

The Disabled Throwing Shoulder: Spectrum of Pathology Part III: The SICK Scapula, Scapular Dyskinesis, the Kinetic Chain, and Rehabilitation

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We use the acronym SICK to refer to the findings one sees in this syndrome (Scapular malposition, Inferior medial border prominence, Coracoid pain and malposition, and dysKinesis of scapular movement). This recently recognized overuse muscular fatigue syndrome is yet another cause of shoulder pain in the throwing athlete who presents with dead arm complaints.¹ The hallmark feature of this syndrome is asymmetric malposition of the scapula in the dominant throwing shoulder, which usually appears on examination as if one shoulder is lower than the other. This statically observable position is suggestive of underlying muscle activation alterations that produce altered kinematics of the scapula upon dynamic use. The altered kinematics fall into 3 clinically recognizable patterns of scapular dyskinesia, 2 of which are most commonly associated with labral pathology: type I, inferior medial scapular border prominence, and type II, medial scapular border prominence.

The type III pattern, which is associated with impingement and rotator cuff lesions rather than labral lesions, displays prominence of the superomedial border of the scapula. In the SICK scapula syndrome, scapular asymmetry is measured statically, but actively produces scapular dyskinesia as the shoulder goes through the throwing cycle. The malpositioned dyskinetic scapula, in turn, dynamically produces altered kinematics of the glenohumeral and acromioclavicular joints and the muscles that insert on the scapula.

Because of these complex interrelationships, scapular dyskinesia, including the SICK scapula syndrome, can cause a spectrum of clinical complaints originating from any or all of these anatomic locations.

A thrower with this syndrome presents with an apparent “dropped” scapula in his dominant symptomatic shoulder compared with the contralateral shoulder’s scapular position. In actuality, the scapula initially protracts, rotating about a horizontal axis, with the upper scapula rotating anteroinferiorly. However, the clinical appearance, with the arms relaxed in adduction at the side, is that the involved scapula is lower than the scapula on the uninvolved side (Figs 1 and 2). Viewing from behind, the inferior medial scapular border appears very prominent, with the superior medial border and acromion less prominent. When viewed from the front, this tilting (protraction) of the scapula makes the shoulder appear to be lower than the opposite side. The pectoralis minor tightens as the coracoid tilts inferiorly and shifts laterally away from the midline, and its insertion at the coracoid becomes very tender.

Symptomatic patients with an isolated SICK scapula may complain of anterior shoulder pain, posterosuperior scapular pain, superior shoulder pain, proximal lateral arm pain, or any combination of the above. In addition, posterosuperior scapular pain may radiate into the ipsilateral paraspinal cervical region or the patient may complain of radicular/thoracic outlet type symptoms into the affected arm, forearm, and hand or any combination of the above. The onset of symptoms with the SICK scapula syndrome is almost always insidious. By far, the most common presenting complaint is anterior shoulder pain in the region of the coracoid, which can easily be confused with anterior

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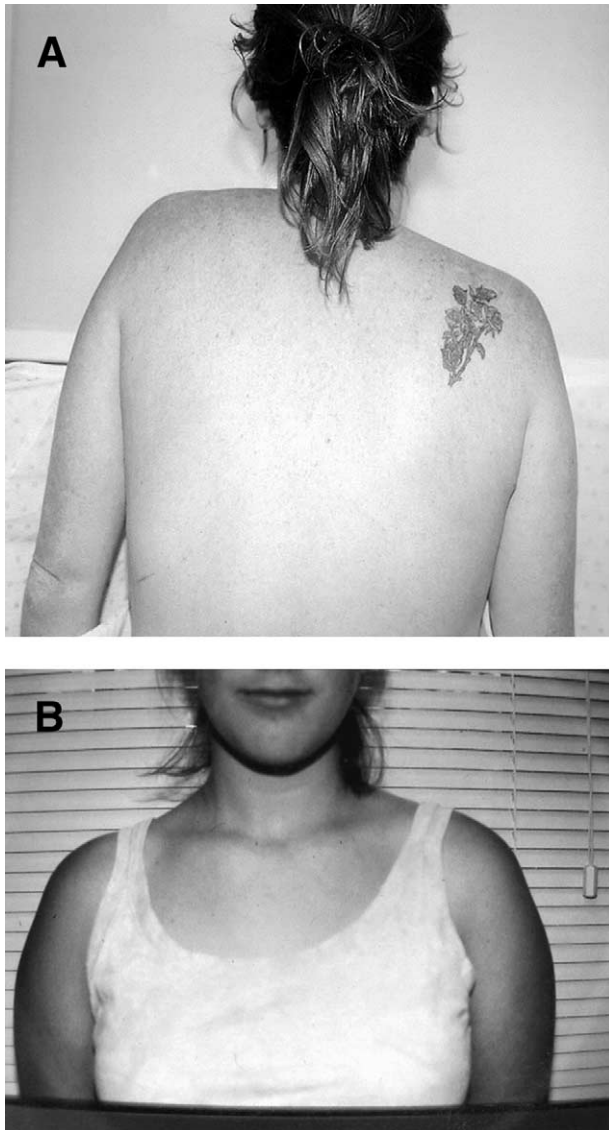


FIGURE 1. (A) A severe SICK right scapula viewed from posterior. The right shoulder appears more than 1.5 inches lower than the left shoulder despite the absence of scoliosis, limb length discrepancy, or pelvic tilt. (B) A SICK right scapula viewed from anterior in a multisport high school overhead athlete.

pain associated with anterior instability if the coracoid is not meticulously examined for tenderness. Postero-superior scapular pain with or without radiation into the paraspinous neck region is next in frequency. Proximal lateral arm pain (subacromial) and superior shoulder pain (acromioclavicular joint) are less frequent, and radicular symptoms (thoracic outlet) are rare.

Of 96 overhead-throwing athletes diagnosed and treated for this isolated syndrome by one of the au-

thors (C.D.M.), 58 were baseball pitchers, 6 were baseball catchers, 20 were tennis players, and 12 were volleyball players. In this series, presenting pain location was as follows: approximately 80% anterior (coracoid) pain, 70% anterior (coracoid) and postero-superior scapular pain, 10% isolated anterior (coracoid) pain, 20% proximal lateral arm (subacromial) pain, 5% acromioclavicular joint pain, and 5% radicular (thoracic outlet) pain radiating into the arm, forearm, and hand.

In throwers presenting with the SICK scapula syndrome, static scapular malposition versus the non-

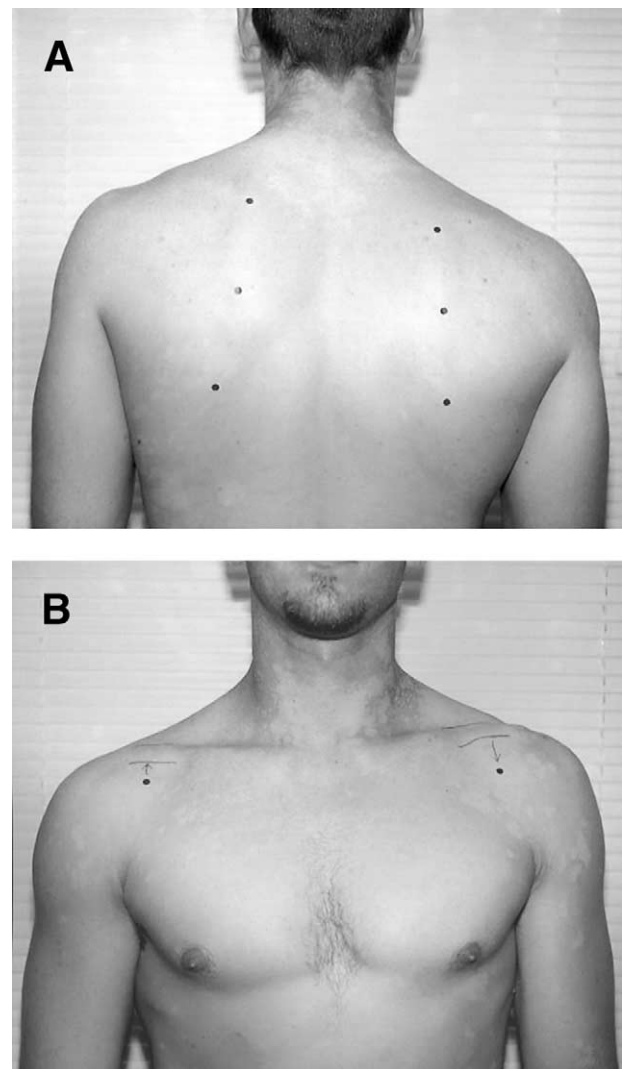


FIGURE 2. (A) A professional right-handed baseball pitcher with a SICK right scapula viewed from posterior. (B) Viewed from the anterior, the apparent inferior position of the lateral clavicle caused by scapular protraction can be seen.

throwing shoulder is objectively measured in 3 categories: (1) *infera*, which is the visual appearance of a dropped scapula due to scapular tilting or protraction; (2) lateral displacement; and (3) abduction. All measurements are made statically with the patients standing erect with arms relaxed in adduction at their side. The measurement of *infera* is the difference in vertical height of the superomedial scapular angle of the dropped scapula in centimeters compared with the contralateral superomedial angle (Figs 3 and 4). This measurement is most accurately performed with a bubble goniometer that uses the same bubble chamber as a carpenter's level. Although the measuring process would appear to quantify linear displacement of the

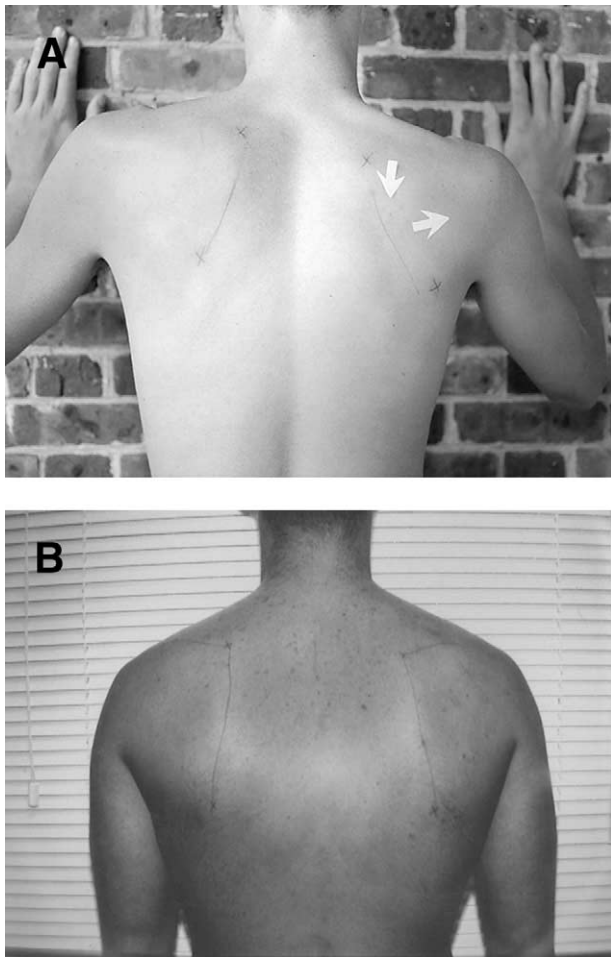


FIGURE 3. Three categories of static scapular malposition seen with the SICK scapula syndrome. (A) All 3 malpositions—*infera*, lateral protraction, and abduction—are illustrated in a high school quarterback's right shoulder. (B) One or any combination of the 3 malpositions may occur, as seen in the right shoulder of a college baseball pitcher with an isolated abduction component of the SICK scapula syndrome.

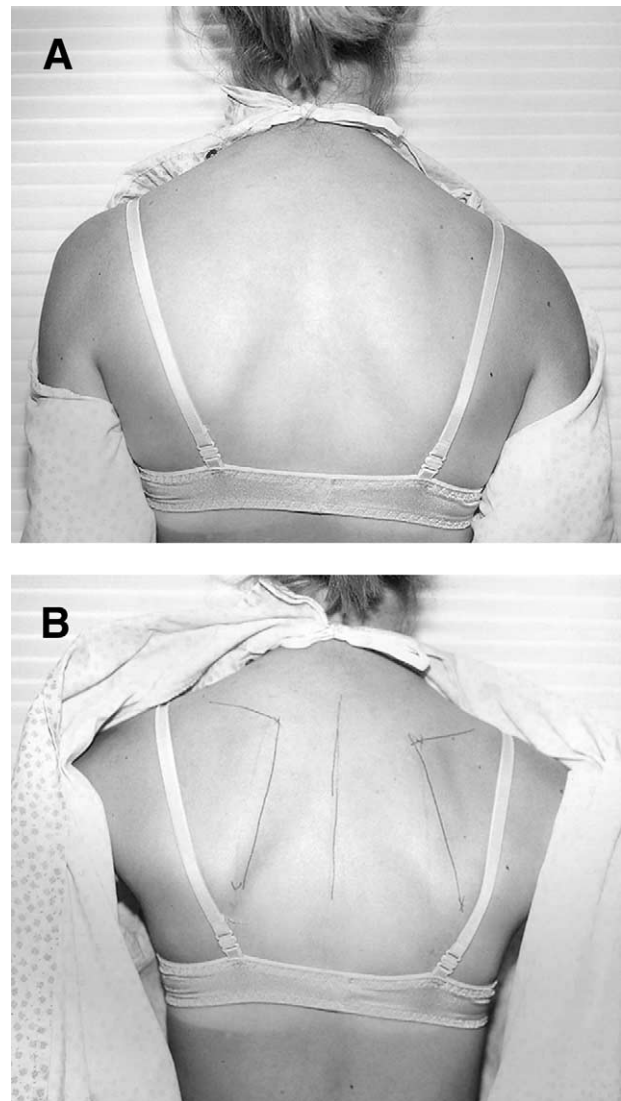


FIGURE 4. (A) A high-level woman tennis player with a SICK right scapula. (B) Note that the severity of the problem is more evident when the scapular margins are outlined.

scapula inferiorly, the malposition is actually a rotational displacement (forward tilting, protraction). Even so, the linear measurements are useful in estimating how severe the dysfunction is and allowing objective measurement of recovery during rehabilitation. Scapular lateral displacement is the difference in centimeters of the superomedial scapular angle from the midline between the SICK and contralateral scapula (Figs 3 and 4). Scapular abduction is the difference in angular degrees of the medial scapular margin from plumb midline between the SICK and contralateral scapula measured with a goniometer (Fig 5).



FIGURE 5. Scapular abduction is measured with a goniometer on the affected medial scapular margin in reference to vertical midline versus the uninvolved medial scapular margin.

Most throwers with the SICK scapula syndrome present with static scapular malposition in all 3 categories, but single or dual combinations of any of the 3 have been seen. Although one of the authors (C.D.M.) has devised a grading system for the severity of scapular malposition based on these measurements, we recognize that the use of superficial landmarks can make these measurements less reliable and less reproducible than we would like. Nonetheless, they give us a qualitative sense of the severity of this dyskinetic syndrome and a method of measuring progress with a rehabilitation program (Table 1).

On physical examination, patients with anterior shoulder complaints and a SICK scapula are found to have marked coracoid tenderness, more medial than lateral on the coracoid tip, at the point of insertion of the pectoralis minor tendon. Throwers with anterior coracoid pain can easily be confused with throwers with other causes of anterior shoulder pain, such as anterior instability or SLAP lesions, unless the coracoid is meticulously examined. SICK scapula patients with coracoid pain usually lack full forward flexion on the affected side and have accentuated coracoid pain with attempts at gaining maximum passive forward flexion by the examiner (Fig 6). With manual repositioning of the scapula in retraction and posterior tilt by the examiner (the scapular retraction test), full forward flexion without coracoid pain is usually attained, which is diagnostic of this syndrome (Fig 6B).

The pathophysiology behind why the dropped SICK scapula presents with coracoid pain is explained by the coracoid static malposition and the dyskinesia that it produces. Because of the ellipsoid shape of the thorax, as the scapula tilts anteriorly, protracts, and abducts, it tends to ride “up and over” the top of the thorax.^{2,3} As it does, the coracoid tilts anteroinferiorly and moves laterally from the midline. The pectoralis minor and short head of the biceps become adaptively tight. This tightness increases the scapular malposition, lowers the leading edge of the acromion, and decreases the ability to achieve full forward flexion of the arm. Impingement-like symptoms result from the anteroinferior angulation of the acromion because of scapular protraction.

Overhead athletes with SICK scapulas who present with posterosuperior periscapular and lower paracervical pain are usually found to have marked tenderness at the superomedial angle of the affected scapula in the area of insertion of the levator scapulae muscle. As the scapula tilts and rotates laterally, traction on the levator scapulae creates pain and muscle spasm (Fig 7). This can often be relieved on physical examination by correcting the tilted scapula by means of the scapular retraction test.

TABLE 1. SICK Scapula Rating Scale: Static Measurements: 0–20 Points



DATE _____ SPORT POSITION _____
 NAME _____ PRESENTING SX^s _____
 AGE _____

SUBJECTIVE		PAIN		YES	NO	SCORE
	Coracoid			1	0	
	AC Joint			1	0	
	Periscapular			1	0	
	Prox. Lat. Arm			1	0	
	Radicular			1	0	
OBJECTIVE						
	Coracoid			1	0	
	AC Joint			1	0	
	Sup. Med. Scap. Angle			1	0	
	Impingement Test			1	0	
	Scapular Asst. Test			1	0	
	Tos Paresthesias			1	0	
SCAP. MALPOSITION						
		0cm	1cm	2cm	3cm	SCORE
	Infero	0	1	2	3	
	Lateral Protraction	0	1	1	3	
	Abduction	0°	5°	10°	15°	
		0	1	2	3	
TOTAL SCORE						

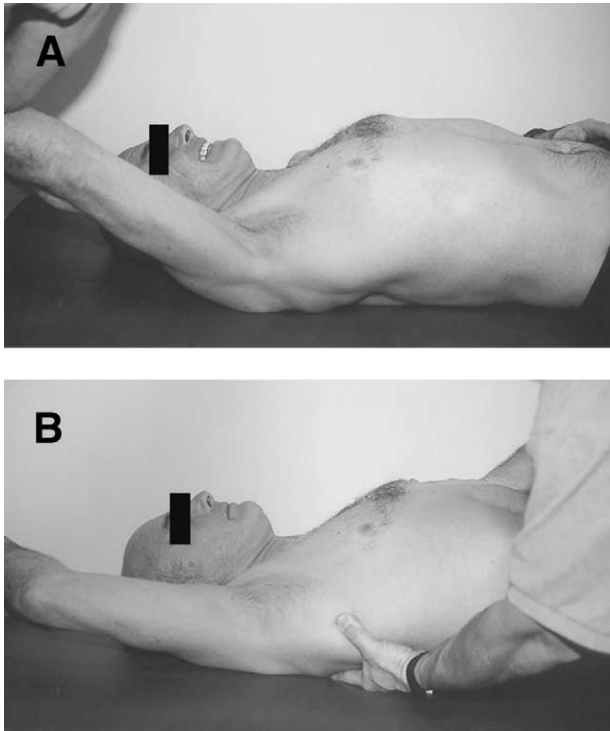


FIGURE 6. The scapular retraction test. (A) SICK scapula patients with infera and abduction components and resultant coracoid pain present with lack of full forward flexion because of pseudotightness of the pectoralis minor caused by coracoid malposition. (B) With manual repositioning of the scapula (retraction) the coracoid pain resolves and full forward flexion is attained.

Subacromial origin pain in the SICK throwing shoulder will present with positive subacromial impingement tests that bring out proximal lateral arm pain. However, the true cause of these findings is a malpositioned dyskinetic acromion resulting from scapular protraction during all phases of the throwing cycle, rather than true mechanical subacromial impingement produced by a type III acromion with an anterior osteophyte. Likewise, acromioclavicular joint pain is caused by a relatively incongruous position of the distal clavicle in reference to the acromion as a result of scapular malposition. As the scapula tilts and protracts, its acromion process moves anterior, decreasing the acromioclavicular angle and increasing compressive stress to the acromioclavicular joint.⁴ The altered kinematics at the acromioclavicular joint (and occasionally the sternoclavicular joint) cause pain with repetitive overhead use.

Finally, the rare thrower with radicular or thoracic outlet symptoms associated with a malpositioned SICK scapula develops these symptoms due to a shift

in position of the clavicle in reference to the upper chest wall, particularly the first rib. As the scapula shifts, the lateral clavicle also drops anteroinferiorly, resulting in a decreased subclavian chest wall space. This space restriction may impinge the brachial plexus as it crosses this zone resulting clinically in the picture of thoracic outlet syndrome.

A 20-point clinical rating scale for the SICK scapula syndrome has been developed by one of the authors (C.D.M.) to statically assess the severity of the syndrome at the time of presentation and to objectively monitor clinical improvement during the treatment phase (Table 1). The rating scale awards points for subjective complaints and objective findings in the categories previously discussed, as well as points for the presence and severity of static scapular malposition in the 3 modes ("apparent" infera, lateral translation, and abduction). A healthy symmetrical asymp-

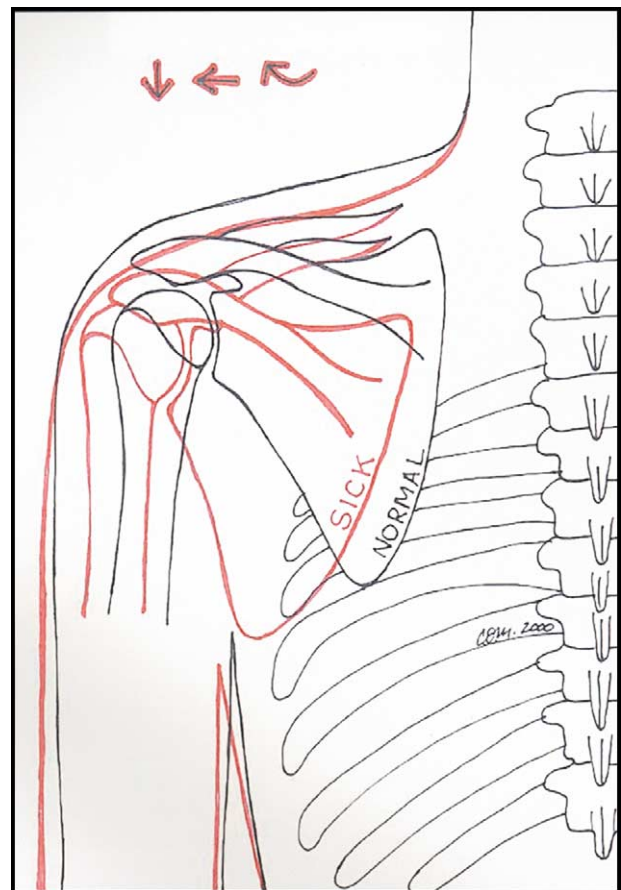


FIGURE 7. Levator scapulae insertional pain caused by a SICK scapula. As the scapula abducts and protracts, the insertion of the levator scapulae at the superomedial angle of the scapula becomes overtensioned and produces a painful tendinopathy in this area.

tomatic scapula receives a score of 0, and the worst SICK malpositioned scapula with all the pathologic clinical components is scored as 20.

In an attempt to determine if components of scapular malposition could be a normal adaptive phenomenon in the throwing athlete, a group of asymptomatic professional baseball pitchers who denied previous shoulder problems were prospectively evaluated for presence or absence of SICK scapulas and scored on the SICK scapula rating scale (Table 1) during spring training and at the end of 2 consecutive pain-free baseball seasons (P. Donley, J. Cooper, personal communication, 2000). Nineteen pitchers meeting these criteria were studied and found to have scores of 0 during the entire testing period (P. Donley, J. Cooper, personal communication, 2000). These findings support the concept that the healthy throwing shoulder exhibits no component of the SICK scapula syndrome (no scapular asymmetry), and that this syndrome is abnormal and predisposes the throwing shoulder to pathologic symptomatology as previously discussed.

Treatment for the SICK scapula syndrome regardless of presenting symptoms or severity of scapular malposition is nonoperative and focused on scapular muscle rehabilitation. Initially, the thrower is restricted from all throwing and begun on a regimented daily strengthening and re-education program for all the scapular stabilizer muscles (P. Donley, J. Cooper, personal communication, 2000). Details of a scapular rehabilitation program are discussed in the section dealing with rehabilitation. During the treatment period, scapular position is monitored on a weekly basis. When the affected scapula is 50% or more improved in position from its initial pathologic position, the thrower is begun on an interval throwing program, if asymptomatic, and continues the scapular program until the scapula is symmetric with the other side (Fig 8). At that time, return to sport and unrestricted throwing is allowed and the thrower is strongly encouraged to maintain an every-other-day scapular muscle-strengthening program to prevent recurrence of the syndrome.

In general, most throwers with symptomatic SICK scapulas present with scores between 10 and 14. Interval throwing usually begins with scores in the 4 to 6 range, and return to sport at the thrower's previous level of performance is attained when the score drops between 0 and 2.

In an adherent patient who commits to doing the rehabilitation exercises 3 times per day, the 50% repositioned scapula can be routinely attained within 2 to 3 weeks. Completion of the interval throwing pro-

gram usually takes 3 to 4 weeks, and complete symmetrical scapular repositioning usually takes 3 months. In general, the anterior tilt (apparent infera) component is the first to resolve, the lateral translation goes away second, and the abduction component (loss of protraction control) is the last and most difficult to resolve. Of the 96 overhead athletes treated for this syndrome and followed up for more than 1 year, all successfully returned to asymptomatic throwing at their preinjury level of performance by 4 months.

Resolution of symptoms during the treatment period was directly proportional to the rehabilitation program's ability to reposition the scapula symmetrical to the other side. In addition, compliance with an every-other-day scapular stabilizer muscle-strengthening program prevented recurrence of the syndrome. Nine of the 96 patients developed recurrence of the syndrome after 3 to 4 months of symptom-free throwing. However, all 9 of these throwers admitted to total nonadherence with the maintenance scapular program for more than 3 months before developing symptoms related to recurrent SICK scapula syndrome.

PRE-SLAP PRODROME AND THE "SHOULDER AT RISK"

On persistent questioning, most throwers with arthroscopically proven posterior type II SLAP lesions and the picture of internal impingement admit to a pre-SLAP prodrome of ill-defined symptoms that they ignored. During the early prodromal phase, the thrower senses tightness in the back of his or her dominant shoulder, oftentimes described as an inability to "get loose." As the player tries to "play through" these prodromal symptoms and continues to throw, the posteroinferior capsular contracture that caused the tight symptomatology gets worse, to a point where posterosuperior discomfort is now present with the sense of tightness or stiffness. As the magnitude of the capsular contracture continues to increase with continued throwing in the face of these prodromal symptoms, the SLAP event will then occur at which time the shoulder develops sudden onset of mechanical symptoms that were absent during the prodrome. Once mechanical symptoms are present, treatment becomes a surgical issue directed toward SLAP repair and correction of the internal rotation deficit as discussed in the section dealing with posterior inferior capsular contracture.

The "shoulder at risk" of developing dead arm complaints is the asymptomatic shoulder that exhibits small to moderate amounts of either a throwing-ac-

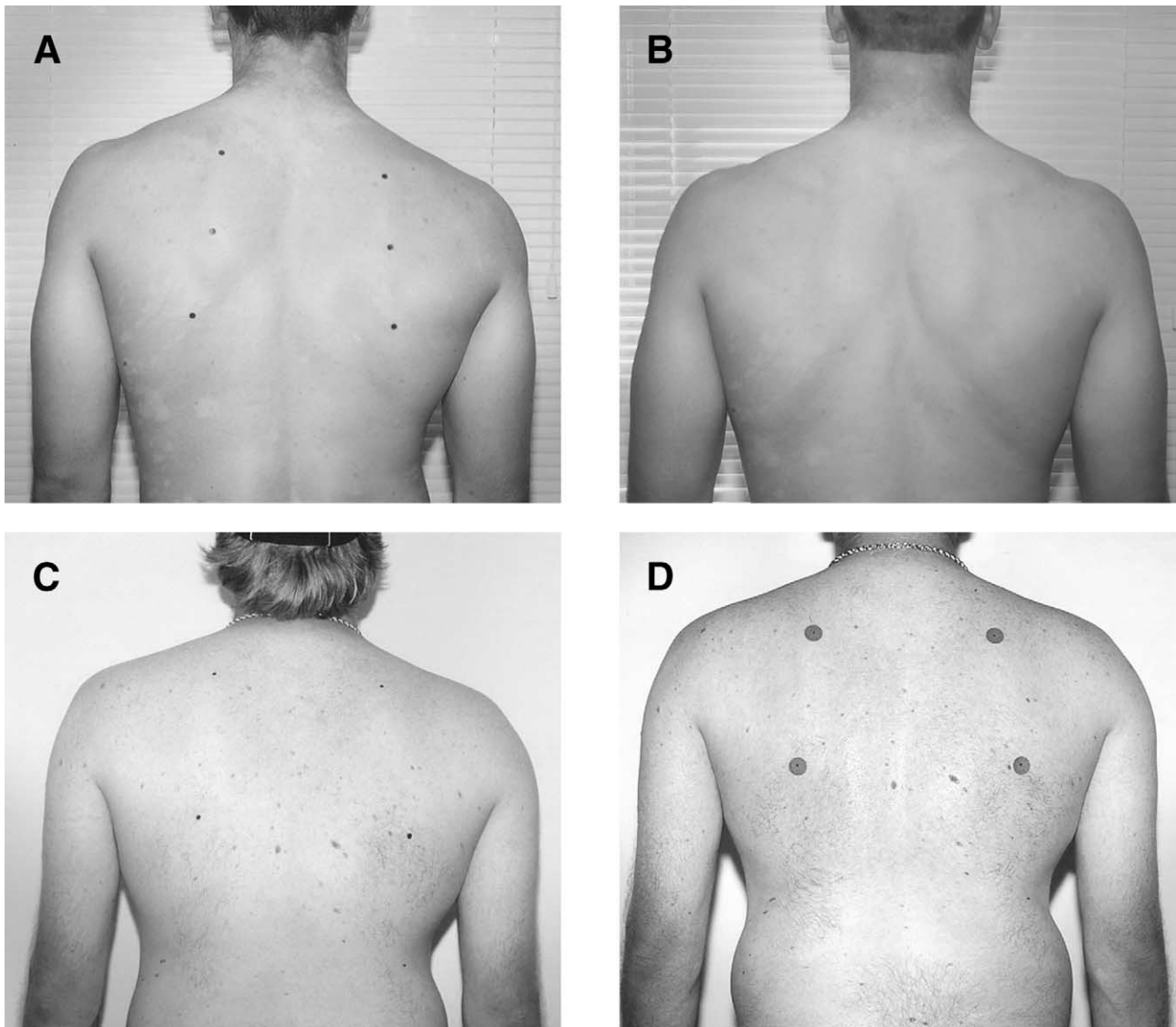


FIGURE 8. The SICK scapula before and after treatment. A professional right-handed baseball pitcher with a symptomatic SICK right scapula seen (A) at the time of initial presentation and (B) 6 weeks after scapular rehabilitation. The 1999 National League All Star starting pitcher and 2001 World Series Most Valuable Player seen in the late 1999 season with a (C) symptomatic SICK right scapula and (D) 6 weeks after scapular rehabilitation.

quired glenohumeral internal rotation deficit (GIRD), a malpositioned SICK scapula, or both.

In our experience, if the athlete with a shoulder at risk keeps throwing, the magnitude of the shoulder dysfunction will increase to a point at which intra-articular structural damage occurs and the patient becomes symptomatic. Unfortunately, at this point the problem has usually become a surgical issue. The shoulder most at risk is one with a SICK scapula and GIRD. This combination is particularly dangerous to the posterolateral labrum, the undersurface of the

posterior supraspinatus tendon, and the anterior inferior capsular structures. The reason for this is that both problems cause the thrower to abduct in extension (toward second base), rather than in the plane of the scapula, and hyperangulate in external rotation with a low arm body angle (below the horizontal) during the late cocking phase of throwing (Fig 9). This hyperangulation is made more acute if the dyskinetic scapula is in a position of protraction and glenoid antetilting, thereby bringing the posterior edge of the glenoid toward the humerus. More proper mechanics seen in

healthy shoulders consist of abduction in the plane of a well-positioned scapula and a greater arm-body abduction angle during the late cocking and early acceleration phases of the throwing cycle (Fig 10).

Early recognition of the shoulder at risk and institution of internal rotation stretching and scapular stabilizer strengthening protocols have been shown to be highly effective in converting the shoulder at risk to the shoulder not at risk and, in so doing, avoiding the shoulder at surgery. For these reasons, we encourage and recommend screening examinations to find these problems at the beginning and during each season for all overhead athletes. In addition, these athletes need to be educated by their respective athletic trainers and doctors regarding these issues so that they are willing to commit to the stretching and strengthening that will keep their shoulders healthy.

THE ROTATIONAL UNITY RULE

The healthy throwing shoulder has normal rotational kinematics without any form of glenohumeral instability throughout the throwing cycle as long as its GIRD is less than or equal to its external rotation gain. However, if the GIRD exceeds the external rotation gain (ERG) with a GIRD/ERG ratio greater than 1, the shoulder then becomes headed for trouble because of a posterosuperior shift of the glenohumeral rotation point with abduction and external rotation during the late cocking phase of throwing as previously discussed. The risk of structural injury is directly proportional to the increase in the GIRD/ERG ratio past unity. Although the absolute rotational numbers in degrees will vary from patient to patient because of variability in congenital laxity, GIRD that exceeds 10% of the contralateral shoulder's total rotation arc will usually produce a GIRD/ERG ratio significantly greater than 1. It is important to stabilize the scapula with the arm at 90° abduction when measuring internal and external rotation.

KINETIC CHAIN CONTRIBUTIONS TO DEAD ARM

Shoulder function in throwing requires contributions from all body segments to generate the forces necessary to propel the ball and position the bones of the joints to minimize the loads each joint structure must bear, and pass the forces and loads to the distal segments.⁵ This coordinated sequencing of the segments is termed the kinetic chain. In the normal kinetic chain of throwing, the ground, legs, and trunk act

as the force generators; the shoulder acts as a funnel and force regulator; and the arm acts as the force delivery mechanism. The labrum is a key structure in providing glenohumeral stability in this sequence and may be injured by excessive or imbalanced forces that may occur at the shoulder if regional or distant areas of the kinetic chain are abnormal.

Clinical studies have shown that alterations in flexibility or muscle imbalance are common in patients with shoulder injury. Kinetic chain alterations have been documented in shoulder impingement,^{6,7,8} rotator cuff injury,^{6,9} and instability.^{9,10} One study specifically evaluated distant findings that were present in throwers with arthroscopically proven posterosuperior labral tears.¹¹ Of 64 patients, 38 had isolated posterosuperior labral tears, and 26 had combined anterior and posterior lesions. On physical examination, 64 of 64 (100%) had restricted goniometrically measured internal rotation (with the shoulder at 90° abduction and the scapula stabilized) of more than 25° compared with the opposite side (mean, 32.6°; range, 26° to 58°). A total of 60 of 64 (94%) had patterns of dynamic scapular dyskinesis, or asymmetric motion or position of the scapula on rest or abduction motion.^{6,12}

The SICK scapula is an extreme form of scapular dyskinesis. A total of 41 subjects had the type I dyskinetic pattern of winging of the inferomedial scapular border and depression of the acromion with rest or motion, whereas 19 showed the type II pattern of true lateral slide, in which the entire medial scapular border is winged with the scapula protracted and laterally translated with rest or motion.¹³ The mean lateral slide asymmetry measurement¹² in 60 patients was 2.2 cm (range, 0.7 to 3.1 cm). A total of 46 of 64 patients (72%) showed weakness of the infraspinatus or teres minor on resisted external rotation. A total of 31 of 64 (48%) exhibited lower-back inflexibility, with reach deficits of greater than 5 cm on sit-and-reach examination. A total of 28 of 64 (44%) were unable to complete a nondominant leg stability sequence of one-legged stance with no Trendelenburg sign, 1-legged squat with pelvic stability, and 1-legged step-up and step-down with pelvic stability. Finally, 25 of 64 subjects (39%) had asymmetric decreased internal rotation on the nondominant hip.

These alterations in normal kinetic chain motions and positions affect the shoulder by altering proximal force generation and altering distal joint positioning. These alterations are especially important at the shoulder, which does not have a large force-generating



FIGURE 9. This pitcher shows abduction in extension, with angulation of the arm posterior to the plane of the scapula rather than in the plane of the scapula. Note the “dropped elbow” in this pitcher, causing the arm-body angle to drop below the horizontal.

capability and whose bony anatomy is dependent on body positioning and muscular activation to control excessive joint translations.

Leg and Trunk

The leg and trunk are important to provide a stable base for arm motion,^{13,14} provide rotational momentum for force generation,^{14,15} and generate 50% to 55% of the total force and kinetic energy in the tennis serve.³ Inflexibility of the nondominant hip or of trunk rotation, or weakness of hip abductors or trunk flexors “breaks the kinetic chain.” This breakage increases lumbar lordosis in acceleration, which places the arm behind the body. This “slow arm” creates a hyperabduction/external rotation position at the shoulder and increases posterior compression loads on the structures including the labrum. It moves the arm out of the safe zone of glenohumeral angulation that has been advocated by Jobe et al.¹⁶ and shown by Happee¹⁷ to be the most stable angle for the joint.

Scapula

The scapula plays many roles in throwing or serving that may affect labral injury.⁹ First, the glenoid must be positioned and stabilized in 3-dimensional space to act as a congruous socket for the humeral head as it rotates at the high velocities necessary for throwing or serving. Second, it must smoothly retract and protract around the thoracic wall as the arm moves from cocking through full cocking and then into acceleration and follow-through. The scapula must move in relation to

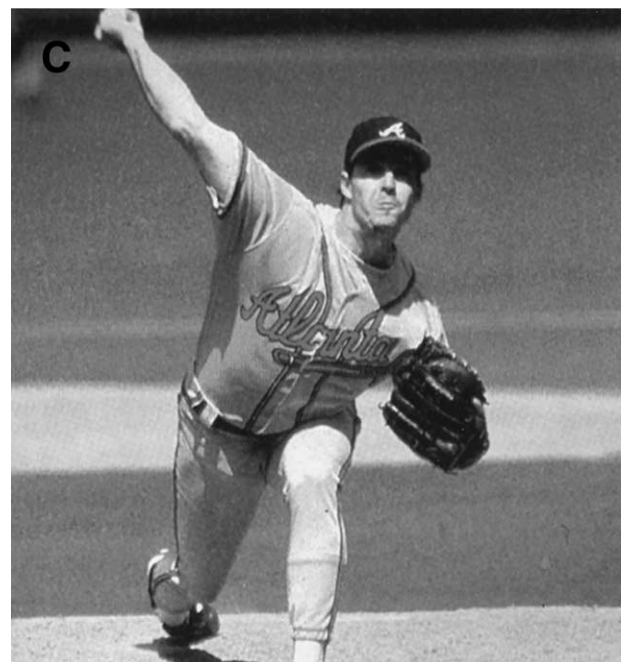
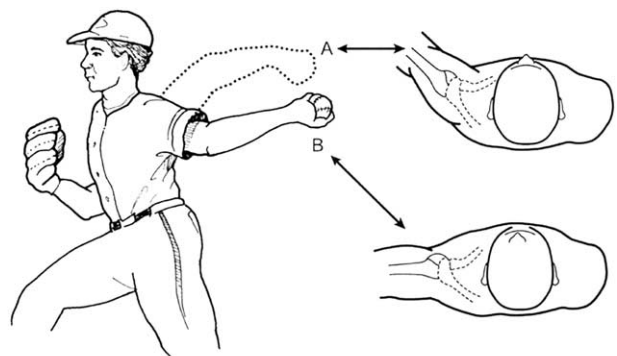


FIGURE 10. Ideal mechanics involve abduction in the plane of the scapula (A, dotted line) with the elbow high enough to keep the upper arm at or above the horizontal plane. (B) With a “dropped elbow” (solid line), the upper arm hyperangulates posterior to the plane of the scapula. (C) This pitcher has excellent mechanics, with the arm abducted in the plane of the scapula and positioned above the horizontal plane.



FIGURE 11. Type I scapular dyskinesis, with prominence of the inferomedial scapular border.

the moving humerus to maintain a safe zone of glenohumeral angulation and avoid hyperangulation of the humerus on the glenoid.¹⁶ Finally, it acts as a stable base for origin of the extrinsic and intrinsic muscles that control arm motion and provide glenohumeral compression. These roles require active positioning and active motion by the periscapular muscles. Alterations of normal position or motion, which have been termed scapular dyskinesis,¹³ can be caused by inflexibility, weakness, or activation imbalance of the muscles.

Two patterns of dynamic scapular dyskinesis are associated with posterosuperior labral lesions. The type I pattern consists of inferomedial scapular border



FIGURE 12. Type II scapular dyskinesis, with prominence of the entire medial border.

prominence at rest, with increasing prominence, lack of acromial elevation, and lack of full retraction on cocking (Fig 11). It is associated with inflexibility of the pectoralis major and minor, and weakness of the lower trapezius and serratus anterior. The type II pattern consists of entire medial border winging at rest, which becomes more prominent with cocking or elevation (Fig 12). It is associated with upper and lower trapezius and rhomboid weakness, with little anterior inflexibility. Both patterns create an abnormal position of excessive scapular protraction at rest and an abnormal motion of decreased scapular retraction and decreased acromial elevation during cocking and early acceleration. The type III pattern, which is not associated with superior labral lesions, displays prominence of the superomedial border of the scapula. It is associated with impingement and rotator cuff symptoms.

Scapular dyskinesis that creates excessive protraction and decreased cocking and elevation alters normal scapular biomechanics and plays a role in pos-



FIGURE 13. Scapular assistance test. The examiner stabilizes the upper scapular border and assists upward rotation of the inferomedial border.

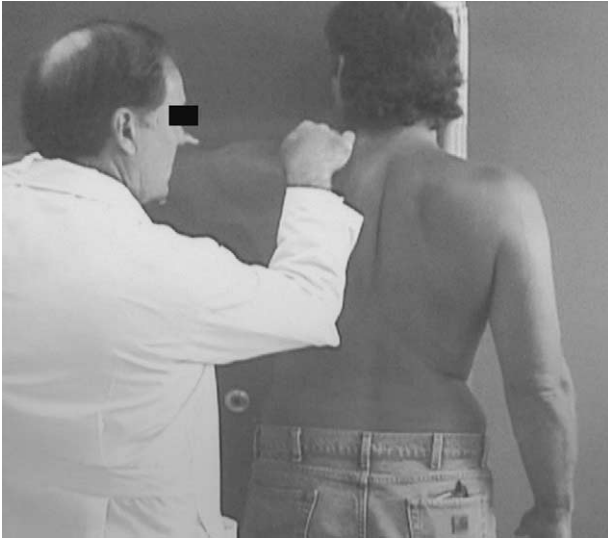


FIGURE 14. Scapular retraction test. The examiner stabilizes the retracted scapula against the thorax.

terosuperior labral injuries. Excessive protraction increases glenohumeral angulation outside of the safe zone, creating excessive anterior tension and posterior compression. Increased glenohumeral angulation also may increase humeral external rotation in cocking and acceleration as the arm lags behind the body, increasing the peel-back effect. Decreased retraction increases the posterior glenohumeral compression on the labrum and rotator cuff and decreases the scapular role as a stable base for muscle origin, thereby effectively decreasing muscle strength. Decreased acromial elevation due to scapular protraction creates rotator cuff impingement in cocking and acceleration as the arm is abducted.

Clinical Implications

Evaluation of athletes with suspected superior glenoid labral lesions should include tests to check kinetic chain functioning. History taking should include questions about previous leg or back problems, especially on the nondominant side. A screening exami-



FIGURE 15. One-leg stance, trunk rotation, and scapular retraction combined movements.

nation for the legs and trunk should include a posture examination for legs and trunk, seated range-of-motion examination of both hips and knees, the one-leg stability series (i.e., 1-legged stance with no Trendelenburg sign and 1-legged squat with no pelvic tilt or rotation), low back flexion and extension and side-bending, and situps and back extensions. Further testing is indicated for patients with positive tests.

The screening scapular examination should include a posture check for cervical and thoracic areas, scapular symmetry at rest and on ascending and descending arm motion in flexion and abduction, active scapular retraction and elevation, lateral slide measurements, and glenohumeral internal rotation measurements.

Special scapular tests include the scapular assistance test (SAT) and the scapular retraction test (SRT). The SAT (Fig 13) involves assisting scapular upward rotation by manually stabilizing the upper medial border and rotating the inferomedial border as the arm is abducted. The test is positive when it gives relief of symptoms of impingement, clicking, or rotator cuff weakness.¹³ The scapular retraction test (Fig 14) involves manually positioning and stabilizing the entire medial border of the scapula. This test is helpful in 2 groups of patients. The first group has decreased retraction and apparent rotator cuff weakness. The test is positive when retesting reveals increased muscle strength with the scapula in the stabilized position. The second group has a positive Jobe relocation test for posterosuperior labral injury. The test is positive when scapular retraction decreases the pain or impingement associated with the Jobe relocation test. When positive, these tests show that specific scapular alterations are present and should be addressed as part of the rehabilitation.

REHABILITATION OF THE OVERHEAD ATHLETE

Rehabilitation of patients with superior glenoid labral lesions and scapular dyskinesis should also include the kinetic chain. This aspect of the rehabilitation may be started early, even while shoulder evaluation and treatment is being done. Leg, back, and trunk flexibility and strength should be normalized, and exercises that emphasize kinetic chain activation of the leg, trunk, and scapula should be instituted.^{13,18} Useful combinations of movements to allow activation include trunk extension and scapular retraction, trunk rotation and scapular retraction, and 1-legged stance and diagonal trunk rotation and scapular retrac-

tion (Fig 15). All of these exercises facilitate lower trapezius muscle activation.

Scapular exercises start with scapular punches and isometric scapular retractions. A very safe exercise is the "low row" (Fig 16), an exercise that involves trunk extension, scapular retraction, and arm extension as the patient pushes against resistance in a posterior direction. More advanced closed-chain exercises include the scapular clock (Fig 17), in which the hand is placed on the wall, eliminating the weight of the arm, and moving the scapula in elevation and depression (the 12 and 6 o'clock positions) and retraction and protraction (the 9 and 3 o'clock positions). These exercises are generally safe and do not seem to increase labral injury. All preoperative patients may be placed on this program, similar to knee exercises before anterior cruciate ligament reconstruction. By stabilizing the kinetic chain and scapular base, the



FIGURE 16. The "low-row" trunk extension, scapular retraction, and arm extension. This can be initially done as an isometric exercise, progressing to an isotonic movement.



FIGURE 17. Scapular clock: The hand is placed on the wall or a ball, with varying degrees of abduction and flexion.

patient can move more rapidly through the early stages of rehabilitation, regain the normal patterns of trunk-scapula-arm integration, eliminate the “shrug” that is common when arm abduction is attempted, and move more rapidly into rotator cuff strengthening.

Rotator cuff exercises can be integrated into the rehabilitation program after the proximal base has been established.^{18,19} Maximal rotator cuff activation requires a stable scapular base, since all the rotator cuff muscles have their origin on the scapula; adequate scapular elevation, to eliminate impingement; closed-chain activation to eliminate excessive shear in the early stages of rehabilitation; and coordinated activation of all of the components in force couples, rather than isolated activation of individual muscles. Closed-chain rotator cuff exercises include humeral head depressions and rotations on a ball (Fig 18),

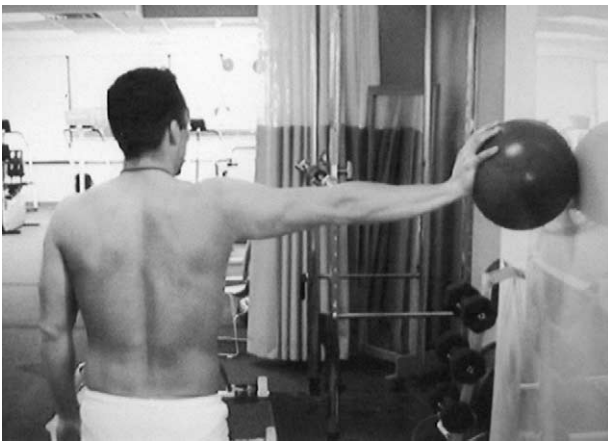


FIGURE 18. Humeral head depressions and rotations with the hand on a ball.

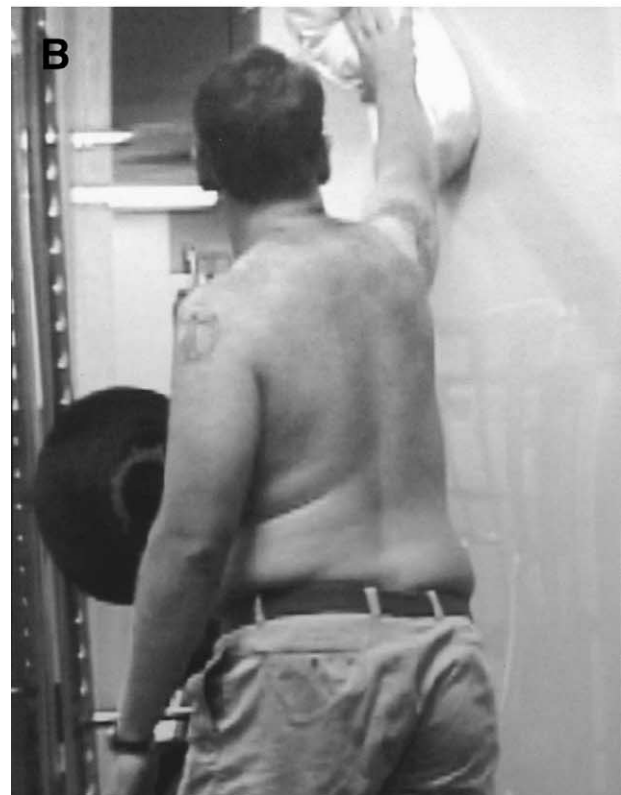
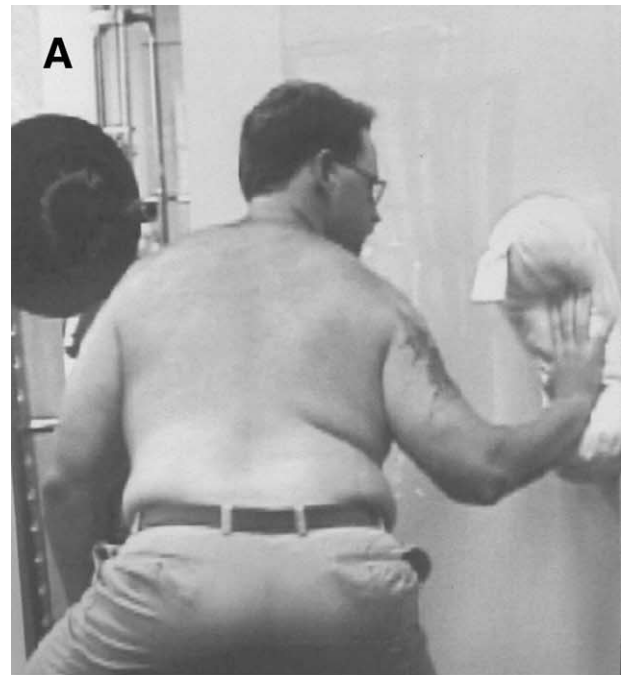


FIGURE 19. “Wall washes”: (A) Starting position and (B) movement pattern. The arm is then brought back to the starting position.

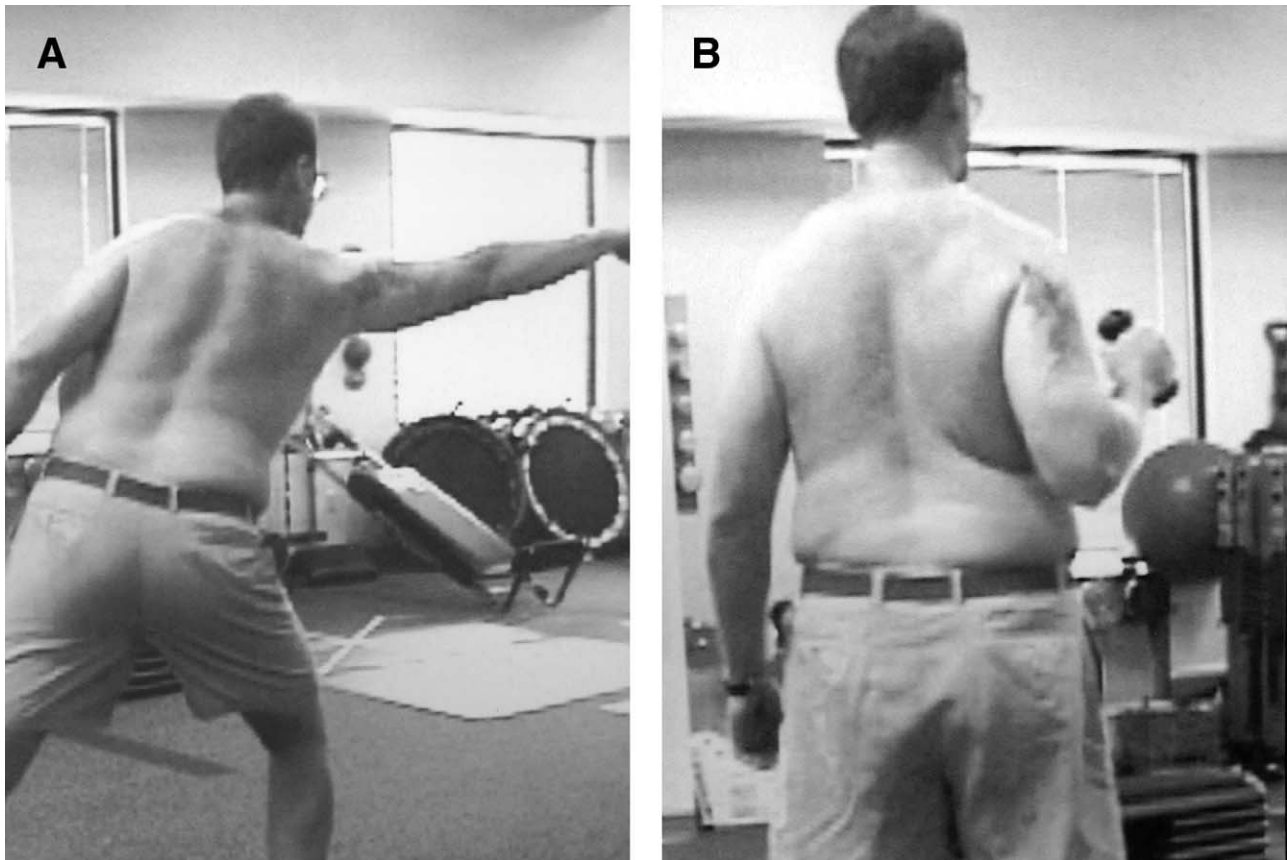


FIGURE 20. Punches: (A) Punch out. The motion may be varied-diagonal, upward, or downward. (B) Return position should always be “elbows in the back pocket” to facilitate scapular retraction.

“wall washes,” which combine trunk and scapula activation with rotator cuff activations (Fig 19), and punches, which combine closed-chain shoulder activation with open-chain arm motion (Fig 20). This progression of exercises creates a progression of challenge to the rotator cuff, and a resulting progression of muscle activation (Table 2).^{20,21} Closed-chain exercises require less activation than open-chain exercises,

and vertical patterns with the arm closer to the body, creating a shorter lever arm, requires less activation than diagonal patterns, with a long lever arm. This progression allows a more rapid but safe progression of rehabilitation that can be characterized as “accelerated,” similar to the accelerated anterior cruciate ligament programs that take advantage of the same physiologic and biomechanical principles of stable fixation, early protected range of motion, closed-chain activation of cocontraction force couples, and early activation of muscles in their physiologic positions.²²

TABLE 2. *Progression of Muscle Activation*

Exercise	Percent of Maximal Muscle Activation	
	Supraspinatus	Infraspinatus
Humeral head rotations (Codman exercise on a ball)	<10	<10
Vertical wall wash	12.8	8.6
Diagonal wall wash	18.0	10.4
Vertical punch	16.9	9.8
Diagonal punch	21.6	14.6

Rehabilitation for the SICK Scapula

The SICK scapula, as well as other types of scapular dyskinesis, must be aggressively treated with a focused scapular rehabilitation program. Rehabilitation consists of both stretching and strengthening.

The 2 areas of tightness that accompany the SICK scapula are (1) pectoralis minor tightness anteriorly and (2) posteroinferior capsular tightness with GIRD.



FIGURE 21. Pectoralis minor tightness is treated by placing a rolled towel between the shoulder blades of the supine patient and pushing posteriorly on the shoulders.

The anterior tightness is treated by placing a rolled towel between the shoulder blades of the supine patient and steadily pushing posteriorly on the shoulders to stretch the pectoralis minor (Fig 21). The postero-inferior capsular tightness is treated by “sleeper stretches” in which the athlete lies on his side with the shoulder in 90° flexion and the elbow in 90° flexion. The shoulder is passively internally rotated by pushing the forearm toward the table around a fixed elbow, which acts as the pivot point (Fig 22).



FIGURE 22. Sleeper stretch: (A) The patient lies on the side with the involved arm against the table and perpendicular to the body. The elbow is flexed 90°. (B) The patient pushes the forearm toward the table, stretching the postero-inferior capsule. (See Fig 4 in Part I of this Current Concepts in the April issue for other variations of the “sleeper stretch.”)

Strengthening for the SICK scapula patient consists of exercises to regain control of scapular protraction, retraction, depression, elevation, and rotation. Closed-chain exercises without weight are initiated to regain scapular control (Fig 23). Open-chain forward and lateral lunges and diagonal pulls are added, first without weights and then with 2- to 3-lb wrist weights or dumbbells (Fig 24). Blackburn retraction exercises are used to strengthen the scapular retractors and posterior rotator cuff (Fig 25). Seated push-ups strengthen scapular depressors or retractors, triceps, latissimus dorsi, and teres major (Fig 26). Rowing exercises, both standard row and low row, strengthen scapular retractors and the posterior rotator cuff. The low row is more specific for strengthening the serratus anterior (Fig 27).

SUMMARY OF THE ENTIRE CURRENT CONCEPTS: PARTS I, II, AND III

The disabled throwing shoulder comprises a spectrum of pathology. At the most dramatic and severe end of this spectrum is the dead arm, a pathologic shoulder condition in which the thrower is unable to throw with preinjury level velocity and control. The most common cause of the dead arm syndrome is a type II SLAP lesion, although the SICK scapula may cause a reversible type of dead arm with different clinical findings from those of the SLAP lesion.



FIGURE 23. Closed-chain scapular control exercises are best done in front of a mirror so that the patient can observe that he is performing the proper maneuver with his scapula. (A) Protraction, (B) retraction, (C) elevation and retraction, (D) depression and retraction, (E) internal rotation and elevation, and (F) external rotation and depression.

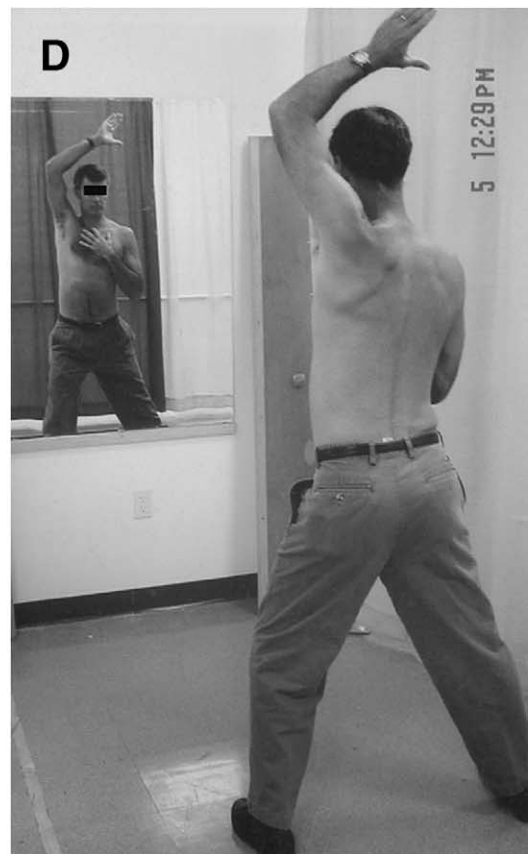
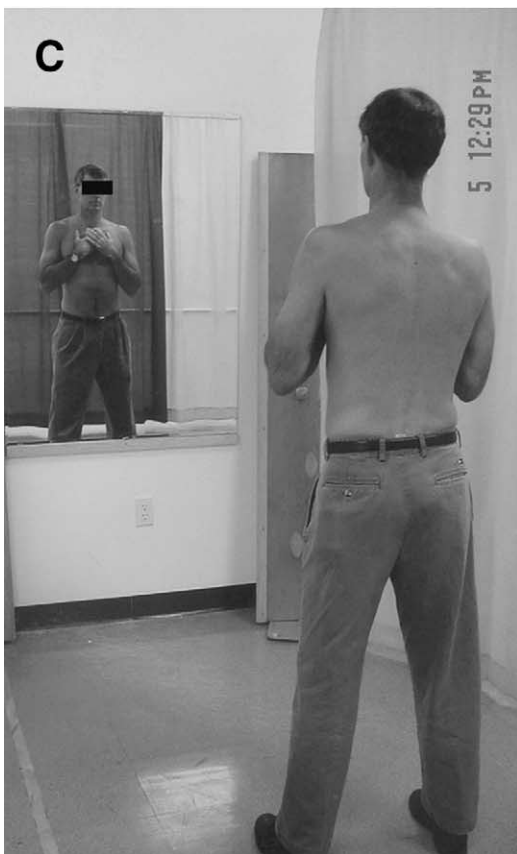


FIGURE 24. Open-chain scapular strengthening exercises. (A, B) Forward lunges to strengthen scapular protractors and retractors. (C, D) Lateral lunges to strengthen scapular retractors and upward rotators (upstroke) as well as scapular depressors and downward rotators (downstroke). (E, F) Diagonal pulls (lawnmower pulls) to strengthen scapular protractors and depressors as well as retractors and elevators.



FIGURE 24 (Continued).

The culprits in development of the dead arm are:

1. A tight posterior-inferior capsule causing GIRD and a posterosuperior shift in the glenohumeral rotation point, with a resultant increase in the shear stress applied to the posterosuperior glenoid labrum;
2. Peel-back forces in late cocking that add to the already-increased labral shear stress to cause the SLAP lesion;
3. Hyperexternal rotation of the humerus relative to the scapula caused by the shift in the glenohumeral rotation point that increases the clearance of the greater tuberosity over the glenoid and reduces the humeral head cam effect on the anterior capsule; and
4. Scapular protraction.

The ultimate culprit that initiates the pathologic cascade to the SLAP lesion is the tight posteroinferior capsule, which probably develops in response to the

loads that act on it during follow-through. The mechanics responsible for this pathologic cascade can be represented by means of a reciprocal cable model.

Hyperexternal rotation causes a hypertwist phenomenon that can, over time, result in (1) fatigue failure of posterosuperior rotator cuff fibers due to tensile, torsional, and shear overload, overshadowing any damage caused by direct abrasion of the cuff against the posterosuperior glenoid (internal impingement); and (2) torsional overload of the inferior glenohumeral ligament, causing elongation of anterior stabilizing structures. It should be emphasized that fatigue failure of the inferior glenohumeral ligament occurs mainly in veteran elite pitchers and that anterior instability as a part of the dead arm syndrome is very unusual, particularly in younger athletes.

We believe that microinstability is not a cause of the dead arm and that pseudolaxity from SLAP lesions coupled with a reduction in the cam effect (as a result of the posterosuperior shift of the glenohumeral con-

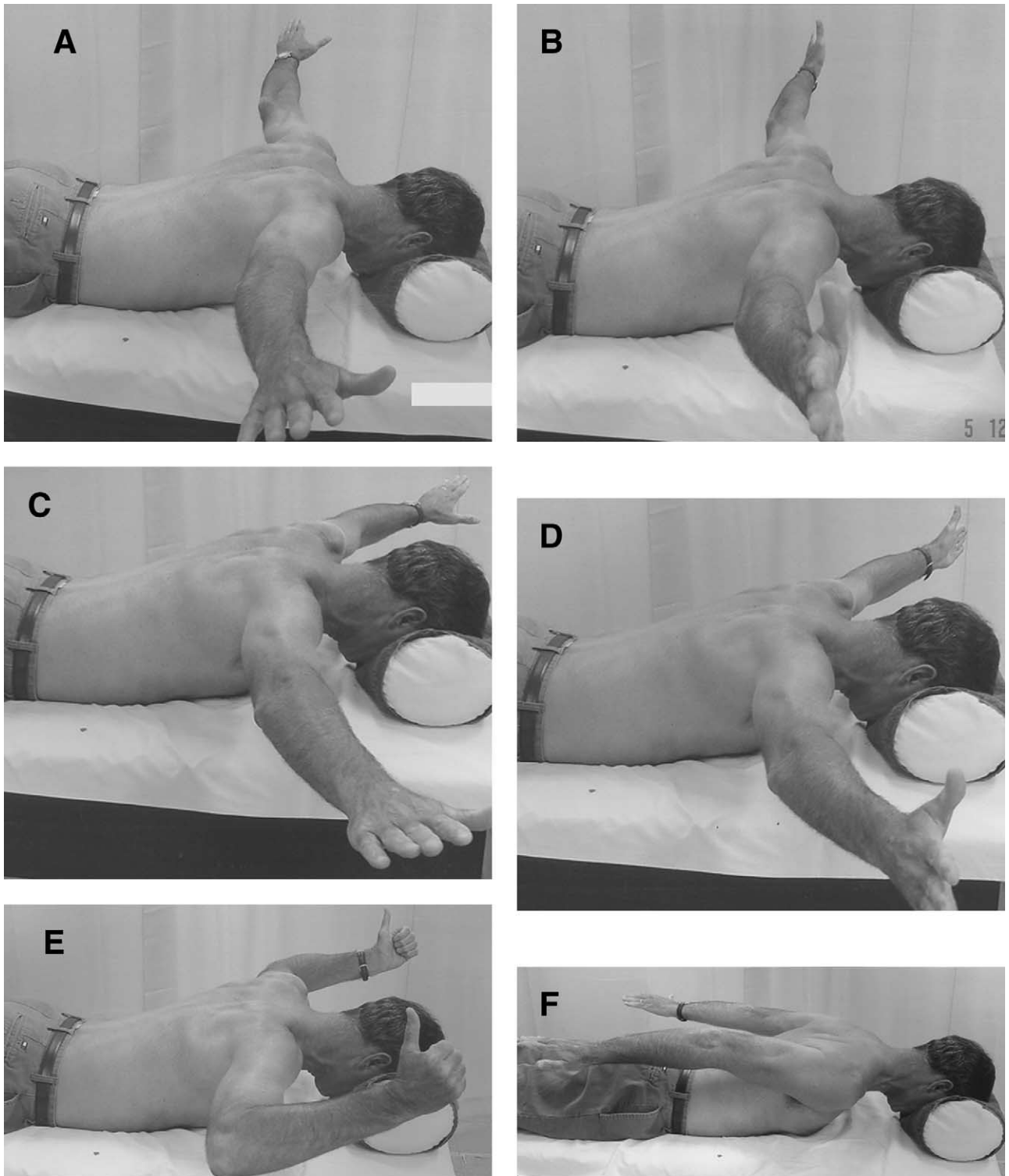


FIGURE 25. Blackburn exercises to strengthen scapular retractors and posterior rotator cuff. (A) Position 1, (B) position 2, (C) position 3, (D) position 4, (E) position 5, and (F) position 6.

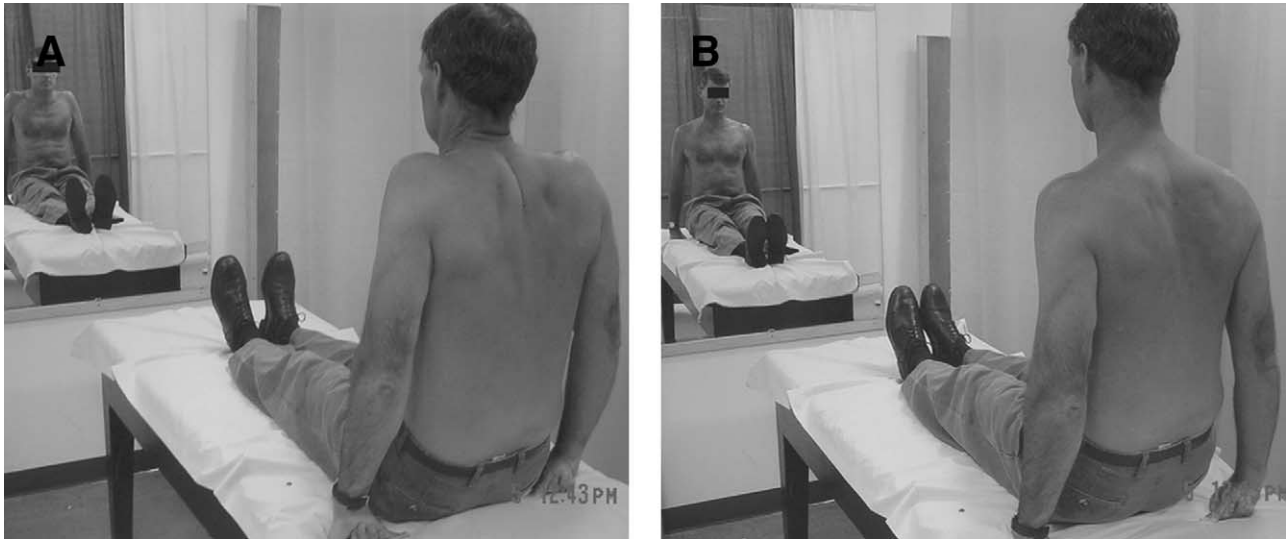


FIGURE 26. Seated push-ups: (A) Strengthening scapular retractors and elevators. (B) Strengthening scapular retractors and depressors.

tact point) has been misinterpreted as microinstability. Furthermore, we believe that internal impingement is a normal phenomenon that is not usually pathologic in the throwing shoulder.

For a thrower with a dead arm and a SLAP lesion, repair of the SLAP lesion combined with an ongoing stretching program of the posteroinferior capsule is usually curative, returning the thrower to his preinjury

level of competition in 87% of cases.²³ For successful SLAP repair, the surgeon must arthroscopically confirm elimination of the peel-back sign and elimination of the drive-through sign. For throwers who have been “stretch nonresponders,” the surgeon may consider performing an arthroscopic release of the posteroinferior capsule. If there is greater than 130° of external rotation with the scapula stabilized, one should consider electrothermal shrinkage versus arthroscopic capsular plication of the anterior band of the inferior glenohumeral ligament.

The SICK scapula syndrome, an extreme form of scapular dyskinesis, can be a cause of dead arm. Extreme protraction and anterior tilting of the scapula gives the impression that it is inferiorly displaced. This syndrome has unique clinical characteristics and generally responds to a focused rehabilitation of the shoulder.

Scapular biomechanics are vitally important to the throwing athlete and can be adversely affected by derangements at any point in the kinetic chain, including lower extremity function. The surgeon who treats throwing athletes must have a thorough understanding of the kinetic chain as well as an appreciation for the need for a well-focused rehabilitation program in restoring these athletes to the high level of function that their sport demands. In addition, closed-chain exercises can restore function while decreasing stresses to damaged tissues, resulting in more rapid rehabilitation.

The biomechanical and anatomic factors responsi-

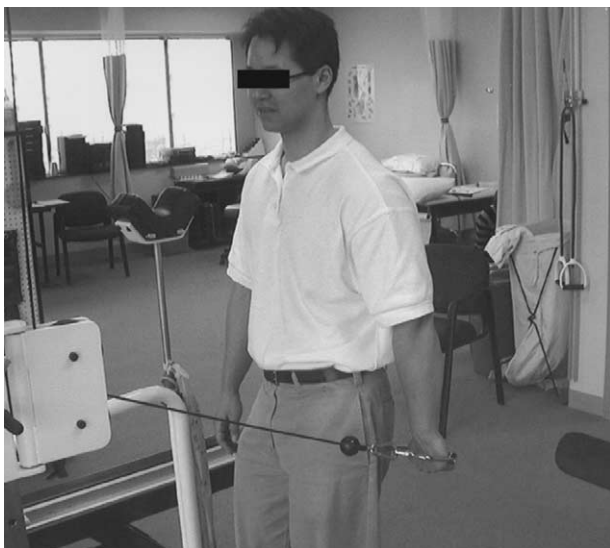


FIGURE 27. The low row is performed with the patient pulling posteriorly (shoulder extension and retraction) with the elbow locked in full extension. This exercise strengthens scapular retractors, particularly the serratus anterior.

ble for the dead arm have only recently been identified. As a result, appropriate surgical and nonsurgical treatments, including rehabilitation, can now be more precisely directed at specific pathophysiologic elements. The dead arm syndrome, so mysterious and so elusive for so long, is finally giving up its secrets.

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