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Shoulder Proprioception: Function and Role in Rehabilitation

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I. Introduction

Shoulder pain and dysfunction is a common problem among the general population. An estimated 50% of the general population suffers with shoulder pain at some point in their life and up to 50% of those individuals seek medical attention (30). Physical therapy is ordered in approximately 24% of individuals seen in primary care for shoulder pain (30). Approximately 33% of patients seeking medical attention reported an injury associated with their shoulder pain and 21% were work-related injuries (30). The most common diagnosis codes from primary care include rotator cuff syndrome (9.6%), shoulder pain (8.5%), osteoarthritis (5.6%), tendonitis (4.8%), but 99 different diagnosis codes were utilized by primary care physicians in this study (30). Shoulder instability is one cause of shoulder pain that will often result in a referral to physical therapy. Shoulder instability is a clinical syndrome in which joint laxity produces symptoms (8) and is often but not always associated with subluxations and dislocations. Shoulder instability can be categorized into traumatic or atraumatic instabilities depending on the mechanism of injury and further classified into minor or major instability depending on the severity of tissue damage. Atraumatic instability has also been called microtraumatic instability and can be the cause of secondary impingement (27). Secondary impingement is shoulder pain that occurs without trauma or dislocation but involves excessive humeral head translation that may be due to capsuloligamentous laxity or poor neuromuscular control which will later be described in detail.

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Approximately 95% of shoulder dislocations are from a traumatic event including falling on an outstretched arm or a forceful collision (8). Minor incidents account for the other 5% of dislocations (8). Recurrent dislocations are very common (70%) and usually occur within 2 years of the first dislocation (8). The adolescent population is more likely to suffer from recurrent episodes than the older population (8). No studies were found that reported on the prevalence of atraumatic instabilities or secondary impingement. One possible reason for this paucity in literature is difficulty in defining and accurately categorizing atraumatic shoulder instabilities.

Proprioception is a term to describe our ability to sense joint position, or detect movement (kinesthesia) and force application (2, 20) through feedback from mechanoreceptors in and around the joint to our spinal cord and central nervous system. Proprioceptive deficits in the presence of shoulder instability have been clearly established (15,21,29,31). More recently, deficits in proprioception have been linked to osteoarthritis (4) and impingement (18,27). Shoulder rehabilitation often includes exercises and techniques designed to address proprioceptive deficits. This paper will 1) discuss components of shoulder stability including the role of proprioception, 2) describe common clinical presentations that often require proprioceptive training during rehabilitation, and 3) review evaluation and treatment of proprioception in the shoulder complex.

II. Definitions and Anatomical Bases of Passive and Active Stabilizing Structures

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Shoulder joint stability is defined as proper alignment of the humeral head in the glenoid fossa, regardless of arm position, through equalization of forces from passive structures (e.g. joint capsule and glenohumeral ligaments) and dynamic structures (e.g. muscles of the rotator cuff) (19). Shoulder instability occurs when the integrity of these structures are compromised and will be referred to as passive or active instability depending on which structures are at fault. Additionally, shoulder stability relies on a neuromuscular control system, deficits in this system will be referred to as functional instability. Each of these instabilities is further defined below.

Passive instability refers to impairments in the static stabilizers of the shoulder including the glenoid labrum, the glenohumeral ligaments, and the joint capsule (16). The labrum

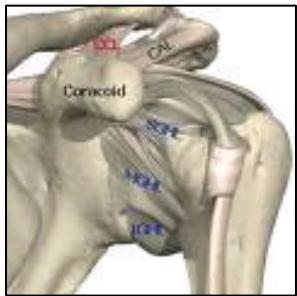


Figure 1. Ligaments of the glenohumeral joint.

provides stability by increasing the depth of the socket, enlarging the contact area for the humeral head, and serving as an attachment point for the glenohumeral ligaments (16). The glenohumeral ligaments are thickenings in the joint capsule and provide stability by preventing excessive translation of the

humeral head (1) (See Figure 1). The inferior glenohumeral ligament consists of three components: the anterior band, the axillary pouch and the posterior band (16). It is often referred to as a complex. The anterior and posterior bands are thought to limit anterior, posterior and inferior translations depending on arm position (5,16). The inferior glenohumeral ligament complex is the primary passive restraint against anteroinferior subluxation (5). The superior glenohumeral ligament arises from the anterosuperior labrum and inserts above the lesser tubercle (16). This ligament, along

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with the coracohumeral ligament, limits external rotation and abduction (5) and acts as a stabilizer against inferior subluxation from 0 to 50 degrees of abduction (5,22). The middle glenohumeral ligament runs from the anterosuperior labrum to the lesser tubercle where it blends with the subscapularis tendon just medial to its insertion (5,16). It is thought to provide anterior stability between 45 and 60 degrees of abduction (5,16). At neutral, the joint capsule of the glenohumeral joint is quite loose allowing one inch of distraction of the humeral head from the fossa in the absence of ligament or muscular restraints (22). In a healthy joint, movement of the arm into combined abduction and external rotation will cause the joint capsule and glenohumeral ligaments to become taut and provide passive restraint to anterior and inferior translation of the humeral head (22). This same position is often symptomatic when there has been damage to the passive restraints of the glenohumeral joint (5). Passive instability may occur secondary to macrotrauma (e.g., dislocation) (8, 15, 23, 31) or microtrauma (e.g., repetitive use) (6, 8, 25). Atraumatic laxity may present as a developed instability but have an underlying genetic predisposition (6).

Active stability is achieved in three ways: 1) muscle contraction of the rotator cuff provides joint compression, 2) direct attachments of the rotator cuff to GH ligaments enhances the stabilizing function of the ligaments, and 3) coordinated contraction of the rotator cuff and scapular muscles maintain good alignment of the humeral head in the glenoid fossa during active movement (16). The rotator cuff includes the supraspinatus, infraspinatus, subscapularis, and teres minor muscles. Important scapular muscles include the trapezius (upper, mid and lower), serratus anterior and the rhomboids. These muscles

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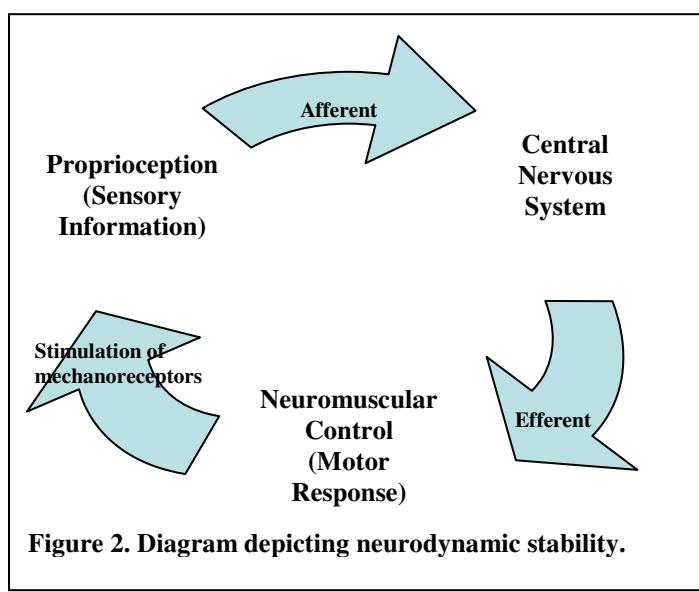
in addition to a few others (e.g. the deltoid and the long head biceps) work together in various combinations called force couples. A force couple is when opposing forces create a pure rotation (22). For example, the subscapularis, infraspinatus and teres minor muscles provide an inferior translation in opposition to the strong superior translation of the deltoid during elevation (22). If the rotator cuff is not working properly to oppose the superior translation of the deltoid, the humeral head will not maintain a proper position in the glenoid fossa (22). The humeral head will superiorly translate, likely impacting the coracoacromial arch in mid-range and potentially contribute to shoulder dysfunction and pain (22). Another example of a force couple in the shoulder complex is the coordinated contraction of the trapezius and serratus anterior muscles. These muscles can be called scapular stabilizers. They affect the position of the scapula and therefore the position of the glenoid with upper extremity movement. For example, during shoulder elevation these muscles work simultaneously to produce upward rotation of the scapula (22). Disruption of these synergistic actions may lead to alterations in the centers of rotation and excessive excursion of the humeral head in the glenoid fossa. Many force couples contribute to coordinated movement of the shoulder and the reader is referred to the cited reference (22) for further descriptions. Active instability occurs when there are impairments in the function of the muscular system controlling the position of the humeral head and/or the glenoid during movement. These muscular impairments may include weakness, fatigue, and/or alterations in muscle synergies (2, 22).

III. Neuromuscular Determinants of Joint Stability

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As with many joints, passive and active stability of the shoulder rely on a neuromuscular feedback mechanism between the peripheral joint and the central nervous system (CNS). The interpretation of sensory information being relayed from the peripheral joint to the CNS along afferent pathways is called proprioception. The motor response of the CNS to sensory input is referred to as neuromuscular control (15,20). Functional instability occurs when there are impairments in this neuromuscular feedback mechanism resulting in symptoms. The definitions for functional instability vary in the literature but this is how it will be used throughout this paper unless otherwise specified.

As stated above, clinically the term proprioception is used to describe our ability to detect



joint position sense and kinesthesia (2,20). These two sensations, in addition to force application is the sensory information relayed to the CNS via mechanoreceptors located in the joint capsule, glenohumeral ligaments and rotator cuff tendons (20). A mechanoreceptor

is a sensory neuron that is stimulated by mechanical deformation, either tensile or compressive forces. In the glenohumeral joint two common types of mechanoreceptors are found: ruffini receptors and pacinian corpuscles. Pacinian corpuscles are the most common in the glenohumeral ligaments (20). Both of these mechanoreceptors are

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sensitive to tensile force while the pacinian corpuscles are also stimulated by compression (20). Mechanoreceptors transform information about joint position, movement and force into neural input which is sent to the CNS via afferent pathways in the spinal cord (20). The CNS responds with efferent (motor) signals that produce coordinated movement patterns at the shoulder (20). Additionally, proprioceptive information may synapse directly with alpha motoneurons or gamma motoneurons in the spinal cord. Alpha motoneurons send signals to the extrafusal fibers of the muscle (17). A direct activation of alpha motor neurons would result in a reflexive recruitment of peripheral musculature at the shoulder, which has been shown to exist (7,19). However, it is not clear if this spinal reflex could functionally contribute to stability as the latency of the reflex arc may be too long and would require a very large force to elicit an alpha motor neuron response (20). Another likely player in neuromuscular stability is the direct activation of gamma motor neurons (20). Gamma motoneurons send signals to the intrafusal muscle fibers that make up the muscle spindle (17). Muscle spindles are sensory organs in muscle that respond to changes in muscle length (17). When gamma motoneurons fire they stimulate contraction of the intrafusal fibers making the muscle spindle more sensitive (17,20). Increased sensitivity in muscle spindles improves the potential to resist sudden joint displacements and diminishes the burden on the glenohumeral ligaments to resist these destabilizing forces (20). While we still do not have a full understanding of this feedback mechanism it is well accepted that proprioceptive deficits in the shoulder result in faulty neuromuscular control and shoulder dysfunction in the form of functional instability (20,21).

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IV. Causes of Deficits in Proprioception

Deficits in proprioception are present in conditions of capsuloligamentous laxity, attenuation or complete disruptions (8,15,28,31). Tissue laxity or damage may be from a traumatic event often involving a dislocation in which a tear in the labrum or the ligaments occur. Lephart et al (15) compared proprioception in healthy, unstable and surgically repaired individuals. The unstable group consisted of 30 individuals that suffered traumatic and chronic anterior instability and had failed a rehabilitation program. In this group the authors found significant deficits in both kinesthesia and position sense in the involved shoulder compared to the uninjured side. When testing reproduction of passive positioning there was a significant deficit moving from external rotation to internal rotation or vice versa, but not from neutral to either rotated position. This is not surprising as shoulder proprioception has been shown to be more accurate at end-ranges rather than mid-ranges (10). At end-ranges there is maximum tension on capsuloligamentous structures in addition to muscles, tendons and skin. This increased tension and subsequent stimulation of mechanoreceptors and muscle spindles may explain the greater position sense acuity at end-range positions (10).

Microtraumatic capsuloligamentous laxity may be developed with sustained or repeated tension at end-ranges over time. This is often seen in overhead throwing athletes and may or may not be symptomatic (6, 25). Safran et al (25) tested joint position sense and kinesthesia of 21 baseball pitchers with no history of shoulder instability or surgery. They found significant deficits in joint position sense when moving from 75% of maximal external rotation into internal rotation in the dominant pitching arm compared to the non-

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dominant side. They did not find any significant differences in other joint positions or in kinesthesia in any position. Six pitchers with recent shoulder pain did exhibit significant deficits in kinesthesia of the involved shoulder compared to the uninvolved side. The position sense deficit in external rotation in this particular group is consistent with the idea that capsuloligamentous laxity reduces proprioceptive sense as this group would repeatedly overstretch into this position but not necessarily spend as much time at the end-ranges of other positions which did not exhibit any deficit. The six symptomatic players that did exhibit deficits in kinesthesia indicate a possible relationship between symptoms and proprioceptive loss. Dover et al (6) found significant differences in external rotation joint position sense in female softball players when compared to non-throwing athletes but no difference from non-dominant to dominant side. The authors in this study discussed a trend in research where impairments in joint position sense and laxity are seen bilaterally in overhand throwing athletes which may suggest a congenital predisposition to this presentation (6).

Deficits in proprioception are also seen under conditions of muscle fatigue (3,12). Carpenter et al (3) tested healthy subjects for initial detection of movement into rotation (both internal (IR) and external (ER)) before and after exercise to fatigue the shoulder rotators. They found that ER was detected before internal rotation and likely due to increased capsular tightening with ER versus IR. They also found that the detection to movement worsened by 73% after exercise. Lee et al (12) examined proprioception before and after muscle fatigue and found active repositioning in shoulder external rotation was significantly worse after muscle fatigue. They did not find any differences

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with passive repositioning and they did not look at detection of movement. Both studies suggest there is altered proprioception after muscle fatigue in the shoulder. These two studies support a theory that proprioception is made up of information from muscle receptors in addition to the joint receptors (3,12). Another possibility is that muscle fatigue reduces the sensitivity of joint receptors (3). In either situation, it has been shown that muscle fatigue reduces proprioception in the shoulder and may therefore diminish shoulder function leading to poor performance or predisposition to injury, particularly instability (3).

V. Clinical Presentations Often Associated with Proprioceptive Deficits

A. Traumatic Instability

Several authors have shown proprioceptive deficits to be present after chronic anterior subluxation and/or dislocations (15,23,31) often resulting in structural damage to the joint capsule, ligaments and labrum. In addition to pain, paraesthesia or anesthesia may be present most commonly in the lateral deltoid region, if the axillary nerve was affected. Complaints of painful clicking with elevation and anxiety with certain movements are common. Mechanisms of injury often include positions of combined abduction and external rotation or falling on an outstretched arm. Clinical tests usually reveal positive instability tests (e.g. drawer tests, apprehension, relocation, clunk, crank), hypermobile physiological and accessory motions unless apprehensive muscular guarding is present, and scapular winging or atrophy of shoulder musculature (9). Most traumatic dislocations occur in the anterior inferior direction.

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B. Primary or Secondary Impingements

Impingement is a common term among practitioners to describe shoulder dysfunction. Classically, it is thought of as an outlet stenosis of the acromiohumeral space (27) resulting in abnormal stress and friction to the rotator cuff and subacromial bursa. More recently impingement has been further classified so that this original description will be referred to as primary impingement. Secondary impingement can be defined as atraumatic shoulder pain secondary to excessive superior translation of the humeral head resulting in similar stress to the rotator cuff and subacromial bursa (9). Secondary impingement is often associated with microtraumatic passive instability (9) but could also occur due to faulty neuromuscular control, dynamic instability or an overlap of all three. Secondary impingement often occurs in overhead athletes, usually under the age of 35 (27) but could also be seen in individuals with occupational overhead activities, or individuals with genetic ligamentous laxity (9). Clinically, secondary impingement may present with signs of excessive joint play and positive instability tests (e.g. anterior drawer, relocation test) although they may be subtle (9). Sorenson and Jorgenson (27) feel that instability in the shoulder may be present before clinical detection is possible and hypothesized that microtraumatic instability could easily be misdiagnosed as primary impingement.

Machner et al (18) looked at kinesthesia in patients classified as Neer's type II impingement that were planned to undergo surgical subacromial decompression. Neer's type II impingement is when fibrosis and tendonitis are present (9). These authors did not mention instability. They found the affected shoulder exhibited deficits in kinesthesia

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compared to the uninvolved limb which then improved at the post-surgical six month follow-up. This article failed to report specific numbers at follow-up but does suggest a few possibilities: 1) proprioceptive deficits are present in primary impingement or 2) the fifteen patients in this study did in fact have an undetected instability as described by Sorenson and Jorgenson (27). It is worthwhile to note that the patients in this study underwent a twelve week post-operative rehabilitation program which could also explain the improvements in proprioception.

C. Osteoarthritis

Osteoarthritis of the shoulder often involves the glenohumeral and acromioclavicular joints. It results in cartilage and bone destruction, often with development of osteophytes. Muscular atrophy and soft tissue changes often accompany this degenerative process. The changes in tissue alter the congruency of the joint structure and mechanics (4). Clinically the shoulder exhibits a loss of active and passive range along with reduction in accessory motions. Cuomo et al (4) recently tested proprioception in the arthritic shoulder and compared the results to the uninvolved side, age-matched controls and again six months after a total shoulder replacement surgery. They found significant deficits in the arthritic shoulder compared to both age-matched controls and the uninvolved side. At six months following a total shoulder arthroplasty, proprioception was restored to the point that no significant differences were detected between the uninvolved side and age-match controls (4). They studied both detection to passive movement and joint position sense. Both qualities improved following surgical intervention and post-operative rehabilitation (4).

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VI. Evaluation of Proprioception

Proprioception of a joint is determined by testing the ability of the shoulder to detect movement, which is called kinesthesia, and also by the ability to reproduce a joint position, this is called position sense. Position sense can be active or passive. Active repositioning is when a person is first passively placed in a set position than removed and asked to actively put their arm in the previous position. Active repositioning is theorized to test proprioception by stimulation of the muscle receptors (muscle spindles and GTOs). Passive repositioning is when the person's shoulder will be moved passively the second time until they indicate that the arm is in the same position as previously held. Passive repositioning is theorized to stimulate mechanoreceptors in the joint versus muscles. Most studies examining proprioception of the shoulder are using equipment that stabilizes the trunk and shoulder in a set position, then allows movement to occur in one plane either passively or actively. The equipment additionally allows the examiners to measure the exact position where the patient either detects movement or matches a previous position. Some clinics or sports medicine facilities may have access to equipment that would allow them to accurately test proprioception in this manner however; many clinicians do not have access to this type of set-up. While not yet examined for reliability or validity a clinical test for proprioception may include eyes-closed mirroring in which the examiner would passively move one arm and the patient would actively imitate the movement with the opposite arm. This is similar to the angle velocity reproduction test (AVRT) described by Jerosch et al (11) but without the equipment for exactness. In the absence of reliable clinical tests understanding the nature of proprioceptive deficits and recognizing the common presentations that research has

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shown us often result in deficits will assist in determining the appropriateness of including proprioceptive training in our rehabilitation programs.

VII. Interventions to Restore Proprioception

Treatment for the unstable shoulder may include reconstructive surgery with rehabilitation or conservative rehabilitation without surgery. One of the goals with either rehabilitation option is to restore proprioceptive sense and functional stability while preventing recurrence of injury or progressive joint degeneration (2). Surgical intervention attempts to restore structural mechanisms (2) and has been shown to additionally restore proprioception by one-year follow-up (15,31).

A. Surgical Treatment

Lephart et al (15) were the first to test proprioception in healthy, unstable and surgically repaired shoulders. They found in persons with a history of traumatic anterior shoulder instability the involved shoulder was less accurate than the uninjured side for both initial detection of movement and repositioning for internal and external rotation. They also found that individuals undergoing surgical repair tested 6 months after surgery exhibited symmetrical proprioceptive accuracy on the surgical side compared to the uninjured side. The authors concluded that the re-tensioning of the capsuloligamentous structures resulted in restoration of the neuromuscular feedback mechanism and therefore improved proprioception. They theorized that increased tension in the soft tissue structures reduce the threshold for activation of the mechanoreceptors and may stimulate

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growth in population of mechanoreceptors as it has been shown to do after ACL graft reconstruction in the knee (15).

In 2003, Zuckerman et al (31) also studied the effect of surgery on proprioception in individuals with traumatic anterior shoulder instability. However they were able to test subjects prior to surgery and re-test the same subjects at 6 months and 12 months following surgery. These authors tested position reproduction and detection to movement for flexion, abduction and rotation. They found significant deficits in proprioception on the involved side prior to surgery, by six months following the ability to detect initial movement had been restored to the level of the uninvolved side and by one year out both position sense and detection of movement were not significantly different from the uninvolved side. All subjects underwent a standardized postoperative rehabilitation program which was not described in detail (31). The authors concluded that the glenohumeral capsule and ligaments play a large role in proprioception, that shoulder reconstruction and rehabilitation allow activation of the joint and muscle receptors which result in improved neuromuscular control. Because joint proprioception had improved but not entirely at six months after surgery but had recovered fully by twelve months they concluded that rehabilitation likely plays a role in the return of proprioception.

A similar study by Potzl et al (23) examined proprioception in fourteen subjects before surgery and at a long term follow-up five years later. All subjects had suffered recurrent anterior shoulder dislocations prior to surgery. Results were similar in that proprioception deficits were noted prior to surgery and significantly improved to a

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normal level when compared to healthy control subjects (23). Unlike the results in the Lephart study (15), joint position sense of the unininvolved shoulder was significantly worse preoperatively and improved postoperatively. The authors hypothesized the improvements suggest that joint position sense has central level regulations (23).

These three studies (15, 23, 31) similarly examine proprioception before and after surgical repair in traumatic anterior instabilities. No studies were found that looked at surgical outcomes following atraumatic or neuromuscular instabilities.

B. Conservative Approach

Borsa et al (2) described the goals of neuromuscular training in rehabilitation: 1) to improve cognitive appreciation of the shoulder relative to position and motion, 2) to enhance muscular stabilization of the joint in the absence of passive restraints, and 3) to restore synergistic muscle firing and coordinated movement patterns. In addition, neuromuscular rehabilitation should also aim to negate the affects of muscle fatigue on proprioception and performance by including endurance training (3,12). The neuromuscular training exercises are thought to facilitate restoration of proprioception by enhancing mechanoreceptor sensitivity, increasing the number of mechanoreceptors stimulated, or by enhancing compensatory sensations from secondary receptor fields (2).

1. Central Nervous System Considerations

Many authors (2,14) stress the importance of understanding the role of the Central Nervous System (CNS) on motor activities when designing a neuromuscular training

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program. As described earlier the proprioceptive feedback is integrated in the CNS to elicit a motor response (14) and the afferent information contributes to the CNS function on three levels: spinal reflexes, motor control in the brainstem, and motor programming at the highest centers of the CNS (2,14). At the spinal level, activities should include sudden changes in position and co-contraction to encourage reflexive joint stabilization (2). The motor control at the brainstem uses afferent joint information in addition to vestibular and visual information to maintain postural alignment and balance (2). Changing the stability of the standing surface, challenging proper alignment in the neck and trunk, or changing the amount of vision one is using during upper extremity exercises may contribute to successful motor control. The high centers of the CNS involved in motor programming will respond to voluntary movement initiated at the cognitive level but with repetition will develop into unconscious motor programs (2).

2. Neuromuscular and Proprioceptive Training Exercises

Active and Passive Repositioning: These exercises encourage repeated movements both passively and actively and address motor control in the areas of the CNS involved with motor programming (2). Mirroring limb movement with the eyes-open and eyes-closed is one example of this type of exercise. Another would be using passive repositioning equipment as described in many research studies for determining deficits. These exercises should not only be performed in mid-range but also be performed near the end-ranges of movement where articular mechanoreceptors are undergoing maximal deformation and therefore stimulation (2,10). With passive repositioning the articular mechanoreceptors

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are targeted with active repositioning both articular and muscular receptors are stimulated (2). Additionally resistance can be added to further increase muscular receptors (2).



Figure 3. Repositioning, active or passive. (PNF): PNF techniques are designed to improve the neuromuscular response by stimulating the stretch receptors (muscle spindles, golgi tendon organs) in the musculotendinous unit (2). They often involve



Figure 4. Manual resistance for PNF diagonal (D1).

diagonal movement patterns with various forms of manual assistance or resistance applied (see Figure 4). Shimura and Kasai (26) examined the effects of a PNF posture versus a neutral posture on the initiation of voluntary movement and motor cortex excitability using wrist extension. They found that

the PNF posture enhanced the movement efficiency of the joint by inducing changes in muscular activation and reduced the reaction time to initiate movement (26). They felt this study corroborated the beliefs that PNF positively effects neuromuscular control and are mediated by altered levels of excitation of the cortical motor area and corresponding motoneurons (26).

PNF - Rhythmic Stabilization (RS): RS is a form of PNF. These exercises involve the CNS at the spinal level by encouraging reflexive muscular stabilization and co-

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contraction of the rotator cuff and scapular stabilizers (2). Articular and muscular mechanoreceptors are stimulated during RS exercises. They can be performed in functional open chain positions with manual perturbations (see Figure 5) or assistance. They can also be performed in closed chain positions (see Figure 6) with the use of Swiss balls, wobble boards, Bosu balls, or other unstable bases.



Figure 5. Manual RS perturbations.



Figure 6. RS in closed chain.

Plyometrics: These exercises involve an eccentric load or prestretch followed by a



Figure 7. (above) Using a resistive band for plyometric training of external rotators. Figure 8. (below) Open-chain plyometric training with ball toss.

concentric contraction (2). A plyometric training program on female swimmers showed significant improvements in proprioception after 6 weeks (28). Both joint position sense and kinesthesia were measured. The authors theorized that repetitive movement toward the end of shoulder range stimulated the joint mechanoreceptors as well as facilitating muscle spindle activity and decreasing Golgi tendon organ (GTO) activity from the length-tension changes occurring to the musculotendinous structures (28). Desensitizing the inhibitory response of the GTOs is thought to enhance the sensitivity of the muscle



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spindles (28). Both joint mechanoreceptors and muscle spindles are responsible for sending afferent information about joint position and movement to the central nervous system. In addition to the peripheral stimulation the repetitive nature of plyometric training may also affect motor programming centers in the central nervous system resulting in long-term central adaptations affecting proprioception (28). Common plyometric training exercises include throwing motions, trunk motions, resistive band exercises, ball/wall drills and plyometric push-ups (2). (see Figures 7-9)



Figure 9. A plyometric push-up involves pushing up from a lowered position so that both hands leave the ground before landing and re-lowering to the start position. Shown here is an advanced start position with one hand on the ball.

Closed Chain/Open Chain: Many of the above exercises can be performed in either an open or closed chain manner and both have been shown to be beneficial in improving proprioception (24). Rogol et al (24) tested proprioception before and after a rehabilitation program. One group was trained with open-chain exercises and another group underwent closed-chain exercises. Both groups exhibited improved joint reposition sense following either 6 week exercise program. Another interesting observation from the results of this study suggest there may be carryover to improved proprioception in an open-chain task following closed-chained training as proprioception was tested in open-chain. Lephart and Henry (13) summarized the characteristics of closed chain exercises as greater compressive forces, joint congruency, decreased shear, stimulation of

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mechanoreceptors and enhanced dynamic stabilization. The characteristics of open chain exercises are distraction and rotary forces, promotion of a stable base, joint mechanoreceptor deformation, concentric acceleration and eccentric deceleration and simulated function (13). It is always important to consider specificity of training when designing an exercise program (i.e. training with open-chain activities to return to open-chain sport).

VIII. Conclusion

Proprioception is a term to describe our ability to sense joint position, detect movement (kinesthesia) and force application (2,20) through feedback from mechanoreceptors in and around the joint to our spinal cord and central nervous system. There is a large base of literature that supports the conclusions that deficits in proprioception are linked to shoulder instabilities and more recently to osteoarthritis and impingement syndromes.

The literature is not consistent in the definitions of shoulder instability which makes it difficult to categorize and research separate types of instability. For this reason most of the research has been done on traumatic instabilities that result in dislocation and obvious capsuloligamentous disruptions. While this population is easily research and the results have given us a better understanding of proprioception and neuromuscular control in the shoulder joint it does not speak to the population with subtler instabilities. It would be beneficial to become more consistent in our definitions of instabilities so that we could begin researching this population with atraumatic instabilities that we see so often in the clinic. There are only a few studies that examine the results of various proprioceptive and neuromuscular training interventions. This paper makes recommendations based on

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the available research and understanding of the mechanisms affected by proprioceptive deficits. However this is another area that needs further research. We do not have any controlled studies that compare standard shoulder rehabilitation programs with and without a neuromuscular training emphasis. At this point we can only hypothesize that we would be more effective in our treatments if we include proprioceptive and neuromuscular training in conditions that are known to have deficits such as shoulder instabilities, osteoarthritis and impingement syndromes. Additionally, it is very possible that our shoulder patients that have come in with pain and subtle undetected instabilities could attain positive outcomes that may not otherwise have been achieved by including neuromuscular training as recommended above.

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