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CERVICAL CONTRIBUTION TO FUNCTIONAL SHOULDER IMPINGEMENT: TWO CASE REPORTS

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ABSTRACT

Background: Subacromial impingement is a common condition among overhead athletes. The cause of subacromial impingement can be multifactorial and often involves impaired rotator cuff function.

Case Description: The following cases outline the presentation, examination and intervention of two overhead athletes, a high school football quarterback and a collegiate swimmer, each presenting with signs and symptoms of subacromial impingement. The unique feature in each case was the manifestation of the cervical spine as the apparent source of rotator cuff weakness, which contributed to functional subacromial impingement although other overt signs of cervical or associated nerve root involvement were absent.

Outcome: Subsequent to this finding, the athletes demonstrated a rapid recovery of rotator cuff strength and resolution of impingement symptoms in response to cervical retraction and retraction with extension range of motion exercises along with posture correction. They both returned to unrestricted sporting activities within a week, with maintenance of strength and without reoccurrence of symptoms.

Discussion: The signs of functional subacromial impingement often include weakness of the supraspinatus and infraspinatus. The cause of the weakness in the two cases appeared to be the result of stresses associated with forward head posture contributing to a possible intermittent C5 nerve root compression. The findings in the two cases would suggest the cervical spine should be considered as a potential cause of rotator cuff weakness in individuals presenting with subacromial impingement. Future research should examine the influence of cervical postures and shoulder muscle strength.

Level of Evidence: 4

Keywords: Cervical posture, functional subacromial impingement, rotator cuff strength

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BACKGROUND AND PURPOSE

Subacromial impingement (SAI) and shoulder pain are common in those who participate in overhead sporting activities. Pitchers, quarterbacks and swimmers are particularly vulnerable to SAI due to the repetition and velocity of the overhead motions inherent to participation in their respective sports.[1,2,3,4] Impaired shoulder mechanics can lead to the approximation of the humeral head and coracoacromial arch, thereby encroaching upon the intervening structures, resulting in tissue injury. The subacromial bursa, the rotator cuff tendons and the long head of the biceps brachii are often injured due to their occupation of the subacromial space.[5]

Extrinsic and intrinsic factors have been identified as contributing to the development of SAI. Bigliani and Levine report extrinsic factors as those extratendinous conditions that compromise the subacromial space. Extrinsic factors may include space occupying bony anomalies, such as a hooked (Type III) acromion or eburnation and bone spur formation at the distal clavicle, projecting into the subacromial space. Glenouhumeral joint capsular influences offer potential extrinsic SAI factors through either posterior capsular tightness or general capsular laxity. Posterior capsular tightness may contribute to impingement by encouraging anterior humeral head translation toward the coracoacromial arch during shoulder elevation.[6,7,8] On the other hand, general capsular laxity, particularly in the overhead athlete, may result in altered mechanics and subacromial impingement to compensate for subtle subluxations.[5] Lastly, enlargement of the coracoclavicular ligament may compromise the subacromial space, thus providing an additional extrinsic factor, which may contribute to SAI.[5]

Intrinsic factors have been described as those that are intratendinous in nature and may be the result of rotator cuff weakness or fatigue.[5] The repeated exposure of the rotator cuff tendons to high velocity shoulder movements, such as those accompanying throwing and swimming, may lead to overuse strain or tear due to frank tendon overload. Impaired rotator cuff function as a result of strength deficits, endurance limitations, or injury may permit excessive superior humeral head translation during shoulder elevation, resulting in SAI.[5,6,7,8,9,10,11]

SAI resulting from the intrinsic factors of deficient rotator cuff strength, endurance or control, resulting in impaired dynamic stability at the glenohumeral joint and excessive superior migration of the humeral head into the coracoacromial arch has been described by Janda as *functional impingement*.[8] Functional SAI is delineated from *structural impingement* which is the result of the physical narrowing of the subacromial space due to extrinsic factors.[5, 12]

The symptoms associated with functional SAI often readily resolve once the rotator cuff strength deficit has been addressed and normal dynamic stability has been restored to the glenohumeral joint.[5,12, 13]

The causes of rotator cuff weakness in the overhead throwing athlete may include: rotator cuff fatigue secondary to overuse, rotator cuff inhibition due to pain, muscle strain associated with frank tissue overload, and/or periscapular muscle dysfunction, resulting in rotator cuff length-tension issues.[5,8] The author contends that cervical nerve root compression should also be considered as a potential contributor to rotator cuff weakness and, therefore, to functional SAI in the overhead throwing athlete. This will be discussed later in greater detail.

Regardless of the cause of the rotator cuff weakness, the clinical manifestation of functional SAI is often shoulder pain made worse with overhead use, passive shoulder range of motion (ROM) within normal limits, a painful arc of active abduction between 60-120°, and pain and weakness with isometric testing of shoulder abduction and/or external rotation.[5,14,15] Suspicion of functional SAI is often confirmed with a painful response to a battery of special tests, including Neer's impingement maneuver, the Hawkins-Kennedy test and Empty Can (Jobe) test.[14-18] This cluster of symptoms often leads to the clinical diagnosis of functional SAI and the clinician would expect to see impairments of rotator cuff strength, painful range of motion and functional limitations, including compromised performance of overhead movements, particularly throwing and swimming.

The conservative clinical management of functional SAI often involves protection of symptomatic tissues from pain provoking activity, maintenance of shoulder passive ROM and gradual restoration of rotator cuff strength and endurance, while attempting to address any observed possible contributing factors such as muscle length imbalances, impaired scapular muscle control, posterior capsular tightness, poor postural habits or faulty throwing mechanics.[5,12,15]

The following are two cases in which athletes involved in overhead sporting activities presented with symptoms and signs consistent with functional SAI in which the cause for the muscle weakness appeared to be cervical in origin. The unique feature in both cases was that the only apparent sign of cervical involvement was that of weakness throughout the C5 myotome without complaints of radiculopathy, paresthesia, overt pain or restriction of cervical motions.[21,22] Therefore, the purpose of these two case reports is to discuss the presentation, diagnostic process, intervention and outcome of the two cases of functional SAI attributable to cervical dysfunction.

CASE DESCRIPTIONS

Case One

A 5'9", 165 pound, 16 year-old, left-handed, high school varsity quarterback, who was also a pitcher on the varsity baseball team, presented with left shoulder soreness and complaints of a "dead-arm" after participating in routine pre-season passing drills 24 hours earlier. He recalled his shoulder becoming progressively fatigued and sore after making 10-15 medium to maximum velocity throws of 10-20 yards. He did not recall a specific throw or incident leading to his symptoms nor did he experience sensations of sudden or sharp pain, tearing, popping, catching, instability or parasthesias. He denied a history of significant left shoulder symptoms prior to the onset of his current episode. He had not received prior imaging studies or diagnostic testing. His general health was unremarkable. He noted a history of recurrent episodes of cervical stiffness and acknowledged being a habitual cervical "self-manipulator" in that he would manually with overpressure rotate his cervical spine to end range in a rapid and forceful manner.

He also reported having been involved in an extensive off-season conditioning program that had included core, rotator cuff and scapular stabilizing muscle strengthening exercises. Additionally, he had participated in a progressive football throwing program in preparation for the pre-season. He also acknowledged having donned his football helmet for the first time of the season during practice that day.

Observation

The subject was of a mesomorphic build with evidence of left latissimus dorsi and pectoralis major hypertrophy, not uncommon in the dominant side of a throwing athlete.[8] He also presented with mild scapular abduction, suggestive of pectoralis minor/major tightness and a moderate forward head, suggestive of possible suboccipital and upper trapezius tightness.[8] No gross asymmetries were noted with regard to his sagittal spinal alignment.

Physical Examination

The initial physical examination consisted of active ROM with passive overpressure of the cervical spine and shoulders performed in standing.[19] This was followed by shoulder, elbow and wrist resisted isometric strength testing per Cyriax.[19] Active cervical range of motion was within normal limits in all planes of motion with the exception of retraction and retraction with extension, which were both mildly limited. Active cervical motions and active cervical motions with passive overpressure failed to produce symptom complaints. He demonstrated active left shoulder abduction that was visually estimated at 0-175° with a painful arc from 85-95° during both concentric raising and eccentric lowering. Passive left shoulder range of motion (ROM) was visually estimated to be abduction 0-180°, flexion 0-180°, external rotation 0-115° and internal rotation 0-60° all with capsular end feels. Resisted isometric strength testing with his shoulder in neutral tested weak and mildly painful for both the left shoulder external rotators (infraspinatus/teres minor) and abductors (deltoid/supraspinatus). All other left shoulder isometric strength tests were strong and painless. The neutral position of the shoulder is advocated by Cyriax in an attempt to selectively tension the potential contractile tissues at fault without placing the tendons of the rotator cuff in a compromising position or placing undo tension on other inert structures.[19] No left shoulder glenohumeral joint symptoms, asymmetry or instabililty were noted with ligamentous testing. Ligamentous examination included Load and Shift testing for anterior and posterior glenohumeral joint translation and Sulcus sign testing for inferior glenohumeral joint translation. Ligamentous testing was followed by confirmatory special testing including the Empty Can (Jobe) test, Hawkins-Kennedy test and Neer's impingement sign, each of which was positive for symptom reproduction.[14,15](Tables 1 and 2)

The cluster of positive clinical findings, including a painful arc of active abduction, weakness and pain with resisted external rotation and a positive empty can (Jobe) test strongly suggest the likelihood of SAI.[14] This conclusion was further supported by the positive findings with the Hawkins-Kennedy and Neer tests.[15] The painful arc, Hawkins-Kennedy and Neer tests are intended to compress subacromial tissues, while resisted external rotation and the Empty Can (Jobe) test tension the rotator cuff tendons with emphasis to the infraspinatus and supraspinatus, respectively.

Clinical Impression #1

Preliminary clinical diagnosis was of left shoulder symptoms associated with impaired rotator cuff strength (supraspinatus/infraspinatus), resulting in functional subacromial impingement with overhead activities, most notably repeated throwing motions.

Having identified a clinical cluster of symptoms consistent with SAI associated with rotator cuff weakness, further investigation was indicated to search for factors which may have contributed to the impingement beyond the apparent obvious factor of rotator cuff overuse secondary to throwing a football. The patient's comments regarding donning his helmet for the initial time in the season supported the idea that further examination of the cervical spine was warranted, in spite of the lack of apparent significant findings with the initial cervical scan of active motions and overpressures.

The continuation of the cervical examination proceeded with repeated cervical motion testing per McKenzie with the intent to discern the influence that repeated cervical motions may have had on the baseline signs and symptoms previously estab-

Table 1. *Common Responses to Resisted Isometrics (RI) and Interpretation[19]*

Common Responses to Resisted Isometrics	Interpretation
Strong and Painless	No lesion to contractile unit
Strong and Painful	Minor lesion of muscle or tendon
Weak and Painless	Musculotendinous rupture (3^{rd} degree) or peripheral nerve or nerve root involvement
Weak and Painful	Severe soft tissue lesion or fracture with pain induced inhibition.

Table 2. *Testing Results Pre-Post Repeated Cervical Motion Interventions**

Examination Test	Pre-Testing Case One	Pre-Testing Case Two	Post-Testing Case One	Post-Testing Case Two
Painful Arc	Positive	Positive	Negative	Negative
Isometric Shoulder External Rotation	Weak and (mild) painful	Weak and (mild) painful	Strong and painless	Strong and painless
Isometric Shoulder Abduction	Weak and (mild) painful	Weak and painful	Strong and painless	Strong and painless
Empty Can Test (Jobe)	Positive	Positive	Negative	Negative

***Repeated cervical motion testing: Case One= retraction 3x10 reps; retraction with extension 2x 10 reps Case Two= retraction 2x10 reps; retraction with extension 2x 10 reps**

lished.[20] Because no acute distress was noted with initial retraction testing, repeated end range cervical retraction (chin tuck) for three sets of ten was performed since the retraction movements were accompanied with end range tightness that lessened with repetition. Cervical repeated motion testing for retraction ceased at this point due to the resolution of the complaint of end range cervical tightness. This was followed by repeated end range cervical retraction with extension that also demonstrated end range tightness, which lessened with repetition of two sets of ten. Testing for cervical retraction with extension was also discontinued at this point due to the resolution of the complaints of end range tightness with the movement. Repeated cervical motion testing was performed in the standing position.

At this point a re-examination of his left shoulder strength and motion was performed to determine if the repeated cervical movements had influenced the patient's symptoms and signs. Upon re-examination, active left shoulder abduction was performed through full ROM with no painful arc. Resisted isometric testing for left shoulder abduction and external rotation each re-tested as strong and painless. The Empty Can (Jobe) test also re-tested strong and painless.

Clinical Impression #2

It was concluded that the cervical spine had contributed to the patient's left shoulder strength deficits, resulting in a functional SAI. Consequently, both the cervical retraction (chin tuck) and cervical retraction with extension movements would play a major role in the rehabilitative process of this patient.

Intervention

Initial intervention included instruction to avoid head forward postures as much as possible for the ensuing 24 hours. This was implemented due to the acknowledged favorable response the repeated cervical retraction movements had on the patient's symptoms and strength. Accordingly, modifications were made in his standing, sitting and sleeping postures to promote a more neutral position of his cervical spine. Similarly, he was also instructed to perform end range cervical retraction and retraction with extension (10 times each) on an every one to two hour basis in order to reinforce the favorable response demonstrated in the clinic.[20] (Figures 1 and 2).

Figure 1. *Retraction*

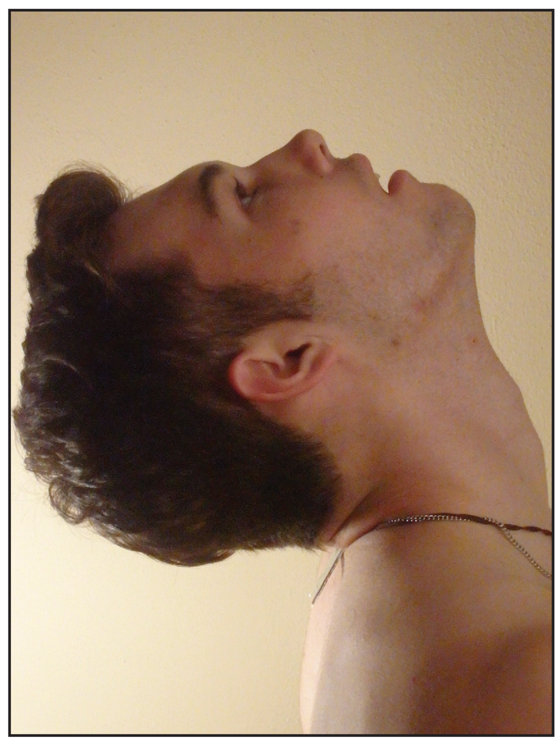

Figure 2. *Retraction with Extension*

He was seen for follow up within 24 hours, and it was noted that his improvement with regard to symptom-free left shoulder motion and normal strength had been maintained. He resumed full participation in football practice, including throwing drills without event that day. A follow up one week after the initial examination revealed that he had maintained his improvement, and he continued with full participation in pre-season football practices without event. He acknowledged ongoing compliance with his prescribed active cervical ROM exercises and posture modifications.

Clinical Diagnosis

Left shoulder symptoms with impaired rotator cuff strength, resulting in functional SAI with overhead activities, most notably throwing, likely due to faulty cervical postures and excessive lower cervical flexion stresses.

Case Two

A 6'- 4'', 185 lb, 22 year old collegiate swimmer presented with bilateral shoulder pain and dysfunction that he associated with a weight lifting session he had performed the previous day as part of his off-season conditioning program. He reported having performed intense straight bar squats, in addition to a series of upper extremity strength training exercises. He denied a specific exercise or movement that led to his symptoms, but believed his symptoms were, in fact, related to the above activities. He denied a significant history with regards to his shoulders. He had no complaints of joint instability or upper extremity parasthesias. He had not received prior imaging studies or diagnostic testing. His general health was unremarkable.

Observation

The subject presented with a mesomorphic build. His standing posture was characterized by a marked forward head, moderately rounded shoulders and a moderate thoracic kyphosis. No deviations were noted with regards to his sagittal spinal alignment.

Physical Examination

Examination of the cervical spine and upper quarter was performed as described by Cyriax with additional cervical examination proceeding with repeated motion testing as described by McKenzie.[19,20] Active cervical range of motion was within normal limits in all planes of motion with the exception of retraction and retraction with extension, which were both mildly limited. Active cervical motions and active cervical motions with passive over-pressure failed to produce symptom complaints. He demonstrated active bilateral shoulder abduction that was visually estimated to be 0-180° with bilateral painful arcs between 80-100° during concentric raising and eccentric lowering. Passive shoulder ROM was full with capsular end feels. Excessive external rotation and normal internal rotation was noted bilaterally. Resisted isometric strength testing, with his shoulder in neutral, tested weak and painful for bilateral shoulder abductors. Bilateral shoulder external rotators were also weak and mildly painful. All other shoulder isometric strength tests were strong and painless. There were no apparent glenohumeral joint instability issues with ligamentous testing of his shoulders bilaterally. Ligamentous testing included Load and Shift testing for both anterior and posterior joint capsules. Inferior translation of the glenohumeral joint was assessed using the Sulcus sign. The Empty Can (Jobe), Hawkins-Kennedy, as well as Neer's Impingement tests were all positive bilaterally.

Clinical Impression #1

Preliminary clinical diagnosis was: bilateral shoulder symptoms associated with impaired rotator cuff strength (supraspinatus greater than infraspinatus), resulting in functional SAI with overhead activities, most notably weight lifting.

Having arrived at an initial physical therapy diagnosis, the examination proceeded in an attempt to identify additional contributing factors to the previously stated impairments. Given his noted postural faults, additional examination of the cervical spine was warranted, in spite of a lack of significant symptom production or apparent associated symptoms with the initial cervical scan. End range cervical retraction was accompanied with end range tightness, which lessened with 20 repetitions. This was followed by end range cervical retraction with extension that was also accompanied with end range tightness that lessened with 20 repetitions. Examination of both cervical retraction and retraction with extension ceased once end range was attained and reports of tightness ceased.

Re-examination of bilateral active shoulder abduction was performed with a noted absence of a painful arc. The Empty Can (Jobe) test, Hawkins-Kennedy and Neer's tests were each now noted to be negative bilaterally. Resisted isometrics for bilateral shoulder abductors and external rotators each were strong and painless on re-examination. (Table 2) The patient was asked to perform repeated end range active cervical protrusion times twenty repetitions in an attempt to reproduce the patient's strength deficits and associated symptoms. Protrusion is a cervical movement that accentuates the forward head posture to end range followed by a return to the neutral position. This was done in a slow, repetitious fashion to assess the effect of the cervical motion opposite to that which was found to have a favorable impact on his symptoms and strength, namely cervical retraction. No cervical discomfort was reported during the performance of the repeated active cervical protrusion, however, upon completion, the bilateral painful arc returned, the Empty Can (Jobe) test was once again positive and resisted isometrics for the shoulder abductors and external rotators were again found to be weak and painful.

The performance of an additional twenty end range cervical retraction movements and twenty end range cervical retraction with extension movements resulted in the abolishment of the bilateral painful arc, the return of normalcy for the Empty Can (Jobe) test and a return of abductor and external rotator strength that was strong and painless.

Intervention

In a manner similar to the intervention described in Case One, the patient was instructed to sit, stand and sleep in postures that reinforced a more neutral cervical position as opposed to his habitual head forward posture. He was encouraged to use a lumbar roll to assist in the maintenance of his lumbar lordosis when sitting. He was also asked to perform end range cervical retraction and end range cervical retraction with extension exercises for ten repetitions each on an hourly basis throughout the day. He was seen for follow up within 24 hours, and his improvement for symptom reduction and rotator cuff strength was maintained. Subsequent follow up one week later revealed that he had maintained his improvement and had stated ongoing compliance with his prescribed cervical active ROM exercises and posture modifications.

Clinical Diagnosis

Final clinical diagnosis for this subject was bilateral shoulder symptoms associated with impaired rotator cuff strength, resulting in functional SAI with overhead activities, most notably recreational weightlifting, likely due to faulty cervical postures and excessive lower cervical flexion stresses.

DISCUSSION

The observation that movements of the cervical spine influence upper quarter signs and symptoms and function is not novel. A number of authors have described characteristic patterns of symptoms, myotomal strength deficits, and hypotonic reflex changes which affect the upper extremity to varying degrees when a cervical nerve root has been irritated.[21-25]. The characteristics of the symptoms and signs present in the upper quarter are dictated by the cervical nerve root level that is involved. Typically, cervical active movements are restricted and produce symptoms of pain and/or paresthesia that may extend or radiate into the upper extremity. Cervical extension and both cervical lateral bending and rotation to the side of the irritated nerve root are likely to produce or increase symptoms due to the narrowing of the intervertebral foramen which accompanies each movement.[26] The most common cervical nerve root levels involved in radiculopathy are C6 and C7, followed by C5.[22,27]

Consequently, the characteristic symptoms and signs associated with involvement of the C6 nerve root may include pain over the lateral forearm into the thumb and 2nd digit, sensory changes over the thumb and 2nd digit, strength deficits in the biceps brachii and wrist extensors, and reflex changes of the biceps brachii.[22,27]

Characteristics of C7 nerve root irritation may include pain that may extend into the medial scapular region, dorsum of the forearm and 3rd digit, sensory changes that extend over the dorsum of the forearm and 3rd digit, potential strength deficits of triceps brachii and wrist flexors, and reflex changes of the triceps brachii.[22,27]

Characteristic of C5 nerve root involvement symptoms and signs may include pain over the medial scapula and lateral aspect of the arm, sensory changes present over the lateral aspect of the arm, strength deficits of the deltoid, supraspinatus and infraspinatus, and reflex changes of the supinator.[22,27]

What is of particular interest to the author in the two described cases is the clinical expression of the apparent cervical involvement. The cervical motions themselves were not particularly limited and failed to demonstrate aberrant results that would have initially suggested the cervical spine was involved or at fault. Nor were the cervical motions accompanied by pain during the movement or at end range of any particular movement that would have indicated involvement. Additionally, the cervical movements failed to be accompanied with any of the characteristic upper extremity symptoms indicative of cervical nerve root involvement, namely, pain or parasthesia in the noted distributions.[22,27] Admittedly, reflex testing was omitted from the exam for expediency, and extensive sensory testing was not performed due to each patient's subjective report of normalcy. The primary clinical sign that appeared to be associated with the cervical spine was that of shoulder muscle weakness that appeared to dominate the C5 myotome (deltoid, supraspinatus, infraspinatus). It was, in turn, surmised that the noted strength deficit had compromised normal shoulder mechanics, resulting in a functional SAI. The clinical sign of shoulder muscle weakness of a cervical origin without cervical pain or reports of associated sensory impairment is the unique feature in these cases, and this warrants discussion.

The rather immediate improvement in rotator cuff strength and normalization of shoulder function in response to repeated end range cervical retraction followed by end range cervical retraction with extension range of motion exercises is in need of an explanation. The author contends a plausible explanation is that muscle weakness was produced through an intermittent C5 nerve root compromise induced by forward head postures, resulting in a possible transient conduction block to the C5 myotome. Compressive forces between 20-30 mm Hg can impair neural blood flow, and subsequently, may result in compromised nerve function.[28] The reduction in blood flow is believed to reverse once the compression is removed without apparent residual nerve damage.[28] However, compressive forces of 50 mm Hg, for periods as little as two minutes duration, have been shown to result in damage to the myelin and axon.[28] Therefore, if an intermittent compromise of the C5 nerve root is the cause of the rotator cuff weakness in the above cases, it is surmised that the compression was of a magnitude sufficient to impair nerve *function*, yet not to cause nerve *damage*.

Periods of accentuated forward head postures and mild limitations in cervical retraction and retraction with extension AROM were common to the subjects in both cases and may suggest that each was vulnerable to a form of cervical nerve root compression. The cause of the compression is open to debate, particularly given the proposed involvement of the C5 nerve root and the paucity of information regarding segmental vertebral kinematics involving the cervical spine in either the protruded (forward head) or retracted (chin tuck) positions.

The literature suggests the most common causes of cervical nerve root compression are from either posterior bulging of an intervertebral disc or by intervertebral foraminal stenosis. [22-24,27] The following discusses the plausibility of the C5 nerve root being compromised under either condition.

Considering that the C5 nerve root exits the spinal column superior to the C5 vertebra, a disc bulge would likely occur at the C4-C5 level in order to compromise the C5 nerve root. Although the C5-C6 and C6-C7 levels are more likely to demonstrate posterior disc protrusion, resulting in nerve root compression on the C6 and C7 nerve roots respectively, a number of studies indicated the C4-C5 disc level is subject to degeneration and protrusion, and therefore, a potential source of compression to the C5 nerve root.[25,27,29,30] Matusmato et al[30] in an MRI study of asymptomatic subjects noted that 15% of the subjects demonstrated posterior disc protrusions at the C4-5 level. Okada et al[29] in a 10-year longitudinal study, using MRI, reported that 25% of the C4-C5 posterior disc protrusions, originally identified, progressed in severity over the course of the study. Wainner et al[27] in their study reported that 2 of the 20 cervical radiculopathies identified likely involved the C5 nerve root. Furthermore, Kim et al's[25] study of 1,305 consecutive patients undergoing primary cervical surgery reported 16.6% of those subjects under the age of 40 had C5 nerve root involvement. In the same study, 39.4% of patients between ages 40-60 and 48.1% of those over 60 had procedures for C5 nerve root involvement.

A disc bulge, resulting in nerve root compression, would likely be due to flexion at the C4-C5 motion segment, causing the annulus to deform and bulge posteriorly. A radiographic study by Orway et al, analyzing the segmental vertebral kinematics of the cervical spine in the protruded (head forward) position, suggests that the C4 vertebra is in a position of relative flexion to the C5 vertebra of 6.3$\pm$ 4.1°.[31] The flexed position places an offset flexion load on the C4-C5 disc level, thus creating a potential mechanism for the nucleus pulposus of the disc to migrate, possibly contributing to a posterior disc bulge. Cervical retraction, in the same study, was demonstrated to begin to extend C4 relative to C5, however, the study suggests the motion segment remains flexed 4.5$\pm$4.4°.[31]

Orway et al[31]demonstrated that the cervical motion of retraction followed by extension results in extension of C4 relative to C5 of 9.5$\pm$ 3.9°. This provides additional theoretical evidence for decreasing a potential C5 nerve root compression (caused by a mildly bulging disc) by encouraging the extension of C4 on C5.[31]

Stenosis of the intervertebral foramen has also been presented as a potential source of cervical nerve root irritation, and is typically thought of in an aging population. Although stenosis is generally associated with degenerative changes that accompany aging, and the two cases involved young adult males, a study by Anderst et al offers information that may shed insight on a potential explanation for C5 nerve root compression through an intervertebral foramenal stenosis mechanism. Their study evaluated segmental cervical kinematics during active flexion and extension in asymptomatic subjects (46$\pm$9 yrs) using a bi-planar X-ray system. An anterior shear of C4 on C5, on the magnitude of 33%, was reported to occur preceding end range cervical flexion.[32] This anterior shear is likely to result in a narrowing of the anterior/posterior dimension of the intervertebral foramen as the inferior articular process of C4 moves toward the posterior aspect of the C5 uncovertebral joint as the C4 body shears anteriorly. Cervical flexion is not identical to the protrusion, which accompanies the head forward posture, but similarities do exist in the lower cervical spine. Ordway et al demonstrated C4 to flex on C5 9.5 $\pm$ 3.1° during full flexion, while C4 flexed on C5 6.3 $\pm$ 4.1° during full protrusion in their kinematic study. It is reasonable to assume that an anterior shear of C4 on C5 would also occur during the lower cervical flexion that accompanies protrusion and the forward head posture, however, to a lesser degree than observed during full flexion.[31] Consequently, a foraminal stenosis at C4-C5 may be created by end range lower cervical flexion resulting from time spent in a head forward posture, which, in turn, may be a potential source of C5 nerve root compression.

The potential for the cervical retraction movement to reduce a C5 nerve root compression due to stenosis is supported by Lentell et al,[33] who suggested an increase in both the vertical and transverse dimensions of the C4-C5 intervertebral foramen after moving from the neutral to the retracted cervical posture. Cervical retraction resulted in an 11% increase in foramenal area compared to the neutral cervical position. Their study examined the cervical spines of 20 healthy, asymptomatic 22-25 year old subjects, using MR imaging in the supine position.[33] Cervical retraction, according to data provided by Lentell et al, increases the space available for the C5 nerve root to exit the spinal column, possibly reducing compression of a compromised nerve root in a young adult population.

Evidence has been provided supporting the incidence of the C5 nerve root being involved in cervical pathology. Evidence has also been presented in an attempt to explain the unique findings of the two case reports. The author of this case series has proposed that either a mildly, posterior bulging cervical intervertebral disc encroaching on the C5 nerve root or a temporary, mild intervertebral canal stenosis is the potential mechanism of C5 nerve root compression. Nerve root compression from either source could potentially result in the supraspinatus and infraspinatus muscle weakness, as was seen in these two cases, and can help explain the changes in the shoulder symptoms and signs previously reported.

Therefore, both retraction and retraction with extension are cervical movements that could potentially reduce irritation to a mildly compressed nerve root, regardless of etiology. Retraction has two potential mechanisms by which this may occur, first, with cervical retraction, C4 begins to extend on C5. This cervical extension, though limited in magnitude and failing to reach neutral, may be sufficient to counteract a mild disc bulge pushing on the C5 nerve root.[31,32,35,36] Second, the increase in intervertebral foraminal area that accompanies retraction as suggested by Lentell, may be adequate to decompress a C5 nerve root that has been compromised by a stenosis-like mechanism.[31,32]

The retraction with extension movement is likely to further counter the stress to a mildly bulging posterior annulus by encouraging anterior migration of the nucleus pulposus through offset loading.[31,32,35] The converse was demonstrated by Tampier, using a porcine model, where the cyclic loading of a cervical motion segment into flexion resulted in anterior compression of the annulus fibrosis and posterior movement of the nucleus pulposus, resulting in herniation.[34] Anderst et al in vivo and Skrzypiec et al in vitro each demonstrated cervical extension that resulted in compression of the annulus posteriorly.[32,34] It is then theorized that extension of a cervical motion segment, (assuming a disc with an intact hydrostatic mechanism) may possibly result in the movement of the nucleus pulposus in an anterior direction, in response to cervical extension, as has been demonstrated to occur in the lumbar spine.[36]

In the first case, the donning of the football helmet for the initial time of the season could have contributed to the lower cervical flexion stresses. Thus, the potential for posterior nucleus pulposus migration or an intervertebral foraminal stenosis was increased.

The subject in the Case Two had been performing straight bar squats as part of his weight lifting regimen prior to the onset of his symptoms. The lifting technique he described, with the bar resting across his shoulders posterior to his cervical spine, likely reinforced his forward head posture and may have contributed to a posterior disc bulge or an intervertebral foraminal stenosis. This, combined with the examination finding of a decrease in muscle strength following performance of repeated end range cervical protrusion and an increase in muscle strength and reduction of associated impingement signs following performance of repeated end range cervical retraction and retraction with extension, would suggest a lower cervical extension exercise preference and support for a temporary avoidance of flexed lower cervical postures.

The age of the subjects in the current cases should be discussed. The subjects were age 16 and 22. The typical ages of those diagnosed with cervical radiculopathy have been reported to be in their 30's and 40's, with incidence peaking in their early 50's.[22,37] However, Matsumoto, in a MRI evaluation of asymptomatic cervical intervertebral discs, reported posterior disc protrusions in 17% of male subjects in their twenties. The most common cervical level to demonstrate protrusion was C5-C6 and C6-C7, followed by C4-C5.[30] If muscle weakness in the associated myotome is the only sign of nerve compression, as suggested in these cases, it is difficult to determine what the actual incidence of occurrence is or at what age the weakness or other symptoms commence.

An acknowledged factor that should be mentioned, which may have influenced the outcome in the current cases, is the effect of posture on the subacromial space. Borstad and Ludewig[38] reported a reduction in subacromial space in the presence of pectoralis minor tightness. In both cases, pectoralis minor tightness was suspected, due to the varying degree of rounded shoulder posture observed during the initial examination. Posture instruction, in the form of attempting to maintain an erect cervical posture as much as possible, was part of the intervention in each case. Seitz et al[39] reported an increase in subacromial space during active upper extremity elevation, in response to a scapular repositioning manuever. Lewis et al[40] reported an increase in active forward flexion and abduction ROM in both asymptomatic and symptomatic shoulders, in response to taping and posture correction. Consequently, the posture correction that accompanies cervical retraction, which was prescribed as a part of the exercise plan in these two current cases, could have played a role in shoulder posture thereby affecting the painful arc.

The explanation for the clinical observation of improved rotator cuff strength and shoulder function in apparent response to the performance of repeated cervical retraction and retraction with extension range of motion movements warrants further investigation. If, in fact, a nerve root conduction block resulted in the sole neurologic sign of rotator cuff weakness leading to functional SAI, as suggested by the author, clinicians should be mindful to thoroughly examine the cervical spine and look for cause and effect on shoulder strength and function, even though the cervical movements may not be associated with cervical pain or marked cervical ROM impairments. Otherwise, the impairment of rotator cuff weakness becomes a principle focus of the rehabilitation, while the underlying contribution of the cervical spine may go undetected, making reoccurrence of functional SAI likely.

Additionally, since these patients, once identified, respond in a rapid fashion, the benefits from both outcome and economic perspectives are important.

CONCLUSION

Although weakness of the rotator cuff has long been associated with SAI and has been the direct focus of intervention, the cause for the weakness and the remedy in a number of cases may, in fact, be found by looking more closely at the cervical spine, even when outward symptoms and signs may suggest otherwise. The author contends a population exists that experiences a significant loss of rotator cuff strength following periods in protruded cervical (forward head) postures. Similarly, the author suggests research focusing directly on the effect of various cervical postures on rotator cuff strength be conducted to shed further light on the relationship between the cervical spine and shoulder function.

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