the tendon(s) of one or more rotator cuff muscles. The term *tendinopathy* is preferred to its predecessor, tendinitis, as it includes conditions of the rotator cuff with and without inflammation. Strictly speaking, tendinitis is a misnomer because inflammatory infiltrates within the rotator cuff tendon are not a predominant feature. The difficulty in adequately and consistently defining rotator cuff tendon injuries stems from a lack of routine tissue histological evaluation because patients with tendinopathies are generally treated nonoperatively.[67] This may explain why reviews undertaken to assess the effectiveness of interventions, such as therapeutic exercise in managing rotator cuff tendinopathies, have produced mixed results.[68–72]

Not surprisingly, the clinical presentation of patients with rotator cuff tendinopathy varies, depending on (1) which of the four rotator cuff muscles is involved, (2) where along the muscle and/or tendon the insult has occurred (i.e., muscle belly, musculotendinous junction, or tenoperiosteal attachment), (3) the etiology of the injury (i.e., trauma, overuse, or secondary to a primary injury), and (4) the severity of the pathology (i.e., first, second, or third degree). Treatment of rotator cuff tendinopathy should be based on information pertinent to these four points, as well as a thorough subjective and objective assessment of the entire shoulder girdle.

Determinants for Treatment of Rotator Cuff Tendinopathy

- Which of the four rotator cuff muscles is involved?
- What is the cause of the injury?
- What part of the tendon has been injured?
- How severe is the injury?

The majority of patients with rotator cuff tendinopathy describe shoulder pain as aggravated with overhead activities and relieved with rest. ROM is generally preserved, with possible decrease at the extremes of motion. Resisted strength testing reproduces pain and, depending on the severity of tendon injury, possible weakness. Patients' chief complaints are usually related to diminished function lasting longer than 3 months.[68] Tendinopathy of the rotator cuff has been shown to demonstrate similar pathological changes to tendon disorders in other areas of the body in which loaded (i.e., against gravity or resistance) exercise has proven beneficial. Littlewood et al.[68,73] evaluated the effectiveness of loaded exercise programs for rotator cuff tendinopathy and concluded that the role of loaded exercise in treating rotator cuff tendinopathy was promising, but it required further high-quality research.

Impingement Syndromes

The term *impingement syndrome* was popularized by Neer in 1972, in an evaluation he performed on 100 dissected scapulae.[74] He described 11 as having a "characteristic ridge of proliferative spurs and excrescences on the undersurface of the anterior process of the acromion," which he attributed to repeated contact or impingement of the rotator cuff and the humeral head, with traction of the coracoacromial ligament (Figure 7-12). Of special interest was his discovery that the anterior lip and undersurface of the anterior third of the acromion were involved in all cases. In this region, the supraspinatus tendon inserts onto the greater tuberosity, lying anterior to the coracoacromial arch with the shoulder in the neutral position. With forward flexion, the structures must pass beneath the arch, providing the opportunity for impingement. This early description of shoulder impingement syndrome is now commonly referred to as *primary impingement* because the primary cause of the resultant impingement is the actual abutting of soft-tissue structures (e.g., the rotator cuff and glenoid labrum) and bony prominences (e.g., the acromion and coracoid process). Static factors believed to cause impingement include abnormalities of the coracoacromial arch that may lead to areas of higher than normal compression of the rotator cuff.

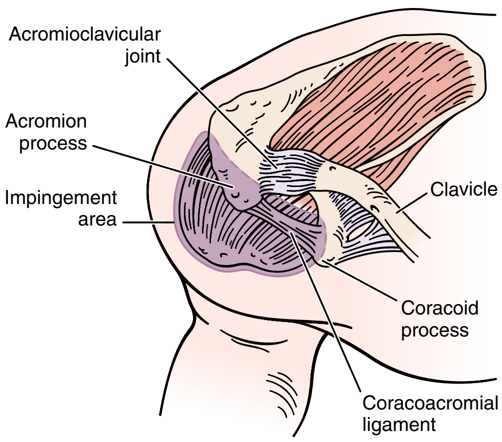

Figure 7-12 The functional arc of elevation of the proximal humerus is forward, as proposed by Neer. The greater tuberosity impinges against the anterior one third of the acromial surface. This critical area comprises the supraspinatus and bicipital tendons and the subacromial bursa. (From Magee DJ: *Orthopedic physical assessment*, ed 6, p 317, St Louis, 2014, Saunders/Elsevier.)

The shape of the acromion has been implicated as one cause of mechanical impingement of the supraspinatus tendon. Bigliani et al.[32] studied the relationship between acromion shape and external impingement by comparing the shape of the acromion to full-thickness supraspinatus tears in cadaveric specimens. Their results concluded that there are three distinct acromion shapes: flat (type I), curved (type II), and hooked (type III). Type II and type III acromion shapes had the strongest correlation with full-thickness tears, leading the authors to conclude that rotator cuff disease is influenced by acromion shape (see Figure 7-3).

Importantly, subacromial impingement is not always the sole cause of rotator cuff disease; in fact, it is more accurately described as the phenomenon that occurs because of the more probable causes of aging and physical loading of the shoulder and arm (Figure 7-13). The process of aging causes degeneration in the rotator cuff, particularly at its insertion points, which leads to fiber thickening and the formation of granulation tissue. The damaged rotator cuff then malfunctions, leading to further disorders in the subacromial area. Excessive loading of the rotator cuff may occur either through a static or "structural" (e.g., hooked acromion) mechanism, or dynamically because of the humeral head and rotator cuff moving against an unyielding structure. Current evidence indicates that both static and dynamic factors act upon the rotator cuff, leading to eventual rotator cuff disease.[33,62] With dynamic causes, the coracoacromial arch and other subacromial structures initially may be normal; however, abnormal motion of the humeral head and the rotator cuff relative to the scapula is the cause of the impingement, leading to sometimes permanent structural changes.[39,75,76] This type of anterior, external impingement is known as *secondary impingement* because the impingement is secondary to the underlying instability and pathology. A third type of impingement, termed *posterior internal impingement*, occurs with the shoulder in 90° of abduction and lateral (external) rotation, and involves compression of the undersurface of the posterior rotator cuff tendons against the posterior-superior glenoid rim (see Figure 7-10). This type of impingement commonly affects individuals involved in overhead work or throwing sports.[28,75]

Rotator Cuff Tears

Rotator cuff tears are among the most common conditions affecting the shoulder, accounting for 4.5 million physician visits in the United States per year.[77] They may

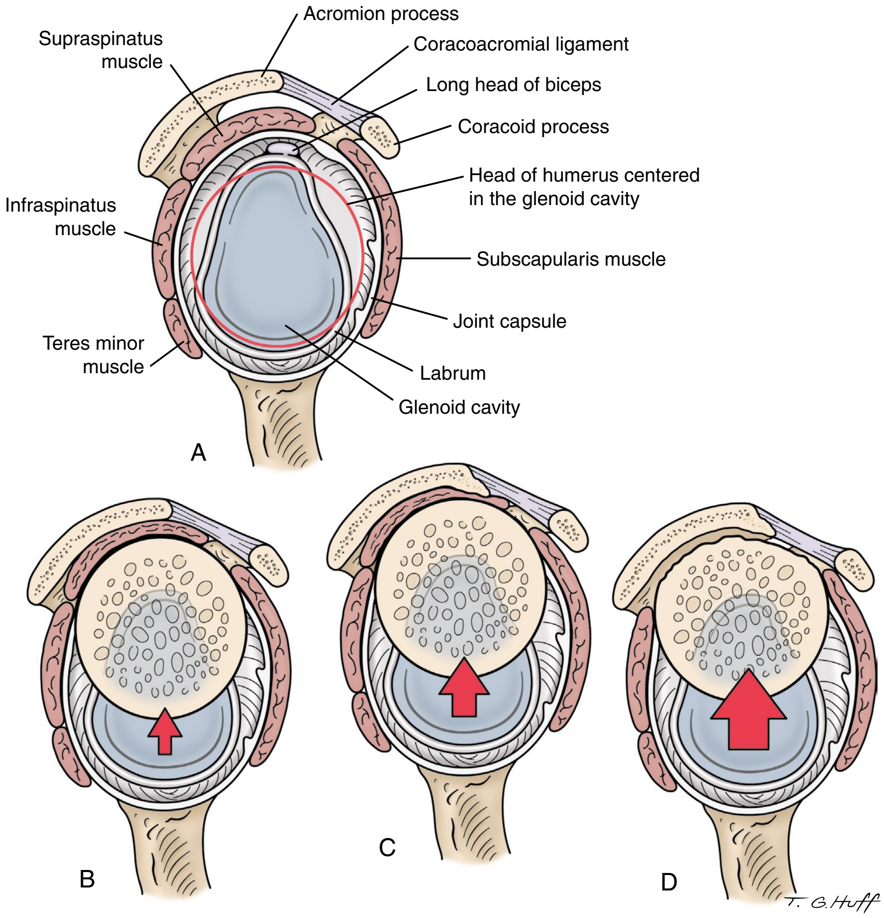

Figure 7-13 Rotator cuff degeneration. **A,** Normal relationships of the cuff and the coracoacromial arch. **B,** Upward displacement of the head, which squeezes the cuff against the acromion and the coracoacromial ligament. **C,** Greater contact and abrasion give rise to a traction spur in the coracoacromial ligament. **D,** Still greater upward displacement, resulting in abrasion of the humeral articular cartilage and cuff tear arthropathy.

be classified as partial or full thickness, acute, chronic, or acute on chronic, and traumatic or atraumatic or degenerative. Tears can range from mild microtearing to total absence of one or more of the rotator cuff tendons. In a young, active patient, rotator cuff tears are almost always the result of a traumatic insult and may occur in combination with other injuries such as an avulsion fracture of the greater tuberosity or injury to the glenoid labrum (Figure 7-14). Degenerative rotator cuff failure, associated with the older individual, typically begins with a partial-thickness defect on the deep undersurface of the supraspinatus as it attaches to the greater tuberosity (Figure 7-15).[78,79] A common misdiagnosis of shoulder pain (e.g., tendinitis, bursitis, and impingement syndrome) may actually represent failure of the deep surface fibers of the rotator cuff. The degree to which the intact fibers may hypertrophy, strengthen, or adapt to stabilize the tear and take up the function of the damaged fibers is unknown. Pettersson[80] used the term *creeping tendon ruptures* to describe a condition in which major rotator cuff defects may occur without symptoms or a recognized injury. He suggested that previous minor, often subclinical, fiber failure could leave the shoulder weaker and the rotator cuff tendons progressively less able to withstand the loads encountered in daily living.

The majority of rotator cuff tears involve the supraspinatus tendon, followed by the infraspinatus and upper subscapularis tendons.[52,54,78] The articular (deep) surface of the supraspinatus tendon has approximately half the strength of the bursal (upper) surface, making it more vulnerable to tearing. The presence of tearing within the anterior supraspinatus or superior subscapularis can also cause injury to the biceps tendon, specifically instability of the long head of the biceps. In addition, long head of biceps dislocations are commonly associated with lesions of the rotator cuff.[29,47,81]

The reported incidence of rotator cuff tears ranges anywhere from 13% to 32%; however, the actual frequency of all tears is unknown because partial-thickness tears are poorly defined and can occur without symptoms.[4,10,82]

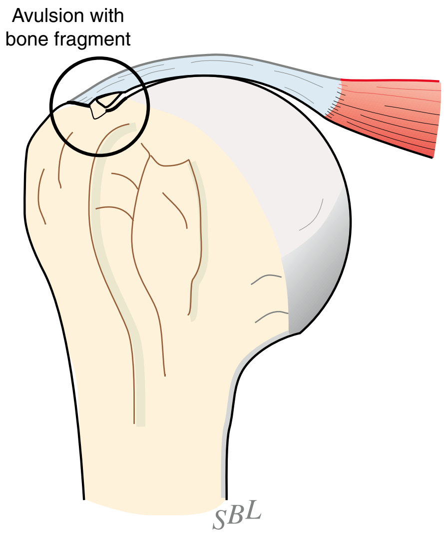

Figure 7-14 Partial-thickness cuff tear with avulsion of a bony fragment from the tuberosity. (From Matsen FA III, Lippitt SB, Sidles JA, et al: *Practical evaluation and management of the shoulder*, Philadelphia, 1994, WB Saunders.)

One group of authors,[82] studying the role age plays in the prevalence of rotator cuff tears, noted that the incidence in cadavers younger than 60 was 6%, compared with cadavers older than 60 years, where it was 30%. Partial-thickness tears appear approximately twice as often as full-thickness defects and can occur on the bursal side, on the articular side, or in the intratendinous region of the rotator cuff. Bursal-sided tears are reported to produce the most severe symptoms due in part to the large number of pain receptors in bursal tissue and the location of the tear within the subacromial space.[7,62,83]

Massive rotator cuff tears are reported as ranging from 10% to 40% of all rotator cuff tears[84] and defined as tears larger than 5 cm in combined areas or a tear involving two or more tendons.[85] The conditions that lead to a massive tear of the rotator cuff are diverse, and its presentation is similarly diverse. A common feature of the shoulder with a massive rotator cuff tear is the subsequent upward migration of the humeral head, which develops over time because of the shear forces created by muscles such as deltoid (Figure 7-16). These tears can cause an uncoupling of forces across the glenohumeral joint and result in unstable shoulder kinematics. Patients typically present with pain, periscapular atrophy, and significant weakness. Rotator cuff tear arthropathy is a pathological condition of the glenohumeral joint in which chronic rotator cuff tearing is associated with structural glenohumeral joint destruction.[86] The joint destruction involves humeral head erosion or collapse, and in some patients, glenoid erosion (Figure 7-17). Rotator cuff tear arthropathy does not include rotator cuff disease in which proximal migration of the humeral head is present without advanced structural changes in the joint surfaces. After a massive rotator cuff tear, the shoulder joint is progressively affected by the profoundly altered biomechanical and biological changes. The corresponding morphological changes were recognized as early as the mid-1800s, when tear-associated joint destruction was compared with localized destructive arthritis. The term *rotator cuff tear arthropathy* was first used by Neer in 1977 to describe shoulder joint destruction secondary to rotator cuff tearing.[87]

Natural History of Rotator Cuff Tears

Natural history refers to the progression of change of the rotator cuff over time in the absence of medical intervention. An understanding of natural history allows for the development of prevention and treatment programs and allows the clinician to inform the patient about what may happen should the disorder go untreated.[88]

As previously noted, rotator cuff tears may be either asymptomatic or symptomatic, and appear to increase in prevalence with increasing age.[89] Approximately 50% of

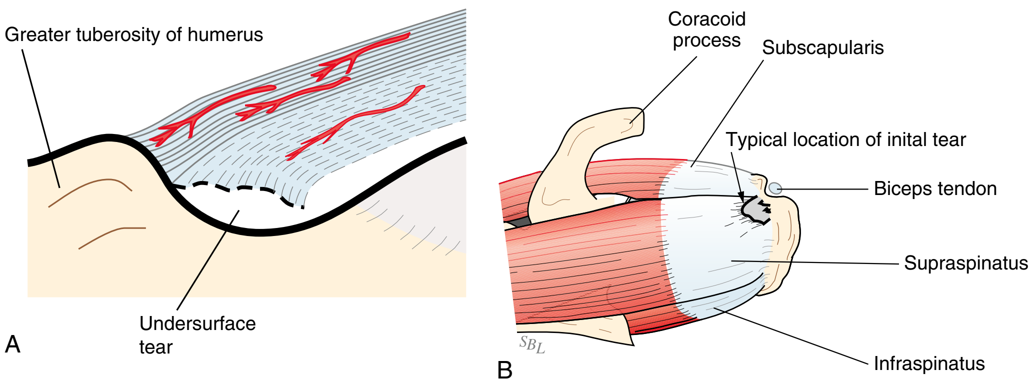

Figure 7-15 In degenerative cuff disease, the tendon fibers fail a few at a time, usually starting at the deep surface of the supraspinatus near its insertion, close to the long head of the biceps. **A,** Anterior view. **B,** Posterior view. (From Matsen FA III, Lippitt SB, Sidles JA, et al: *Practical evaluation and management of the shoulder*, Philadelphia, 1994, WB Saunders.)

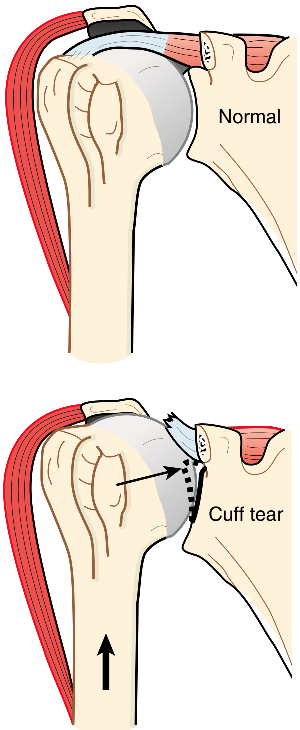

Figure 7-16 In chronic cuff deficiency, erosion of the upper glenoid may leave the shoulder with a permanent tendency toward superior subluxation that cannot be reversed by cuff repair surgery. (From Matsen FA III, Lippitt SB, Sidles JA, et al: *Practical evaluation and management of the shoulder*, Philadelphia, 1994, WB Saunders.)

Figure 7-17 Collapse of the humeral head in combination with massive cuff deficiency because of cuff tear arthropathy. (From Walch G, Edwards TB, Boulahia A, et al: Arthroscopic tenotomy of the long head of the biceps in the treatment of rotator cuff tears: clinical and radiographic results of 307 cases, *J Shoulder Elbow Surg* 14(3):238-246, 2005.)

asymptomatic, untraumatized shoulders, belonging to patients between 60 and 90 years of age, were reported as having partial- or full-thickness rotator cuff defects.[12,89–91] Thus, when describing the natural history of rotator cuff tears, the course of the asymptomatic tear and the symptomatic tear both must be examined.

It remains unclear why some asymptomatic rotator cuff tears become symptomatic. To answer this question, two longitudinal studies have assessed a series of patients with asymptomatic tears. The first series of 45 patients were followed for an average of 5.5 years, with 23 returning for repeat ultrasound assessment. Over a mean of 2.8 years, 51% developed symptoms, and there was an association with a significant increase in pain and decrease in the ability to perform activities of daily living in patients who developed symptoms. It also appeared that the onset of symptoms was associated with an increase in tear size.[14]

A second series of 195 patients, followed with ultrasound, demonstrated an association between pain and the progression in tear size and a greater likelihood that larger tears were more likely to develop pain in the short term compared with smaller tears.[92] Of note, both of these studies examined individuals with a symptomatic rotator cuff tear in the contralateral shoulder. The authors acknowledged that there may have been differences between the patient population studied and individuals without involvement of the contralateral shoulder.

No longitudinal studies exist that examine the untreated symptomatic rotator cuff tear. A series of 59 shoulders receiving nonoperative treatment and followed with serial magnetic resonance imaging suggested that full-thickness tears were more likely to progress in size compared with partial-thickness tears, and that this rate of progression was more prevalent after 18 months than it was during the first 18 months. Additionally, the authors also demonstrated greater progression in individuals over 60, atrophy only with full-thickness tears, and a 17% rate of development or progression of fatty infiltration.[93]

Increased use of diagnostic imaging, such as magnetic resonance imaging and arthrography, over the past decade has identified a seemingly "normal" indicator of aging tissue. Furthermore, rotator cuff defects have been found to be compatible with normal, painless, functional activity. Yamanaka and Matsumato[94] used arthrography to study the progression of 40 untreated partial-thickness tears in asymptomatic patients with a mean age of 61 years. Their 1-year results revealed overall improvements in their subjects' shoulder function scores, despite arthrography findings that showed greater than 50% of subjects had either an enlargement of their tear or a progression to a full-thickness tear.

General Treatment of Rotator Cuff Tears

The management of rotator cuff tears is the subject of substantial debate among clinicians and researchers. In a number of studies, the clinical results of rotator cuff repairs in symptomatic patients, who were followed for as long as 10 years, were good to excellent in a high percentage of cases, even though rerupture of the rotator cuff was known to occur in 20% to 65% of cases.[94–97] Interestingly, a massive, irreparable rotator cuff tear is not incompatible with good overhead function. Observations such as these have made clinical decision making relative to the treatment of symptomatic rotator cuff tears challenging. Historically, treatments have consisted of rehabilitation and surgical repair such as subacromial decompression without repair, tendon transfers, and tendon substitution techniques. The most commonly reported prognostic factors for rotator cuff repair are patient age, chronicity of tear, and size of tear.[28,97–100] Rerupture is clearly a major concern in these patient populations, and it has been correlated with tear size, tear chronicity, and patient satisfaction.

Some current controversies in the management of rotator cuff tears include the role of rehabilitation, the indications for and timing of surgical repair, the method of surgical repair, and the management of irreparable defects. Important factors that must be considered in the decision between surgical and conservative management of a patient with a symptomatic, full-thickness rotator cuff tear are the patient's age and expected activity level and whether muscle or tissue retraction and rotator cuff muscle atrophy and fatty replacement are present.[100–102] Surgery is almost always suggested for patients in the fourth or fifth decade of life who have suffered a traumatic injury that resulted in a full-thickness tear. Patients in the sixth, seventh, or eighth decade of life may or may not be well suited for surgical repair, depending on the chronicity of the tear and the quality of the remaining tendon and muscle tissue, as well as other health issues.[97,98] If the tissue is less than optimal, failure to heal is a significant possibility. Most patients in the older age groups are more interested in eliminating shoulder pain and achieving a functional ROM than in having powerful overhead use of the arm. However, the average age of the general population is increasing, and a sizable proportion of these individuals are remaining active. Therefore, the clinician is likely to be faced with challenging questions about the best way to manage rotator cuff tears and maximize a patient's functional goals and requirements.

CLINICAL PRESENTATION OF ROTATOR CUFF PATHOLOGY

Rotator cuff pathology manifests itself clinically in a variety of ways, depending on the type and severity of pathology, as well as the individual patient characteristics (i.e., concurrent injuries, co-morbidities, and activity level). Common complaints include pain worsened with overhead activities, strength limitations, and functional impairments. Limitations in mobility are related to muscle weakness; therefore, active ROM is usually affected more than passive ROM is. Pain is reported at the end point of motion and is generally greater in abduction and lateral (external) rotation planes of motion. Patients with subacromial impingement or rotator cuff tears, however, may present with limitations in both active and passive ROM because of altered anatomy and/or biomechanics of the shoulder girdle. Patients with subacromial impingement also may possess a characteristic painful arc of movement between 60° and 120° of abduction (Figure 7-18). Patients with rotator cuff tendinopathies and partial-thickness tears tend to have more pain because of the intact sensory components, as compared with patients with full-thickness tears. Functional impairments commonly described by this patient population include difficulty with reaching overhead and behind one's back, lifting (especially with an extended elbow), and lying on the affected shoulder.

Weakness and pain are the primary causes of functional limitations associated with rotator cuff pathology. The degenerative tendon fibers may be weakened and may fail without a specific clinical manifestation.[40,53,54] Patients with partial-thickness tears have substantially more pain on resisted muscle action than those with full-thickness tears have, again because of the often intact nervous tissue, and bursal-sided tears seem to be more symptomatic than deeper tears because of the resultant problems with roughness of the articulation between the upper surface of the rotator cuff and the undersurface of the coracoacromial arch.[62,83,103] Some authors and clinicians believe that a subacromial injection of local anesthetic can be used to differentiate weakness caused by painful inhibition from weakness caused by a tendon defect.[104]

Rotator cuff tears may cause shoulder instability because of a loss of efficient centering of the humeral head in the glenoid. Acute tears of the subscapularis, although less common, may contribute to recurrent anterior instability. Chronic loss of the normal compressive effect of the rotator cuff mechanism and of the stabilizing effect of the superior rotator cuff tendon interposed between the humeral head and the coracoacromial arch may contribute to superior glenohumeral instability (and secondary impingement).[105]

Patients with rotator cuff tears frequently complain of a symptomatic crepitus during passive glenohumeral ROM. This roughness often is caused by bursal hypertrophy, secondary changes in the undersurface of the coracoacromial arch, loss of integrity of the upper aspect of the rotator cuff tendons, or degenerative changes in the tuberosities of the humerus.[28,34,78,103] These factors culminate in a condition known as **subacromial abrasion** (Figure 7-19). Neer et al.[87] used the term *rotator cuff tear arthropathy* to describe a similar phenomenon, which they believed was a cause of this roughness associated with rotator cuff defects.

Patient History

Clinical evaluation of the patient with suspected rotator cuff pathology begins with a detailed medical history, as well as a comprehensive objective examination of the shoulder girdle and adjacent spine and ribs. Shoulder pain can be caused by injury to the osseous and/or soft tissue of the

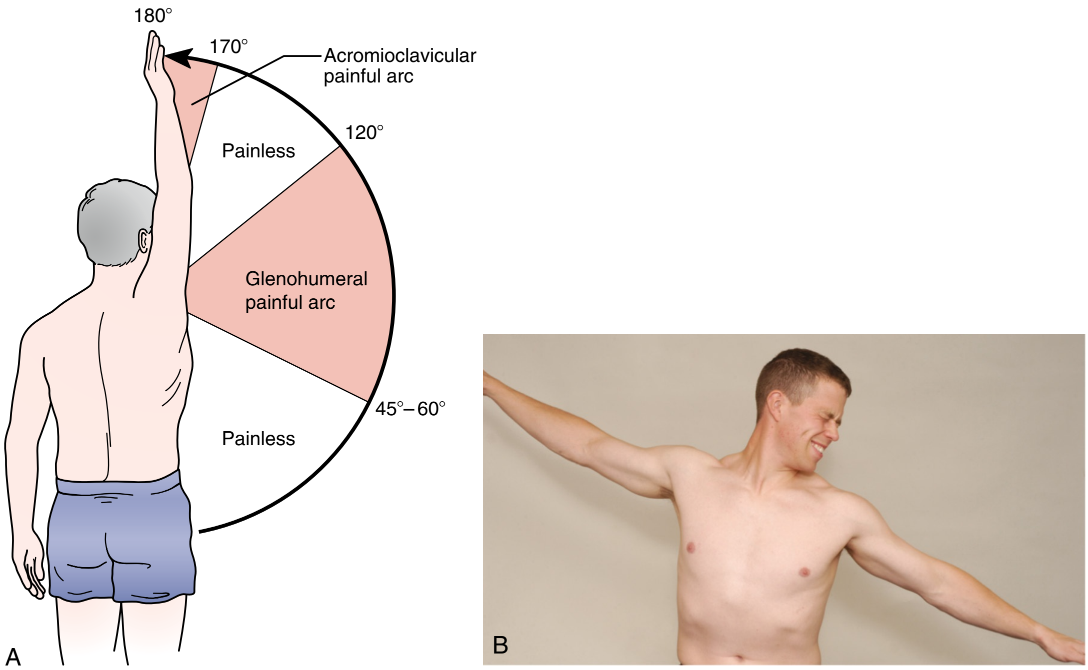

Figure 7-18 Painful arc in the shoulder. **A,** Painful arc of the glenohumeral joint. In the case of acromioclavicular joint problems only, the range of 170° to 180° would elicit pain. **B,** Note the impingement causing pain on the right at approximately 85°. (From Magee DJ: *Orthopedic physical assessment*, ed 6, p 273, St Louis, 2014, Saunders/Elsevier. **A,** Modified from Hawkins RJ, Hobeika PE: Impingement syndrome in the athletic shoulder, *Clin Sports Med* 2:391-405, 1983.)

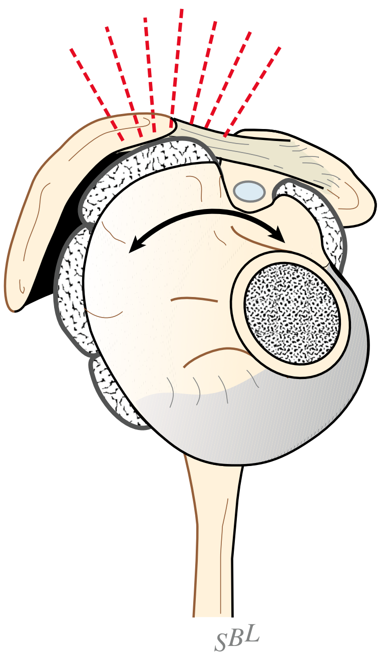

Figure 7-19 Abrasion sign. Symptomatic subacromial crepitance on rotation of the arm elevated to 90° with respect to the thorax, a position in which the capsule and ligaments are normally not under tension. (Modified from Rockwood CA, Matsen FA, editors: *The shoulder*, p 824, Philadelphia, 1998, WB Saunders; Matsen FA III, Lippitt SB, Sidles JA, et al: *Practical evaluation and management of the shoulder*, Philadelphia, 1994, WB Saunders.)

glenohumeral joint, dysfunction about the scapulothoracic region, or it may originate within the cervical or thoracic spine regions. Clinicians must be cognizant of primary and secondary causes of rotator cuff pathology and include all suspected regions within a patient's assessment. The subjective history should follow a systematic approach, similar to all other musculoskeletal assessments, with particular attention given to the following factors.

Patient Profile

Factors such as patient age, occupation, and activity level are key in understanding the etiology and pattern of rotator cuff injuries. Injuries are common in young, active individuals, as well as in patients over 65 years of age who possess normal, degenerative processes at the glenohumeral joint. Younger patients with rotator cuff injuries are often involved in some type of overhead sport or an occupation that places excessive force and repetitive demand on the shoulder, especially during overhead activities. The rotator cuff is particularly susceptible to conditions such as subacromial impingement and partial tears in patients over 50 years of age, where normal degeneration of the shoulder joint occurs. The prevalence of subacromial impingement and rotator cuff tears has been shown to increase significantly in this population, but it is not limited to older adults—partial-thickness tears are also reported in younger athletes.[5] Age does, however, seem to be most consistently related to the incidence of full-thickness tears, with older patients being significantly more affected.[12,16,79] Activities that are repetitive and occur overhead, place individuals at greater risk of rotator cuff injury. These include daily life, work, and/or recreational activities. Minagwa et al.[10] performed a population study that showed an increased prevalence of full-thickness tears in people who performed heavy labor. The nature of work performed may also influence frequency of partial-thickness tears, but no studies have looked at this as a risk factor.

Mechanism of Injury

Rotator cuff injuries can occur as a result of an acute traumatic mechanism, such as a fall on an outstretched hand ("FOOSH" injury), or they can be atraumatic in nature, often because of repetitive overload or degeneration. Many patients possess a combination of mechanisms ("acute on chronic"), describing one incident that caused the eventual injury on a shoulder that was previously injured to a lesser degree and/or was always "sore." The literature indicates that a memorable incident of trauma is related to degree of tearing. Of patients with a full-thickness tear, 65% were able to describe a defined incident that caused their shoulder injury, whereas only 47% of patients with partial tears, and 37% with tendinitis were able to recall a specific onset.[7] Differences between grades of lesion and mechanism have also been reported: patients with a well-defined injury more often had full tears (65%), versus 53.3% of patients with partial tears, who had no episodic injury.[95]

Aggravating and Easing Factors

Information related to what aggravates and eases a patient's injured shoulder is helpful in establishing the type and degree of rotator cuff pathology involved. In addition, these factors form important, patient-directed functional outcome measures. Typical aggravating factors include movements and positions that place the arm overhead and behind the plane of the body. Both of these positions are made worse if load is added to the arm and the elbow is extended, resulting in greater leverage forces directed on the shoulder joint. Activities performed in abduction are worse than flexion and scaption (i.e., movement in the scapular plane), and most patients with rotator cuff pathology describe impingement-type symptoms when their arm is medially (internally) rotated and elevated to around 90° of elevation, especially in combination with horizontal adduction.

Pain

Pain almost certainly accompanies the patient with rotator cuff pathology. It typically presents in the anterolateral aspect of the shoulder with a common referral pattern to the deltoid insertion point. A deep ache that intensifies to a sharp pain when the arm is placed in aggravating positions is commonly noted in this patient population. Night pain is also a common complaint for both partial-thickness and full-thickness tears, with pretears and partial-thickness tears generally more painful than full-thickness tears are.[7] Pain is a strong motivator for patients seeking care and the most common symptom of a rotator cuff tear.[15] Radicular pain is not indicative of rotator cuff pathology, and if reported, it should direct the clinician to perform a cervical spine examination.

Function and Health-Related Quality of Life

Rotator cuff disease has a high incidence rate, and active individuals are often affected.[106] As noted, common clinical features are pain at rest and at night, motion loss, and weakness.[26,33,51] These symptoms can lead to disruption of activities of daily living, work, and leisure, with overall poor quality-of-life experience.[17] The functional disability of rotator cuff injuries affects not just the physical aspect of a patient's life but also the mental and social aspects.[17,18] Therefore, clinicians should include outcome measures related to upper-extremity functional capacity and quality-of-life measures to understand and monitor the level of effect a rotator cuff injury has on the patient's livelihood.

Physical Examination

The objective physical examination of the patient with suspected rotator cuff pathology should include the entire shoulder girdle, as well as the proximal (cervical and thoracic spine), ribs, and distal segments (elbow, wrist, and hand) of the upper-extremity kinetic chain. Diagnostic imaging (i.e., ultrasound and magnetic resonance imaging) and arthroscopic surgery may or may not be included in the diagnostic procedure. Objective assessment of the shoulder consists of a series of tests that measure how the shoulder and related tissue(s) move, as well as "special tests" designed to discern the type and severity of rotator cuff involvement. Despite this systematic approach, accurate diagnosis of an injured rotator cuff is notoriously difficult. Partial-thickness tearing is especially troublesome to diagnose because its signs and symptoms mimic those of other shoulder conditions such as rotator cuff tendinopathy, impingement syndromes, and atraumatic instability.[107–109] Open surgery is considered the gold standard for rotator cuff disease diagnosis; however, this is clearly impractical for use in every patient with suspected pathology. Magnetic resonance imaging and ultrasound are considered reference standards.[110] However, there is uncertainty regarding the accuracy of these tests in identifying conditions such as partial-thickness tears.[107] Special tests for the rotator cuff, although easy to apply and inexpensive,[110] generally have high sensitivity but low specificity.[109,111,112] Most research into the diagnosis of rotator cuff pathology concludes that no single test (physical or imaging) is accurate on its own,[113] reinforcing the importance of considering all components of the patient's assessment (i.e., subjective, objective, and imaging tests) when determining a patient's diagnosis.[114]

Observation

Observation starts at the beginning of the patient-clinician interaction and continues throughout the entire examination. The patient should be observed at rest and with movement, from the anterior, lateral, and posterior views (Figure 7-20). Ideally, the patient should be observed in positions he or she reports as aggravating, as well as while performing his or her functional activities. Careful inspection of the scapulothoracic region and adjacent spine and ribs is important because these regions may play a role in the cause and effect of the rotator cuff pathology. In the young patient, there may be evidence of scapular dyskinesis or muscle imbalance seen, specifically the lower trapezius (i.e., decreased) and the upper trapezius muscles (i.e., increased) (Figure 7-21). The older patient with rotator cuff disease may have an altered or reversed scapulohumeral rhythm because of significant rotator cuff weakness and/or pain, and this may be in combination with muscle atrophy of one or more of the rotator cuff muscles. The thoracic and lumbar spine regions may be important to inspect depending on the patient's history and/or initial presentation. The young, athletic patient with suspected rotator cuff pathology should be carefully observed for postural abnormalities of the spine and hypomobility of the ribs and lower kinetic chain that may be contributing factors to his or her shoulder injury. Postural abnormalities such as shoulder girdle rounding, chin poking, and thoracic spine kyphosis are common in older patients with shoulder pain.

Mobility

Mobility testing assesses several components involved in movement: a patient's willingness to move, joint range, and muscle power.[115] Active and passive ROM testing of the scapulothoracic and glenohumeral joints, as well as selective testing of tissue known to be tight in this population, should all be included in this portion of the examination. Because the primary tissue at fault (i.e., rotator cuff) is contractile, active ROM should be affected more than passive ROM is; however, this will depend on whether the tissue surrounding the rotator cuff is involved (i.e., coracoacromial arch). Common limitations include: (1) glenohumeral abduction affected more than flexion, (2) greater than 90° elevation positions affected more than those less than 90°, and (3) active ROM affected more than passive ROM. Patients with impingement or acute rotator cuff pathology may demonstrate a painful arc of movement from approximately 60° to 120° of abduction and a compensatory shoulder hike pattern (i.e., reversed scapulohumeral rhythm). Overhead athletes with an injured rotator cuff should be carefully assessed for deficits of medial (internal) rotation ROM at 90° of abduction, particularly of their dominant shoulder (Figure 7-22). Loss of this movement, with a concurrent increase in lateral (external) rotation ROM, is believed to occur as a result of osseous and soft-tissue changes about the shoulder, and it has been linked to shoulder pathology.[50,116,117]

Limitations in movement about the shoulder can occur as a result of different mechanisms. They can be as a direct result of the injured rotator cuff, as seen in the patient with decreased abduction from a supraspinatus tear, or a painful arc of motion in the patient with subacromial impingement. Motion can also be altered because of a compensation pattern adopted by the patient to avoid pain, impingement, or instability at the glenohumeral joint.

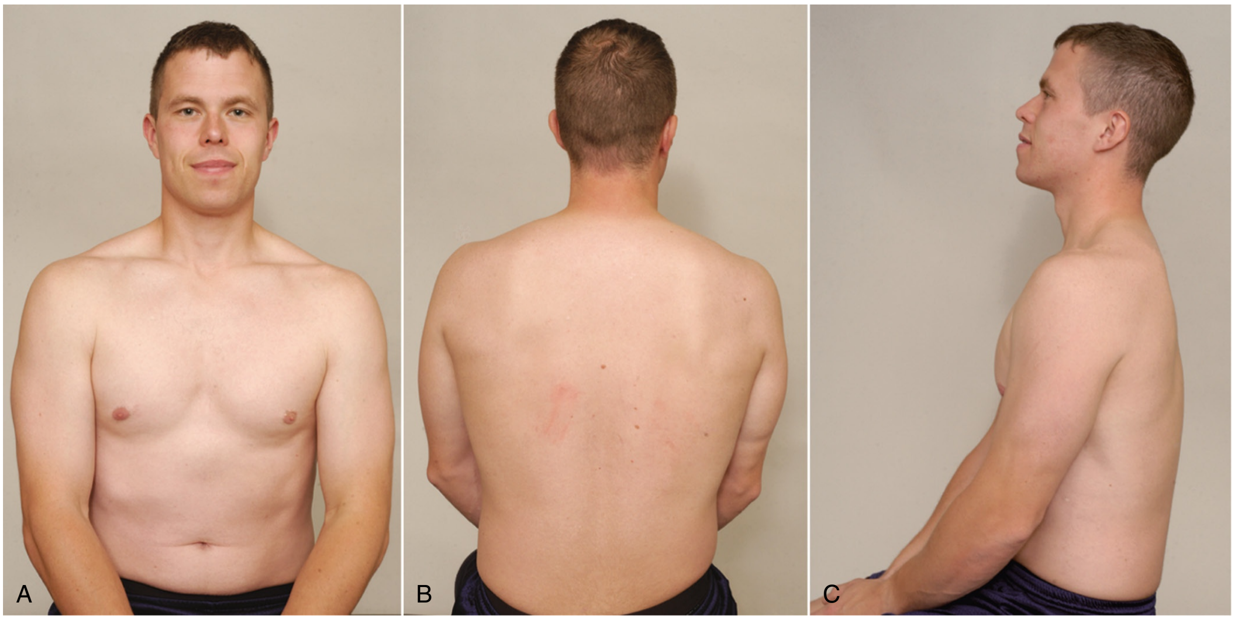

Figure 7-20 Views of the shoulder. **A,** Anterior. **B,** Posterior. **C,** Side. (From Magee DJ: *Orthopedic physical assessment*, ed 6, p 265, St Louis, 2014, Saunders/Elsevier.)

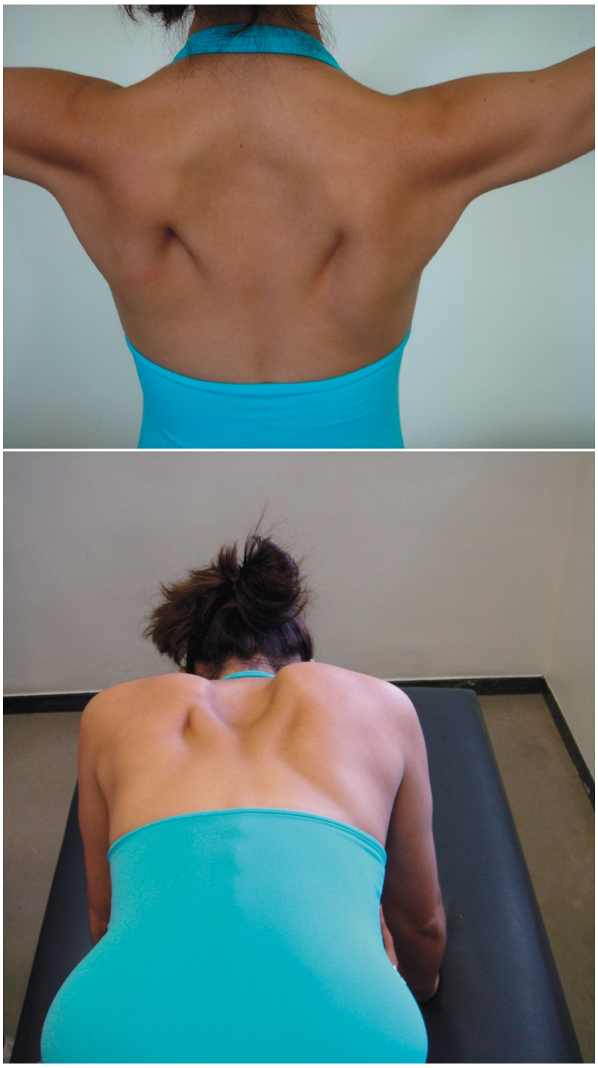

Figure 7-21 Imbalance pattern of the upper and lower trapezius. Note the overdevelopment of the upper trapezius and lower trapezius working to prevent rotary winging. (From Magee DJ: *Orthopedic physical assessment*, ed 6, p 269, St Louis, 2014, Saunders/Elsevier.)

An example of this is the patient who uses a reversed scapulohumeral rhythm (i.e., shoulder hike) to elevate his or her arm because of significant rotator cuff weakness or the patient who uses scapular retraction instead of lateral (external) rotation to avoid painful motion or instability at the glenohumeral joint (Figure 7-23). Finally, abnormal ROM may be part of the contributing cause of the rotator cuff pathology. Common clinical features associated with patients with rotator cuff pathology include scapular instability (i.e., in the young patient) and decreased thoracic spine extension (i.e., in the older patient). Distal ROM alterations such as decreased hip and trunk rotation can affect the efficiency of the kinetic chain and have a detrimental effect on the shoulder and rotator cuff in individuals who use their arm in overhead positions.[118] Clinicians must be aware of all of the interrelationships

Figure 7-22 Glenohumeral medial rotation passive ROM measurement using stabilization of the scapula by holding the coracoid process and the scapula down. (From Magee DJ: *Orthopedic physical assessment*, ed 6, p 277, St Louis, 2014, Saunders/Elsevier.)

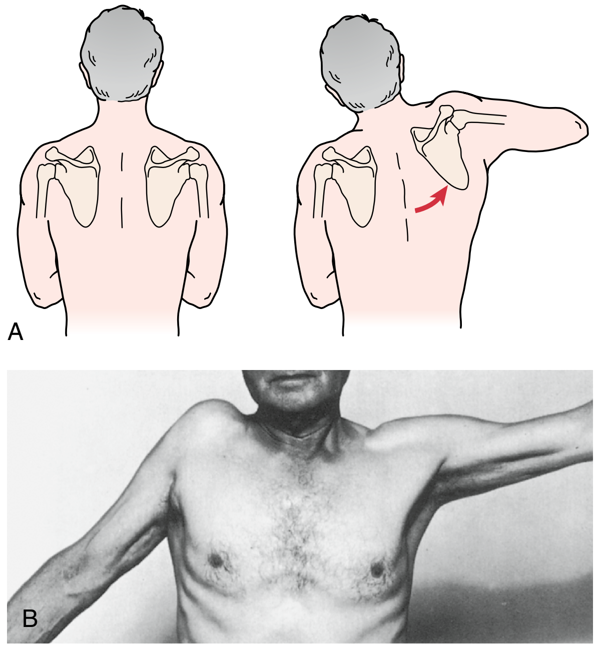

Figure 7-23 Reverse scapulohumeral rhythm (notice shoulder hiking) and excessive scapular movement. Examples include **A,** frozen shoulder or **B,** tear of rotator cuff. (**A,** From Magee DJ: *Orthopedic physical assessment*, ed 6, p 276, St Louis, 2014, Saunders/Elsevier. **B,** From Beetham WP, Polley HF, Slocum CH et al: *Physical examination of the joints*, p. 41, Philadelphia, 1965, WB Saunders.)

of the kinetic chain during shoulder movement and be careful to differentiate which of the three categories the limitation falls into for successful treatment planning.

Strength

Strength testing should include the muscles of the entire shoulder girdle, not just the rotator cuff. Muscles that attach to and control the stability and movement of the scapulothoracic articulation are often weakened because of rotator cuff injury or they are part of the cause of the rotator cuff failure in the first place. Furthermore, shoulder rehabilitation programs emphasize the strength of the scapular muscles; therefore, establishing baseline strength measures of these muscles is essential for determining outcome. Rotator cuff strength should be assessed with the glenohumeral joint in neutral, as well as in abduction (at 90°), if pain allows. In young, athletic individuals with rotator cuff pathology, information related to the rotator cuff muscles' endurance, as well as strength, may prove more useful. Most patients with rotator cuff pathology present with weakness of lateral (external) rotation (i.e., primarily infraspinatus) and abduction (i.e., supraspinatus) movements of the glenohumeral joint.[115] McCabe found all grades of rotator cuff tears had strength deficits in most movements, but 10° abduction showed a distinction between partial and full thickness—full-thickness tears had a greater loss of strength.[16] The most common finding relative to the scapulothoracic joint includes weakness of the lower trapezius and serratus anterior muscles with a concurrent increase of overactive upper trapezius muscle.[119,120]

Special Tests

Special tests are designed to confirm or rule out a tentative or preliminary diagnosis. Although strongly suggestive of a particular injury or condition when they yield positive results, they do not necessarily rule out the injury or condition when a negative result is obtained. Ever since Codman's original description of rotator cuff disease, special tests have been developed to identify lesions of the rotator cuff. Tests most commonly included in the shoulder examination of a patient with suspected rotator cuff pathology are those that measure rotator cuff integrity (e.g., empty can test) and detect impingement (e.g., Neer test) (Figure 7-24). Shoulder instability tests should also be included when examining a young, overhead athlete. A comprehensive description of the special tests for the shoulder may be found in *Orthopedic Physical Assessment*.[115] Systematic reviews evaluating many of the special tests used to diagnose rotator cuff disease have found the tests to be adequate at ruling out rotator cuff lesions but less accurate for ruling them in (Table 7-1).[4,109,110,121–123]

Diagnostic Imaging

Although the importance of a thorough history and physical examination for a patient with suspected rotator cuff pathology cannot be understated, the suspicion of an acute rotator cuff tear or the failure of nonoperative treatment supports obtaining objective imaging of the rotator cuff. Imaging helps to confirm or refute a suspected diagnosis, and it assists the clinician with planning further nonoperative and potential operative management. Imaging may also assist the clinician in better assessing the prognosis of a rotator cuff tear.

Figure 7-24 Impingement sign. **A,** A positive Neer impingement sign is present if pain and its resulting facial expression are produced when the examiner forcibly flexes the arm forward, jamming the greater tuberosity against the anteroinferior surface of the acromion. **B,** An alternative method (Hawkins-Kennedy impingement test) demonstrates the impingement sign by forcibly medially rotating the proximal humerus when the arm is forward flexed to 90°. (From Magee DJ: *Orthopedic physical assessment*, ed 6, p 318, St Louis, 2014, Saunders/Elsevier.)

TABLE **7-1**

Sensitivity and Specificity of Special Tests for Diagnosis of Rotator Cuff Pathology

Test	Sensitivity	Specificity
Neer's	0.89	0.32
Hawkin's	0.92	0.2
Jobe	0.84	0.58
Empty can	0.89	0.5

Data from Dinnes J, Loveman E, McIntyre L et al: The effectiveness of diagnostic tests for the assessment of shoulder pain due to soft tissue disorders: a systematic review, *Health Technol Assess* 7:iii, 1-166, 2003.

TABLE **7-2**

Sensitivity and Specificity of Diagnostic Tests

Test	Sensitivity	Specificity
MRI: FTT	0.89	0.93
MRI: PTT	0.9	0.44
US: FTT	0.87	0.96
US: PTT	0.67	0.9

MRI, Magnetic resonance imaging; *US,* ultrasound; *FTT,* full-thickness tears; *PTT,* partial-thickness tears

Data from Dinnes J, Loveman E, McIntyre L et al: The effectiveness of diagnostic tests for the assessment of shoulder pain due to soft tissue disorders: a systematic review, *Health Technol Assess* 7:iii, 1-166, 2003.

Many imaging options exist, including x-rays, arthrography, computed tomography arthrography, ultrasound, and magnetic resonance imaging without or with contrast. However, it is critically important to assess imaging in the context of clinical findings. Imaging is highly sensitive, and management based solely on imaging findings may lead to unnecessary or overtreatment.[124]

Multiple studies exist assessing the accuracy of the various imaging modalities for rotator cuff tears. Regional variations exist in the preference of one modality over the other, with computed tomography arthrography being more prominent in Europe than in North America. Studies assessing the accuracy of imaging compared with surgical findings generally provide the highest level of evidence; however, the assessment of atrophy and fatty infiltration cannot be done surgically, which is why these studies provide a measure that is less defined.

Studies with a higher level of evidence exist for the assessment of ultrasound and magnetic resonance imaging, compared with surgical findings, as well as for the comparison of ultrasound, magnetic resonance imaging and magnetic resonance imaging with contrast, and surgical findings. The literature does demonstrate a trend for imaging to define full-thickness tears better, compared with partial-thickness tears and tendinosis, with the accuracy of ultrasound and magnetic resonance imaging decreasing with tears that are less severe. As with all imaging, the results require interpretation and are thus dependent on the skill and experience of the interpreter.

For the evaluation of the rotator cuff specifically, ultrasound should be considered as the imaging modality of choice for the initial detection of full- and partial-thickness rotator cuff tears in patients with a history and clinical findings that do not suggest any other intra-articular disorder.[125] Magnetic resonance imaging may be reserved for cases with equivocal findings and in patients with involvement of anatomical structures not well defined by ultrasound.[126] Ultrasound has the advantage of being noninvasive, well tolerated, inexpensive with minimal risk, and it can be used for bilateral comparisons. However, it does not reveal pathological conditions in bone or intra-articular lesions, and there is a significant learning curve before obtaining adequate results.[127] In most cases, the use of both modalities is redundant, and their use in combination with the use of magnetic resonance imaging with contrast should be reserved for cases where the desired information can only be provided by the specific modality or combination of modalities.

All of these tests, however, have limitations when compared with the gold standard of arthroscopic surgery. Magnetic resonance imaging has fairly high sensitivity and specificity for full-thickness tears, but for partial-thickness tears, sensitivity is much lower with continued high specificity.[109] Although magnetic resonance imaging is acceptable for ruling in any rotator cuff tears, cost and wait times for testing are additional issues.[108] Ultrasound is also more accurate for full-thickness tears but only fair for evaluating partial-thickness tears (Table 7-2).[109]

TREATMENT OF ROTATOR CUFF PATHOLOGY

Treatment of the patient with rotator cuff pathology is based upon the specific nature of the injury or condition, as well as the individual patient's goals and functional requirements. Care must be taken to understand and address the underlying pathology, as well as the causal factors that led to or are secondarily contributing to the injury. Finally, treatment plans should be based upon information obtained from the patient history and physical examination.

The initial treatment of most rotator cuff injuries, including partial-thickness and some full-thickness tears, is nonoperative. The literature, however, is diverse with respect to best practice recommendations, specifically regarding which patients require surgical intervention and when and what nonoperative interventions are most effective in managing the various rotator cuff pathologies.

Operative versus Nonoperative Management

As mentioned, the majority of rotator cuff injuries benefit from nonoperative management for a period of at least 3 months, before surgical consideration. Exceptions to this include an acute, traumatic tear, especially in a young, active individual or chronic full-thickness tears in patients younger than 65 years of age.[99,101,128] Early operative intervention may also be considered in cases with mitigating factors such as severe recalcitrant pain, inability to work because of symptoms, or timing of surgery according to the start or end of an athlete's season to minimize time lost for recovery.[28] Comparative outcome studies of operative versus nonoperative interventions in rotator cuff patients have yielded inconclusive results. Seida et al.[69] studied the benefits and harm of conservative and surgical treatments on clinically important outcomes in adults with full- and partial-thickness rotator cuff tears. They identified five studies in their review that compared nonoperative with operative treatments. Four of these studies included physical therapy (treatment components not specified) with or without the addition of steroid injections, oral medications, activity modification, or manual therapy.[102,128–130] One study examined the use of shockwave therapy.[131] All study groups showed statistically significant improvements regardless of the intervention; however, the reviewers noted that the evidence was too limited to draw conclusions regarding comparative effectiveness. Decisions regarding which patients with rotator cuff pathology ultimately require surgery and at what stage remain unclear.

Nonoperative Management

The state of evidence, relative to physical therapy management of patients with rotator cuff disease, is limited and of low-moderate methodological quality.[68,102,128–130,132–134] Very little evidence exists to support its effectiveness in treating patients with rotator cuff injuries. Reports of treatment programs are inconsistent and often lacking in detail regarding specific interventions, exercise dosage parameters, and treatment delivery methods. In fact, "physical therapy" is often labeled as the intervention with no further description provided. Very little work has examined the effect of physical therapy on specific rotator cuff pathologies. Most studies combine tendinopathies, impingement syndromes, partial-thickness tears, and full-thickness tears within their study populations, despite evidence that some rotator cuff injuries (i.e., full-thickness tears) require surgical repair.[69,84,85] Conversely, some studies investigate a specific physical therapy intervention (e.g., laser) but have applied it to a heterogeneous sample of patients with varying types of shoulder pathologies.[69,71] Despite this, physical therapy remains a mainstay of treatment for rotator cuff injuries, and clinicians should continue to deliver treatments grounded in general shoulder rehabilitation principles. Goals of treatment include (1) protecting and encouraging optimal healing of the injured rotator cuff muscle or tendon(s) and surrounding tissue, (2) educating patients (including how to avoid repeated injury), (3) managing pain, (4) restoring normal mobility or tissue length of the shoulder girdle and adjacent kinetic chain segments, (5) restoring normal strength with emphasis on the affected and nonaffected rotator cuff muscles and scapular stabilization muscles, (6) including or encouraging a component of aerobic exercise to the overall treatment, and (7) educating patients on modifying work and recreational activities that may interfere with achievement of the optimum outcome.

Pain is a powerful inhibitor of muscle activation at the shoulder. This is particularly problematic given the number of muscles that act to coordinate shoulder girdle motion, stability, and function. Efforts should be taken to relieve or manage pain in the patient with rotator cuff pathology. This can be accomplished through the use of pain-relieving modalities, patient education, and active exercise. Complete rest from activity is seldom recommended; instead, exercises should be modified by reducing the intensity and/or frequency and altering factors such as arm position (e.g., neutral versus overhead), leverage (e.g., bent elbow versus straight elbow), and degree of motion (e.g., small arc versus large arc exercises). Ideally, exercises should be done within a pain-free ROM, and patients should be instructed on how to manage and monitor pain responses.

Rotator cuff injuries are commonly associated with local and distant deficits in flexibility, strength, and muscle balance that can affect the entire kinetic chain. For example, poor upper-body or trunk posture is often observed in individuals with chronic rotator cuff impingement syndrome. Exercises to correct poor posture should be implemented early in the rehabilitation process, and patients should be instructed that all subsequent exercises begin with establishing "proper posture." Reduced hip and lumbar spine flexibility (i.e., rotation) can lead to increased stress on the shoulder of an overhead athlete. To maintain the maximum force output, the athlete compensates for the loss of hip and trunk flexibility by overextending his or her shoulder into greater degrees of abduction and lateral (external) rotation—a position known to stress the rotator cuff. A breakdown or deficit of any part of the kinetic chain may cause shoulder injury or may occur because of the injury. Regardless, all deficits of the kinetic chain should be noted on assessment and incorporated into the overall treatment plan.

The stabilizing muscles of the scapula should always be included in a strengthening program for the rotator cuff. Scapular winging, abnormal periscapular muscle recruitment, and disruption of the scapulohumeral rhythm are all common in patients with rotator cuff pathology.[116,135] The lower trapezius and serratus anterior muscles are often affected and become "underactive," whereas the upper trapezius compensates and becomes "overactive." The resultant loss of scapular control can contribute to or worsen shoulder impingement, acromioclavicular dysfunction, and rotator cuff disease.[135,136] Proper shoulder rehabilitation should include careful evaluation and treatment of any abnormalities in this region. Patients should be first taught how to perform an isolated contraction of the weakened scapular muscle(s), followed by a sequence of progressions that encourage the muscle(s) to work in a coordinated, synchronous manner with other scapular muscles, postural trunk muscles, and finally muscles of the glenohumeral joint and arm. Proximal stability (i.e., scapular control) must be achieved early in the rehabilitation process, before distal mobility (i.e., glenohumeral and arm movement) is encouraged.

It is important to remember that activation of the rotator cuff muscles involves a concentric movement, whereas stabilization is both static (i.e., isometric) and dynamic (primarily eccentric). Exercise programs should reflect all three of these types of muscle contractions. As previously mentioned, patients must be taught to stabilize their scapula while performing rotator cuff exercises, to ensure a proper base of support for the working muscles. Initially, rotator cuff exercises should be performed with minimal resistance in low-risk positions (i.e., neutral position with a 90° bent elbow) that encourage proper muscle firing without compensation, pain, and/or impingement (Figure 7-25). When appropriate, progressions toward more complex, functional movement patterns that incorporate the rotator cuff should be initiated. Examples include increasing the amount of glenohumeral elevation, eventually performing rotation in overhead positions; increasing volume and resistance; incorporating muscles and joints from the rest of the upper kinetic chain (e.g., proprioceptive neuromuscular facilitation [PNF] patterns); altering the body's base of support to engage the trunk and lower kinetic chain (e.g., sitting on a physioball, standing on one leg); and proprioception and balance types of exercises (e.g., Body Blade® and closed kinetic chain exercises). The patient's signs and symptoms, as well as his or her ability to perform the exercise(s) properly, are always the best guide for progressing to more challenging levels. The goals of rehabilitation for a patient with a symptomatic, full-thickness rotator cuff tear are a combination of education about activity modification, pain management, and ROM and strengthening exercises that focus on the remaining muscles of the rotator cuff and the supporting musculature.[137,138]

The shoulder complex, particularly the glenohumeral joint, is prone to stiffness after trauma, injury, or immobilization.[139] Early intervention through a combination of gentle stretching and joint mobilization techniques may

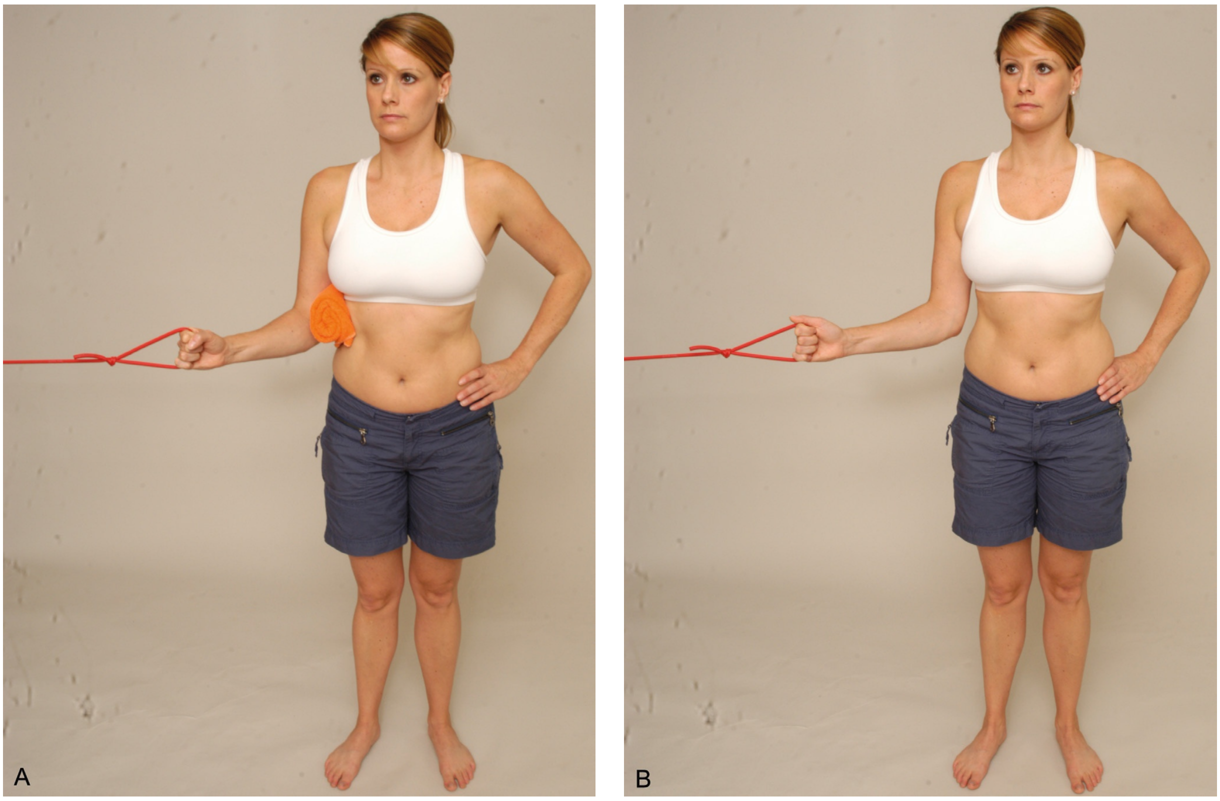

Figure 7-25 Examples of tubing exercises into medial rotation, with the arm at the side, both with (**A**) and without (**B**) a towel at the elbow. Note that the clinician should be cautious in using a towel to hold the elbow against the side. If the patient does not have good control of the scapula, stabilization through the elbow occurs, and using a towel reinforces the incorrect pattern.

prevent this restriction, reduce associated pain, and allow the shoulder to function normally. Special consideration must be given to the adaptive tightening of the glenohumeral posterior capsule, which presents clinically as a medial (internal) rotation ROM deficit, and to the anterior shoulder muscles (e.g., pectoralis minor), which also tend to shorten because of postural and scapular malpositioning.[116,117,140] The sleeper stretch (for posterior capsule tightness) (see Figure 6-31) and the open book stretch (for pectoralis minor tightness) are two examples of appropriate stretches that may be useful with this patient population. Exercises such as the pendular arm swing and the active-assist stick or cane may be used initially to increase ROM. Once a patient is able to actively move his or her shoulder with proper scapulohumeral rhythm (i.e., no upper trapezius shoulder hiking) and minimal pain, he or she should be progressed to active ROM exercises. Eventually, patients with rotator cuff pathology should be performing a variety of ROM exercises—in both open and closed chain positions, with and without load—that mimic functional movement patterns (see Figure 6-37). The goal of nonsurgical treatment of partial-thickness tears specifically is to ensure that the scar collagen that forms in the defect becomes as supple as a normal tendon. If this does not happen, scar contracture tends to concentrate the load of the rotator cuff on the scar, leading to recurrence and further propagation of the injury.[141,142]

Operative Management

Surgical intervention is an option for symptomatic rotator cuff tears that fail a trial of nonoperative management, which has included an active exercise program.[143] When determining whether surgery is appropriate, both patient and rotator cuff–specific factors should be considered. Patient-specific factors include age, medical comorbidities, worker compensation status, and ability or willingness to follow instructions and participate in postoperative physical therapy.

The primary goal of rotator cuff repair surgery is pain control, and although function can be improved, patients need to be appropriately counseled that even though the shoulder will be significantly better after surgery, it will likely still not be normal. Patients may be unable to return to their previous line of work or sport if they are heavy laborers or overhead or throwing athletes. Surgical outcomes tend to be better in patients who are younger than 65, nonsmokers, and are free of other medical comorbidities such as diabetes and osteoporosis.[143–148] Worker compensation patients demonstrate worse outcomes.[149] Managing patient expectations in terms of length of time to recovery is also important because full recovery may take up to 1 year. Physical therapy is recommended to aid in this recovery, and patients who are unwilling or noncompliant in following postoperative instructions should be counseled to avoid surgical management.

Rotator cuff–specific factors to be considered include tear size, chronicity, and status of the rotator cuff musculature. Large, retracted tears that are associated with significant atrophy of the involved muscles have a worse prognosis with surgical intervention.[143,148,150] For acute traumatic tears, early surgical management should be considered, providing the absence of muscle atrophy and retraction.[143,151]

Surgical repair of the torn rotator cuff can be performed through an arthroscopic, mini-open, or an open approach. The repair involves securing the torn edge of the rotator cuff back down to its native insertion on the proximal humerus. The tendon can be reattached using suture anchors or through transosseous tunnels with heavy suture material. Tears can be repaired either through a formal open incision, a smaller mini-open approach, or an all-arthroscopic technique. Over the past decade, the latter technique has significantly increased in popularity. At the time of surgery, concomitant pathology of the long head of biceps, labrum, and acromioclavicular joint can be treated if warranted. At present, evidence is lacking for routine subacromial decompression or removal of bone from the undersurface of the anterolateral acromion.[152] Furthermore, the type of surgical repair that has the best outcome has yet to be elucidated, with all approaches and techniques providing significant improvement in disease-specific and health-related quality-of-life outcome measures.[153–155]

Postoperative Management

A patient who has undergone rotator cuff repair surgery is managed initially with a period of sling and swath immobilization, followed by a progressive regimen of ROM, strength, and functional exercises.[155–157] The length of time patients are immobilized varies, but for most, it is 4 to 6 weeks, depending on the specific surgical procedure and patient adherence.[156] Most postoperative protocols are divided into phases with specific goals and treatment interventions for each phase. Phases generally coincide with tissue-healing timeframes: phase I extends from 0 to 4 to 6 weeks after surgery (depending on the immobilization period); phase II from 4 to 6 weeks to 12 weeks; and phase III from 12 to 24 weeks. Primary goals of phase I are to encourage optimum healing of the repaired rotator cuff and decrease pain and inflammation. Clinicians must be mindful of the equally important secondary goals during this initial phase, which are protecting glenohumeral joint ROM, scapulothoracic stabilization, kinetic chain exercises, and general health and wellness. Patients should be considered ready to progress to phase II of their rehabilitation if pain is significantly reduced at rest, there has been no disruption to normal tissue healing, and the patient has been able to manage the exercises within phase I. The goals of phase II, following rotator cuff repair, include increasing glenohumeral joint ROM and shoulder girdle strength, while still protecting the healing rotator cuff tissue. During this stage of recovery, patients should begin to resume normal ADL and should be able to engage in increased integration of the whole kinetic chain. Clinicians must remain careful not to disrupt the repair through aggressive stretching or loading of the healing tissue during phase II. By the end of phase II, it is expected that most patients will be able to achieve approximately 120° of active shoulder elevation in either flexion or scaption and about 40° of lateral (external) rotation. Active ROM should be achieved with minimal or no pain and with proper scapulohumeral rhythm.

The primary goals of phase III are directed primarily toward improving strength and endurance of the shoulder girdle. Full, functional ROM of the glenohumeral joint and the entire upper-extremity kinetic chain is also expected by the end of this stage of rehabilitation. The majority of patients achieve these goals and are able to use their affected arm in most to all ADL by 6 months' time. The decision regarding when to return to heavy work and/or overhead sporting activities is generally at the discretion of the surgeon and physical therapist; however, most patients resume these high-demand activities sometime between 6 and 9 months.

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CHAPTER 8

Shoulder Arthroplasty

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INTRODUCTION

According to the American Academy of Orthopaedic Surgeons, approximately 4 million people in the United States seek medical care each year for shoulder problems. These conditions range from injuries, such as dislocations and fractures, to chronic debilitating diseases, such as arthritis. Shoulder arthroplasty has been available to patients with severe, chronic, debilitating shoulder pathology since the early 1960s (Figure 8-1).[1–3] Like other joint replacement surgeries, it is designed to remove diseased portions of bone and joint and replace them with a prosthesis, thereby reducing the friction caused by disease and improving range of motion (ROM), eliminating the associated pain.

PREVALENCE OF SHOULDER ARTHROPLASTY

The prevalence of total shoulder arthroplasty in the United States is less than that of total hip or knee arthroplasty but more than total elbow replacements. Shoulder arthroplasty has become the treatment of choice for many patients with a glenohumeral articular disease. The frequency of shoulder arthroplasties has increased substantially, from approximately 10,000 in 1990 to 20,000 in 2000.[4] From 1990 to 1993, fewer than 5000 total shoulder arthroplasties were performed each year in this country; in comparison, combined hip and knee replacement operations exceeded 500,000 per year. According to an orthopedic news source, the prevalence of total shoulder arthroplasty in 1998 was 15,266, of which 8556 were hemiarthroplasties and 6710 were total shoulder athroplasties.[5] As the average age of the population increases and the outcome of shoulder arthroplasty continues to improve, the prevalence of this procedure will continue to rise.

ANATOMY AND JOINT DESIGN

Because of its anatomy and design, the glenohumeral joint does not lend itself to mechanical overload and cartilaginous breakdown. This non–weight-bearing joint is a ball and socket joint. The top of the humerus widens and forms the humeral head, which fits into a shallow socket of the scapula (glenoid fossa). During motion the humeral head moves upon the glenoid fossa, providing a wide ROM. Because of the fit of these two bones, very little surface area of the bones is actually in contact at any given moment, which may contribute to site-specific cartilage breakdown. The stability of the glenohumeral joint relies primarily on the joint capsule and the rotator cuff musculature. The tendons of the rotator cuff help create dynamic stability to the glenohumeral joint by connecting the humerus to the scapula. The primary force producer of the glenohumeral joint is the large deltoid muscle, which creates a force couple with the rotator cuff to allow normal kinematics to occur.

A layer of articular cartilage covers the head of the humerus and the surface of the glenoid fossa; in a healthy joint, this cartilaginous layer protects the bones against friction during movement. In addition, the glenoid labrum surrounds the periphery of the glenoid fossa and provides a slightly deeper socket for the humeral head to rest in. The joint capsule is lined with a synovial membrane, which provides synovial fluid to help reduce friction. In a normal glenohumeral joint, these parts work together to provide stability during a wide ROM. When any of these joint surfaces become diseased, the normally smooth surface becomes rough, causing friction and pain (Figure 8-2). Arthritis leads to the breakdown of cartilage, allowing the bones to rub against each other. When this happens, scar tissue and bone spurs may also develop, leading to further pain and stiffness.

INDICATIONS FOR SHOULDER ARTHROPLASTY

Many different conditions can affect the glenohumeral joint and lead to a discussion about total shoulder replacement. (A hemiarthroplasty replaces the humeral head only, whereas a total shoulder arthroplasty involves replacing the glenoid fossa as well.) Some of these

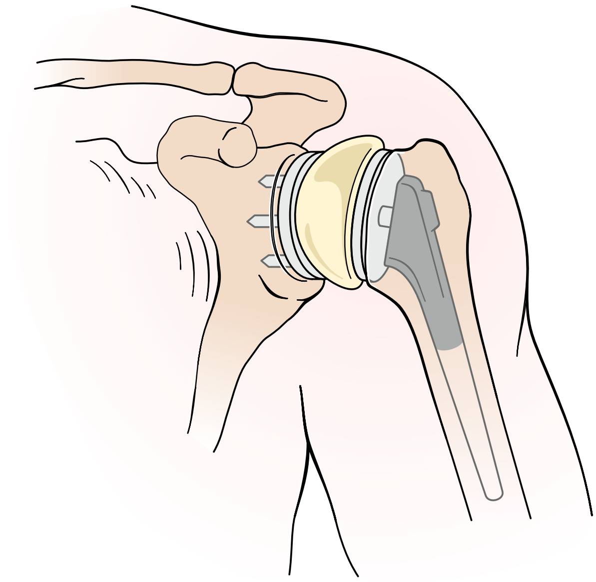

Figure 8-1 Components of a total shoulder arthroplasty.

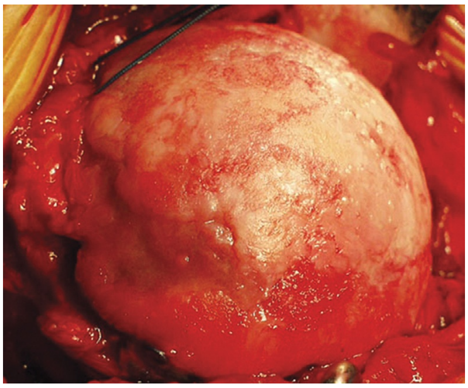

Figure 8-2 Humeral head with severe degenerative disease.

conditions include rheumatoid arthritis, osteoarthritis, rotator cuff arthropathy, avascular necrosis, fractures of the shoulder region, and a failed shoulder prosthesis (from a previous joint replacement).[6–9] Some proximal humeral fractures (e.g., displaced anatomical neck fractures, four-part fractures, fracture-dislocations, and head-splitting fractures) are also indications for shoulder replacement (Figure 8-3).[10–13]

Deterioration of the glenohumeral joint after capsulorrhaphy for recurrent instability is more common in athletes; this condition arises more commonly after surgical procedures such as Bristow, Magnuson-Stack, and Putti-Platt and less commonly after the Bankart and capsular shift procedures.

Pain relief is the primary indication for total shoulder arthroplasty. Most surgeons are cautious about recommending shoulder replacement for improved ROM and function. However, the most common reason for

Figure 8-3 Proximal humeral fracture requiring shoulder arthroplasty.

Figure 8-4 Inferior glenohumeral dislocation.

total shoulder arthroplasty is arthritis. Because arthritis causes the cartilage to break down, it can cause a number of associated problems, such as scar formation and osteophytes. Posttraumatic arthritis is a form of osteoarthritis that develops after an injury, such as a fracture or dislocation of the shoulder (Figure 8-4). Arthritis also can develop after a rotator cuff tear. Rheumatoid arthritis is a systemic inflammatory condition of the joint lining that can affect people of any age and that usually affects multiple joints on both sides of the body.

EVALUATION OF THE ARTHRITIC SHOULDER

History

Total shoulder arthroplasty has become the treatment of choice for most glenohumeral arthritides. A systematic, reproducible approach to each patient ensures comprehensive information gathering and therefore the proper diagnosis and choice of surgical procedure. Initially the patient's age, hand dominance, occupation, athletic activities, medical history, and family history are recorded.

Hand dominance is important to treatment recommendations because the dominant arm of an active individual provides a different therapeutic challenge than the nondominant shoulder of a sedentary individual. Most arthritic shoulders and rotator cuff tears affect the dominant shoulder.[14] Although a patient's age is not necessarily diagnostic, full-thickness rotator cuff tears are found almost exclusively in patients more than 50 years of age. A complete general medical history should be obtained, with special attention given to any systemic or rheumatological disorders. In addition, a family history of generalized ligamentous laxity is important in the treatment of an arthritic patient with multidirectional shoulder instability. A previous incident of trauma can be the precursor of an arthritic shoulder; this can be an important piece of information in presurgical planning.

The patient's chief shoulder complaint usually consists of some element of pain, weakness, or loss of motion. Documenting the pattern of these symptoms is important, most notably the duration, severity, provocation, and location. Shoulder arthritis can arise from a traumatic event, but it often is atraumatic in origin, arising from repetitive loading. Severe night pain and pain at rest are common complaints in patients with mechanical shoulder pathology and usually are due to glenohumeral arthritis; however, infection and tumor must be ruled out.

Weakness of the affected shoulder is a frequent complaint, but splinting from pain commonly contributes to this lack of strength. Patients with concomitant large rotator cuff tears often report weakness or fatigue with overhead use, but they also can have surprisingly good motion and function once the arthritic shoulder has been replaced. Patients also may complain of "crackling" in their shoulders with motion; this can be due to a variety of disorders, including full-thickness tears (which produce crepitus when the greater tuberosity comes into contact with the undersurface of the acromion), glenohumeral osteoarthritis, and acromioclavicular (AC) arthritis. However, when the "crackling" is accompanied by severe pain on movement, glenohumeral osteoarthritis needs to be ruled out.

Physical Examination

Physical examination of the arthritic shoulder should proceed in an organized, reproducible manner. The examination consists of five basic parts: inspection, palpation, ROM, strength testing, and provocative tests. The shoulder often is the site of referred pain, commonly from the cervical spine. Therefore the cervical spine must be thoroughly examined for any coexisting or referred pathology. Often a patient with shoulder arthritis has some degree of cervical spine arthritis and some degree of referred pain to the shoulder. The cervical spine is brought through an ROM, including flexion, extension, and lateral rotation. Pain and crepitation with motion should be noted. Spurling's test, which is effective in distinguishing cervical spine radiculopathy from intrinsic shoulder arthritis, is performed with gentle cervical extension and rotation toward the affected shoulder with axial compression; a positive test result is indicated by posterior shoulder pain and radiculopathy. The deep tendon reflexes should be assessed bilaterally, and a thorough dermatomal sensory and motor evaluation should be performed.

Inspection (Observation)

The patient should be examined with both shoulders exposed, which allows access anteriorly and posteriorly. A thorough inspection of the shoulder should note any muscular atrophy, hypertrophy, asymmetry, or deformity, as well as bony prominences. An arthritic shoulder sometimes presents as an extremely large mass on inspection. The examination may reveal a prominent scapular spine resulting from spinati atrophy; this often indicates a long-standing rotator cuff tear but may also be present with suprascapular nerve entrapment. The shoulder is evaluated for any deformity of the biceps muscle because the long head of the biceps tendon often is ruptured in patients with rotator cuff disease and severe glenohumeral arthritis. Bony prominences and the contour of the shoulder also are noted because a patient with glenohumeral arthritis loses normal contour and shows squaring and anterior fullness.

Palpation

Palpation around the shoulder joint should be done systematically so that each muscle, joint, and bony prominence is evaluated. The sternoclavicular joint; the clavicle; the AC joint; the anterior, lateral, and posterior acromion; the anterior and posterior joint lines; and the biceps tendon each should be tested for discrete tenderness. Localized tenderness over the AC joint often is overlooked as a source of symptoms and may be implicated in rotator cuff pathology or degenerative joint disease. Pain often is elicited by palpation of the greater tuberosity with rotator cuff pathology, but it also may be due to glenohumeral arthritis, and the finding should be correlated with rotational radiographs of the humerus (Figure 8-5). Tenderness in the bicipital groove may be present with involvement of the biceps tendon and can be implicated in both rotator cuff pathology and glenohumeral instability. Anterior and posterior joint line tenderness may be present with glenohumeral instability, whereas posterior joint line tenderness commonly is noted in patients with glenohumeral arthritis.

Range of Motion

The general motion of the shoulder complex is observed for glenohumeral rhythm, scapulothoracic motion, and overall synchrony of motion. The American Shoulder and Elbow Surgeons professional organization currently recommends that four functionally necessary arcs of motion be measured both actively and passively: total elevation, lateral rotation at neutral abduction, lateral rotation

Figure 8-5 Radiograph confirming the diagnosis of severe glenohumeral arthritis.

at 90° abduction, and medial rotation.[15,16] Patients with limited motion in a single or multiple planes must be examined carefully to differentiate the possible etiologies, such as a rotator cuff tear, adhesive capsulitis, and glenohumeral arthritis.

In this patient population, total elevation, including both glenohumeral and scapulothoracic motion, may be more reproducibly measured than attempting to isolate glenohumeral motion and is more functionally relevant. However, both measurements are important for determining which joint is contributing to a functional loss. Passive elevation is more accurately measured in the supine position, whereas active elevation is measured erect. Lateral rotation is tested with the arm at the side, the patient supine (to eliminate trunk rotation), and with the arm at 90° abduction.

Functional rotation ranges of motion are also documented. Functional medial rotation is measured according to the highest vertebral level an upright patient can reach with the thumb. Apley's scratch test evaluates the available functional lateral rotation ROM.

Strength Testing

The strength and integrity of joints, muscles, and tendons are important aspects of surgical planning. Manual strength testing may be difficult to assess quantitatively because of coexistent pain. Crepitation with motion from the glenohumeral joint, subacromial space, or scapulothoracic joint should be noted. Manual muscle testing of the supraspinatus is performed with the arm in 90° forward elevation and 20° medial rotation with the elbows extended (i.e., the empty can test). Manual muscle testing in lateral rotation is performed with the elbow flexed and the arm at the side to prevent deltoid contribution; weakness in this position is a common finding with a tear involving the infraspinatus tendon. Weakness in lateral rotation at neutral may indicate a long-standing rotator cuff tear,[17] and with concomitant weakness in shoulder abduction it shows a statistically significant correlation with the size of the tear.[18]

Manual muscle testing of the shoulder in lateral rotation above 45° abduction is performed primarily to assess the teres minor. Patients with large or massive tears involving the infraspinatus often are unable to maintain the arm in lateral rotation at neutral and when the arm is abducted. The lateral rotation lag sign is designed to test the integrity of the supraspinatus and infraspinatus tendons; it is performed by passively flexing the elbow to 90° and the shoulder to 20° elevation and near maximum lateral rotation. The patient then is asked to maintain this position while the clinician releases the wrist; a positive result is a lag or angular drop.[19] The drop sign is designed to assess infraspinatus function; for this test, the patient is seated with his or her back to the clinician, who holds the affected arm at 90° elevation and maximum lateral rotation with the elbow at 90° flexion. The patient is asked to maintain this position while the physician supports the elbow and releases the wrist; the result is positive if a lag occurs.

Patients with subscapularis tears have an increased passive lateral rotation ROM and weakness of medial rotation. The competence of the subscapularis muscle can be assessed using the lift-off test, which has been shown to be both sensitive and specific for a tear in the subscapularis tendon. Originally the lift-off test was determined to be positive (i.e., poor subscapularis function) when a patient could not lift the hand posteriorly off the lumbar region; however, the test was modified to achieve even greater sensitivity for subscapularis tears.[20] In the modified lift-off test, the examiner places the arm in maximum medial rotation by passively lifting the patient's arm posteriorly off the lumbar region; the test result is positive (poor subscapularis function) if the patient is unable to maintain that position and the arm falls or "springs back" onto the back.

The internal (medial) rotation lag sign is used to assess subscapularis competency. It is similar to the modified lift-off test except that the examiner releases the wrist at maximum internal (medial) rotation, maintains support of the elbow, and then measures the lag between the maximum internal (medial) rotation position and the lag position (Figure 8-6).

The abdominal compression test (Figure 8-7) also is used to assess subscapularis weakness or deficiency, especially when passive medial rotation is difficult to perform during the lift-off test or internal rotation lag sign. A positive abdominal compression test is indicated by an inability to keep the palm of the hand compressed against the abdomen while bringing the elbow anterior to the scapular plane. A side-to-side difference between the affected and unaffected shoulders during the abdominal compression test may indicate subtle subscapularis weakness or a tear and warrants further investigation. Medial rotation strength and integrity must be taken into consideration during surgery for proper intraoperative muscle balancing.[21]

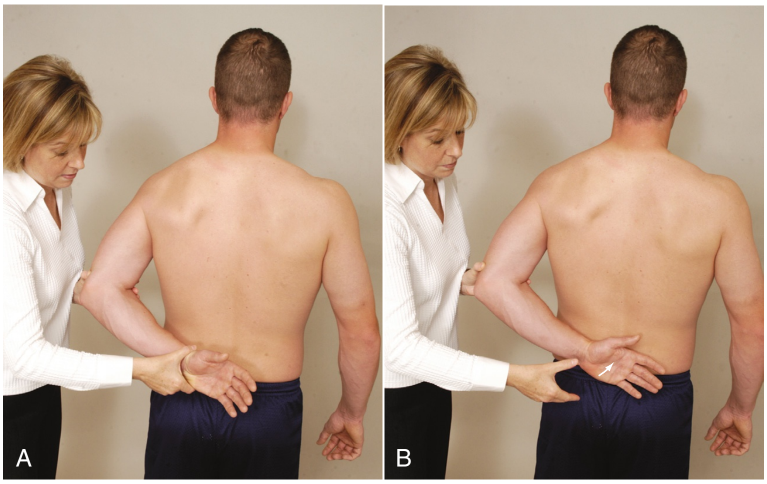

Figure 8-6 Internal (medial) rotation lag sign (also called the subscapularis spring back or lag test). **A**, Start position. **B**, Patient is unable to hold the start position and hand springs back toward the lower back. (From Magee DJ: *Orthopedic physical assessment*, ed 6, p 339, St Louis, 2014, Elsevier/Saunders.)

Figure 8-7 Abdominal compression test. (From Magee DJ: *Orthopedic physical assessment*, ed 6, p 334, St Louis, 2014, Elsevier/Saunders.)

To ensure maximum shoulder function after arthroplasty, a thorough assessment of shoulder motion and strength must include evaluation of the scapular muscle stabilizers. Some of the key muscles tested before shoulder arthroplasty that may affect the final outcome include the serratus anterior, trapezius, and rhomboids. Weakness in the serratus anterior muscle can cause scapular winging and disruption of the synchronous glenohumeral motion. Scapular winging, which can be detected by having the patient flex to 90° or do a wall push up, is commonly the result of a palsy of the long thoracic nerve. Trapezius muscle function is tested with a shoulder shrug, and weakness or atrophy of this muscle may be due to a palsy of the spinal accessory nerve. The motor strength of the rhomboid muscles is tested with the patient prone, shoulder abducted 90°, and arms extended.

When assessing function in shoulder arthritis and arthroplasty patients, the clinician should be aware of the possible existence of contractures as well as generalized ligamentous laxity. In addition, identification of a pattern of instability or contracture is a key component in planning for a shoulder arthroplasty.[16,22] The presence of glenohumeral instability affects the surgical procedure. For example, if excessive posterior laxity and anterior contracture are factors, a posterior capsulorrhaphy and anterior capsular releases can be performed. Conversely, if the patient has anterior laxity, precise soft tissue balancing and prosthetic positioning are needed to prevent functional deficits.

DIAGNOSTIC MODALITIES

Imaging studies, such as radiographs, computed tomography (CT), arthrography, and magnetic resonance imaging (MRI), are used to confirm and further define the pathological process. Early in the development of glenohumeral arthrosis, subtle radiographic findings may suggest the diagnosis when the symptoms are mild. Later in the progression of joint destruction, imaging studies are crucial to the planning of prosthetic arthroplasty. Subsequent to arthroplasty, imaging studies can identify coexisting or potential problems that might correlate with the clinical outcome.

Plain Radiographs

Plain radiographs or x-rays are part of a comprehensive history and physical examination for a patient with shoulder arthritis. All patients should have anteroposterior

(AP) views in the scapular plane in neutral, medial, and lateral rotation, a lateral view in the scapular plane, and an axillary view. Although these radiographs usually are normal in early rotator cuff disease or glenohumeral instability, they are essential to the diagnosis of glenohumeral arthritis, tumors, fractures, or dislocations. Cervical spine films should be ordered for patients with concomitant neck and shoulder pain.

With more advanced glenohumeral arthritis and rotator cuff disease, AP radiographs may demonstrate degenerative changes of the greater tuberosity, AC joint, or anterior acromion, as well as the glenohumeral joint. Radiographic criteria for evidence of a rotator cuff tear, such as the acromiohumeral interval, can also be assessed on the AP view; a decrease of more than 7 mm is considered abnormal and suggestive of chronic cuff pathology.[23] Rotational AP views are valuable for detecting calcium deposits in the rotator cuff. The lateral rotation AP view shows the greater tuberosity in profile, which may reveal sclerosis, cysts, or excrescences. These tests are included for a complete evaluation of the surgical candidate and to allow the surgeon to visualize any other pathology or conditions that may be encountered before the start of the surgical procedure.

Axillary radiographs are useful for demonstrating glenoid degenerative changes or fractures associated with instability, as well as posterior dislocations that may be overlooked on routine AP views. Special views to visualize the AC joint often are obtained using an underpenetrated, or soft tissue, technique in the AP and cephalic tilt AP views. These views often demonstrate osteolysis of the distal clavicle, arthritic changes, and inferior osteophyte formation, as well as fractures. Although patients have shoulder arthroplasty primarily for pain relief, the arthritic changes on radiographs and degree of degeneration can be key factors in the decision on whether to have surgery. Changes visualized on the radiographs can also assist the clinical evaluation in determining the source of pain if no degenerative changes appear on the radiographs.

Key Factors That Determine the Need for Arthroplasty

- Pain
- Arthritic changes on radiographs (decreased joint space, decreased acromiohumeral interval, degenerative changes to greater tuberosity, acromion, osteophytes, subchondral sclerosis, bone deformation)[24,25]
- Degree of degeneration

Arthrography

Long considered the gold standard for imaging full-thickness rotator cuff tears, the arthrogram is an invasive procedure with significant risks, such as infection, allergic reaction, radiation exposure, and pain. Double contrast arthrography is inexpensive and easily performed; however, it has a limited ability to diagnose partial cuff tears accurately and provide information about their size and location. Despite this, arthrography remains an excellent modality for diagnosing full-thickness rotator cuff tears that may accompany glenohumeral arthrosis (Figure 8-8).

Figure 8-8 Infiltration of dye into the subscapularis fossa indicates a possible rotator cuff tear.

Computed Tomography

When an arthritic patient is suspected of having glenohumeral instability, CT images can be obtained to delineate bony and soft tissue intra-articular pathology. CT images also can be useful in a standardized technique to determine the humeral torsion angle and landmarks that can be used during surgery.[26,27] Three-dimensional CT scans (Figure 8-9) can further delineate complex proximal humeral fractures and assist planning for a hemiarthroplasty.[28,29] Hernigou et al.[26] found that retroversion with CT is more accurate than palpating the epicondylar axis or using the forearm as a goniometer during surgery. CT is useful for measuring the amount of rotation of the humerus with a malunited fracture or severe arthritic deformity.

Magnetic Resonance Imaging

MRI is an excellent, noninvasive, radiation-sparing modality that is useful in the diagnosis of rotator cuff pathology and glenohumeral instability but is not always necessary for patients with shoulder arthritis. Soini et al.[30] demonstrated that in severely destroyed rheumatoid shoulders scheduled for arthroplasty, MRI soft tissue findings were not consistent with tissue changes at the time of surgery. The integrity of tendons could not readily be elucidated with MRI because of an inflammatory process and scarred

Figure 8-9 CT scan showing signs of glenohumeral arthritis, which is important in preoperative planning.

Figure 8-11 MRI demonstrating humeral head migration superiorly with rotator cuff arthropathy.

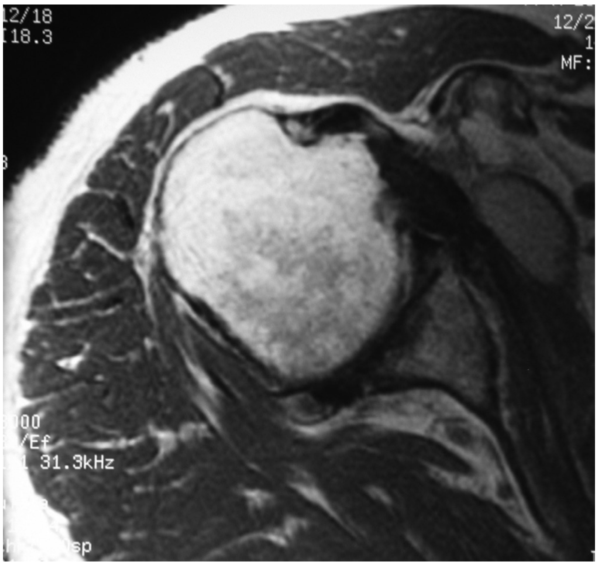

Figure 8-10 MRI scan showing increased signal in the glenoid and humeral head in addition to joint space narrowing; these findings indicate severe degenerative disease.

tissues. Often, during surgery, the changes were difficult to categorize. MRI scans of severely destroyed rheumatoid shoulders before arthroplasty were shown to be of only minor importance. However, this may not be true for patients with osteoarthritis of the glenohumeral joint (Figures 8-10 and 8-11).

Other Modalities

When systemic disorders are suspected, routine blood tests to evaluate for rheumatological disorders, infection, and tumors should be considered. A complete blood count with differential, sedimentation rate, C-reactive protein level, chemistry profile, latex fixation test, serum protein electrophoresis, and an acid phosphatase level should be obtained as part of preoperative testing (Table 8-1). This panel allows the surgeon to rule out any other significant factors and pathology and provides a baseline for comparison after surgery, if necessary.

YOUNG SHOULDER ARTHROPLASTY PATIENTS

Shoulder arthroplasty is becoming more common in young patients. Although trauma to the glenohumeral joint is associated with a dislocation episode, little is known about the incidence of shoulder arthroplasty in patients with shoulder instability. The long time span between dislocation and the development of arthritis makes determining any cause and effect relationship difficult.

Severe damage to the humeral head cartilage can occur after a dislocation. Taylor and Arciero[31] reported on 63 young patients under 24 years of age with first-time, traumatic anterior shoulder dislocations who were evaluated arthroscopically within 10 days of dislocation. Fifty-seven patients had Hill-Sachs lesions. However, osteochondral lesions of the humeral head were identified in 34 patients, and chondral lesions were noted in an additional 24, providing evidence that humeral head damage occurs. Hovelius et al.[32] followed 245 patients 10 years after primary anterior dislocations of the shoulder. The patients' ages at the time of dislocation ranged from 12 to 40 years. Radiographs taken on 185 shoulders at the time of primary dislocation demonstrated a Hill-Sachs lesion in 99 shoulders. This finding was associated with a significantly worse prognosis with regard to recurrent instability but had no effect on the development of arthritis. Radiographs made for 208 shoulders at the 10-year follow-up examination were evaluated for

TABLE 8-1

Preoperative Laboratory Tests with Normal Ranges

CBC with Differential and PLT		Complete Metabolic Panel	
WBC	3.8-10.83/μL	Glucose	65-139 mg/dL
RBC	3.80-5.106/μL	Sodium	135-146 mmol/L
Hemoglobin	11.7-15.5 g/dL	Potassium	3.5-5.3 mmol/L
Hematocrit	35%-45%	Chloride	98-110 mmol/L
MCV	80-100 fL	Carbon dioxide	21-33 mmol/L
MCH	27-33 pg	Urea nitrogen	7-25 mg/dL
MCHC	32-36 g/dL	Creatinine	0.5-1.2 mg/dL
RDW	11%-15%	BUN/creatinine ratio	6-25
PLT	140-4003/μL	Calcium	8.5-10.4 mg/dL
MPV	7.5-11.5 fL	Protein, total	6.0-8.3 g/dL
Total neutrophils, %	38%-80%	Albumin	3.5-4.9 g/dL
Total lymphocytes, %	15%-49%	Globulin, calculated	2.2-4.2 g/dL
Monocytes, %	0%-13%	A/G ratio	0.8-2
Eosinophils, %	0%-8%	Bilirubin, total	0.2-1.3 mg/dL
Basophils, %	0%-2%	Alkaline phosphatase	20-125 units/L
Neutrophils, absolute	1500-7800 cells/μL	AST	2-35 units/L
Lymphocytes, absolute	850-3900/μL	ALT	2-40 units/L
Monocytes, absolute	200-950 cells/μL		
Eosinophils, absolute	15-550 cells/μL		
Basophils, absolute	0-200 cells/μL		

ALT, Alanine aminotransferase; *AST,* aspartate aminotransferase; *BUN,* blood urea nitrogen; *CBC,* complete blood count; *fL,* fluid liter; *MCH,* mean corpuscular hemoglobin; *MCHC,* mean corpuscular hemoglobin concentration; *MCV,* mean corpuscular volume; *MPV,* mean platelet volume; *PLT,* platelet count; *RBC,* red blood cell; *RDW,* red cell distribution width; *WBC,* white blood cell.

posttraumatic dislocation arthritis. Twenty-three shoulders (11%) had mild arthritis, and 18 (9%) had moderate or severe arthritis. No further instability was reported in some of the shoulders that had arthritis.

The most defining study to date was published by Marx et al.,[33] who used a case-control study to examine whether shoulder dislocation was associated with the development of arthrosis. Patients with osteoarthrosis who had undergone hemiarthroplasty or total shoulder arthroplasty were asked whether they had ever sustained a shoulder dislocation. Ninety-one patients who had undergone shoulder arthroplasty and 282 control subjects responded. The authors concluded that the risk of developing severe arthrosis of the shoulder is 10 to 20 times greater for individuals who have had a dislocation of the shoulder. It is evident that some degree of chondral injury and hemarthrosis may be associated with a shoulder dislocation and is not beneficial to the glenohumeral joint. Whether the hemarthrosis predisposes this patient population to arthritis and an eventual shoulder arthroplasty remains unclear.

One thing that is clear is the effect of overconstraining the glenohumeral joint during surgery for shoulder instability. Sperling et al.[34] reported on the intermediate to long-term results of shoulder arthroplasty performed to treat osteoarthritis after instability surgery in 33 patients with glenohumeral arthritis. The mean age at the time of the shoulder arthroplasty was 46 years. Twenty-one patients who had a total shoulder arthroplasty and 10 who had a hemiarthroplasty were followed for a minimum of 2 years. Shoulder arthroplasty was associated with significant pain relief and significant improvement in lateral rotation and active abduction. Interestingly no significant difference was seen between the hemiarthroplasty group and the total shoulder arthroplasty group with regard to postoperative lateral rotation, active abduction, or pain. This study suggests that shoulder arthroplasty for the treatment of osteoarthritis of the glenohumeral joint after instability surgery in a group of young patients provides pain relief and improved motion. However, it may be associated with a higher incidence of revision surgery. Three patients in the hemiarthroplasty group and eight patients in the total shoulder arthroplasty group underwent revision surgery. In contrast, this was not the case in a series of 22 shoulder arthroplasties followed up over an average of 6 years. Burroughs et al.[35] reported no evidence of accelerated deterioration of shoulder function, and only two patients underwent revision surgery.

Given this conflict in the literature, little information is available to guide clinical decision making with regard to shoulder arthroplasty after instability surgery. The management of these patients requires a thorough preoperative evaluation and a careful review of diagnostic modalities. The severity of glenoid wear should be assessed using CT scans to determine the need for glenoid bone grafting. In addition, the surgeon should obtain the previous operative reports to determine the type of stabilization performed; this allows the surgeon to recognize frequently distorted anatomy and facilitates safe, effective soft tissue releases. The decision to proceed with a shoulder arthroplasty in a patient who has had a previous stabilization procedure should be made with caution. The surgeon should be prepared to address associated soft tissue contracture and potential bone deficiency.

Shoulder arthroplasty in the young patient for osteoarthritis of the glenohumeral joint after instability surgery provides satisfactory pain relief and improvement in motion but may be associated with a high rate of revision surgery and unsatisfactory results as a result of implant failure, instability, and painful glenoid arthritis.[35]

PREOPERATIVE REHABILITATION

Accepting differences in expectations, patient goals, physician goals, and physical therapist goals is essential. The physical therapist ideally should meet with the patient and the patient's caregiver before the surgery. At this time the rehabilitation team can set reasonable goals for both early and eventual outcomes (Table 8-2). At this point, when the patients are not under the influence of pain medications, feeling ill, or experiencing pain, they can better

TABLE 8-2

Rehabilitation Guidelines after Shoulder Arthroplasty

Immediate Postoperative Phase (Days 0 to 7)	
Goals	Patient understands surgery, precautions, and pain-relief methods Protect the surgery Improve ROM at shoulder and maintain ROM of proximal and distal joints Improve scapular stability
PROM	Pendulums, CPM (facility dependent) PROM flexion and lateral rotation (if allowed) by therapist
AAROM	Pulleys for sagittal plane flexion and scaption Cane exercises for flexion and lateral rotation
AROM	AROM of hand, wrist, elbow, scapula, and cervical spine Gripping
Patient education	Sling use (full time for community activities, as needed at home) or as instructed by physician Arm can be used for light, waist-level activity Patient understands that activity should not increase pain level drastically Patient should ice shoulder to reduce pain Patient should sleep sitting up (in chair)
Precautions	Do not force lateral rotation No medial rotation isometrics, isokinetics (no medial rotation muscle activation) Exercises should not cause severe pain Watch for and minimize muscle guarding
Early Postoperative Phase (Weeks 1 to 6)	
Goals	Continue to restore motion, scapular stability Begin light rotator cuff activation Continue pain modulation
PROM	Pendulums for warm-up and pain modulation
AAROM	Pulleys for sagittal plane flexion, medial rotation, and scaption Cane exercises for flexion, horizontal adduction and abduction, and punches
AROM	Continue cervical, scapular, elbow, wrist, and hand motion Continue gripping Begin submaximum isometrics for external rotators (i.e., pushing against towel or ball on wall) Begin submaximum isometrics for deltoid if no pain is present Begin upper body exercises at 4 weeks (shoulder flexion below 80°) Begin serratus strengthening if done below 90° shoulder flexion (punches, dynamic hugs, scaption)
Patient education	No heavy pushing, pulling, or lifting for 6 weeks No weight bearing for 6 months
Precautions	Patient feels fine, but healing is not complete. *Protect the joint.*
Intermediate Postoperative Phase (Weeks 6 to 12)	
Goals	Ensure stable prosthesis and joint stability ROM goals, active and passive Minimal pain Begin improving strength and endurance Improve function

(Continued)

TABLE 8-2

Rehabilitation Guidelines after Shoulder Arthroplasty—cont'd

Intermediate Postoperative Phase (Weeks 6 to 12) *cont'd*	
PROM	Mobilizations as needed Posterior capsule stretching/sleeper stretch
AAROM	As needed (cane, pulleys) Posterior capsule stretching/sleeper stretch
AROM	Muscle activation exercises: • Dusting • Active lateral rotation/medial rotation within pain-free motion Open chain stability exercises: • Flexbar 0°, then 45°, and finally 90°, as tolerated • Body Blade at 0° • Rhythmic stabilizations (at 8 to 10 weeks, higher level patients) Closed chain stability exercises: • Ball stabilization • Perturbation training with therapist • PWB through shoulder Strengthening exercises: • Medial rotation/lateral rotation pain free (e.g., with Thera-Band) • Side-lying lateral rotation with weight • Lateral rotation at 45° with elbow supported (8-9 weeks) • Prone extension • Prone horizontal abduction • Biceps and triceps curls • Anterior deltoid strengthening • Strengthening of all scapular muscles (e.g., punches, lower and middle traps, rows, extensions with Thera-Band)
Patient education	No heavy lifting No sports
Precautions	Do not initiate base strengthening until pain is minimal
Return to Activity Phase (Weeks 12 to 24)	
Goals	Full ROM in all planes Strength 80% or greater of uninvolved side Improved endurance and neuromuscular control Patient confident in shoulder Full return to activity
PROM/AAROM	PROM as needed
AROM	Strength testing: • Isokinetic Continue activation, stability, and strength exercises; progress as tolerated: • Lateral rotation 90/90 • Overhead wall taps • Light ball toss; chest pass, overhead • Plyometrics up to 90/90 position Functional exercises: • Thera-Band golf/tennis • Golf chipping and putting • Breast stroke
Patient education	Patient very familiar with HEP to maximize strength and ROM gains
Precautions	Avoid increasing inflammation Do not strengthen in abduction Do not strengthen with high weights; use low weights and medium repetitions Use caution with overhead activity until all pain has resolved If pain requires use of treatments, reduce exercise or intensity of ADLs

AAROM, active-assisted range of motion; *ADLs,* activities of daily living; *AROM,* active range of motion; *CPM,* continuous passive motion; *HEP,* home exercise program; *PROM,* Passive range of motion; *PWB,* partial weight bearing; *ROM,* range of motion.

comprehend the rehabilitation program, precautions, and expectations. This allows the patient and physical therapist to establish a relationship that will foster better cooperation during the sometimes painful and frustrating rehabilitation process. After the educational process, the preoperative assessment should include the available ROM, strength, and the presence of visible atrophy, sensation, pain, and functional evaluation.

The educational process is essential for informing the patient of the risks and complications of surgery and anesthesia.[36] These risks for shoulder replacement surgery include injury to nerves and blood vessels, stiffness or instability of the shoulder joint, loosening of the prosthetic parts (requiring additional surgery), tearing of a rotator cuff tendon, and fracture of the humerus. In addition, total shoulder replacement carries with it the normal risks of any elective surgery, such as complications from anesthesia, excessive bleeding, infection, blood clots, and ultimately death.[37–39] The surgical procedure and the components of shoulder arthroplasty also are explained.

The educational process is essential for informing the patient of the restriction on the use of the shoulder and the impact on activities of daily living for at least the next 6 weeks. Because the subscapularis muscle is cut to expose the shoulder joint, no active medial rotation motion is permitted. For the patient, this means no violent squeezing, hugging, or slamming of car doors. However, the patient should be encouraged to use the upper extremity for light functional activities such as eating, brushing the teeth, or putting on a hat; the patient should avoid lifting anything heavier than 0.91 kg (2 lb). The patient also should be instructed to avoid immobilization.

The importance of a postoperative rehabilitation program is reviewed, and the importance of the first 6 weeks of rehabilitation also is emphasized. The immediate and early program is geared toward regaining the motion achieved at the time of surgery. The patient needs to appreciate that this time frame is the most important period of the rehabilitation process, a period in which significant changes and gains in ROM and maintaining optimum neuromuscular function can be accomplished. The home environment is discussed to determine whether the patient is one of approximately 50% of patients who require home nursing assistance, home physical therapy, or, infrequently, discharge to an extended care facility after hospitalization. In addition, special considerations need to be identified for patients who require assistive devices for ambulation, the visually impaired, or those who use wheelchairs.

Patient and family education is essential for achieving consistently good outcomes after shoulder arthroplasty. It is essential that family members be made aware of restrictions on activity and the importance of frequent home exercise. The patient and caregivers must understand that being active, but using only the arms at the side, does not replace stretching overhead to prevent contracture or stiffness. The recommendation for long-term active ROM exercise is 5 times a day for the first 6 weeks, 2 times a day for the next 6 weeks or until goals are achieved, then once a day for a lifetime. Patients who are involved and enthusiastic about their management tend to recover and regain shoulder function rapidly.[40]

Clinical Note

The patient must take an active role because the patient is the most valuable member of the multidisciplinary team.

Risks and Complications of Arthroplasty[37–39]

- Injuries to nerves and blood vessels
- Glenohumeral joint stiffness
- Glenohumeral or scapular instability
- Loosening of prosthesis
- Additional surgery
- Rotator cuff tear
- Humeral fracture
- Ectopic ossification
- Excessive bleeding
- Infection
- Blood clots
- Death

Although age is not a limiting factor, the patient must be able to cooperate in a postoperative rehabilitation program, which is crucial to the success of the surgery.[40] Patients are expected to obtain medical clearance from their primary care physician with regard to any systemic medical conditions, along with preoperative testing (e.g., laboratory tests, electrocardiography [ECG]). Patients should discontinue nonsteroidal anti-inflammatory medications or aspirin-containing medications 7 to 10 days before the scheduled surgery to minimize postoperative bleeding and complications with coagulation. For total shoulder arthroplasty, 1 unit of autologous blood may be donated in anticipation of the surgery, which can result in a blood loss of 1 to 2 units, especially in complicated cases.

The clinical examination includes shoulder ROM and strength measurements. Limited abduction and lateral rotation movements and decreased rotator cuff muscle strength are common.[40–42] Preoperative motions that should be measured include forward elevation, lateral rotation in a supine position, and medial rotation with the hand reaching up the back. Deficits of active versus passive motion also provide information about the function of the rotator cuff, and these data are supplemented with manual muscle testing of abduction and lateral rotation strength against resistance.

A demonstration of therapeutic exercises by a physical therapist can be part of the preparation for the surgical procedure. Preoperative demonstration of therapeutic exercises in the rehabilitation program is associated with better patient understanding and a more rapid progression after surgery.[40] Unfortunately, most insurance companies do not provide for preoperative therapy or a preoperative home health assessment, despite their intuitive benefits.

HEMIARTHROPLASTY VERSUS TOTAL SHOULDER ARTHROPLASTY

Arthroplasty is the most definitive method for managing the pain associated with severe glenohumeral arthritis. Since 1893 when the first shoulder replacement was performed, shoulder arthroplasty has continued to improve with advances in component materials, instrumentation, and surgical techniques.[1] Good candidates for shoulder arthroplasty are older patients who will place minimal demands on the shoulder and have no rotator cuff damage. A total shoulder arthroplasty involves the replacement of both sides of the glenohumeral joint (the humerus and glenoid).[22] A hemiarthroplasty replaces the humeral head only and is the treatment of choice when replacement of the glenoid is not advised.[43] The decision is made based on the nature and degree of the patient's arthritis. Individuals who should not have shoulder arthroplasty at all are contact athletes and heavy laborers. They are not good candidates for shoulder replacement because of the long-term demands they place on the shoulder. In addition, total shoulder arthroplasty is not always appropriate for patients with large, inoperable rotator cuff tears. Because the rotator cuff muscles hold the humeral head tightly to the glenoid, these massive tears would allow excessive movement of the humeral component, leading to abnormal loading and loosening of the glenoid component.[44] Hemiarthroplasty is recommended for patients with isolated humeral head arthritis and normal glenoid cartilage. Patients with poor glenoid bone quality need to consider a hemiarthroplasty. Patients with an active infection in the shoulder joint should not have shoulder arthroplasty.[45]

Patients Unsuitable for Shoulder Arthroplasty

- Athletes in contact sports
- Heavy laborers
- Patients with large, inoperable rotator cuff tears
- Patients with an active infection

Another subset of patients in whom primary hemiarthroplasty is indicated are patients with proximal humeral fractures. Hemiarthroplasty has become the gold standard for the treatment of such fractures when the humeral head is deemed to be nonviable or not amenable to reconstruction with internal fixation techniques.[46] The current indications for primary hemiarthroplasty include a displaced and translated four-part fracture, with or without associated dislocation of the humeral head, and a head-splitting fracture with involvement of more than 40% of the articular surface.[46] Outcomes with this procedure have been mixed. The shoulder usually is free of pain, but the overall functional result at 1 year varies in terms of ROM, function, and power. A better functional outcome can be anticipated for a younger individual than for elderly patients.[47] However, total shoulder arthroplasty should be considered in these cases; Carroll et al.[48] reported on long-term follow-up of 16 patients after conversion of painful hemiarthroplasty to total shoulder arthroplasty. The authors concluded that revision of a failed hemiarthroplasty to a total shoulder arthroplasty is a salvage procedure, and the results are inferior to those of primary total shoulder arthroplasty. Recently, in patients with rotator cuff arthropathy, a "reverse" shoulder prosthesis was attempted. The premise is that a "reverse" shoulder prosthesis resists glenohumeral subluxation and potential component loosening by preventing superior displacement of the humeral head (Figure 8-12).[49–53] Modern implant designs emphasize a medial center of rotation to recruit more deltoid muscle fibers which aid with active elevation.[54] In a 10-year follow-up study performed by Guery et al.,[55] survival rates for the prosthesis were 91% and 84% among 80 patients with mostly massive rotator cuff tears. However, the authors also found that the protheses were prone to functional deterioration over time; a statistic they attributed to aseptic glenoid loosening.

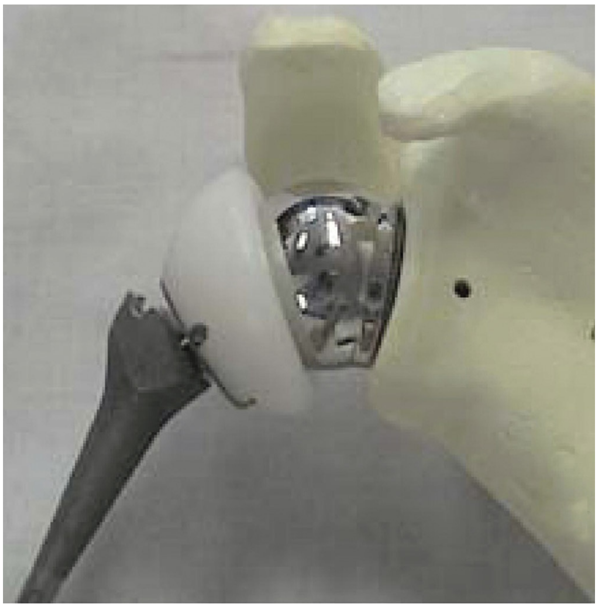

Figure 8-12 Example of a reverse total shoulder replacement prosthesis. (From Harman M, Frankle M, Vasey M et al: Initial glenoid component fixation in "reverse" total shoulder arthroplasty: a biomechanical evaluation, *J Shoulder Elbow Surg* 14[1 suppl S]:162S-167S, 2005.)

FACTORS ASSOCIATED WITH SUCCESSFUL OUTCOMES

The outcome of a shoulder arthroplasty can never be predicted with certainty; however, a number of preoperative, intrasurgical, and rehabilitative factors are associated with a successful outcome.[16] Factors observed at the time of surgery, such as the quality of the cartilage, bone, and rotator cuff, are a recognized influence on the result of arthroplasty, but preoperative information also is associated with greater improvement in patient-assessed function after shoulder arthroplasty (Table 8-3). Preoperative decision making by patients and their surgeons can influence the final outcome. It was found that higher surgeon and hospital case volumes led to improved perioperative metrics with all shoulder arthroplasty procedures, including reverse total shoulder arthroplasty.[56] The success of shoulder arthroplasty also depends on postoperative rehabilitation.[15,36,40,57,58]

The first preoperative predictor of outcome is the presence of systemic disease. Numerous research studies have demonstrated that patients with Parkinson's disease, systemic lupus erythematosus (SLE), and rheumatoid arthritis have poorer outcomes after shoulder arthroplasty.[59–61] In the case of SLE and rheumatoid arthritis, the soft tissues needed for muscle balancing may be severely compromised. The tissue also may have become friable and thin because of excessive use of steroids.[62] In addition, patients with preexisting diabetes are at a higher risk for perioperative morbidity and mortality following shoulder arthroplasty.[63] The progressive multisystem involvement seen in many diabetic patients may be the cause of the higher perioperative mortality encountered in patients with this condition.

Hettrich et al.[64] examined preoperative factors associated with improvements in shoulder function after hemiarthroplasty in 71 shoulders. These researchers were able to demonstrate that shoulders with rheumatoid arthritis, capsulorrhaphy arthropathy, and rotator cuff tear arthropathy had the least functional improvement, whereas those with osteonecrosis or primary and secondary degenerative joint disease had the greatest improvement. The patient's age and gender did not significantly affect the outcome. Shoulders that had not had previous surgery had greater functional improvement than those that had had previous surgery. These results were supported by Kay and Amstutz,[65] who found better results in patients with osteonecrosis compared with those who had a fracture. Trail and Nuttall[66] found that an intact rotator cuff was associated with a better outcome in patients with rheumatoid arthritis. Levy and Copeland[67] found poorer outcomes in patients with cuff tear arthropathy and posttraumatic arthropathy than in those with other diagnoses. As these studies show, patient selection plays an important role in determining the final outcome. In a recent study, Bot et al.[68] noted that patients with preoperative psychological illness were found to be at an increased risk for perioperative morbidity and posthospitalization care following shoulder arthroplasty.

Preoperative ROM can also have an effect on the postoperative outcome. Iannotti and Norris[69] found that patients with less than 10° of passive lateral rotation before surgery had substantially less improvement in lateral rotation after hemiarthroplasty. Thirteen of 128 shoulders had a repairable full-thickness tear of the supraspinatus tendon, but these tears did not affect the overall outcome, decrease in pain, or patient satisfaction. The authors' experience has been that patients scheduled for shoulder arthroplasty who lack passive medial rotation and have a tight posterior capsule have a very difficult time regaining the motion postoperatively and report poorer outcomes. Surgeons and patients thinking of shoulder arthroplasty need to consider identifying these preoperative factors.

Shoulder arthroplasty is considered among the most technically demanding of the current joint replacement procedures because of the glenohumeral joint's lack of intrinsic stability.[21] The tension of the rotator cuff and glenohumeral capsule must be balanced for mobility and stability. The surgeon is faced with many decisions regarding the choice of implant, implant fixation, soft tissue management, and options for glenoid resurfacing.[70,71] In general, if the precise cause of the arthritic condition has been identified, the choices become more straightforward. For advanced osteoarthritis of the shoulder joint in an older patient with asymmetrical posterior erosion of the glenoid, total shoulder arthroplasty renders the best relief of pain and improvement in motion. Similarly, for

TABLE 8-3

Factors Associated with Successful Outcomes

Stage	Factors
Preoperative	Cartilage quality Bone quality Rotator cuff quality Glenohumeral instability Systemic disease Posterior glenoid wear Heterotropic ossification
Intrasurgical	Soft tissue balancing Prosthetic component positioning Intraoperative fracture Nerve injury Infection Inadequate range of motion
Postoperative	Infection Contracture Lack of range of motion Inadequate strength recovery Prosthetic component loosening

advanced rheumatoid arthritis in a patient with an intact rotator cuff, total shoulder arthroplasty results in the best pain relief. If the rotator cuff is deficient and irreparable, an anatomically sized humeral head replacement is appropriate.

Many types of implants are available. Biomechanical and anatomical studies suggest that a better technical result can be achieved with the most recent implant design, which is able to recreate accurately the proximal anatomy of the humerus.[72,73] Nevertheless, complications are inevitable in shoulder replacement surgery (incidence of approximately 14%). Numerous intraoperative complications have been identified. In order of decreasing frequency, they include instability, rotator cuff tear, ectopic ossification, glenoid component loosening, intraoperative fracture, nerve injury, infection, and humeral component loosening.[16,36,74,75] Successful treatment of these surgical difficulties requires careful identification and surgical planning, and the surgeon must always keep in mind that the etiology often is multifactorial.

OPERATIVE TECHNIQUE FOR TOTAL SHOULDER REPLACEMENT

Patient Positioning

Appropriate patient positioning is essential in total shoulder surgery. The patient is placed in a modified beach chair position with a headrest that allows access to the superior part of the table (Figure 8-13). The head of the table should be raised to approximately 25° to 30° to reduce venous pressure, and an arm board should be used on the side of the table that will be raised and lowered as necessary throughout the procedure. A mild sedative and an interscalene block are administered.[76]

Incision and Exposure

The first step is to demarcate all bony landmarks, including the clavicle, and the coracoid process. The incision will be made from the lateral aspect of the coracoid process at the level of the clavicle and extend in line with the deltoid toward the midaspect of the humeral shaft and along the deltopectoral groove. The subcutaneous flaps are dissected from the deltoid fascia to expose the deltoid and pectoralis major muscles. The skin is retracted with self-retaining retractors, and the deltopectoral interval is developed. The pectoralis major is retracted medially, and the deltoid is retracted laterally. The cephalic vein, which delineates the deltopectoral groove, is retracted either medially or laterally (preferably laterally) because this minimizes bleeding from the deltoid muscle or cephalic vein (Figure 8-14). The upper 1 to 2 cm (0.5 to 1 inch) of the pectoralis major tendon is released. The interval between the deltoid, the coracobrachialis, and the short head of the biceps is developed, and a retractor is placed deep to it. The superior portion of the subacromial space is exposed, and the anterior aspect of the coracoacromial ligament is resected. The subscapularis major tendon is identified, as are both the superior and inferior margins of the muscle. The tendon is divided just medial to the bicipital grove and is removed from the lesser tuberosity. Care is taken to avoid the axillary nerve. The subscapularis muscle is retracted medially, exposing the articular surface of the humeral head. As a unit, the capsule and subscapularis tendon are taken laterally off the lateral aspect of the humerus (Figure 8-15). Care must be taken to avoid the axillary nerve during this approach. Lateral rotation of the humerus at this point is helpful for protecting the axillary nerve.[77]

Figure 8-13 Patient is placed in a modified beach chair position for shoulder arthroplasty.

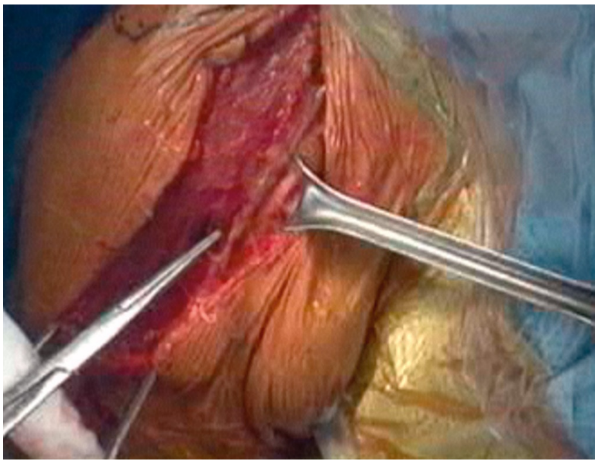

Figure 8-14 Pectoralis major is retracted medially, and the deltoid is retracted laterally. The cephalic vein, which delineates the deltopectoral groove, is retracted to minimize bleeding.

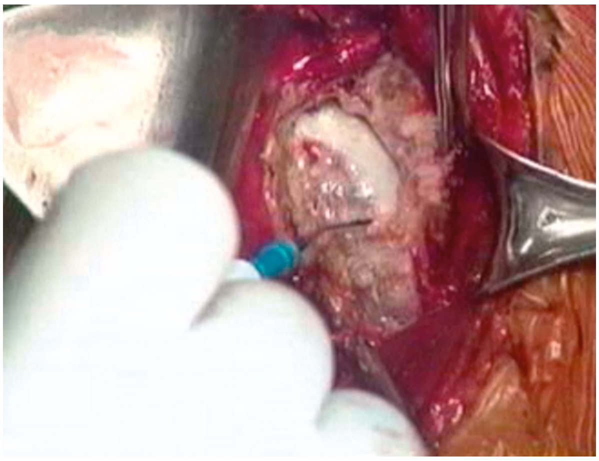

Figure 8-15 Capsule and subscapularis tendon are reflected, and the humeral head is exposed.

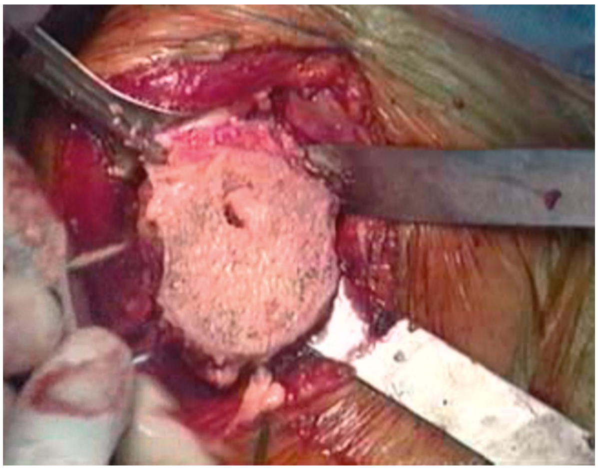

Figure 8-17 Humeral head is removed.

Preparation of the Humerus

Once the subscapularis and capsule have been retracted sufficiently, the arm is placed into extension and lateral rotation to facilitate dislocation of the humeral head. Once the humeral head has been dislocated, arthritic osteophytes are removed so that the anatomical neck of the humerus junction of the articular cartilage and the cortical bone can be identified. An intramedullary reamer (drill) is placed just posterior to the bicipital groove, and a starter hole is made. Sequential reaming then is performed, using a tapered reamer in the intramedullary canal.

The humeral head is then resected with a cutting block technique (Figure 8-16). The cut is made with proper retroversion, which is 20° to 40° retroversion. The resected head is removed (Figure 8-17) and used to size the prosthesis appropriately.[10]

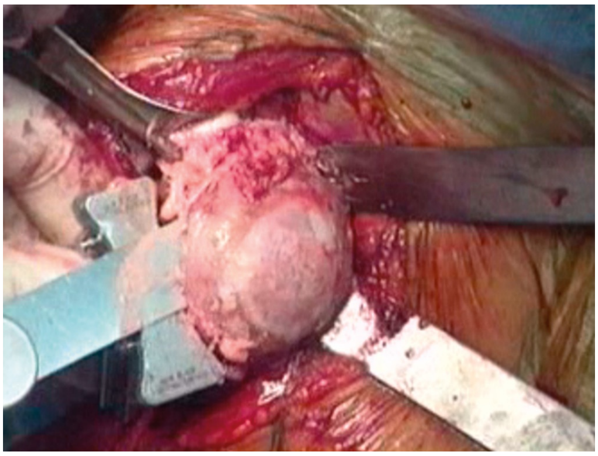

Figure 8-16 Humeral head is resected using a cutting block and oscillating saw.

A provisional humeral stem is used and correlated to the last size of the largest reamer used initially. Retroversion is additionally checked at this point. It is critical that the humeral head be centered relative to the rotator cuff.[43] Many of the current prostheses allow offset head placement; this allows the margin of the humeral head to rest immediately adjacent to the rotator cuff insertion superiorly on the greater tuberosity and slightly overhang the calcar spur medially. ROM could be limited postoperatively if the humeral component is placed too low. If the humeral component is placed too high, the rotator cuff muscles will be under too much tension, and loosening of the glenoid component may occur.[78]

Preparation of the Glenoid

Preparation of the glenoid is essential to a successful outcome in total shoulder arthroplasty. It is essential that the surgeon have a good understanding of the patient's glenoid anatomy before implantation of the component; this involves using either an axillary radiograph of the glenoid or a CT or MRI scan to assess for anterior and posterior wear. It is important to identify the center of the glenoid cavity. Osteophytes that may be obstructing the center of the glenoid need to be removed. If an inadequate or deformed glenoid prevents proper placement of the glenoid component, the surgeon should consider performing a hemiarthroplasty rather than a total shoulder arthroplasty. Any cartilage remaining on the glenoid should be removed, and the glenoid must be sized appropriately.

A centering hole should be made in the glenoid (Figure 8-18). Whether a keeled or pegged glenoid component is used depends on the surgeon's preference.[79] Regardless of the type of component chosen, the appropriate placement for drilling and reaming into the glenoid must be followed. Reaming to the appropriate depth is critical, as is taking care not to remove a

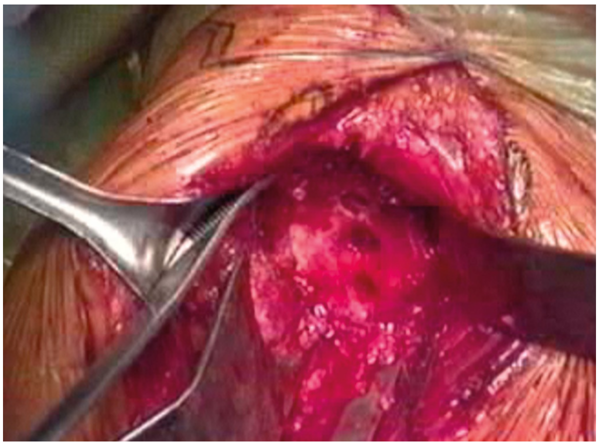

Figure 8-18 Centering and pegged glenoid holes are drilled into the glenoid.

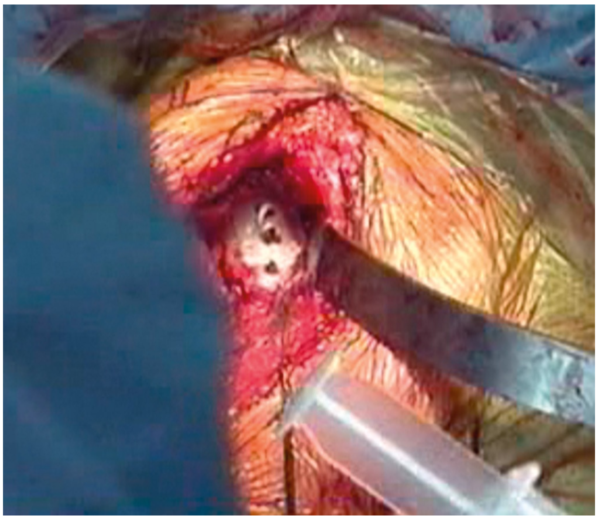

Figure 8-19 Glenoid prosthesis is inserted.

substantial amount of subcortical bone, because this could affect glenoid stability and long-term preservation of the arthroplasty.[80] Reaming is performed by hand and a drill is not used so as to preserve as much subcortical bone as possible. The glenoid then is sized. The optimum glenoid component should not overhang any perimeter of the glenoid.[81]

The humeral head prosthesis is then selected. The prosthesis should be sized according to the removed humeral head. Standard offset heads may be used. The humeral head and its stem as well as the glenoid are then tried together. The joint is reduced and put through a ROM.

Implantation of the Prosthesis

The glenoid component is implanted first. Cement is introduced into the keel or peg holes with a pressurized syringe. The cement is placed, pressurized, and held. The glenoid prosthesis then is inserted and impacted with pressure, which is maintained until the cement has hardened (Figure 8-19). Excess cement is removed.

The humeral component then is prepared for implantation. The surgeon's preference determines whether a press fit or cement is used. Often cement is placed circumferentially around the collar proximally instead of being packed completely into the intramedullary canal.[82]

Various trials should allow for a humeral head that permits 50% of the head to be subluxed posteriorly and inferiorly and to fall back into place once the pressure is released. A prosthetic component that is too large will overstuff the glenohumeral joint and will not allow proper soft tissue balancing.[83]

Before inserting and cementing the final humeral component, the surgeon makes several suture holes through the anterior neck of the proximal humerus. Heavy braided #2 sutures should be passed before cement fixation of the humeral shaft. The suture holes are used to reattach the subscapularis tendon; they often are best placed medially against the neck of the humerus, which effectively lengthens the tendon and thus prevents it from being overtightened.[84] If a cement restrictor plug is used, the proper canal diameter must be determined so that the appropriate-sized cement restrictor can be placed. Before cementing, the intramedullary canal should be thoroughly cleaned and dried. The cement is injected into the humeral canal and finger packed to pressurize it thoroughly. Once the final sizing has been done, the prosthesis is placed and held in position until the cement has dried fully. Bireduction is performed with a modular head. Once the proper sizing has been performed, the humeral taper stem should be thoroughly cleaned and dried. After the proper humeral head has been selected, the humeral head is applied and impacted appropriately (Figure 8-20). The joint then is brought through ROM, and stability is assessed.

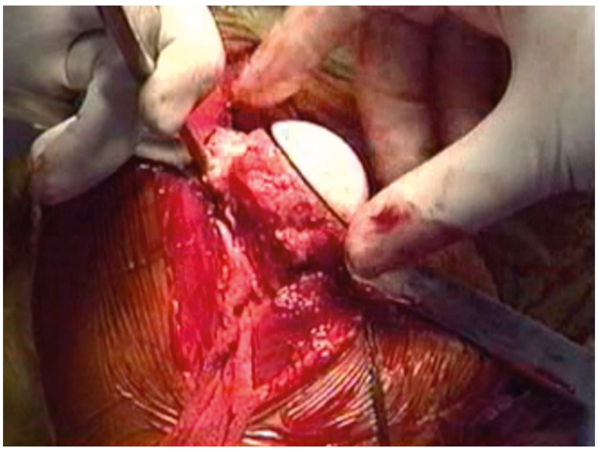

Figure 8-20 Humeral head is replaced.

Closure

The wound is irrigated copiously. Then, beginning inferiorly, the subscapularis is reattached using the sutures that were passed before cementing of the humeral shaft. A Hemovac drain is placed deep to prevent hemarthrosis. Care is taken to avoid all neurovascular structures upon closure. The deltoid and subcutaneous layers are closed, with the subarticular stitches close to the skin (Figure 8-21). Steri-Strips and a sterile dressing are applied. A postoperative radiograph is then taken (Figure 8-22). The patient is kept in a sling and swath until interscalene anesthesia has worn off, and perioperative antibiotics are continued for 24 to 48 hours. Major complications that can arise before discharge include neurovascular injuries and dislocation.

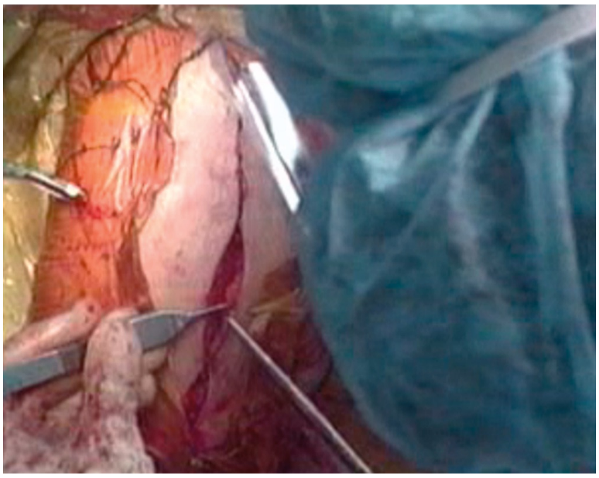

Figure 8-21 Deltoid and subcutaneous layers are closed, with the subarticular stitches close to the skin. Steri-Strips and a sterile dressing are applied.

Figure 8-22 Postoperative radiograph is taken.

READMISSION

Readmission rates for shoulder arthroplasty have not been closely evaluated in very many studies. Fehringer et al.[85] assessed the Veterans Association population for postoperative complications and found that 14-day readmission rates and 30-day mortality rates in total shoulder arthroplasty incidence compared favorably with knee and hip arthroploasty. Mahoney et al.[86] found that the readmission rate for all arthroplasties was 5.9%. For hemiarthroplasty, total shoulder arthroplasty, and reverse total shoulder arthroplasty, 90-day readmission rates were 8.8%, 4.5%, and 6.6%, respectively. Thirty-day readmission rates were significantly more common for hemiarthroplasty and reverse total shoulder arthroplasty.[86]

POSTOPERATIVE REHABILITATION

A successful outcome for total shoulder arthroplasty depends on a well-designed and well-executed physical therapy program. For maximum benefit, the program usually is initiated immediately after surgery and follows a logical pattern of joint mobilization and stretching, followed by muscle strengthening. This design attempts to balance the need to obtain and maintain motion with the need to allow adequate soft tissue healing. Overly protective rehabilitation can result in stiffness, whereas overly aggressive therapy could compromise healing of the subscapularis and rotator cuff musculature and therefore shoulder stability and function.[87]

Immediate Postoperative Phase (Days 0 to 7)

The rehabilitation process should start as soon after surgery as the surgeon allows. Many surgeons begin at least some passive mobility on the same day as surgery, while the patient is still under the influence of a regional nerve block. At the authors' hospital, continuous passive motion is not used for the shoulder; however, some surgeons may prescribe this modality. The patient is seen in the hospital for 2 to 4 days after surgery. Immediate postoperative care involves monitoring pain management and evaluating vital signs and the neurovascular status of the operated arm, as well as caring for the surgical wound site. Pain control is essential during hospitalization. The authors use a regional block to provide effective pain relief for approximately 12 hours. Patients can use an indwelling patient-controlled analgesia (PCA) pump at home for the first 24 hours, but most surgeons want to avoid the extra risk for infection.[88]

After the surgery, the surgeon delineates the extent of soft tissue damage and repair for the physical therapist because these factors determine when exercises can begin. For example, a Z-plasty lengthening, which sometimes is performed on the subscapularis if a long-standing medial rotation contracture is present, may limit the amount of lateral rotation allowed. Also, a massive rotator cuff repair may delay active lateral rotation for up to 8 weeks. Finally, the authors find it extremely useful for the surgeon to inform the physical therapist of the motion achieved at the end of the case.[15,87]

Early mobilization is essential to prevent the formation of adhesions and capsular contracture, especially if a complete capsular release was performed at the time of surgery. Early mobilization exercises (e.g., pendulum exercises) are recommended to relieve pain and prevent adhesion formation. Pendulum exercises produce minimal muscle activity and are considered a safe exercise during this period. The authors have even found that having the patient rest the hand on a physioball and perform pendular exercises makes the patient feel more relaxed and in control (Figure 8-23). Active-assisted shoulder flexion also is initiated at this time in the hospital bed (Figure 8-24). In fact, the electronic bed can be raised or lowered to use the influence of gravity to the patient's benefit. During this period, elbow and wrist ROM and gripping exercises are encouraged.

Milestones the authors look to achieve for discharge and progression to the next rehabilitation phase are (1) review with the patient the surgical procedure, precautions, and expectations during rehabilitation, (2) provide some pain relief, (3) ensure the patient's independence in performing exercises, and (4) accomplish initial ROM goals. With mobilization of the soft tissues, capsular releases, and proper component placement, the hospital therapy goals of 140° of forward elevation and 40° of lateral rotation before discharge can be achieved in many patients.

Immediate Postoperative Phase Milestones

- Review surgical procedure with patient
- Review precautions and expectations during rehabilitation
- Provide pain relief
- Help patient attain independence in performing exercises
- Achieve initial ROM goals (140° forward flexion, 40° lateral rotation)

Early Postoperative Phase (Weeks 1 to 6)

The early postoperative phase focuses on returning motion, restoring scapular stability, and initiating rotator cuff muscle activation. Active-assisted ROM (AAROM) should be initiated, using a pulley for sagittal plane flexion and scapular plane elevation. In addition, a cane, golf club, or umbrella can be used to help the patient regain flexion, abduction, adduction, and lateral rotation at 0°, 45°, and 90° abduction. An early series of exercises the authors prefer is using the cane supine three different ways for a total of 90 repetitions of horizontal adduction, serratus supine, and flexion (Figures 8-25 and 8-26). Unfortunately, the authors have not found a preferred method of AAROM for medial rotation with a cane, nor have they found the towel stretch to be useful for regaining medial rotation ROM. The authors' preferred method is to have the patient use pulleys in the standing position (Figure 8-27) or to perform the sleeper stretch (Figure 8-28) after 6 to 8 weeks of healing.

Joint mobilization and passive ROM can be important adjuncts if ROM difficulties occur. As mentioned, passive motion exercises, such as pendulum exercises, are good for pain relief and preventing the formation of adhesions.

Figure 8-23 Example of the pendulum exercise in the supported position.

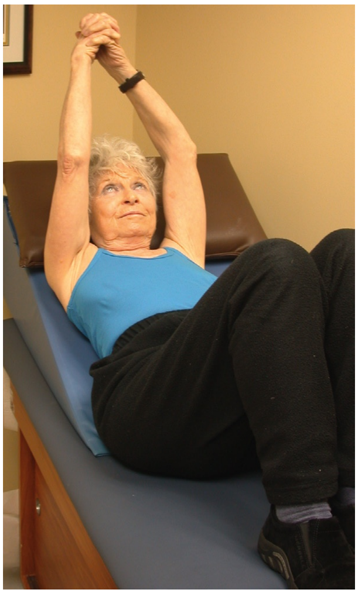

Figure 8-24 Active-assisted shoulder flexion can be done anywhere with the use of the uninvolved upper extremity.

Figure 8-25 Active-assistive shoulder flexion can be accomplished with the use of a cane or stick.

Figure 8-26 Horizontal abduction and adduction can be accomplished with the use of a cane or stick.

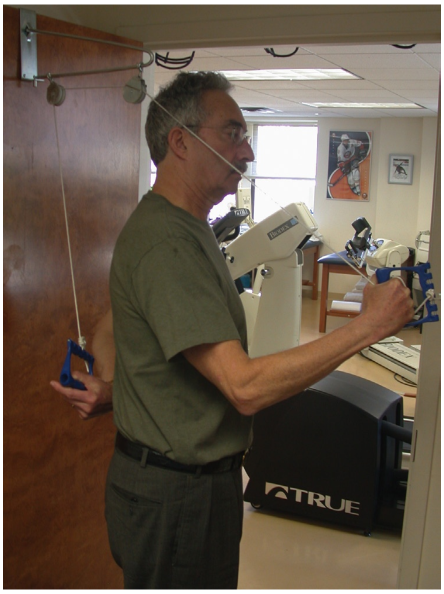

Figure 8-27 Use of pulleys to regain functional medial (internal) rotation ROM.

Figure 8-28 Sleeper stretch.

At this time, the mobility of the sternoclavicular joint, AC joint, and scapulothoracic joint is addressed, and these joints are mobilized if indicated. Once the milestone of mobility of these proximal joints has been reached, manual scapular stabilization is initiated. In the side-lying position, manual resistance can be applied to the scapula to resist elevation, depression, protraction, and retraction. The authors have found that pain can be a limiting factor when starting scapular stabilization and rotator cuff isometrics. However, submaximum pain-free isometrics for lateral rotation can be started as early as 7 days after surgery provided there is no rotator cuff involvement (Figure 8-29).

Isometric deltoid activity may also be initiated during this phase, provided pain is not a limiting factor. An arm upper body ergometer (UBE) using light resistance can be beneficial at this time (4 weeks) to facilitate ROM and initiate active muscular control of the shoulder. The axis of rotation of the UBE should remain below the level of the shoulder joint so that forward flexion is not forced above 80°. The authors also stress the retro component of performing the UBE. By performing retro UBE (going backward), the authors hypothesize, the patient mobilizes the scapula and encourages proper posture.

Early strengthening of the serratus anterior muscle also is encouraged, provided the arm is maintained slightly below 90° of shoulder flexion and movement is pain free. Subsequent atrophy of the serratus anterior muscle, as a result of disuse, may allow the scapula to rest in a downwardly rotated position, causing inferior border

Figure 8-29 Early lateral (external) rotation strengthening.

Figure 8-30 Serratus anterior strengthening in the supine position.

prominence. Decker et al.[89] used electromyography (EMG) to determine which exercises consistently elicited the highest percentage of the maximum voluntary contraction (MVC) of the serratus anterior. They found that the serratus anterior punch out, scaption, dynamic hug, knee push up plus, and push up plus exercises consistently elicited greater than 20% MVC. Most important, they determined that the push up plus and the dynamic hug exercises maintained the greatest percentage of the MVC and maintained the scapula in an upwardly rotated position. Although the early postoperative phase is too early in the rehabilitation process to perform these late exercises, Decker et al.[89] highlighted the serratus anterior punch out as a valuable exercise. Performed in a controlled, supervised setting, this is an excellent choice to initiate early serratus anterior neuromuscular reeducation (Figure 8-30). Progression to the more challenging serratus anterior strengthening exercises can be considered during the intermediate phase of rehabilitation, keeping in mind that the patient is instructed to avoid heavy pushing, pulling, and lifting for the first 6 weeks, and no upper extremity weight-bearing activities are allowed for 3 months.

A delicate balance exists between pushing patients too hard and progressing them as planned. Too often the patient may feel better rather than worse during this early protective phase; therefore the clinician must always respect the laws of tissue healing.

Milestones the authors look to achieve for progression to the next rehabilitation period are (1) toleration of submaximum isometrics of the rotator cuff muscles at 0° abduction; (2) attaining symmetrical mobility of the sternoclavicular, AC, and scapulothoracic joints; and (3) protraction, retraction, elevation, and depression of the scapula against submaximum manual resistance. Adequate passive ROM in flexion, abduction, adduction, and medial rotation at 45° abduction (50% to 75% of the uninvolved side) also should be attained before the patient is progressed.

Early Postoperative Phase Milestones

- Ability to tolerate submaximum rotator cuff isometrics at 0°
- Symmetrical mobility of sternoclavicular, acromioclavicular, and scapulothoracic joints
- Ability to protract, retract, elevate, and depress scapula against submaximum manual resistance
- Adequate passive ROM (50% to 75% of uninvolved side) at 45° abduction

Intermediate Postoperative Phase (Weeks 6 to 12)

During the intermediate postoperative phase of rehabilitation, the patient attempts to regain all available ROM, including passive medial rotation. Initiation of passive lateral rotation ROM at 0°, 45°, and 90° abduction and stretching beyond neutral rotation position are emphasized. Advancement to terminal ranges in all planes of motion is advocated unless otherwise specified by the referring surgeon. Medial rotation submaximum resistive exercise progressions also are initiated during this phase. A traditional rotator cuff isotonic exercise program is started, which includes side-lying lateral rotation, prone extensions, and prone horizontal abduction (which may need to be limited between neutral to the scapular plane position initially, with progression to the coronal plane

Figure 8-31 Rhythmic stabilization in open kinetic chain.

Figure 8-32 Rhythmic stabilization in closed kinetic chain.

as ROM improves). Biceps and triceps curls in the standing position with glenohumeral joint rotation in a neutral resting position are begun. In addition, oscillation exercises with Flexbar or a Body Blade can begin. Rhythmic stabilization in open kinetic chain (Figure 8-31) and closed kinetic chain (Figure 8-32) environments can begin, with caution in shoulder arthroplasty patients who will be placing higher demands on the shoulder.

Intermediate Postoperative Phase Milestones

- Regaining of full ROM
- Rotator cuff isotonic exercise program
- Oscillation exercises
- Open and closed kinetic chain exercises

Return to Activity Phase (Weeks 12 to 24)

The return to activity phase is the stage of rehabilitation in which clinicians begin "to lose" their patients. Often patients lose focus, exhaust insurance coverage, or neglect the importance of fine-tuning the shoulder's function before fully returning to their lifestyle. This phase is designed to prepare patients to return without hesitation to full participation in all activities.

Milestones to complete this phase successfully include (1) acceptable (functional) pain-free ROM and (2) isokinetic or handheld dynamometry strength testing demonstrating less than a 20% deficit in shoulder flexion, abduction, and medial rotation. A deficit greater than 20% may still exist, depending on rotator cuff integrity at the time of surgery. Although not always considered as such, inadequate ROM is a complication that can occur after total shoulder arthroplasty. Acceptable ROM after shoulder arthroplasty has been defined as forward elevation to 140° to 160°, lateral rotation with the arm at the side to 40° to 60°, full extension, and 70° of medial rotation with the arm at 90° in the coronal plane. Inadequate ROM may be due to insufficient soft tissue release or joint overstuffing.

Return to Activity Phase Milestones

- Functional, pain-free ROM
- No more than 20% strength deficit
- Patient has confidence in shoulder

In this phase, the patient continues to work in functional positions, including plyometrics and strengthening at 90° abduction, and gradually returns to full activity, provided the individual is pain free, has nearly full ROM in all planes, confidence in the shoulder, and 85% to 90% of the strength of the opposite side for the motions of internal and lateral rotation. The patient acquires confidence by being able to perform pain-free functional movement in the person's usual activities. In the authors' experience, recreational athletes require 1 to 2 months more to allow the shoulder to accommodate to the sports-specific motion. The authors currently use the American Shoulder and Elbow Surgeons Shoulder Evaluation Form to standardize the documentation of pain, motion, strength, stability, and function. However, enough data have not been gathered to determine a criterion score for return to sports after shoulder arthroplasty. In a review article by Healy et al.,[90] 35 members of the American Shoulder and Elbow Surgeons Society were surveyed about their recommendations for athletics and sports participation for their patients who had shoulder replacement surgery. The surgeons were asked to rate 42 athletic activities as recommended or allowed, allowed with experience, no opinion, and not recommended. The 35 responses were analyzed to obtain a consensus recommendation for each activity. If a valid percentage was not achieved for either a positive or negative recommendation, no conclusion was provided for those activities. These recommendations are presented in Table 8-4.

These rehabilitation guidelines are a continuum of rehabilitation phases based on the effect of surgery on the tissue and surrounding structures. Scientific rationale is applied whenever possible, but as surgical procedures evolve, so must rehabilitation procedures. However, these guidelines are by no means set in stone, nor is every exercise distinct to that phase (see Table 8-2). The goals and exercises must be modified based on the performer, pathology, and performance demands. No exercise prescription should be viewed as protocol, but as a guideline upon which to base rehabilitation.

Key Points in Rehabilitation

- Goals and exercises must be modified based on the individual, pathology, performance demand, and functional outcome the individual hopes to achieve
- No exercise prescription should be viewed as a protocol but instead as a guideline based on individual patient needs and desires for a good functional outcome

OUTCOMES OF SHOULDER ARTHROPLASTY

Diagnosis of the correct indication for shoulder arthroplasty, the preoperative planning, and the postoperative rehabilitation program are essential for a good functional outcome and the keys to physical activity after shoulder arthroplasty. Although the optimum method for measuring the outcome in patients with shoulder arthroplasty has yet to be defined, the ideal assessment should include measures of general health, a shoulder-specific assessment, and an assessment specific to the disease state for which the shoulder arthroplasty was indicated.[91] Each of these levels of sensitivity offers a different perspective on the outcome of shoulder arthroplasty until the ideal universal outcome tool is developed.

Overall, shoulder arthroplasty has been shown to provide predictable pain relief and functional improvement in patients with glenohumeral degenerative arthritis and an intact rotator cuff.[92–94] Norris and Iannotti[95] reported on 133 total shoulder replacements and 43 hemiarthroplasties at an average follow-up of 46 months. They found no differences in postoperative pain, function, American Shoulder and Elbow Surgeons scores, or ROM. Also, no differences were seen between total shoulder arthroplasty and hemiarthroplasty in patients with repairable rotator cuff tears. Total shoulder arthroplasty and hemiarthroplasty for the treatment of primary osteoarthritis result in good or excellent pain relief, improved function, and patient satisfaction in 95% of cases. Boyd et al.[96] compared 64 patients who had hemiarthroplasty with 146 patients who had total shoulder arthroplasty in a retrospective review with a mean follow-up of 44 months. They found similar outcomes in terms of patients' functional improvement. No difference in

TABLE 8-4

Activity after Total Shoulder Arthroplasty: 1999 American Shoulder and Elbow Surgeons Survey

Recommended or Allowed	Allowed with Experience	Not Recommended	No Conclusion
• Cross-country skiing • NordicTrack • Speed walking and jogging • Swimming • Doubles tennis • Low-impact aerobics • Bicycling (road and stationary) • Bowling • Canoeing • Croquet • Shuffleboard • Horseshoes • Dancing (ballroom, square, and jazz)	• Golf • Ice skating • Shooting • Downhill skiing	• Football • Gymnastics • Hockey • Rock climbing	• High-impact aerobics • Baseball, softball • Fencing • Handball • Horseback riding • Lacrosse • Racquetball, squash • Skating (roller, inline) • Rowing • Soccer • Tennis, singles • Volleyball • Weight training

From Healy WL, Iorio R, Lemos MJ: Athletic activity after joint replacement, *Am J Sports Med* 29:377-388, 2001.

overall patient outcomes was observed between hemiarthroplasty and total shoulder arthroplasty.

Secondary to improvements in design and implant survivorship, total shoulder arthroplasty patients strive to return to higher levels of function and sports activity. In a case series conducted in 2010, 100 consecutive patients with unilateral total shoulder arthroplasty were followed for at least 1 year. Of the 55 patients who played sports before shoulder disease, 89% were still playing at a mean follow-up of 2.8 years, whereas more than two thirds (69.4%) reached the same level of intensity as before the shoulder pathology.[97] McCarty et al.[98] exhibited similar findings among 75 patients followed for a minimum of 2 years. Of the 48 patients who stated one of the reasons they opted for surgery was to return to sport, 71% demonstrated an improvement in their ability to play their sport and 50% increased their respective frequency of participation postoperatively (Figure 8-33). Jensen and Rockwood[99] retrospectively evaluated 24 golfers with shoulder replacements and found 23 players (96%) were able to resume playing golf. Three of these patients had bilateral shoulder arthroplasty. Among the 26 shoulder replacements in 24 patients, 6 were hemiarthroplasties and 20 were total shoulder arthroplasties. Of the 18 patients who were able to report a preoperative handicap, an average improvement of five strokes was reported after the operation.

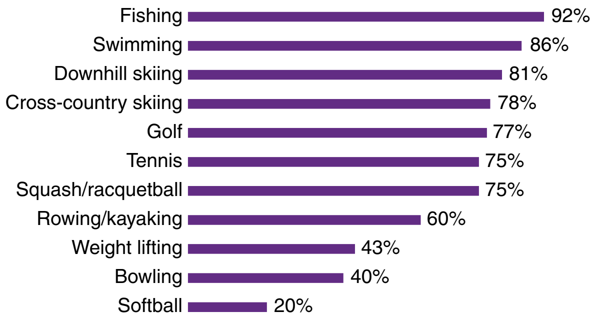

Figure 8-33 Sports participation after shoulder replacement surgery. (From McCarty EC, Marx RG, Maerz D et al: Sports participation after shoulder replacement surgery, *Am J Sports Med* 36(8):1579, 2008.)

SUMMARY

The success of shoulder arthroplasty depends on proper patient selection through a comprehensive history and physical examination, appropriate use of diagnostic modalities, good surgical skill, and effective rehabilitation. A thorough understanding of the pitfalls and complications that can affect patients undergoing shoulder arthroplasty is essential to a successful outcome. Technical advances in shoulder arthroplasty have allowed surgeons the use of this procedure with confidence and have helped optimize results.

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