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Sensorimotor Deficits Contributing to Glenohumeral Instability

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The conventional perspective has been that capsuloligamentous structures act as a mechanical restraint to humeral translation at the shoulder. Although this is true, the capsuloligamentous structures also have a sensorimotor influence on the musculoskeletal system, providing stability at the shoulder. The purpose of the current study was to discuss the sensorimotor role that the capsuloligamentous structures play in providing stability, how these mechanisms are disrupted with glenohumeral instability, and how surgical intervention restores such mechanisms. Proprioceptive information transmitted from the mechanoreceptors embedded within the capsuloligamentous structures influence the coordinated motor patterns, reflex activity, and joint stiffness to provide enhanced joint stability. The capsuloligamentous injury that occurs with shoulder instability not only affects mechanical restraint, but also alters this proprioceptive input to the central nervous system. As a result of these deficits in proprioception, alterations in reflex activity and motor programs as evident by muscle firing pattern alterations manifest. Although the main goal of surgical intervention is to restore the mechanical restraint that is lost with joint dislocation or subluxation, surgical intervention whether through open, arthroscopic, or thermal techniques seem to restore the proprioceptive deficits that exist after joint injury.

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The mechanical role capsuloligamentous structures play in providing glenohumeral joint stability is commonly accepted in the orthopaedic community. Glenohumeral ligaments and the joint capsule cradle the humeral head to provide mechanical restraint to the humeral translation, especially at end ranges of motion.[7,18,45] However, capsuloligamentous structures cannot solely provide sufficient stability for functional activities.[4] Glenohumeral stability is achieved by capsuloligamentous structures working together with well-balanced musculature.[4] As such, capsuloligamentous restraints also influence the behavior of the shoulder musculature through neurologic mechanisms, specifically the sensorimotor system. It is this synergistic relationship between the capsuloligamentous structures and musculature surrounding the shoulder where sufficient stability is provided. The purpose of the current review was to discuss the sensorimotor role that capsuloligamentous structures play in providing joint stability, how these sensorimotor mechanisms are disrupted by glenohumeral instability, and how surgical intervention restores such mechanisms.

Role of the Sensorimotor System

Capsuloligamentous structures influence musculature thereby providing stability to the shoul-

der via the sensorimotor system. The sensorimotor system includes the sensory, motor, and central integration and processing components of the central nervous system.[34] Sensory information provided by the shoulder (proprioception) travels through afferent pathways to the central nervous system, where it is integrated with information from other levels of the nervous system.[33,58] The central nervous system, in turn, elicits efferent motor responses (neuromuscular control) vital to coordinated movement patterns and functional stability.

Although proprioception has gained attention only within the past decade, Sherrington[52] first discussed this concept approximately 1 century ago. Proprioception is defined as the afferent neural input originating from mechanoreceptors about the shoulder.[34,42] Neural input concerning joint position sense, kinesthesia (joint movement sense), and forces application to the joint can be appreciated consciously. However, this information also is received subconsciously and used for joint stability mechanisms described in the current study.[34,42] Mechanoreceptors are sensory neurons present within the ligaments, joint capsule, muscle, tendon, and skin about the shoulder.[15,27,59] These mechanically sensitive neuronal endings transduce mechanical tissue deformation as frequency modulated signals to the central nervous system through afferent pathways.[15] As tissue deformation occurs, either through voluntary movement or joint perturbation (unexpected joint position changes), release of stored sodium by the mechanoreceptors provides neural input to the central nervous system.[15,38] Specifically, at the shoulder, Vangsness et al[59] reported that neural endings exist in the capsuloligamentous structures. 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.[59] Ruffini afferents are thought to be stimulated only in extremes of motion through tensile force, acting as limit detectors.[15] Similar to Ruffini receptors, Pacinian corpuscles respond in extremes of motion, but through compressive and tensile mechanisms, rather than stretching alone.[15] No mechanoreceptors were present in the subacromial bursa or glenoid labrum.[59]

Afferent proprioceptive information originating from the mechanoreceptors travels to the spinal cord through afferent neurons. These afferent neurons may synapse directly with the alpha motor neurons, gamma motor neuron, or interneurons.[10] Many of the interneurons provide the basis for sensory integration and motor control at the spinal level, whereas others form the ascending tracts (spinocerebellar and dorsal lateral) leading to higher central nervous system structures, such as the cerebellum and cerebral cortex. The cerebellum provides control over dynamic restraints by planning and modifying motor activities and comparing the intended movements with the outcome of actual movement whereas the cerebral cortex provides conscious appreciation of tissue deformation about the joint.[10]

Popular opinion has been that the primary function of capsuloligamentous receptors is to elicit direct reflexive activation of the alpha motor neuron. Through direct electrical and mechanical stimulation of joint ligaments, capsule, or both,[3,28,29,46,55,56] several investigators have shown that a spinal reflex exists between fibrous joint capsule and musculature about the glenohumeral joint in felines.[17,29,55,56] Jerosch et al[22] followed up the feline model research by showing a similar reflex arc between the shoulder capsule and the deltoid, trapezius, pectoralis major, and rotator cuff musculature in a human model. Some criticisms exist when describing this capsuloligamentous reflex as a provider of stability. Correlating reflex activity resulting from electrical stimulation of the capsule to normal physiologic function remains speculative and uncertain at best. A common criticism with mechanical stimulation studies is that the relative high loading required to elicit alpha motor neuron responses is above those forces experienced during in vivo situations.[21,24,49] Also, the latency associated with reflexive contraction from the alpha motor neuron stimulation may be too long to provide stability. This latency consists of the interval that exists between application of a perturbing force, and the myoelectrical phenomenon associated with

muscle contraction.[2,24,47,49] Electromechanical delay also must be considered. Electromechanical delay is the latency between myoelectrical onset and the actual force production within the muscle necessary for stability.[9,13,14,30] The force resulting from the reflexive response probably will not sufficiently absorb the energy necessary to protect the joint.[47]

Capsuloligamentous mechanoreceptors also seem to directly influence gamma motor neuron activation, which may be the most functional aspect related to joint proprioception. Using traction forces (< 5 N) below those associated with tissue damage and nociceptor stimulation has produced potent effects on gamma motor neuron by capsuloligamentous mechanoreceptors.[11,23,25,26,41,49,54] The increased gamma motor neuron activation facilitates joint stability by controlling muscle spindle sensitivity and indirectly adjusts muscle stiffness. Increased muscle stiffness yields enhanced joint stiffness and therefore is thought to augment joint stability through an elevated potential to resist sudden joint displacements.[16,24,37,40] This enhanced ability to absorb additional energy from destabilizing forces may shield the ligaments from bearing the responsibility of stability in isolation. In unstable joints with associated ligamentous laxity, stiffer muscles also may reduce the incidence of joint instability.

Using an in vivo perturbation model, the authors' laboratory currently is examining the role that reflex activity plays in joint stiffness for providing glenohumeral stability. Preliminary observations suggest that reflex latencies decreased whereas stiffness increases because of increased muscle activity. These changes in stiffness and reflex latency probably result from the increased intrafusal muscle fiber (muscle spindle) sensitivity existing as a function of coactivation accompanying extrafusal muscle fiber activation.

Sensorimotor Deficits With Shoulder Instability

Disruption of the stabilizing structures, static and dynamic, whether caused by a traumatic or nontraumatic mechanism results in glenohumeral joint stability. This joint instability is accompanied by decreased proprioception as mechanoreceptor stimulation is diminished from either deafferentation or soft tissue lengthening.[32,57] The combination of capsuloligamentous disruption and proprioceptive deficits contribute to functional instability. Figure 1 shows the cyclic role that mechanical instabil-

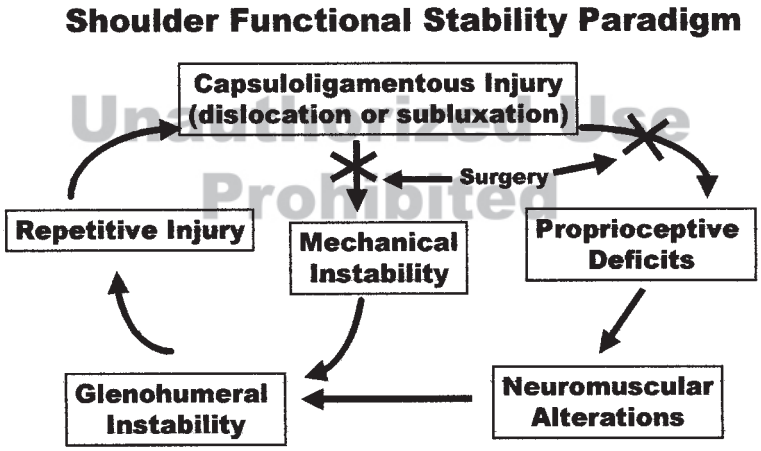

Fig 1. A shoulder functional stability paradigm shows the insidious cycle that results from the combination of mechanical instability, proprioceptive deficits, and neuromuscular alterations of dynamic restraints. Surgical intervention blocks this cycle by restoring the mechanical restraint and proprioceptive mechanisms. (Modified with permission from Lephart S, Henry T: The physiological basis for open and closed kinetic chain rehabilitation for the upper extremity. J Sport Rehabil 5:71–78 1996.)

ity, proprioceptive deficits, and alterations in the neuromuscular mechanisms play in causing joint instability.

Smith and Brunolli[53] were the first to show decreased proprioception (kinesthetic deficits) after shoulder injury using individuals with unilateral instability. Lephart et al[35] compared the subjects' ability to detect passive motion (kinesthesia) and passively reproduce joint positions in healthy individuals, individuals with instability, and individuals who had surgical repair. Significant deficits in kinesthesia and joint position sense were present in subjects with instability as compared with healthy subjects and those who had reconstruction. Forwell and Carnahan[8] showed the inability of individuals with instability to do a manual-pointing task. Using cortical evoked potentials, Tibone et al[57] reported that no significant differences existed between healthy subjects and subjects with instability; indicating that, although the mechanical properties of the capsuloligamentous structures were compromised, the afferent pathways still were intact. These results suggest that capsular laxity alone, rather than mechanoreceptor trauma resulting in deafferentation, is responsible for the proprioception deficits as seen with kinesthesia and joint position sense testing. Blasier et al[5] reported decreased kinesthetic sense in subjects diagnosed with hypermobility but no history of instability or injury. In the absence of mechanoreceptor trauma, the results again indicate that capsular laxity (resulting from hypermobility) decreases proprioception. Allegrucci et al[1] focused on kinesthetic awareness in athletes who participate in overhead activities. Those authors reported decreased kinesthesia in the dominant limb of athletes who participate in overhead activities when compared with the nondominant limb. This decrease may result from the general capsular laxity present in athletes who participate in overhead activities and again indicates that increased capsular laxity may account for proprioceptive deficits.[1] Sainburg et al[50] showed that patients lacking proprioception were unable to do multijoint movements that mimic a slicing gesture. The results suggest that a proprioceptively deficient joint disrupts coordinated movement at other joints along the kinetic chain by altering the motor program.

The resulting deficits in proprioception after joint injury seem to contribute to alterations in the motor program and muscle recruitment patterns during movement in humans. Glousman et al[12] measured muscle activity during pitching using fine wire electromyography in subjects with anterior glenohumeral instability. The authors showed increased compensatory supraspinatus and biceps brachii activity in individuals with instability to accommodate for a lack of glenohumeral stability. In addition, Glousman et al[12] reported decreased subscapularis, pectoralis major, latissimus dorsi, and serratus anterior activity during the late cocking phase of pitching in individuals with instability. This decreased activity may be problematic because the shoulder relies on activation by these muscles for anterior stability especially in positions of vulnerability, such as the late cocking phase of pitching.[12] Kronberg et al[31] showed decreased anterior and middle deltoid activity with shoulder flexion and shoulder abduction in subjects with instability. This disrupted deltoid activity may alter the force couple action that exists between the deltoid and rotator cuff muscle vital to functional stability. McMahon et al[39] showed that individuals with anterior instability have decreased supraspinatus muscle activity during abduction and scaption, and decreased serratus anterior activity during abduction, scaption, and forward flexion. This disrupted activity data suggest that force couple mechanisms existing between the deltoid and rotator cuff and scapular stabilization mechanisms vital to functional stability and coordinated movement patterns are effected. Although proprioceptive deficits and resulting alterations in the motor programs associated with instability have been shown, no data exist that examine alterations in the influence of capsuloligamentous laxity on the stiffness characteristics of the shoulder.

Surgical Restoration of Sensorimotor Mechanisms

Surgical treatment disrupts the insidious cycle of instability by restoring capsuloligamentous in-

tegrity and restoring proprioceptive capabilities (Fig 1). Surgical techniques such as variations of the capsular shift and thermal capsulorraphy address the capsuloligamentous trauma that results from injury, restoring mechanical restraint.[6,48] Surgical treatment also plays a significant role in restoring the proprioceptive capabilities of the shoulder after injury. Surgery retensions the capsuloligamentous structures, facilitating proprioceptive feedback by allowing mechanical stimulation of the afferents present within the joint capsule and ligaments.[35,57]

Restoration of joint position sense and kinesthesia has been shown in individuals with instability who had open or traditional arthroscopic capsular shifts.[35] These data indicate that restoration of capsular tension also restores proprioceptive feedback. Zuckerman et al[60] did a prospective study in which 30 individuals with unilateral glenohumeral instability of traumatic origin were measured with a joint position sense and kinesthetic testing protocol 1 week before surgery, and at 6 and 12 months after surgery. The subjects had a significant decrease in joint position sense and kinesthesia compared with healthy subjects before surgery, partial restoration by 6 months, and full restoration 12 months after surgery.[60]

A contemporary surgical procedure gaining popularity in the orthopaedic community is the use of thermal energy through radiofrequency devices and/or lasers to address mechanical instability (thermal capsulorraphy).[6,44] Although thermal capsulorraphy has been received with much enthusiasm, data concerning its effectiveness are anecdotal. No substantial clinical studies exist addressing the efficacy of this new technique. Given that thermal energy denatures the collagenous infrastructure of the shoulder capsule,[19,20,51] much controversy exists as to whether the mechanoreceptors present within the shoulder capsule also may be altered.

Myers et al[43] evaluated joint position sense, kinesthesia, and shoulder function in subjects who had thermal capsulorraphy for shoulder instability. These data revealed no significant difference in kinesthesia or active and passive reproduction of joint position sense, 6 to 24 months after surgery. These data mimic results previously reported by Lephart et al[35] because subjects treated with thermal capsulorraphy had similar results for joint position sense and kinesthesia measures compared with subjects who had traditional surgical procedures and healthy subjects. In addition, these subjects returned to near normal daily function as measured with a shoulder rating questionnaire[36] at the time of testing. Prospective investigation with long-term followup ($>$ 2 years) of thermal capsulorraphy and its effect on proprioception, neuromuscular control, and function still needs to be addressed. Similarly, no data currently exist to focus on the restoration of joint stiffness properties after surgical intervention.

Stability at the shoulder results from not only the mechanical restraint provided by the capsuloligamentous structures that surround the joint, but also the role these structures play by influencing on the dynamic restraints that surround the shoulder joint. Proprioceptive information transmitted from the mechanoreceptors embedded within the capsuloligamentous structures influence the motor programs, reflex activity, and the stiffness present at the joint to provide stability. Capsuloligamentous injury that occurs with joint subluxation or dislocation not only affects mechanical restraint, but also alters proprioceptive input. From these deficits in proprioception, alterations in the motor program become manifest. Although the main goal of surgical intervention is to restore the mechanical restraint that is lost with joint dislocation or subluxation, surgical intervention (whether through open, arthroscopic, or thermal techniques) seems to restore the proprioceptive deficits that exist after joint injury. Future directions should focus on establishing the role that joint injury has on the reflexive characteristics and stiffness properties associated with joint stability and how well surgical intervention restores such mechanisms associated with joint stability.

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