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Sensorimotor Factors Affecting Outcome Following Shoulder Injury

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When the shoulder is subjected to an injurious mechanism, a cascade of effects results. These effects include tissue pathology and the manifestation of pain. Sensorimotor alterations also manifest, most likely as a result of tissue pathology and pain. The combination of the tissue pathology, pain, and sensorimotor alterations all directly affect outcome following injury, and thus need to be addressed by the clinician treating the shoulder injury to fully restore function. This article discusses how the sensorimotor system contributes to shoulder function and how it is altered with shoulder injury, thereby affecting outcome.

SENSORIMOTOR CONTRIBUTION TO SHOULDER FUNCTION

During activities of daily living, performance of occupational duties, and participation in sport and recreation, more range of motion is required at the shoulder than any other joints. The shoulder range of motion that is observed during functional activities results from the compounding motion achieved by the three joints and one articulation of the shoulder complex, with most of the motion coming from the glenohumeral joint and scapulothoracic articulations. For example, during humeral elevation, if scapulothoracic movement is eliminated, maximum humeral elevation decreases by approximately one third [1–5]. During full humeral elevation, approximately 120 degrees of the overall movement occurs at the glenohumeral joint and approximately 60 degrees occurs at scapulothoracic articulation [1,5]. At the same time, movement at the sternoclavicular joint allows the clavicle to elevate 7 to 15 degrees, retract 15 to 30 degrees, and axially rotate by 15 to 33 degrees [6,7].

To provide this high level of mobility, the glenohumeral joint and scapulothoracic articulation have limited osseus stability [8]. The glenohumeral joint compensates for this limited osseous stability by relying on the other mechanical stabilizing mechanisms achieved by the static structures, including negative

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intra-articular pressure, the glenoid labrum, tenomuscular structures, and capsuloligamentous restraints. The static mechanical restraints alone are not enough to provide adequate stability. The shoulder complex therefore relies heavily on dynamic action of the musculature that crosses both the glenohumeral and scapulothoracic articulations to maintain stability while still allowing high mobility.

These static and dynamic mechanisms of stability do not happen independently. The static structures (especially capsuloligamentous structures) influence activation of the muscles that cross the shoulder complex, thus providing stability, through stimulation of mechanoreceptors within the sensorimotor system. The sensorimotor system includes the sensory, motor, and central integration/processing components of the central nervous system (CNS) [9]. Mechanoreceptors are sensory neurons present within joint capsules, ligaments, muscles, tendons, fascia, and skin about a joint [10–12]. Mechanoreceptors are mechanically sensitive and transduce mechanical tissue deformation as frequency-modulated neural signals to the CNS through afferent sensory pathways [10]. Vangsness and colleagues [12] reported that neural endings exist in the shoulder's capsuloligamentous structures. The spiral tightening of the capsule that occurs with abduction and external rotation sequentially tightens the capsuloligamentous structures, and is therefore considered to stimulate those mechanoreceptors [13]. Low-threshold, slow-adapting Ruffini afferents were most abundant overall, except in the glenohumeral ligaments where low-threshold, rapid-adapting Pacinian-type afferents outnumber Ruffini afferents [12]. Ruffini afferents are believed to be stimulated only in extremes of motion through tensile force, acting as limit detectors [10]. Like Ruffini receptors, Pacinian corpuscles respond in extremes of motion, but through combination of compressive and tensile mechanisms, rather than stretching alone [10]. No mechanoreceptors were present in the subacromial bursa or glenoid labrum [12].

Sensory information from these mechanoreceptors travels from the shoulder joint through afferent pathways to the CNS where it is processed and integrated with input from the other levels of the nervous system (central processing), eliciting contractile muscle responses vital to coordinated movement patterns and joint stability [9]. This information is termed proprioception and 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 [9,14,15]. Proprioception has three submodalities, which include joint position sense, kinesthesia, and sensation of force [14,15]. 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 [16]. Sensation of force is the ability to appreciate and interpret force applied to or generated within a joint [16].

These submodalities of proprioception are used by the CNS to elicit appropriate neuromuscular control mechanisms important for joint stability and

coordinated movement of the shoulder complex. Neuromuscular control is defined as 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 and function [16]. The 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 [16,17].

Coordinated muscle activation refers to the muscle activation patterns necessary to properly perform the desired functional task, including the combined effort of concentric contraction by the agonist muscles, eccentric contraction or reflexive inhibition of the antagonist muscles, and activation of synergistic muscles about the shoulder complex. Proper coordination of the muscles that cross the shoulder complex results in fluid, coordinated completion of the desired task while achieving dynamic stability of the joint.

Coactivation of the dynamic restraints at the shoulder joint is vital to shoulder stabilization. The rotator cuff is essential for dynamic stability by centralizing the humeral head within the glenoid fossa, thus preventing excessive humeral translation [18]. For example, contraction of the subscapularis counteracts contraction of the infraspinatus/teres minor, controlling the excessive humeral translation in the frontal plane, while contraction of the deltoid counteracts contraction of the lower rotator cuff muscles (infraspinatus, teres minor, and subscapularis), controlling the excessive humeral translation in the transverse plane [19]. The resultant force exerted by the rotator cuff muscles produces glenohumeral joint compression, which in turn increases congruency of the articulating surfaces [20]. In addition to the synergistic action of glenohumeral musculatures, the common insertion of the rotator cuff tendons within the joint capsule provides an element of dynamic capsular tension. As the cuff muscles simultaneously contract, the forces generated in their tendinous insertion apply tension to the joint capsule [21–23]. The result is increased capsular tension that aids in drawing the humeral head into the glenoid fossa, supplementing joint stability.

Muscle reflexes also play an essential role in dynamic stability of the shoulder. Jerosch and colleagues [24] arthroscopically demonstrated that a reflex arc (ligamento-muscular reflex) exists between the shoulder capsule and the deltoid, trapezius, pectoralis major, and rotator cuff musculature in the human shoulder. Traditionally, the ligamento-muscular reflex research has centered around the alpha motor neuron being the efferent pathway to the muscle. Although these afferent alpha motor neuron reflexes do exist, the gamma motor-muscle spindle system is also a plausible mechanism that mediates shoulder stability by way of reflex. Specifically, gamma motor-muscle spindle system modulates sensitivity of the alpha motor neuron, thus affecting muscle stiffness and ultimately joint stability [25–28]. Joint stiffness is defined as resistance provided by tissue, joint, or limb to a change in shape and position [29]. It provides the first line of defense for joint stability when force is applied to the joint [30–35]. It provides an immediate and substantial response to perturbation

and decreases the latency of the reflexive response, thus improving joint stability [36]. The stiffness provided by the muscles about the shoulder plays a substantial role in how effectively external forces imposed on the musculoskeletal system are transmitted to the CNS [37]. Muscle stiffness is strongly influenced by the level of contraction present [38]. As the intensity of muscle contraction increases, so does stiffness [32,39–42]. Mechanoreceptors play a significant role in regulating muscle stiffness. The muscle spindle system contributes to preprogramming muscle stiffness [27]. Ligament mechanoreceptors can also regulate stiffness by heightening muscle spindle sensitivity by way of increased gamma motor neuron excitation, which influences the amount of muscle stiffness and quickening the stiffness achieved from reflexive muscle activation [25–28,43,44].

EFFECTS OF INJURY ON SENSORIMOTOR SYSTEM

When shoulder injury occurs, tissue pathology and pain result. It is hypothesized that sensorimotor alterations also manifest, resulting from the tissue pathology and pain. The combination of the tissue pathology, pain, and sensorimotor alterations all are believed to directly affect shoulder function and ultimately outcome following injury (Fig. 1). Most of the research investigating the affects of injury on sensorimotor system has been targeted toward examining the sensorimotor alteration in patients who have shoulder instability. Effects of rotator cuff diseases, such as impingement and rotator cuff tears, on the sensorimotor system have also been studied.

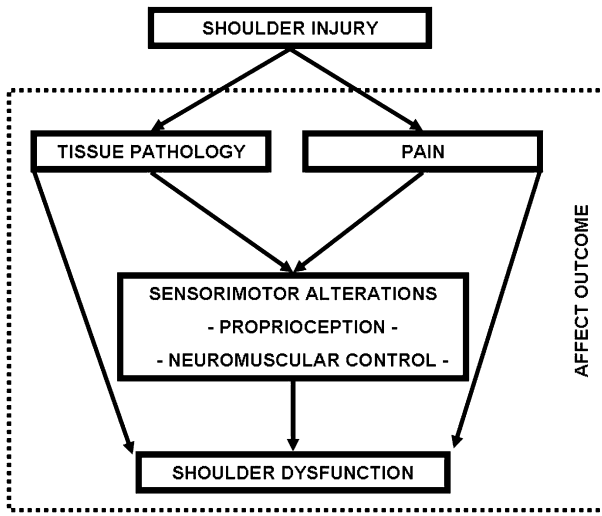

Fig. 1. Effects of shoulder injury on shoulder function.

Sensorimotor Alterations with Shoulder Instability

Sensorimotor alterations have been identified in patients who have various levels of shoulder instability. With traumatic glenohumeral instability, it is believed that the mechanical stimulation of the mechanoreceptors is suppressed because of tissue deafferentation from the trauma or the tissue lengthening (both ligamentous and muscular) [20,45]. Specifically, proprioception deficits, as measured with joint position sense and kinesthesia assessments, have been demonstrated in patients who have shoulder instability [46–48]. A review of the proprioception literature suggests that the tissue lengthening associated with instability is the prime culprit for the sensorimotor deficits. For example, Tibone and colleagues [45] reported that no significant differences in proprioception were present between normal subjects and subjects who had instability, using cortical evoked potential. Given that joint capsule mechanoreceptors were stimulated with electrical potentials rather than from tissue deformation, these results suggest that decreased mechanoreceptor stimulation from capsular laxity, rather than the deafferentation, may be responsible for proprioception deficits in patients who have shoulder instability. In addition, proprioception deficits have been identified in individuals who have atraumatic multidirectional instability [49]. This finding again suggests that the decreased mechanical stimulation rather than tissue deafferentation is the culprit. Patients who have undergone either open or arthroscopic repair to restore capsular tension also demonstrate improved proprioception after surgery [47,48,50–52], suggesting the tissue lengthening and subsequent lack of mechanoreceptor stimulation are associated with the proprioception deficits seen in patients who have instability. Although proprioception is typically improved after surgery, deficits still potentially exist. Fremerey and colleagues [53] demonstrated persisting proprioceptive deficit and an altered EMG pattern following capsulolabral reconstruction. Interestingly, Barden and colleagues [49] demonstrated bilateral deficit in joint position sense in subjects exhibiting unilateral instability. Potzl and colleagues [52] found an improvement in proprioception bilaterally following unilateral surgical intervention. These results suggest that alterations in the central processing mechanisms may also be present.

Neuromuscular control deficits have also been identified in individuals diagnosed with various degrees of instability. Several investigators have assessed the neuromuscular control components of dynamic joint stability in subjects presenting with anterior glenohumeral instability [54–57]. Alterations in coordinated muscle activation pattern were identified in patients who had glenohumeral instability during simple elevation tasks [55,56] and while throwing a baseball [57]. Deficits in coactivation of the rotator cuff and primary humeral movers were present in these patients, possibly leading to compromised dynamic joint stability and further exacerbation of the existing instability [54]. Similarly, Myers and colleagues [54] assessed reflexive characteristics of the shoulder muscles in patients diagnosed with anterior glenohumeral instability. The patients who had instability demonstrated suppressed pectoralis major and biceps brachii mean reflexive activation, significantly slower biceps brachii

reflex latency, and suppressed supraspinatus-subscapularis coactivation. The results suggest that in addition to the capsuloligamentous deficiency and proprioceptive deficits present in patients who have 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 who have glenohumeral instability. To date, there is no research that specifically examines the effects of injury on shoulder muscle stiffness characteristics.

The altered neuromuscular control mechanisms seen in patients who have instability are believed to be related to altered joint function. For example, individuals who have multidirectional instability demonstrate proprioceptive deficits [49], neuromuscular alterations (altered muscle firing patterns) [56,58,59], and altered movement patterns [60,61]. The coexistence of these conditions in patients who have instability suggests strong association and potential cause-and-effect relationship among the conditions. The glenohumeral instability and the dysfunctional neuromuscular control may together contribute to the dysfunction associated with instability.

Sensorimotor Alterations with Rotator Cuff Disease

Sensorimotor alterations have been identified in patients who have various degrees of rotator cuff disease ranging from subacromial impingement to rotator cuff lesions. From a review of the literature, the most likely culprit of the sensorimotor dysfunction is the pain associated with the impingement or rotator cuff tear. Assessment of proprioception in patients who have rotator cuff disease has been limited. Machner and colleagues [62] 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 because of the compression and pain [62]. Safran and colleagues [63] demonstrated that throwers who have shoulder pain have proprioception deficits and suggested that increased nociceptor activity in the painful shoulder overrode proprioceptive input.

Muscle activation abnormalities associated with subacromial impingement and rotator cuff lesions have also been identified [64–69]. Common findings include altered activity of the primary humeral movers, 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 and colleagues [66] assessed activation of the rotator cuff during functional tasks and demonstrated that patients who have painful rotator cuff tears exhibit activation alterations in muscles around the injured shoulder that may limit functional performance compared with both the asymptomatic side and shoulders of normal participants, suggesting that pain is the major contributor to the altered muscle activation patterns seen in patients who have rotator cuff tears. Bandholm and colleagues [68] induced experimental shoulder pain by a bolus injection of

6% hypertonic saline into the supraspinatus muscle of healthy participants and demonstrated altered activation of the middle deltoid and infraspinatus and lower trapezius. These results demonstrate the potent effect pain has on muscle activation patterns. Research using anesthesia models to examine the role of pain on shoulder function has demonstrated muscle activation patterns, strength, and movement patterns that better reflect normal muscle activation and movement in patients who have rotator cuff tears following lidocaine injection [70–73].

Like shoulder instability, the sensorimotor alterations associated with rotator cuff disease are believed to result in decreased function. For example, individuals who had subacromial impingement and rotator cuff tears demonstrated altered scapular movement patterns, including decreased scapular upward rotation, increased scapular anterior tipping, increased scapular retraction, and decreased scapulohumeral rhythm, during a functional overhead task [64,72,74,75]. Bandholm and colleagues [69] demonstrated deficits in force steadiness (defined as maintenance of submaximal isometric contraction) and maximum strength deficits in patients who had subacromial impingement or rotator cuff tears. Patients who have rotator cuff pathology have strength deficits in shoulder elevation and rotation movement patterns [71,76].

SUMMARY

The shoulder complex relies heavily on dynamic action of the musculature that is mediated by the sensorimotor system to maintain stability and allow for coordinated action by the four articulations involved with shoulder motion. When the shoulder sustains an injury, tissue pathology and pain result. Sensorimotor alterations also manifest, most likely a result of the tissue pathology and pain. Sensorimotor deficits in the form of proprioception and alteration in neuromuscular control have been demonstrated in shoulders with various degrees of instability and rotator cuff disease. The combination of the tissue pathology, pain, and sensorimotor alterations directly affect outcome following injury, and thus need to be addressed by the clinician treating the shoulder injury to fully restore function.

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