

Original article

Sensorimotor contribution to shoulder stability: Effect of injury and rehabilitation[☆]

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Abstract

Shoulder joint stability is the humeral head remaining or promptly returning to proper alignment within the glenoid fossa. This is mediated by both mechanical and dynamic restraint mechanisms. Coordination of these restraint systems is required for shoulder joint stability. The sensorimotor system is defined as all of the sensory, motor, and central integration and processing components involved in maintaining joint stability. The sensorimotor system is comprised of several components including proprioception, joint position sense, kinesthesia, sensation of force, and neuromuscular control. With joint injury, not only are the mechanical restraints disrupted (joint capsule, glenoid labrum, etc.) but also, the sensorimotor system is affected. Restoration of the sensorimotor system has been shown to occur through both surgical and conservative intervention and rehabilitation. Surgery has been shown to restore both mechanical restraints and the sensorimotor system. Specific rehabilitation techniques have also been effective at improving the sensorimotor system in healthy and pathological patients.

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1. Introduction

Stability is defined as the state of remaining unchanged, even in the presence of forces that would normally change the state or condition (Thomas, 1993). Applying this definition to the shoulder, glenohumeral stability is the state of the humeral head remaining or promptly returning to proper alignment within the glenoid fossa through an equalization of forces. Joint stability is mediated by both mechanical and dynamic restraints. Mechanical restraints include the glenohumeral joint capsule, glenohumeral and extracapsular ligaments, glenoid labrum, bony geometry and intra-articular pressure. Dynamic restraint results from

activation and force production by the muscles surrounding the shoulder.

Functional joint stability is defined as possessing adequate stability to perform functional activity and results from the interaction between the mechanical and dynamic restraints mentioned above (Lephart et al., 2000). As separate entities, neither the mechanical nor dynamic restraints can act alone in providing functional joint stability, but requires a mechanical-dynamic restraint interaction to achieve a stable shoulder. This mechanical-dynamic restraint interaction is mediated by the sensorimotor system.

2. Sensorimotor system

The sensorimotor system is defined as all of the sensory, motor, and central integration and processing components involved in maintaining joint stability

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(Riemann and Lephart, 2002a,b). For example, the mechanical restraints about the shoulder not only provide physical restraint to the humeral head, but also contribute to stability by providing neural feedback (proprioception) to the central nervous system (CNS) where it is integrated with other somatosensory, vestibular, and visual input, and ultimately results in the generation of efferent control over the dynamic restraints about the shoulder joint (neuromuscular control). Proprioception is defined as the afferent information, arising from peripheral areas of the body (including the mechanical and dynamic restraints about the shoulder) that contributes to joint stability, postural control, and motor control (Lephart et al., 2000; Riemann and Lephart, 2002a,b). Proprioception has three submodalities including joint position sense, kinesthesia, and sensation of force (Riemann and Lephart, 2002a,b). Joint position sense is the appreciation and interpretation of information concerning one's joint position and orientation in space. Kinesthesia is the ability to appreciate and interpret joint motions (Myers and Lephart, 2000). Sensation of force is the ability to appreciate and interpret force applied to or generated within a joint (Myers and Lephart, 2000). Proprioceptive information originates at the level of the mechanoreceptor. Mechanoreceptors are peripheral afferent sensory neurons present within muscle, tendons, fascia, joint capsule, ligaments, and skin about a joint (Kikuchi, 1968; Grigg, 1994; Vangness et al., 1995). Mechanoreceptors are mechanically sensitive receptors that transduce mechanical tissue deformation as frequency modulated neural signals to the central nervous system (CNS) through afferent sensory pathways (Grigg, 1994). Mechanoreceptors including pacinian corpuscles, ruffini endings, golgi tendon organs, and muscle spindles have been identified at the shoulder (Vangness et al., 1995; Solomonow et al., 1996; Gohlke et al., 1996; Ide et al., 1996; Gohlke et al., 1998).

Neuromuscular control is the subconscious activation of the dynamic restraints about the shoulder in preparation and in response to joint motion and loading for the purpose of maintaining joint stability (Myers and Lephart, 2000). These neuromuscular control mechanisms include coordinated muscle activation during functional tasks, coactivation of the shoulder musculature (force coupling), muscular reflexes, and regulation of muscle tone and stiffness (Myers and Lephart, 2000, 2002). The forces provided by the muscles about the shoulder maintain centralization of the humeral head within the glenoid while still allowing for a high degree of mobility.

3. The effects of injury on the sensorimotor system

Injury to the stabilizing structures of the shoulder (capsuloligamentous, articular, and musculotendinous)

whether caused by a traumatic or atraumatic mechanism, results in mechanical instability. Accompanying physical disruption of the mechanical stabilizers is decreased capsuloligamentous-musculotendinous mechanoreceptor stimulation thus altering the sensorimotor contribution to dynamic restraint and joint stability (Lephart and Henry, 1996). This combination of mechanical deficits and sensorimotor alterations contribute to deficits in functional stability (Lephart and Henry, 1996). Ultimately, the deficient function may contribute to reinjury patterns often seen at the shoulder joint. For example, with an acute glenohumeral dislocation-subluxation, the mechanical restraints including glenohumeral joint capsule, glenohumeral ligaments, and glenoid labrum are compromised. Yet within those structures are mechanoreceptors that contribute proprioceptive information to the sensorimotor system that ultimately provides neuromuscular control over the dynamic restraints about the shoulder. Thus with joint injury, not only are the mechanical restraints affected, but also the sensorimotor contribution to dynamic stability is affected.

Several studies have shown that instability at the shoulder has deleterious effects on proprioception (Smith and Brunolli, 1989; Lephart et al., 1994; Zuckerman et al., 2003; Barden et al., 2004). Both joint position sense and kinesthesia are altered in patients diagnosed with glenohumeral instability (Smith and Brunolli, 1989; Lephart et al., 1994; Zuckerman et al., 2003; Barden et al., 2004). Accompanying the disruption of the mechanical stabilizing structures, it is believed that decreased capsuloligamentous mechanoreceptor stimulation resulting from tissue deafferentation and/or the increased tissue laxity limiting mechanoreceptors stimulation, thus decreasing proprioception (Lephart and Henry, 1996; Tibone et al., 1997). Barden et al. (2004) demonstrated errors bilaterally in joint position sense in subjects exhibiting unilateral instability. These results suggest that alterations in the central processing mechanisms may also be present. Interestingly, Tibone et al. (1997) reported that no significant differences existed between normal subjects and subjects with instability, using cortical evoked potential. Given that joint capsule mechanoreceptors were stimulated with electrical potentials rather than tissue deformation, these results suggest that capsular laxity alone rather than mechanoreceptor trauma resulting in deafferentation is responsible for proprioception deficits.

Proprioceptive deficits have also been identified in patients diagnosed with osteoarthritis (Cuomo et al., 2005). Proprioceptive deficits were attributed to decreases in shoulder muscle activity levels combined with local muscle atrophy (Cuomo et al., 2005). Additionally, the increased afferent signals sent by pain receptors about the shoulder were believed to override and subsequently decrease proprioception afferents. The

work by Safran et al. (2001) supports the role of pain in adversely affecting proprioception. These results demonstrated that throwers with shoulder pain have decreased proprioception most likely due to increased nociceptor activity in the painful shoulder of baseball players Safran et al. (2001). Subacromial impingement has also been linked to proprioceptive deficits. Machner et al. (2003) demonstrated decreased kinesthesia in subjects diagnosed with unilateral stage II subacromial impingement. The authors theorized that the subacromial bursa was deficient in relaying the movement sense signals (Machner et al., 2003).

Given the proprioceptive deficits associated with shoulder joint injury, neuromuscular control is hypothesized to be altered as well (Myers and Lephart, 2000, 2002). Several investigators have assessed the neuromuscular control component of dynamic joint stability in subjects presenting with glenohumeral instability (Glousman et al., 1988; Kronberg et al., 1991; McMahon et al., 1996; Myers et al., 2004). Muscle activation alterations were identified in patients with glenohumeral instability during both simple elevation tasks (Kronberg et al., 1991; McMahon et al., 1996) and while throwing a baseball (Glousman et al., 1988). Deficits in coactivation of the rotator cuff and primary humeral movers were present, possibly leading to compromised dynamic joint stability and further exacerbating the existing instability. Our laboratory recently assessed reflexive characteristics of the shoulder muscles in patients diagnosed with anterior glenohumeral instability (Myers et al., 2004). The patients with instability demonstrated suppressed pectoralis major and biceps brachii mean reflexive activation, significantly slower biceps brachii reflex latency, and suppressed supraspinatus-subscapularis coactivation. The results suggested that in addition to the capsuloligamentous deficiency and proprioceptive deficits present in patients with anterior glenohumeral instability, muscle activation alterations are also present. The suppressed rotator cuff coactivation, slower biceps brachii activation, and decreased pectoralis major and biceps brachii mean activation may contribute to the recurrent instability episodes seen in patients with glenohumeral instability.

Muscle activation abnormalities associated with subacromial impingement and rotator cuff lesions have also been identified (Ludewig and Cook, 2000; Reddy et al., 2000; Kelly et al., 2005). Common findings include increased activity in the middle deltoid, decreased activity in the supraspinatus, infraspinatus, and subscapularis, decreased coactivation of the rotator cuff musculature, and suppressed scapular stabilization by the trapezius and serratus anterior muscles during elevation. Kelly et al. (2005) assessed activation of the rotator cuff during functional tasks and demonstrated that patients with symptomatic rotator cuff tears exhibit activation alterations that may limit functional perfor-

mance compared to both asymptomatic and normal subjects. Our laboratory recently identified that patients with subacromial impingement exhibited less subscapularis-infraspinatus, supraspinatus-subscapularis, and supraspinatus-infraspinatus coactivation (Myers et al., 2003). Increased middle deltoid and latissimus dorsi activity was exhibited by the impingement patients. The results indicate that patients with subacromial impingement exhibit suppressed rotator cuff coactivation and abnormal humeral mover alterations during humeral elevation. These demonstrated muscle activation alterations may contribute to impingement of the subacromial structures and subsequent pain during overhead elevation in patients with subacromial impingement.

4. Sensorimotor restoration

There is evidence to suggest that the sensorimotor contributors to joint stability can be restored. For example, surgical intervention to restore mechanical stability has a demonstrated benefit in restoring proprioception (Lephart et al., 1994, 2002; Zuckerman et al., 2003; Potzl et al., 2004; Cuomo et al., 2005). The main goal of surgery for glenohumeral instability is to reestablish mechanical restraint to the humeral head. Yet as reported by several investigators, the surgery was also successful at restoring proprioception (Lephart et al., 1994, 2002; Zuckerman et al., 2003; Potzl et al., 2004; Cuomo et al., 2005). It is believed that by reestablishing tension with the glenohumeral joint capsule and ligaments, that mechanoreceptor stimulation is also reestablished (Lephart et al., 1994, 2002; Zuckerman et al., 2003; Potzl et al., 2004; Cuomo et al., 2005). Mechanoreceptors may also repopulate the joint capsule allowing reinnervation following surgery as a normal part of the histological healing process (Lephart et al., 1994, 2002). Potzl et al. (2004) found an increase in proprioception bilaterally following unilateral surgical intervention, thus hypothesizing an alteration in central mediation of proprioception may also contribute to normalization of proprioception.

Subacromial decompression was also found to restore proprioception in patients with subacromial impingement (Machner et al., 2003). It was suggested that the painful subacromial bursa (and subsequent resection) was the cause for the initial deficit and subsequent restoration of proprioception. These results are supported by Cuomo et al. (2005) who found that both measures of kinesthesia and joint position sense returned to normal levels following total shoulder arthroplasty (Cuomo et al., 2005). It was suggested that a decrease in pain afferents with greater mechanoreceptor afferent activity following surgery was the mechanism for improved proprioception (Machner et al., 2003; Cuomo et al., 2005). Other potential mechanisms for

restoration of proprioception following surgery included retensioning of the joint capsule and surrounding musculature, and restoration of anatomical alignment through greater joint congruence following arthroplasty (Cuomo et al., 2005).

As with any injury, rehabilitation should address inflammation and pain reduction, a return to normal range of motion and flexibility, and restoration of strength through traditional rehabilitation exercises. Yet return to vigorous physical activity and athletic participation requires additional rehabilitation considerations. Lephart and Henry (1995) promote the use of functional rehabilitation for return to athletic and highly demanding activities of daily living (Lephart and Henry, 1996). A large component of functional rehabilitation is the ability to replicate the demands placed on the joint in a controlled manner to decrease the initial impact upon return to physical activity. Some of the expected benefits of functional rehabilitation include increasing proprioceptive awareness, increasing dynamic stabilization, eliciting preparatory and reactive muscle activation, and restoration of functional movement patterns (Lephart and Henry, 1996). Proprioceptive awareness training is believed to reestablish afferent pathways from the mechanoreceptors to the central nervous system, and facilitate supplementary afferent pathways as a compensatory mechanism for proprioceptive deficits that resulted from joint injury (Myers and Lephart, 2000). Dynamic stabilization is paramount in restoring functional joint stability and should focus on restoring both coordinated muscle activation patterns during functional tasks as well as muscle coactivation and the resulting force coupling restraint. Elicitation of preparatory and reactive muscle activation around the shoulder helps to establish reflex loops and muscle stiffness around the joint thus creating stability during destabilizing events. To further ease the transition from rehabilitation to the functional demands of sports or occupation, allowing controlled simulation of tasks is beneficial. Recreating the activities which will be required of the joint during sports in the clinical setting allows a controlled environment to practice and evaluate techniques prior to actual specific performance.

There is some evidence of the effectiveness of exercise in restoring sensorimotor mechanisms at the shoulder. Shoulder plyometric training has been shown to increase proprioception in swimmers (Swanik et al., 2002). It was theorized that repeated eccentric loading and subsequent length/tension changes in the shoulder stabilizers at end-range of motion, created increased proprioceptive awareness of by both the mechanical and dynamic stabilizers (Swanik et al., 2002). Additionally, increases in central processing may have resulted from performing the repeating, perturbing plyometric tasks. This creates increased muscle tension in preparation to the task being performed, which may have increased awareness of joint

position (Swanik et al., 2002). Furthermore, both open and closed kinetic chain exercises have been shown to cause improvements in joint position sense at the shoulder (Rogol et al., 1998).

It has also been shown that closed kinetic chain upper extremity activities facilitates co-activation of the muscles around the shoulder, increasing functional joint stability (Ubinger et al., 1999; Henry et al., 2001). By utilizing closed chain exercises, an increase in joint stability can be obtained by creating greater joint congruency and stimulation of articular mechanoreceptors (Ubinger et al., 1999; Henry et al., 2001). There also appears to be a central component trained during closed chain exercise, as increases were in both shoulders in subjects training unilaterally (Ubinger et al., 1999).

It has also been shown through a randomized controlled trial that enhancing neuromuscular control through exercises designed to enhance coactivation about the shoulder leads to faster recovery of chronic shoulder pain than natural course of recovery (Ginn and Cohen, 2005). It was also shown to be equivalent in recovery time as steroid injection and physical modalities. The authors advocate retraining as it is more cost effective than modalities and has less inherent risk than injection (Ginn and Cohen, 2005).

5. Summary

Functional joint stability results from a coordination of both mechanical restraints (provided by capsuloligamentous, articular, and musculotendinous structures) and dynamic restraints that result from contraction of the musculature that surrounds the joint. Acting independently, neither the mechanical nor dynamic restraints alone can provide joint stability. There must be coordination between the mechanical and dynamic restraints. The coordination between the mechanical and dynamic restraints is mediated by the sensorimotor system. The sensorimotor system includes the sensory, motor, and central integration/processing components of the central nervous system that contribute to joint stability. Sensory information provided by the joint (proprioception) travels through afferent pathways to the central nervous system, where it is integrated with information from other levels of the nervous system. The central nervous system, in turn, elicits efferent motor responses (neuromuscular control) vital to coordinated movement patterns and functional stability. With joint injury, not only are the mechanical restraints disrupted (instability, lesion, etc.) but also, the sensorimotor system is affected. Demonstrated deficits in both proprioception and neuromuscular control accompany joint injury. Both surgical intervention and rehabilitation have been demonstrated to restore not only the

mechanical restraints that are disrupted with injury, but also the sensorimotor contributors to joint stability.

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