Scapular Dyskinesis and Its Relation to Shoulder Injury

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Abstract

The scapula plays a key role in nearly every aspect of normal shoulder function. Scapular dyskinesis—altered scapular positioning and motion—is found in association with most shoulder injuries. Basic science and clinical research findings have led to the identification of normal three-dimensional scapular kinematics in scapulohumeral rhythm and to abnormal kinematics in shoulder injury, the development of clinical methods of evaluating the scapula (eg, scapular assistance test, scapular retraction test), and the formulation of rehabilitation guidelines. Primary scapular presentations such as scapular winging and snapping should be managed with a protocol that is focused on the scapula. Persons with associated conditions such as shoulder impingement, rotator cuff disease, labral injury, clavicle fracture, acromioclavicular joint injury, and multidirectional instability should be evaluated for scapular dyskinesis and treated accordingly.

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Optimal scapular function is a key component of all shoulder function. It is critical to proper alignment and function of the glenohumeral and acromioclavicular (AC) joints. Physiologically, it is important in scapulohumeral rhythm, the coupled and coordinated movement between the scapula and the arm that allows placement of the arm in the optimum position and achievement of the proper motion to accomplish tasks. Biomechanically, the scapula provides a stable base for muscle activation and a moving platform to maintain ball-and-socket kinematics. It also serves as an efficient link between the core, which develops force, and the arm, which delivers the force.

Alterations in these roles and motions are associated with most types of shoulder pathology, including shoulder impingement, rotator cuff disease, labral injury (eg, superior labral anterior-posterior [SLAP] lesion), clavicle fractures, AC joint pathology, and multidirectional instability (MDI). Regardless whether these alterations cause or are caused by the shoulder pathology, they have an impact on maintaining and/or exacerbating the functional consequences of the shoulder pathology.

Management of shoulder pathology should include evaluation and management of the scapula and scapular motion. Frequently, findings related to scapular motion and position provide information that is helpful in determining management options, rehabilitation protocols, and return to activity.

Scapular Function

In normal scapular function, three-dimensional (3D) scapular motions and translations integrate and coordinate with arm and trunk motions to enable task-specific activities involving the shoulder and arm. Basic science studies using both motion tracking systems and indwelling bone pins have been able to accurately and reproducibly demonstrate these motions. These motions have been categorized according to standards developed by the International Society of Biomechanics as individual motions (rotations around axes) and translations (sliding along a surface).[1,2]

The three motions are upward/downward rotation around an axis perpendicular to the scapular body, internal/external rotation around a vertical axis along the medial border, and anterior/posterior tilt around a horizontal axis along the scapular spine.[1] In a study incorporating indwelling bone pins, Ludewig et al[2] demonstrated that the resting position of the scapula in relation to the thorax averages 5.4° of upward rotation, 41.1° of internal rotation, and 13.5° of anterior tilt. As the arm moves to maximum elevation, the scapula moves in all three motions. The scapula rotates upward, tilts posteriorly, and moves into first internal and then external rotation, with a net change toward external rotation.[2] The largest part of these motions occurs with arm elevation >80°.

Two types of scapular translation can occur in the presence of an intact clavicular strut and AC joint: upward or downward sliding of the scapula on the thorax as the result of upward or downward clavicular motion at the sternoclavicular (SC) joint, and anterior or posterior sliding around the curvature of the thorax as the result of anterior or posterior clavicular motion at the SC joint. Although these descriptors and conventions provide a basis for more detailed understanding of scapular motion, their clinical application is limited.

Clinically, scapular movement is a composite of the three motions and two translations. The motions and translations are usually coupled to describe common clinical patterns. The coupling of scapular external rotation, posterior tilt, upward rotation, and medial translation is called retraction. The coupling of internal rotation, anterior tilt, downward rotation, and lateral translation is called protraction. The coupling of upward translation, anterior tilt, and internal rotation is seen as a shrug.

The clavicle and the SC and AC joints are among the most important components in achieving the individual and composite scapular positions, motions, and translations. The clavicle is the only bony connection of the scapula to the axial skeleton. To maximize scapular movement and scapulohumeral motion during maximal arm elevation, the clavicle retracts, elevates, and posteriorly rotates on its long axis.[2] All these motions are dependent on the SC joint. AC joint motions are the result of acromial motion on the clavicle.[2] These constrained motions create reproducible motion about the screw axis (ie, rotational axis and translation) between the clavicle and scapula through the AC joint, which allows the 3D motion.[3]

The scapula has relatively limited bony attachment; thus, it is dependent mostly on muscle activation for mobility and stability. This anatomic construct allows a great degree of mobility and accommodates many demands in different arm positions, but it also requires considerable eccentric muscle activation to withstand high distraction loads in activities involving forward motion of and loading on the arm.

The upper and lower trapezius muscles and the serratus anterior muscle are the greatest contributors to scapular stability and mobility.[4,5] Force coupling of the trapezius and serratus anterior muscles initiates upward rotation and posterior tilt.[4] As the arm elevates to >90°, the lower trapezius serves to increase and maintain scapular upward rotation and the serratus anterior stabilizes the medial border of the scapula against the thorax, acting as a scapular external rotator. Activation of the lower trapezius muscle is also important in the descent of the arm from a position of maximum elevation. This muscle is activated eccentrically to control excessive anterior tilt. Other intrinsic muscles (ie, rhomboids, pectoralis minor) play important but not primary roles. The rhomboids assist the trapezius in stabilizing the scapula, particularly in regard to controlling medial and lateral translation. The pectoralis minor assists the serratus anterior muscle in anterior tilt, internal rotation, and protraction when the arm is in lower levels of elevation (ie, <60° of abduction). Extrinsic muscles, chiefly the latissimus dorsi and pectoralis major, affect scapular motion in their role as prime movers of the arm. Humeral motion also can create scapular motion by placing tension on the glenohumeral capsule and muscles, especially in the presence of glenohumeral internal rotation deficit.

Normal Shoulder Function

The scapula contributes to efficient scapulohumeral rhythm in several ways. First, it facilitates congruency in the glenohumeral ball-and-socket configuration through the full ranges of arm motion by maintaining glenohumeral alignment within physiologic limits, thereby maximizing the concavity compression capability of the joint. Second, the scapula provides a stable base for optimal activation of the scapular muscles. Demonstrated rotator cuff strength can be improved when the scapula is stabilized in a position of neutral retraction.[6] Increases in strength ranging from 13% to 24% have been

Figure 1

Clinical photograph demonstrating scapular dyskinesis in which the medial inferior angle is prominent and the scapular position is protracted and internally rotated.

Figure 2

Clinical photograph demonstrating scapular windup as a result of glenohumeral internal rotation deficit. Because of tight posterior soft-tissue structures, the scapula moves with the humerus as the arm is internally rotated.

reported.[7] Third, the scapular motion causes the acromion to elevate on arm elevation. This requires scapular upward rotation and posterior tilt to allow maximum arm flexion.[8]

Finally, the scapula plays a critical role in the kinetic chain. The scapula facilitates optimal force transfer from the site of largest force development (ie, the core) to the most common force delivery site (ie, the hand). The shoulder acts as a funnel, and dynamic stability is required to achieve efficient transfer of energy. This dynamic stability is created by the actions of the scapular stabilizers, which are maximized when hip and trunk strength is maximized.[9]

Scapular Dyskinesis

Dyskinesis (dys [alteration of] kinesis [motion]) is a general term that is used to describe loss of control of normal scapular physiology, mechanics, and motion. The term dyskinesia is often used interchangeably with dyskinesis. Typically, the term dyskinesia is applied to abnormal active (ie, voluntary) movements that are mediated by neurologically controlled factors (eg, tardive dyskinesia). Because many other factors exist that can cause altered position and motion (eg, clavicle fracture, AC joint separation, muscle detachment), we use the more inclusive term dyskinesis in this article.[10]

Dyskinesis in and of itself is not an injury, it does not result in injury in all cases, and it is not always directly related to a specific injury. Clinically, it can be characterized by prominence of the medial or inferomedial border, early scapular elevation or shrugging on arm elevation, and/or rapid downward rotation on lowering of the arm[10] (Figure 1). The alteration of motion reduces the efficiency of shoulder function in several ways, with resulting changes in 3D glenohumeral angulation, AC joint strain, subacromial space dimensions, maximal muscle activation, and optimal arm position and motion. Each of these scapular components individually can generate symptoms. Alternatively, they may interact with other shoulder pathology to exacerbate dysfunction and thereby affect the management protocol and outcomes.

Causes of Scapular Dyskinesis

Multiple causative factors exist for dyskinesis. Bony causes include thoracic kyphosis, clavicular fracture nonunion, and shortened clavicular malunion. Joint-related causes include high-grade AC instability, AC arthrosis and instability, and glenohumeral joint internal derangement. Neurologic causes include cervical radiculopathy and long thoracic and spinal accessory nerve palsy.

The most common causative mechanisms of scapular dyskinesis involve alterations in the soft tissues, whether in the form of inflexibility or intrinsic muscle pathology. Inflexibility and stiffness of the pectoralis minor and short head of the biceps muscles create anterior tilt and protraction as a result of their pull on the coracoid.[11] The most common form of soft-tissue inflexibility is glenohumeral internal rotation deficit, which creates a "windup" of the scapula on the thorax with arm internal rotation or horizontal abduction (Figure 2).

Alterations in periscapular muscle

activation are common in patients with scapular dyskinesis. Serratus anterior activation and strength are reduced in patients with impingement and shoulder pain; this contributes to loss of posterior tilt and upward rotation, causing dyskinesis.[12] In addition, the force couple of the upper and lower trapezius muscles may be altered; delayed onset of activation in the lower trapezius muscle alters upward rotation and posterior tilt.

The end result of most of these causative factors is a protracted scapula with the arm at rest or an excessively protracting scapula with arm motion. This position of scapular protraction is unfavorable for every shoulder function except the "plus" position (ie, maximum forward motion of the arm and scapula) in weight lifting. It results in decreased subacromial space and increased impingement symptoms, decreased demonstrated rotator cuff strength,[6,7] increased strain on the anterior glenohumeral ligaments, increased risk of internal impingement,[13] and increased strain on the scapular stabilizing muscles. Most of the chief goals of management of scapular dyskinesis relate to regaining functional retraction capability.

Clinical Evaluation of the Scapula

Physical examination of the scapula is done to establish the presence or absence of scapular dyskinesis; evaluate joint-related, muscular, and bony causative factors; and employ dynamic corrective maneuvers to assess the effect of correction of dyskinesis on symptoms. The results of the examination are used to establish a comprehensive diagnosis and guide management and rehabilitation.

The scapular examination should be done mostly from the posterior aspect. The resting posture should be checked for side-to-side asymmetry. In particular, the examiner should assess for evidence of a SICK position (Scapular malposition, Inferior medial border prominence, Coracoid pain and malposition, and dysKinesis of scapular movement) and prominence of the inferomedial or medial border.

The SC and AC joints should be evaluated for instability, and the clavicle should be evaluated for angulation, shortening, or malrotation. AP laxity of the AC joint is evaluated clinically by stabilizing the clavicle with one hand while grasping and mobilizing the acromion in an AP direction with the other hand (Figure 3).

Figure 3

Clinical photograph demonstrating assessment of acromioclavicular joint laxity in a patient with suspected scapular dyskinesis. The clavicle is stabilized with the left hand, and anterior- and posterior-directed forces are applied to the distal end of the acromion with the right hand.

Dynamic examination of scapular motion can be reliably performed by clinical observation of the motion of the medial border as the arm elevates and descends. With a 3- to 5-lb weight in each hand, the patient raises the arms in forward flexion to maximum elevation and then lowers them to the starting position. This exercise is done three to five times. Prominence of any aspect of the medial scapular border on the symptomatic side is recorded as "yes" (prominence detected) or "no" (prominence not detected). Clinical observation of medial border prominence in symptomatic patients has been correlated with biomechanically determined dyskinesis. This method is reliable enough to be used as the basis for determination of the presence or absence of dyskinesis (sensitivity, 78% [arm flexion] and 74% [scaption]; positive predictive value, 76% [arm flexion] and 78% [scaption]).[14]

The scapular assistance test (SAT) and scapular retraction test (SRT) are corrective maneuvers that can alter the injury-related symptoms and provide information on the role of scapular dyskinesis in the dysfunction that accompanies shoulder injury. The SAT is used in evaluating scapular contributions to impingement and rotator cuff strength, and the SRT is used to evaluate contributions to rotator cuff strength and labral symptoms.

In the SAT, the examiner applies gentle pressure to push on the inferior medial scapular angle to assist scapular upward rotation and posterior tilt as the patient elevates the arm (Figure 4). In our practice, we have found that the chief biomechanical effect of the SAT is in increasing scapular posterior tilt by 7° to 10° throughout the entire arc of arm elevation. A positive result is indicated by relief of painful symptoms related to the arc of impingement and on increased arc of motion. This test has been shown to have acceptable interrater reliability.[15]

The SRT is used to grade supraspinatus muscle strength following standard manual muscle testing procedures and evaluate labral injury in association with the dynamic labral shear (DLS) test.[7,16] The examiner places the scapula in a retracted position and manually stabilizes it, then repeats the test to evaluate strength or DLS (Figure 5). Our own data indicate that the biomechanical effects are a combination of increased external rotation and posterior tilt. The test is positive when the demonstrated supraspinatus strength is increased or the symptoms of internal impingement in the labral injury are relieved with the scapula in the retracted position.

Scapular Dyskinesis and Shoulder Injury

Scapular dyskinesis may be the primary instigator in the pathologic process that results in shoulder dysfunction; an associated condition that contributes to injury causation, exacerbates shoulder symptoms, or adversely influences management or outcomes; or an adaptive condition that arises to compensate for other injury or discomfort (Table 1). The relationship between dyskinesis and shoulder symptoms is not always clear. In cases of nerve injury, fracture, AC separation, and muscle detachment, the injury creates the dyskinesis, which in turn affects shoulder function. In other cases, such as rotator cuff disease, labral injury, and MDI, the dyskinesis may be causative, creating pathomechanics that predispose the arm to such injuries. Alternatively, dyskinesis may be a response to injury, creating pathomechanics that increase the existing dysfunction.

Figure 4

Clinical photograph demonstrating the position of the examiner's hands and the patient's scapula at the end of the scapular assistance test. The examiner assists serratus anterior and lower trapezius muscle activity by manually "assisting" the scapula in upward rotation as the arm is elevated.

Primary Scapular Involvement

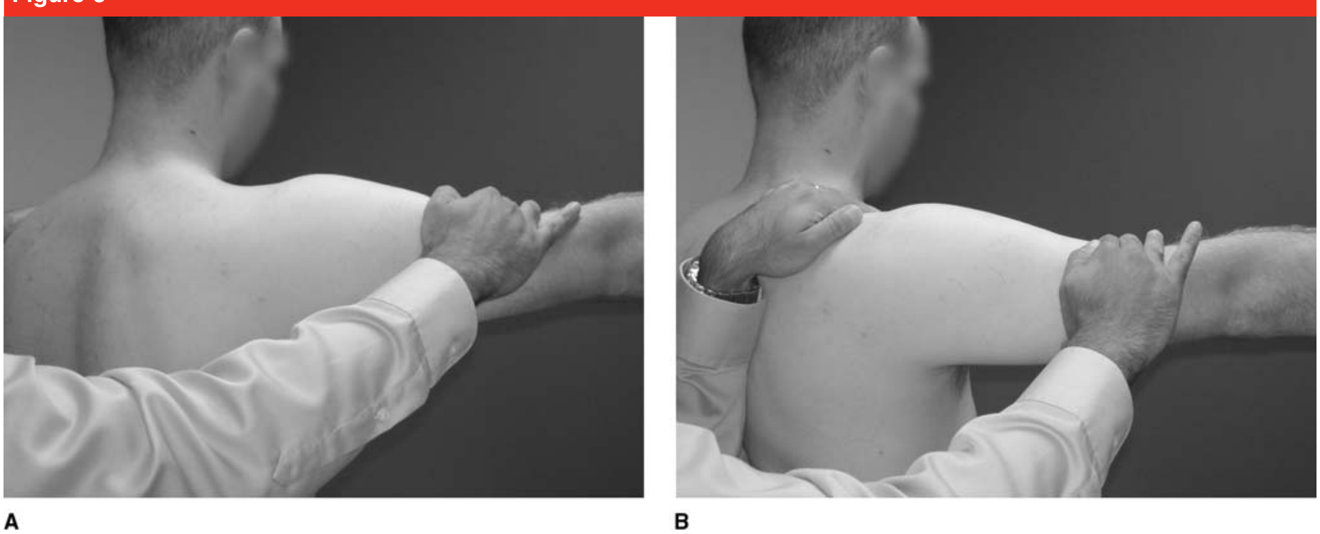
Scapular winging and the snapping scapula have been well reviewed elsewhere.[17,18] Dyskinesis also can be caused by proximal kinetic chain weakness and muscle imbalance.[19]

Table 1

Pathologic Conditions Associated With the Scapula

Primary scapular pathology
Neurologically based scapular winging
Snapping scapula
Kinetic chain–based scapular dyskinesis
Scapular muscle detachment
Associated shoulder injuries
Impingement
Rotator cuff disease
Superior labral injury
Clavicle fracture
Acromioclavicular joint pathology
Acromioclavicular separation
Multidirectional instability

Figure 5



Clinical photographs demonstrating the scapular retraction test. **A,** First, the examiner performs a traditional "empty can" manual muscle test to assess supraspinatus muscle strength. **B,** The examiner then stabilizes the medial scapular border and reapplies the muscle test.

Associated Scapular Involvement

Conditions in which scapular dyskinesis may play a role include shoulder impingement, rotator cuff disease, labral injury, clavicle fracture, AC joint injury, and MDI.

Shoulder Impingement and Rotator Cuff Disease

Studies have demonstrated altered scapular kinematics in persons with rotator cuff weakness,[7,20] rotator cuff tendinopathy or impingement,[21,22] and rotator cuff tear.[20] Studies have almost uniformly identified dyskinesis in patients with rotator cuff impingement or tendinopathy.[21-23] The exact nature of the alterations is varied, with combinations of changes in upward rotation (most showing a decrease), posterior tilt (most showing a decrease), and internal/external rotation (no change or increased internal rotation). All studies of patients with demonstrated rotator cuff tear have shown increased upward rotation of some magnitude, and most also have shown decreased posterior tilt.[20,23]

It is not clear whether dyskinesis causes, is caused by, or develops to compensate for rotator cuff pathology. If dyskinesis is a cause, it could be that decreased scapular upward rotation and posterior tilt alters rotator cuff clearance under the coracoacromial arch, thereby producing mechanical abrasion and wear; that decreased external rotation creates anterior glenoid tilt during arm motion, leading to internal impingement; or that it causes increased strain within the rotator cuff tendon.[23-25] If dyskinesis is an effect, it is likely the result either of the inhibitory effect of pain on individual muscle activation and the subsequent disruption of normal activation patterns, or of the effect of pain avoidance on kinematic patterns. Increased upward rotation in patients with rotator cuff tears may be a compensatory strategy to increase or maximize arm elevation or positioning in the setting of weakened or absent rotator cuff activation. Dyskinesis is associated with low function scores in all of these cases.

The scapular examination in patients with rotator cuff disease should emphasize evaluation of lower trapezius and serratus anterior muscle weakness and the effect of corrective maneuvers (ie, SAT, SRT). A positive SAT confirms that excessive anterior scapular tilt is a factor in the external impingement symptoms. Management should include exercises to increase flexibility in the pectoralis minor and short head of the biceps and to strengthen the serratus anterior muscle (a scapular external rotator) and the lower trapezius muscle (a retractor). Scapular stability exercises are effective in achieving these goals.[26] A positive SRT indicates scapular involvement in the muscle weakness. For these patients, rehabilitation should begin with exercises to enhance scapular stability in retraction rather than with exercises focused on the rotator cuff.

Superior Labral Anterior-posterior Injury

Dyskinesis is frequently seen in association with superior labral injury.[27] The increased internal rotation and anterior tilt alters glenohumeral alignment, placing increased tensile strain on the anterior ligaments, increasing peel-back of the biceps/labral complex on the glenoid,[28] and creating pathologic internal impingement.[27] Evaluation of dyskinesis in patients with suspected SLAP injury can be helpful in determining rehabilitation protocols. Frequently, the symptoms found in the DLS test are corrected on the SRT.[16] This finding confirms the presence of dyskinesis and indicates the need for scapular rehabilitation to improve scapular retraction. Rehabilitation should include mobilization of tight anterior muscles and institution of scapular stability exercises.

Clavicle Fracture

Fracture fragment alignment may disrupt the relationship of the scapula to the axial skeleton, which may in turn affect scapulohumeral kinematics. Alterations in clavicular anatomy include true shortening as the result of fragment overlap or butterfly fragments, anterior/posterior or inferior/superior angulation, or external rotation of the distal fragment. Scapular protraction and tilt may result in altered scapular mechanics. Malunited fractures with 15 mm of shortening have been shown to demonstrate notable scapular protraction and anterior tilting, along with lower subjective scores and notable decreases in strength.[29,30]

In a study comparing plating with nonsurgical management of displaced midshaft clavicle fractures, the surgical group was found to have higher patient satisfaction and significantly better Constant and Disability of the Arm, Shoulder, and Hand scores ($P = 0.001$ and $P < 0.01$, respectively).[31] The factors that correlated most strongly with poor outcomes scores were abduction strength and endurance as well as flexion range of motion, which can been related to dyskinetic position and motion.

Clinical examination of scapular position should be performed when evaluating patients with clavicle fracture. If the examination reveals the medial border prominence that is indicative of dyskinesis, management should be directed toward anatomic restoration of clavicle length, angulation, and rotation. Specialized radiographic views or CT may be required to adequately evaluate the fracture pattern.

Figure 6

Clinical photograph demonstrating altered scapular position as a result of disruption of the acromioclavicular joint. The altered scapular position becomes evident as the patient raises and lowers the arms overhead.

Acromioclavicular Joint Injury

Dyskinesis has been demonstrated in 73% of patients with high-grade AC symptoms (ie, Rockwood types III, IV, and V).[32] High-grade AC separations alter the strut function of the clavicle on the scapula and change the biomechanical screw axis of the scapulohumeral rhythm, thereby allowing excessive scapular internal rotation and protraction as the acromion slides inferior and medial to the clavicle and decreased dynamic acromial elevation on arm elevation (Figure 6). This motion is referred to as the third translation of the scapula on the thorax. The protracted scapular position creates many of the dysfunctional problems associated with chronic AC separation, including impingement and decreased demonstrated rotator cuff strength.

Scapular and shoulder dysfunction can also occur in type II injuries if the AC ligaments are torn. This creates AP AC joint laxity around the axis of the intact coracoclavicular ligaments and can be associated with pain, clicking, decreased arm elevation, and decreased shoulder function.

Lack of dyskinesis on clinical examination is indicative of functional stability of the AC joint, and these patients may progress as rapidly as tolerated through physical therapy. If dyskinesis is demonstrated on the clinical examination, a management protocol should be instituted to correct the scapuloclavicular biomechanical abnormality. Bracing should include retraction of the clavicle and scapula with a figure-of-8 brace. Physical therapy should be directed toward first achieving scapular retraction and external rotation, followed by posterior tilt. Persons who fail a supervised 3- to 6-week program frequently continue to demonstrate dyskinesis and functional symptoms, and they should be counseled regarding surgical options. Surgical management should include not only coracoclavicular ligament reconstruction but also AC ligament reconstruction to restore the screw axis mechanism and stabilize both inferior/superior and AP motion.

Multidirectional Instability

Inherent capsular and ligamentous laxity is only one component of the unstable shoulder in persons with MDI. Many patients have increased scapular protraction and simultaneous humeral head migration away from the center of the joint on arm motion.[33] When patients with MDI elevate the arm, the scapula deviates from the normal kinematic pattern of upward rotation, posterior tilt, and minimal internal rotation and instead follows a pattern of upward rotation, anterior tilt, and excessive internal rotation.[24] This altered position allows the glenoid to face inferiorly and diminishes the bony constraint to inferior translation, which allows the humeral head to translate inferiorly out of the glenoid socket, thereby creating instability. Altered scapular muscle activation patterns produce scapular protraction and increased humeral head motion. Inhibition of activation of the subscapularis, lower trapezius, and serratus anterior muscles, coupled with increased activation of the pectoralis minor and latissimus dorsi muscles, has been shown to place the scapula in a protracted position.[34] The hyperactive latissimus is the main dynamic deforming force that pulls the humeral head inferior. Rotator cuff and biceps activation increases to compensate for this altered scapulohumeral rhythm, which tends to allow the humeral head to migrate away from the joint center, translate inferiorly, and move anterior or posterior.[33]

The seeming paradox of a protracting scapula in the setting of posterior-directed instability is explained by the same mechanical alterations. As the scapula protracts and the posterior cuff muscles are weakened and/or inhibited, the lax capsular structures cannot constrain the action of the latissimus dorsi, which first pulls the humeral head into internal rotation and horizontal adduction and then pulls the humeral head posteriorly. Frequently, patients can reduce the degree of subluxation by externally rotating their arms and placing their scapulae in retraction, thereby achieving dynamic stabilization.

The dyskinetic positions and motions in persons with MDI create and exacerbate altered glenohumeral kinematics and muscle activations, with resulting increases in dysfunction. Evaluation for the presence or absence of scapular dyskinesis should be included as part of a comprehensive examination of the unstable shoulder. By stabilizing the scapula in retraction, the SRT alters the glenoid position to direct it more superior, decreases latissimus dorsi activation and increases rotator cuff activation to minimize humeral head translation, and reduces or eliminates the symptoms of instability associated with arm motion. The patient with a positive SRT should undergo a treatment program that involves strengthening the lower trapezius and serratus anterior muscles and increasing flexibility in the pectoralis minor and latissimus dorsi muscles. Such treatment is typically most efficacious in persons with MDI and posterior instability.

Summary

Scapular dyskinesis may directly cause, contribute to, or be the result of shoulder symptoms. It is a kinematic finding with no specific association to any single shoulder injury, and careful evaluation is required to identify it. Evaluation of the position and motion of the scapula and the results of corrective maneuvers should be a routine part of the overall assessment of all patients with shoulder pathology. Scapular rehabilitation should be included in the treatment of patients with shoulder injury who exhibit scapular dyskinesis. In some cases, this finding will dictate the management protocol.

References

Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, references 1-3, 6, 8, 11, 13, 14, 16, 21-23, 25, 31, and 33 are level I studies. References 7, 9, 20, 26, 27, 32, and 34 are level II studies. References 5, 12, 15, and 29 are level III studies. Reference 18 is a level IV study. References 4, 10, 17, 19, 24, and 28 are level V expert opinion.

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