Shoulder Function During Overhead Lifting Tasks: Implications for Screening Athletes

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ABSTRACT

Overhead exercises are common place in many strength and conditioning programs. An essential element of supporting load above head is the demonstration of optimal joint kinematics at the shoulder complex. This article will consider various dysfunctional movement strategies that may present during overhead lifting exercises, potentially exposing an athlete to pathology. A screening process will also be discussed, providing coaches with a thought-process in which they may assess an athlete’s suitability for overhead lifting exercises based on their movement competency.

INTRODUCTION

Many exercises within the strength and conditioning coach’s toolbox require athletes to assume a hand position above head while supporting load. Variations of overhead lifting within the weight room include vertical pressing, vertical pulling, and derivatives of the Olympic-style lifts. However, athletes competing in sports requiring large volumes of overhead lifting have been shown to be at an increased risk of developing injuries in the upper extremity (8,18,35). As sports involving contact (21,27), striking (72), and throwing (56) already expose athletes to a high risk of shoulder injuries, prescription of overhead exercises may amplify the likelihood of structures becoming pathological. This may be compounded in athletes who use poor upper extremity movement strategies during these motor skills (16,23,39,41,42,44). It is therefore imperative that athletes exhibit optimal shoulder girdle kinematics during overhead lifting tasks to reduce the risk of injury.

Overhead lifting exercises often demand the upper extremity achieve shoulder elevation angles of up to 180°. Numerous joint complexes within the upper extremity provide kinematic contributions that allow for this movement to occur (55). At the shoulder girdle, approximately 120° of shoulder flexion or abduction is provided by the glenohumeral (GH) joint, with the remaining 60° derived from upward rotation of the scapulothoracic (ST) joint (often referred to as ST articulation) (55). These primary movements at the GH and ST joints are complemented with secondary and accessory joint motions that distribute loading evenly across various joint structures (16,44,55,74).

Although many risk factors exist that may predispose an athlete to a shoulder injury (67), disruption of normal joint kinematics at both local (15,23) and global segments (44,49) has been implicated as being important. As such, this article aims to (1) discuss movement strategies that may occur during overhead lifting tasks and (2) to present a thought-process for assessing athlete’s and their suitability for overhead lifting. Readers must be aware that this article does not in any way intend to provide practitioners with the tools to diagnose shoulder pathology. In instances where pathology is suspected, referral to an appropriate medical practitioner is recommended.

OVERHEAD LIFTING AND INJURY MECHANISMS

Although numerous injuries may result from overhead lifting (33), 1 common injury mechanism for shoulder girdle pathology is impingement syndrome (49). Although impingement is a common diagnosis, it merely represents a clinical sign (32). It is the repetitive
exposure to impingement mechanisms that lead to pathophysiological abnormalities at numerous tissues, including the rotator cuff tendons, the superior labrum, the subacromial bursa, or the long head of the biceps brachii (7). This may present in different forms with inflammation or degeneration of the bursa and rotator cuff leading to possible tears and degenerative joint disease of the GH joint (3,52). Treatment of such disorders can be complex and may prove costly for the athlete, both from a financial and performance perspective (49).

Impingements are categorized as either external or internal (32), with both variations being common in overhead athletes (23,59). External impingement refers to any damage to the soft tissue located within the subacromial space, secondary to excessive compression (52). As the shoulder elevates, the subacromial space between the head of the humerus and the coracoacromial arch (consisting of the anterior one-third of the acromion, acromioclavicular joint, and coracoacromial ligament) is compromised, causing compression on the tissue residing within the space (16,52,54). Impingement signs may be due to either primary (53) or secondary factors (26), in which structural abnormalities are present, or instability of the shoulder complex exposes tissues to excessive compression (32), respectively.

Internal impingement describes the entrapment of the under surface of the supraspinatus or infraspinatus tendon against the posterior-superior surface of the glenoid labrum (23). It is believed that this mechanism predominately occurs during extremes of GH abduction when accompanied with external rotation causing uncontrolled anterior glide of the humeral head relative to the glenoid fossa (25). Ludewig and Braman (39) suggest that the angle of shoulder elevation potentially changes the type of impingement an individual may be exposed to. As the athlete moves through 45°–60° of shoulder elevation, the rotator cuff tendon is in close proximity to the coracoacromial arch (2). Beyond this point, the insertion site of the rotator cuff tendons on the greater tubercle actually rotates externally relative to the acromion (2), reducing the potential for rotator cuff impingement. However, as the GH joint moves farther toward full end range, the risk of internal impingement increases as the supraspinatus and infraspinatus tendon approximates the glenoid labrum (39). Although both pathologies warrant a separate treatment protocol as part of a rehabilitation process, abnormal shoulder motion during overhead tasks has been implicated as a risk factor for impingement types (15,23,39,44,46).

SCAPULOTHORACIC JOINT MOTION

Secondary to triplanar contributions from the sternoclavicular and acromioclavicular joints (44), the scapula upwardly rotates around the thorax up to 60°, supplying one-third of total range of motion (ROM) during shoulder elevation (55). Ideal dynamic orientation of the scapula supplies the GH joint with both optimal form and force closure. First, as the scapula reaches approximately 60° of upward rotation during full shoulder elevation, the glenoid fossa is positioned beneath the head of the humerus (14,55). This offers the GH joint structural stability (38), potentially reducing the need for ligamentous and capsular involvement. The active stabilizing system is also optimally set within its length-tension relationship. As the scapula follows the humerus, excessive elongation or shortening of the rotator cuff muscles (depending on their location relative to the GH joint) is prevented through the origin of the muscles being positioned either nearer or farther to their insertion points, respectively (14,63). Therefore, the GH joint function is strongly dependent on optimal ST joint kinematics during shoulder elevation.

Alterations in ST joint motions during dynamic tasks have been defined as scapular dyskinesis (30,31). A significant deficiency in upward rotation at the ST joint has been suggested as a risk factor for GH joint dysfunction (44). Numerous investigations have identified decreased ST upward rotation in individuals who possess shoulder pathology (15,37,41). When ST upward rotation ROM is decreased, total shoulder elevation ROM will be deficient unless a neighboring joint segment increases its motion to offset the deficit. A compensatory movement strategy of increased GH joint flexion/abduction ROM may emerge, resulting in excessive compression on the subacromial tissue as the humeral head glides superiorly within the glenoid fossa (44). A loss of upward rotation may also promote an internal impingement mechanism. Mihata et al. (50) demonstrated during a throwing movement, that reducing ST upward rotation by 10° while maintaining the humerus’ position in space increased the internal impingement area. Reduced ST upward rotation is therefore implicated in both external and internal impingement syndromes, secondary to compensatory GH joint motion.

Conversely, increased ST upward rotation has also been demonstrated in individuals presenting with an impingement syndrome (46). Intuitively, increased ROM during upward rotation may seem to be beneficial, as it allows for an increase in the subacromial space. However, in instances where ST upward rotation has increased beyond the expected norm, the GH joint would theoretically be contributing less to overall motion. As such, it has been suggested by Sahrmann (63) that increased ST mobility may be caused by a lack of extensibility in the posterior structures of the GH joint, rather than overactivity of the ST upward rotator muscles. This increased stiffness is proposed to demand compensation to counteract the loss of GH flexion/abduction, leading to excessive scapula upward rotation. However, tightness in the posterior capsule has been shown to cause excessive anterior and superior translation at the GH joint (20,74), a potential risk factor in both impingement syndromes (9,42). Therefore, it is likely the case that...
too much, or too little upward rotation of the scapula may be problematic for overhead lifting tasks.

During shoulder elevation, upward rotation is combined with posterior tilt of the scapula on the thorax (43,44). Posterior tilt of the scapula in the sagittal plane elevates the anterior aspect of the acromion, increasing the subacromial space (49), thereby reducing the compression on subacromial tissue. During full ROM shoulder elevation, up to 30° of posterior tilt of the scapula has been reported in healthy individuals (47). As subjects with impingement signs demonstrate reduced posterior tilt of the scapula during shoulder elevation tasks (15,41), it is imperative that athletes exhibit similar movement strategies are identified before the prescription of overhead lifting exercises.

**SCAPULOTHORACIC SYNERGISTIC MUSCLE IMBALANCE AND RESTRICTION**

Muscle imbalances around the ST joint complex have been suggested to alter the amount of both upward rotation and posterior tilt of the scapula (44). During shoulder elevation, the upper, middle, and lower trapezius work synergistically with the serratus anterior to move the scapula into full upward rotation (55). Deficiencies in muscle activity or strength of the upward rotator muscles may lead to dysfunctional ST joint kinematics. Ludewig and Reynolds (44) emphasize the synergistic balance in activation between the serratus anterior and trapezius force couple in providing optimal ST upward rotation and posterior tilt. Reduced serratus anterior and lower trapezius activation, along with excessive upper trapezius activity, has been shown to reduce upward rotation and posterior tilt of the scapula (37,41). If serratus anterior and lower trapezius are inhibited, and the upper trapezius muscle attempts to compensate, ST elevation will likely occur. This in turn tilts the scapula anteriorly, as it follows the inclined contour of the rib cage.

Reduced extensibility of the pectoralis minor muscle may prevent both upward rotation and posterior tilt of the scapula. As the pectoralis minor functions to downwardly rotate, protract, and anteriorly tilt the scapula through its attachment to the coracoid process, an inability to lengthen during shoulder elevation has been shown to negatively influence ST joint motion (4). The pectoralis minor muscle is joined in its function as a downward rotator by the rhomboids minor and major, as well as the levator scapula (55). Reduced extensibility of either the rhomboids muscle group or the levator scapula has not yet been correlated with dysfunctional upper extremity movement strategies. Secondary to their function as downward rotators, excessive stiffness in either muscle may also prevent optimal ST joint kinematics.

**GLENOHUMERAL JOINT MOTION**

As previously mentioned, for full shoulder elevation ROM to be achieved, approximately 120° of GH joint flexion or abduction must be present (55). One vital element of GH joint kinematics that occurs as a secondary motion during either flexion or abduction is external rotation (49). Shoulder elevation demands external rotation of the humerus relative to the scapula to allow for the clearance of the greater tubercle under the coracocromial arch (49). External rotation of the humerus occurs throughout shoulder elevation, peaking between 75 and 90° (74), crucially when the greater tubercle is nearest to the coracocromial arch (2). This in turn reduces the approximation between the coracocromial arch and the tendons of both the long head of the biceps brachii and the supraspinatus (5,16). Glenohumeral external rotation also permits full shoulder elevation ROM by slackening the GH capsular ligaments (6).

Optimal arthrokinematics must also be displayed during overhead movements. When the GH joint passes 60° of elevation, inferior translation of the humerus relative to the glenoid fossa is required (17). Inferior translation assists in maintaining a centralized position of the GH joint and provides clearance for the head of the humerus beneath the coracocromial arch (17). Excessive superior and anterior translation of the humerus during shoulder elevation has been theorized as a risk factor for both shoulder impingement syndromes (61,63). Small changes in translation during shoulder flexion have been reported in patients with shoulder impingement, with 1.5 mm of increased superior translation (13) and 3 mm of increased anterior translation (42) being identified.

**GLENOHUMERAL SYNERGISTIC MUSCLE IMBALANCE AND RESTRICTION**

Thoracohumeral muscles possess tremendous potential for limiting ROM during shoulder elevation. Excessive tightness in either the latissimus dorsi or the sternal fibers of the pectoralis major muscle may restrict GH joint movement during shoulder elevation. Excessive tightness in either muscle may also limit GH external rotation (22), preventing the clearance of the greater tubercle under the coracocromial arch during shoulder elevation (63). A lack of extensibility in the both muscles may also compromise the position of the lumbar spine, as they originate from the pelvis, spine, and thorax (22,63). In particular, when the latissimuss dorsi muscle displays reduced extensibility during bilateral shoulder elevation, increases in lumbar extension may take place to permit motion at the GH joint (22). Extensibility of both thoracohumeral muscles should therefore be checked before prescribing overhead activities to ensure that a healthy upper extremity and spinal column integrity are preserved.

The rotator cuff muscles, functioning to maintain the optimal instantaneous axis of rotation between the humeral head and the glenoid fossa, control translation of the GH joint. During the initial 90° of shoulder elevation, the rotator cuff muscles control the superior translation created by the deltoid muscle, reducing mechanical
compression on the subacromial tissues (19). Simultaneously, their synergistic relationship also prevents excessive anterior glide of the humeral head through subscapularis (58). Although weakness of the rotator cuff muscles has been identified in individuals with signs of impingement (1), isometric rotator cuff strength does not predict the occurrence of first-time GH joint instability in healthy individuals (62). Therefore, testing rotator cuff strength may not indicate the muscles’ functional capacity to prevent excessive translation.

As such, qualifying an athlete’s readiness for overhead lifting relative to their ability to control translation may prove difficult. Although tests that assess for GH joint instability are available to the medical community (10), it is likely many strength and conditioning coaches are not trained to perform or interpret results. From a movement dysfunction perspective, attempting to observe excessive translation during dynamic movements can be problematic. However, it may be possible to identify increases in GH joint translation using an indirect approach (49). Reduced extensibility of the posterior capsule and local myofascial tissues situated at the GH joint has been shown to increase superior and anterior translation of the humerus relative to the glenoid fossa (20,74). Assessment of the posterior shoulder cuff may therefore allow practitioners to make inferences about the potential translation that may occur during GH joint motion, especially when ST and GH physiological joint motions are known.

**SPINAL CONTRIBUTION**

Spinal orientation also plays an important role in facilitating optimal scapula kinematics during overhead lifting tasks. During shoulder elevation, extension of the spine, particularly in the thoracic region, promotes efficient shoulder function through its interaction with the scapula (11,12). Spinal extension has been shown to be continuous throughout shoulder elevation (11,69), with a dynamic relationship existing between the upper and lower
thoracic spine during overhead lifting tasks (11). Crosbie et al. (11) revealed upward rotation of the scapula to be phase-locked with upper thoracic extension, demonstrating the significant role of the spine in optimally positioning the scapula.

Thoracic extension is also crucial in permitting the 20–30° of posterior tilt needed at the scapula to alleviate subacromial impingement (49). As the scapula in most cases follows the contours of the thorax, a hyperkyphotic thoracic posture inclines the scapula anteriorly (45). During full shoulder abduction, individuals who remain in a flexed spinal posture have been shown to exhibit a significant reduction in total posterior tilt of the scapula (28). This has the potential to lead to a loss of total ROM during shoulder abduction of up to 23° (28). This disruption of ST joint kinematics has also been shown to lead to significant reductions in isometric force output at 90° of scapular plane abduction (28). Kebaetse et al. (28) proposed that this may be due to disruption of the length-tension characteristics of the ST upward rotator muscles.

Scapula position may also be influenced by the position of the cervical spine (40). Thigpen et al. (70) demonstrated that in individuals with increased forward head posture, anterior tilt of the scapula is increased during overhead reaching tasks. Interestingly, these individuals did not possess excessive thoracic kyphosis when compared with controls, indicating altered head position as the main culprit in distorting scapula kinematics (70). Subjects also demonstrated significantly reduced serratus anterior activation, highlighting the role the cervical spine plays in enabling efficient ST muscle activation during function (70). This finding is further supported by Weon et al. (73), who found decreased serratus anterior activation was compounded by increased upper

<table>
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<tr>
<th>Anatomical region</th>
<th>Ideal posture</th>
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<tbody>
<tr>
<td>Head</td>
<td>Auditory meatus vertically aligned with the center of the glenohumeral joint, greater trochanter of the femur, and lateral malleolus (29)</td>
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<tr>
<td>Thoracic spine</td>
<td>A mild posterior convexity with an even distribution of flexion across the 12 vertebrae (65)</td>
</tr>
<tr>
<td>Scapulothoracic joint</td>
<td>Scapula situated between T2 and T7 with slight anterior tilt (51). Vertebral borders should be parallel to and 3 inches from the thoracic spinous processes (63)</td>
</tr>
<tr>
<td>Glenohumeral joint</td>
<td>The humerus should be oriented vertically in all planes, with the olecranon processes facing posteriorly (63)</td>
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and lower trapezius muscle activation in healthy individuals exhibiting forward head posture. An altered recruitment strategy of this nature would likely correspond with unfavorable ST joint kinematics, potentially resulting in pathology (39).

SCREENING OVERHEAD FUNCTION

When movement faults at the ST or GH joint occur, athletes may be predisposed to pathology. It is important that coaches attempt to distinguish the cause-and-effect relationship of a faulty movement. However, this is a difficult process, as dysfunction in 1 area will potentially demand dysfunction in a corresponding region. For example, limitations in mobility of 1 joint segment may drive compensations of stability at another. Conversely, reduced stabilization capacity around a joint may cause reflexive stiffness in myofascial tissue to provide stability, leading to deficiencies in mobility. Nevertheless, when a movement fault is observed during shoulder elevation, coaches must investigate further to provide tailored corrective training programs.

Based on the review of the literature presented in previous sections, Figure 1 presents a screening process that may allow coaches to qualify an athlete for overhead lifting based on their movement profile. Clearly, this screen is limited as an exclusive tool, ignoring important risk factors such as injury history (57). Nonetheless, combined with other forms of assessment, the screen is designed to provide coaches with a more thorough appreciation of an athlete’s overhead movement profile.

The bilateral shoulder elevation test (BSET) serves as the primary test, in which clear movement compensations may be identified with some consideration for the underlying causes. Clinically, the BSET has been proposed as a fundamental test to identify movement deficiencies around the shoulder complex (63). In identifying alterations in optimal scapula kinematics, McClure et al. (48) established moderate reliability (0.57) when using the BSET with light loads when categorizing athletes as possessing normal, subtle, or obvious scapula dyskinesis. In simplifying this method, Uhl et al. (71) found a high sensitivity (76%) and high predictive value (74%) by assessing scapula dyskinesis during shoulder elevation using a “yes/no” rating criteria. Tate et al. (68) assessed the validity of the clinical BSET by comparing ST and GH joint kinematics using an electromagnetic-based motion capture system in athletes recognized as

Figure 3. Thoracic extension test. The athlete leans against a bare wall with their knees flexed and feet one foot length away from the wall. With the sacrum, upper back, and occiput all in contact with the wall, the athlete posteriorly tilts the pelvis flattening the lumbar spine. From this position, the athlete attempts to tuck their chin to flatten their thoracic and cervical region. If the athlete is unable to achieve upper cervical flexion, poor thoracic extension is indicated.
having either normal or altered ST joint kinematics during live screening. Their investigation provided clear evidence for the validity of the BSET, by illustrating differences in ST joint kinematics between groups (68).

Using the flow diagram presented (Figure 1), further investigation can subsequently be conducted to gain an understanding of the likely cause of a movement restriction. For the purpose of this article, many of the tests will be abbreviated to a brief description. For each muscle length assessment, normative values will be presented where possible. When normative values are not possible, coaches are encouraged to compare bilaterally.

**BILATERAL SHOULDER ELEVATION TEST**

The BSET (Figure 2) replicates many overhead-based movements seen in a traditional exercise program. Fundamentally, assessing shoulder elevation capacity provides coaches with an appreciation of whether an athlete can achieve full shoulder elevation ROM, as well as which joint segments are contributing to the end ROM. This in itself will allow inferences to be made regarding muscle function, based on the literature previously presented. Additional tests may then be used to verify whether a mobility or stability issue is present. In the event of pain being displayed at any point during testing, it is crucial that strength and conditioning coaches refer the athlete for further medical screening. Until a more thorough assessment has been undertaken, interpreting any movement dysfunction without an appreciation for the underlying pathology will likely prove futile.

It is appreciated by the authors that the BSET presents as a low-load assessment. As the low-load motor control system is regulated by separate higher processes within the central nervous system than that of the high load system (24), this test is clearly limited. However, by first performing the test in a low-load environment, practitioners will be able to identify obvious mobility restrictions. A low-load version of the BSET will also reveal if an athlete has the capacity to achieve full shoulder elevation ROM, regardless of strength. Therefore, a low-load BSET will provide coaches with a baseline understanding of an athlete’s upper extremity movement capability. If an athlete’s movement quality deteriorates after the addition of load, it is highly likely that deficiencies in the high-load system are the underlying cause.
INTERPRETING THE BILATERAL SHOULDER ELEVATION TEST

STARTING POSITION
To appreciate an athlete’s movement profile, practitioners should first identify the athlete’s starting position. This may be accomplished through a static postural assessment before the initiation of the BSET. Although static alignment is a poor predictor of injury occurrence (66), it has been shown to possess a strong influence on shoulder mechanics (70). Combined with dynamic assessments, static posture may provide clues as to possible causes of mechanical dysfunction. For example, when a downwardly rotated scapula is observed during a postural examination, a short levator scapula, rhomboids muscle group, and pectoralis minor muscle may be present (63). This in turn has the potential to limit the capacity to upwardly rotate the scapula dynamically (4). Table 1 presents the ideal static alignment for the upper quarter.

Table 2
<table>
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<tr>
<th>Faulty scapulothoracic position</th>
<th>Possible synergistic muscle imbalance</th>
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<tbody>
<tr>
<td>Excessive elevation</td>
<td>Upper trapezius dominance</td>
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<td></td>
<td>Serratus anterior and lower trapezius inhibition</td>
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<tr>
<td>Excessive depression</td>
<td>Lower trapezius dominance</td>
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<tr>
<td></td>
<td>Upper trapezius inhibition</td>
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<td>Excessive protraction</td>
<td>Serratus anterior dominance</td>
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<td>Trapezius inhibition</td>
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<td>Excessive retraction</td>
<td>Trapezius dominance</td>
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<td></td>
<td>Serratus anterior inhibition</td>
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<tr>
<td>Reduced posterior tilt</td>
<td>Upper trapezius dominance</td>
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<tr>
<td></td>
<td>Serratus anterior and lower trapezius inhibition</td>
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**SPINAL MOTION**
Although little research is available in respect to the amount of thoracic spine extension required during a BSET, much depends on the starting position. If the athlete presents with excessive thoracic kyphosis, more extension should be observed relative to an athlete possessing a normal thoracic curvature. With scapula kinematics being heavily dependent on upper thoracic extension (11), the specific region of spinal extension should be noted. A misleading movement strategy when observing spinal extension during the BSET is for the athlete to extend through the thoracolumbar junction, creating the perception of thoracic extension. This is accompanied by an excessive rib flare, as the subcostal angle increases. When this compensatory strategy is displayed, athletes should be cued on “keeping the ribs down,” so their true shoulder elevation capacity can be observed.

Head position should also be noted, as poor cervical spine alignment may negatively impact upper extremity function (70). Throughout the BSET, the auditory meatus should be aligned over the GH joint, representing ideal cervical spine alignment. In many cases, cervical spine alignment will be a compensation for limitations in other segments involved in accomplishing full shoulder elevation ROM. For example, in individuals with excessive thoracic kyphosis, increased flexion at the cervicothoracic junction and upper cervical extension will present in order to allow the athlete to maintain a horizontal gaze (29). The same may occur when poor ST or GH joint mobility is present. In this instance, athletes may choose to assume a forward head position to complete the objective of moving their hands above their head.

When spinal position during the BSET is faulty, coaches must decide whether the deficiency is caused by a mobility restriction, or a motor control dysfunction. This may be accomplished by using the thoracic
extension test against a wall (Figure 3). Athletes who are unable to accomplish a flattening of the lumbar and thoracic region of the spine combined with a tucking of the chin are assumed to have a mobility restriction. If an athlete presents with good thoracic spine mobility, yet remains in a hyperkyphotic position throughout the BSET, it likely a strength or motor control deficit exists. Here, the athlete should be cued on correct thoracic spine motion, repeating the BSET to assess ST and GH function once the athlete can produce the desired extension pattern.

**SCAPULOTHORACIC MOTION**

As previously described, the scapula should achieve 60° of upward rotation and up to 30° of posterior tilt during full shoulder elevation. To measure upward rotation, practitioners must first locate the medial border of the scapula. Normal scapula position with the arms at rest is such that the medial border lies parallel with spinous processes of the thoracic spine (64). At the top position of the BSET, the medial border should reach 60° relative to the vertical spine, indicating full ST upward rotation. Where deficiencies occur, restriction of the downward rotators should first be investigated. Length tests for the pectoralis minor and levator scapula muscle are shown in Figures 4 and 5, respectively.

If each downward rotator muscle seems extensible and is not impeding scapula motion, then poor performance of the scapula upward rotators may be assumed. In this instance, the orientation of the scapula may provide clues as to where the deficiency lies. For example, if the scapula is excessively elevated, the upper trapezius is likely overriding the serratus anterior and lower trapezius. Equally, if the scapula is held in a position of depression, it is likely the lower trapezius that exhibits dominance. These relationships can be deciphered by using an understanding of functional anatomy around the shoulder complex (Table 2).

Serratus anterior contribution to overall motion may be assessed through locating the inferior angle of the scapula relative to the thorax (63). As the serratus anterior is a protractor of the scapula, it is responsible for moving the scapula around the rib cage to follow the humerus. If the inferior angle of the scapula does not reach the midaxillary line, dominance is assumed in the retractor trapezius muscle when restriction in the rhomboids muscle group is absent.
ST motor control is best assessed during the eccentric phase of the BSET (31). Scapula dyskinesis may be identified through dysrhythmia or winging (48). Dysrhythmia presents when the scapula moves through excessive elevation or depression, or exhibits a stuttering movement during either the concentric or eccentric phase of shoulder elevation (48). Winging is exhibited when the scapula no longer follows the contour of the thorax, and the medial border or inferior angle becomes prominent (Figure 6). If at any point during the BSET either scapula dysrhythmia or winging presents, coaches should assume alterations in dysfunctional neuromuscular control of the ST upward rotators. In this instance, a period of motor control and strength exercises for the ST stabilizers should be programmed as part of the corrective strategy. Until scapula dyskinesis has been corrected, coaches should be cautious in prescribing overhead lifting exercises.

GLENOHUMERAL MOTION

During the BSET, external rotation of the humerus on the scapula may be monitored through observing the position of the hand and elbow joint. With the palm facing the midline, the olecranon process faces laterally, it is likely the GH joint is in a position of internal rotation, potentially closing the subacromial space (5,16). It is important to correspond hand position with orientation of the elbow joint, as to prevent the athlete from creating the perception of GH joint external rotation through elbow supination. A lack of external rotation of the humerus may be caused by a number of medial rotators. As such, the latissimus dorsi (Figure 7), the sternal fibers of pectoralis major (Figure 8), and posterior shoulder cuff (Figure 9A) (namely the teres major muscle) should all be tested for mobility restriction.

Were an athlete to present with adequate ST joint motion, yet unable to produce full shoulder elevation ROM, GH joint flexion/abduction ROM is limited. The sternal fibers of pectoralis major, latissimus dorsi, and the posterior shoulder cuff musculature (Figures 9A and 9B) may be restricting GH joint motion. Again, length tests should be performed to differentiate the involved tissue.

CONCLUSIONS

Abnormal mechanics of the spine, ST, and GH joint have been associated with injury mechanisms around the shoulder complex. As each joint segment shares an influential relationship with the other, dysfunction at 1 region will likely impact the function of another. By understanding common kinematic abnormalities and functional anatomy of the shoulder complex, coaches are able to identify movement impairments in athletes that disqualify them from overhead lifting exercises. In using the assessment process presented in this article, coaches should be able to design individualized corrective programs for athletes where shoulder elevation ROM is limited.

Although this article has introduced a specific screening process, coaches should not feel constrained by the approach presented. It was the goal of this article to provide coaches with the tools to appreciate an athlete’s competency during overhead lifting tasks and locate the potential cause of numerous dysfunctional movement strategies. Kinematic impairments in the shoulder complex may just as easily be identified during any overhead lifting exercise. Therefore, screening of the upper quarter may also be accomplished during a standard training session, as long as an evidence-based thought process is used to guide the coach in identifying and correcting the movement impairment.

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