Proprioception of the shoulder joint in healthy, unstable, and surgically repaired shoulders

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Shoulder proprioception was measured in 90 subjects who were assigned to three experimental groups: group 1 (n = 40), healthy college-age subjects; group 2 (n = 30), patients with anterior instability; and group 3 (n = 20), patients who have had surgical reconstruction. Kinesthesia and joint position sense were measured with a specially designed proprioception testing device. The results revealed no significant differences in proprioception between dominant and nondominant shoulders in group 1 for any test condition. Significant differences (p < 0.05) were revealed between the unstable and uninvolved shoulder for both kinesthesia and joint position sense in group 2. No significant mean differences were revealed between the surgical and contralateral shoulder in group 3 under any test condition. This series of studies provides evidence that proprioceptive deficits caused by partial deafferentiation result when capsuloligamentous structures are damaged. Reconstructive surgery appears to restore some of these proprioception characteristics. (J SHOULDER ELBOW SURG 1994;3:371-80)

In addition to their mechanical restraining function, ligaments provide important neurologic feedback that directly mediates joint position sensibility and muscular reflex stabilization about the joint.[33] This sensory afferent feedback mechanism is referred to as proprioception. Proprioception is a specialized variation of the sensory modality of touch and encompasses the sensations of joint motion (kinesthesia) and joint position (joint position sense). Recent investigations involving the knee have drawn attention to the sensory role of articular structures and proprioceptive deficits after injury.[3, 39] In contrast little information exists about proprioceptive sensibility of the shoulder joint.

The perception of joint position and joint motion in the shoulder is essential for placement of the hand in upper limb function. Recent investigations by Vangsness and Ennis[51] and others[42, 56] have provided an anatomic basis for proprioceptive sensibility in the shoulder. On the basis of their observations of neural structures and mechanoreceptors in the capsule and ligaments of the shoulder joint, we hypothesize that a neurologic feedback exists for control of shoulder muscular action. This feedback serves as a protective mechanism against excessive strain in these capsuloligamentous structures. Furthermore we theorize that after joint injury disruption of this proprioception mechanism results in a disruption of the normal neuromuscular reflex joint stabilization and that this disruption may contribute to excessive strain in the capsule and ligaments, thus increasing the potential for continuing shoulder injury (Figure 1).

The clinical relevance of normal joint sensation and the effect of injury have been demonstrated in the ankle[15, 17] and knee.[3, 33, 39, 43] It has also been demonstrated that patients with unilateral, recurrent, traumatic anterior shoulder instability have proprioceptive sensory

GLENOHUMERAL
DISTRACTION

PROPRIOCEPTIVE
DEFICITS

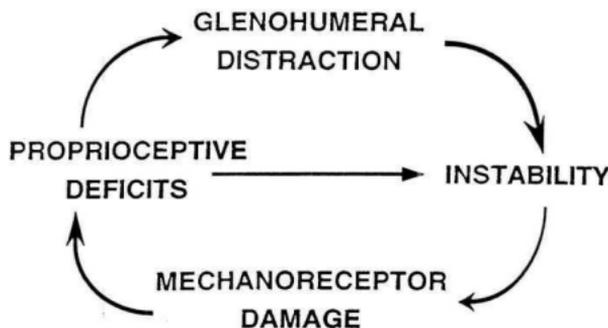INSTABILITY

MECHANORECEPTOR
DAMAGE

Figure 1 Paradigm illustrating relationship between shoulder instability and proprioception.

deficits[48]; however, no analysis has been made of the effect of chronic instability or its surgical treatment on proprioceptive sensibility. The purpose of our studies was to investigate proprioception of the shoulder in groups of individuals with healthy, unstable, surgically repaired shoulders.

METHODS

Study groups. A total of 90 subjects participated in the studies. Three experimental groups were formed. All subjects volunteered to participate and provided informed consent as approved by the Human Subjects Review Board of our institution.

Group 1 (n = 40). This group was the "normal" control group. Forty college-age (19.4 ± 1.2 years) students (26 men, 14 women) who had no history of shoulder injury or pain were studied. All subjects considered themselves athletically active but did not regularly participate in any upper extremity sports (i.e., tennis, baseball, swimming).

Group 2 (n = 30). These subjects were athletically active men (24.3 ± 6.5 years) who subsequent to testing were confirmed during surgery to have chronic, recurrent, traumatic anterior shoulder dislocation or subluxation. Individuals with a history of surgery or clinical findings suggestive of multidirectional instability or voluntary instability were excluded from this study. All these patients had failed a preoperative rehabilitation program aimed at controlling their symptoms. They were tested just before they underwent reconstruction.

Group 3 (n = 20). These subjects were men (30.9 ± 9.6 years) who underwent either open[1] (n = 11) or arthroscopic[55] (n = 9) repair of chronic, recurrent, traumatic anterior shoulder instability. These subjects were tested at least 6 months after surgery (range 7 to 18 months after surgery), when they had completed a prescribed rehabilitation program and had achieved full or nearly full pain-free range of motion.

Proprioception testing. Proprioception was measured with a proprioception testing device (PTD) designed to assess proprioceptive sensibility (Figure 2). The PTD rotated the shoulder into internal and external rotation through the axis of the joint. A rotational transducer interfaced with a digital microprocessor counter provided angular displacement values. The subject was tested in a supine position as in the former studies done on shoulder proprioception by Hall and McCloskey[28] and Smith and Brunolli.[48] The arm of the tested shoulder was positioned at 90° of elbow flexion and 90° of shoulder abduction in the coronal plane. The subject's forearm was positioned in a pneumatic sleeve to reduce the contribution of cutaneous stimuli to the position sense. The pneumatic sleeve was attached to a drive shaft of the PTD. Subjects were blindfolded, and a headset was placed over the ears to negate visual and auditory cues. The test began when the subject gave the investigator a thumbs-up signal. Testing was performed in a single session with the test order of dominant or involved shoulder, starting position, and movement direction being
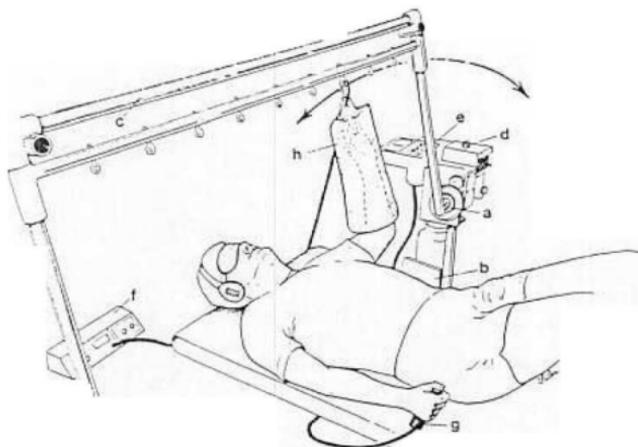
Figure 2 Illustration of proprioception testing device. *a,* Rotational transducer (1:3 mechanical speed-reduction gear-train); *b,* motor; *c,* moving arm; *d,* control panel; *e,* digital microprocessor; *f,* pneumatic compression device; *g,* handheld disengage switch; *h,* pneumatic compression sleeve. Threshold to detection of passive motion is assessed by measuring angular displacement until subject senses shoulder motion.

randomized. Preliminary values on 30 patients revealed a test-retest reliability of $r = 0.92$ for proprioception testing. Instrument reliability was established previously as intraclass correlations, which were calculated with a fixed model and were ranged from intraclass correlations = 0.87 to intraclass correlations = 0.92.

Threshold to detection of passive motion (TTDPM) assessment started with the motor and the shaft of the PTD disengaged. At a random point during the next 10 seconds, shoulder movement was engaged, and the subject was informed to disengage the device by pressing a handheld switch upon sensation of shoulder motion. The PTD moved the shoulder at a constant angular velocity of 0.5°/sec. This speed was chosen because we have found it to be the most reproducible on our test-retest reliability studies. TTDPM of internal and external rotatory movements was measured from starting positions of neutral rotation and 30° external rotation. Three trials were performed from each starting position, moving into both internal and external rotation. We did not test in more extreme positions of external rotation, because we believed that a patient with instability could experience pain or apprehension in these positions.

Reproduction of passive positioning (RPP) for assessment of joint position sense was also performed on groups 2 and 3. RPP was tested to determine accuracy in reproducing both internal and external rotation from starting reference positions of neutral rotation and 30° of external rotation. Limb, direction of rotation, and starting positions were randomized. From each reference angle the shoulder was moved passively 10° in either direction to the presented angle. The angles were presented at variable velocities to reduce any time association. After 10 seconds of static positioning, the shoulder was moved back passively from the presented angle to the reference angle. The subject was then instructed to manipulate the on/off switch to reproduce the previously presented angle. Angular displacement was recorded from the digital microprocessor as the error in degrees between the presented angle and the repositioned angle.

One-way analyses of variance were used to determine significant mean differences between
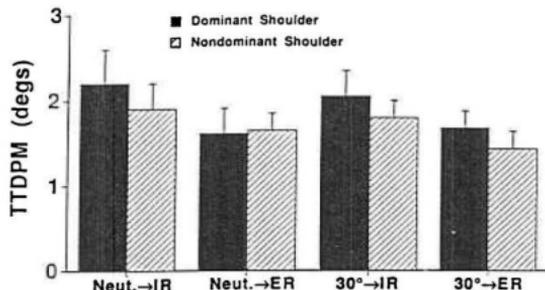
PROPRIOCEPTION OF THE NORMAL SHOULDER

Figure 3 Mean TTDPM (degrees) for dominant and nondominant shoulder from starting positions of neural rotation and 30° external rotation moving into both internal and external rotation (± SE).
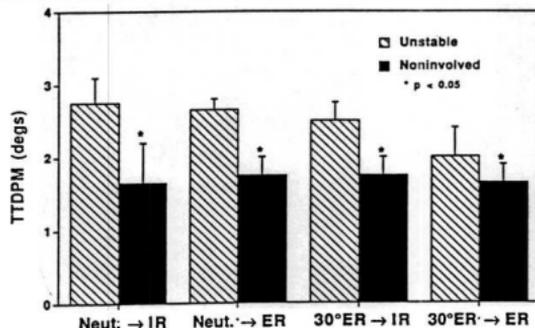
PROPRIOCEPTION OF THE UNSTABLE SHOULDER

Figure 4 Mean TTDPM (degrees) for unstable and uninvolved shoulder from starting positions of neural rotation and 30° external rotation moving into both internal and external rotation (± SE).

dominant or involved shoulder and nondominant or uninvolved shoulder under the four test conditions for both TTDPM and RPP. Significance level was set at a p value of <0.05.

RESULTS

Group 1. Analysis of variance revealed no significant mean differences in TTDPM between dominant and nondominant shoulders for any of the test conditions (Figure 3). Mean values for each of the eight test conditions ranged from $1.43° ± 0.2°$ to $2.20° ± 0.4°$ of shoulder rotation.

Group 2. The unstable shoulders demonstrated significantly longer TTDPM (mean ± SE) compared with the normal contralateral shoulder from a starting position of neutral rotation moving into both internal rotation (IR) ($2.8° ± 0.3°$ vs $1.7° ± 0.6°$; $p = 0.003$) and external rotation (ER) ($2.6° ± 0.6°$ vs $1.8° ± 0.2°$; $p = 0.05$) (Figure 4). The unstable shoulder also demonstrated significantly less accurate RPP (mean ± SE) values compared with the normal contralateral shoulder, from a starting position of 30° ER, in reproducing angles in IR ($4.1° ± 1.0°$ vs $3.3° ± 0.4°$; $p = 0.006$) and ER ($2.8° ± 0.7°$ vs
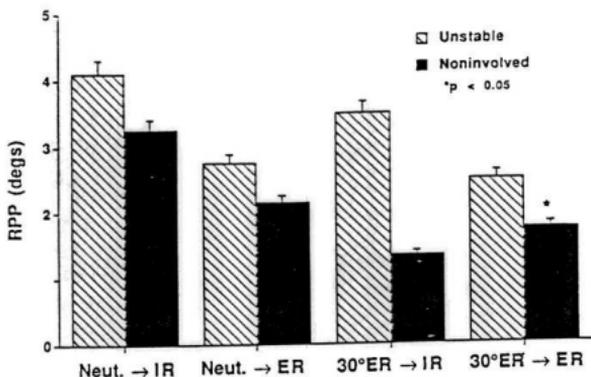
PROPRIOCEPTION OF THE UNSTABLE SHOULDER

Figure 5 Mean error of RPP (degrees) for unstable and uninvolved shoulder from starting positions of neutral rotation and 30° external rotation moving into both internal and external rotation (±SE).

PROPRIOCEPTION FOR THE POST-RECONSTRUCTED SHOULDER

Figure 6 Mean TTDPM (degrees) for reconstructed and uninvolved shoulder from starting positions of neutral rotation and 30° external rotation moving into both internal and external rotation (±SE).

$2.2° \pm 0.4°$; $p = 0.03$). No significant difference was seen between shoulders in RPP, from a starting position of NR, in reproducing the angles in IR ($p = 0.06$) and ER ($p = 0.27$) (Figure 5).

Group 3. No significant mean differences were revealed between the shoulders that had undergone surgery and the normal contralateral shoulders for TTDPM and RPP in any of the test conditions. Mean values for TTDPM ranged from 1.5° to 2.2° and for RPP from 2.0° to 3.5° (Figures 6 and 7).

PROPRIOCEPTION FOR THE POST-RECONSTRUCTED SHOULDER

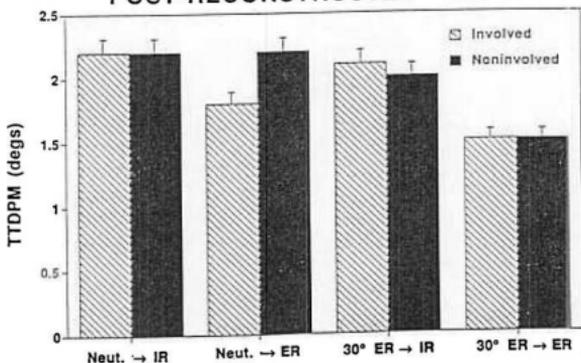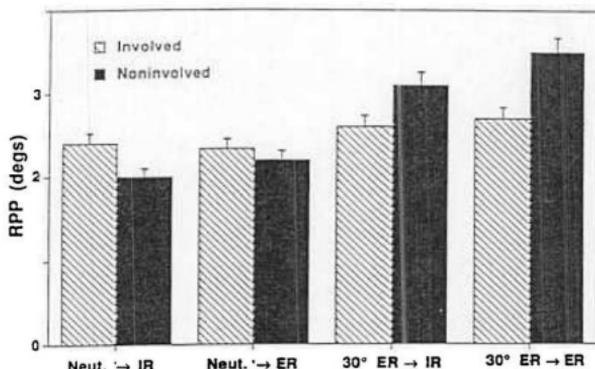**Figure 7** Mean error of RPP (degrees) for reconstructed and uninvolved shoulder from starting positions of neutral rotation and 30° external rotation moving into both internal and external rotation (±SE).

DISCUSSION

Because the glenohumeral joint is minimally constrained by articular anatomy, stability is provided mostly through the combined effect of static and dynamic soft-tissue factors.[31, 50, 52-54] The capsuloligamentous structures function passively as checkreins to excessive translation or rotation of the humeral head on the glenoid but are lax in the midranges of motion. Rotator cuff and long head of biceps contraction dynamically stabilize the joint and are especially important in this midrange of motion where the ligaments are lax.[7, 44] We have hypothesized that proper dynamic stabilization through coordinated muscle contraction is mediated by proprioceptive feedback provided by tension that develops in normal capsule and ligaments of the glenohumeral joint. Injury to these soft-tissue structures may disrupt this neuromuscular mechanism.

Proprioception is mediated by peripheral receptors in articular, muscular, and cutaneous structures. Articular structures (capsule and ligaments of joints) have been found to contain nociceptive (pain) free nerve endings and proprioceptive mechanoreceptors consisting of Pacinian corpuscles, Ruffini endings, and Golgi tendon organlike endings.[37] These three articular mechanoreceptors have recently been identified in the glenoid labrum and glenohumeral ligaments[51]; their presence confirms that the capsuloligamentous structures of the shoulder possess an anatomic potential for perceiving joint motion and position. Mechanoreceptors are specialized neurons that transduce mechanical deformation into electrical signals and thus yield information about joint position and joint motion.[22, 23, 25] Ruffini endings and Golgi tendon organlike endings are slowly adapting mechanoreceptors and thus are more important in giving information about actual joint position or change in position. The Pacinian corpuscles are rapidly adapting mechanoreceptors and therefore function to sense sudden movements of acceleration or deceleration.[47] Stimulation of these receptors also causes reflex muscle contraction about the joint.[5, 6, 49] Muscle spindle receptors, which are slowly adapting receptors, are thought to provide proprioceptive feedback in muscles.

The joint capsule has been identified as the site of the peripheral receptors responsible for joint proprioception.[41, 47] Additionally studies have shown that these capsular receptors respond only to extreme of ranges of motion[24] or during strong stimulus to the joint capsule such as compression or deep pressure, which might occur with translation of the humeral head on

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the glenoid.[10, 26] Some researchers believe that muscle receptors play a more important role in signaling joint position sense.[11, 16, 20] Recent studies have demonstrated that muscle and joint receptors are probably complementary components of an intricate afferent network in which each receptor modifies the function of the other.[5, 14, 27] Both types of receptors have well-established cortical connections to support their combined role in proprioceptive feedback.[29, 30, 47]

Functionally *proprioception* is assessed by measuring threshold to detection of passive motion, and *joint position sense* is assessed by reproduction of passive positioning. Both the relation between injury and disease conditions and proprioception and the implications for function were studied in several joints. Freeman et al.[15] and Glencross and Thorton[17] demonstrated that the unstable ankle had significant proprioceptive deficits after injury. Moreover, they showed that a proper rehabilitation program corrected this deficit. Konradsen and Raven[34] further demonstrated that this proprioceptive deficit resulted in decreased peroneal muscle activity in functionally unstable ankles. We have observed,[37] as have Barrack et al.,[2] proprioceptive deficits in patients who have an anterior cruciate ligament (ACL) injury. It has also been postulated that ballet dancers have reduced position sense resulting from capsular stretching with ankle and knee injuries. This stretching results in damage to neural mechanoreceptors.[2] Furthermore athletic training and bracing appear to partially correct these proprioceptive deficits.[3-5, 39]

Our study is the first to compare shoulder proprioception in groups of individuals with normal, unstable, and surgically repaired shoulders. Previous studies have evaluated only normal shoulder proprioception[28] or instability in a small group of patients.[48] We found insignificant differences in proprioception between dominant and nondominant shoulders in healthy individuals (group 1). This observation is consistent with previous reports.[48] The data reported in this article differ slightly from data presented on these subjects previously by the authors because of a recently noted error in conversion of degrees of angular displacement values. Our subjects were not athletes who participated in sports involving the upper extremities; thus the effects of training on arm dominance cannot be addressed. Our previous work on gymnasts[37] suggests that training may indeed result in enhanced proprioceptive acuity; however, this study was retrospective. Future studies will address this issue of neural adaptation in the shoulder through training.

The instability group (group 2) was found to have significant deficits in proprioceptive sensibility under six of eight test conditions. Though these differences were small (only 1° to 2°), it should be kept in mind that the reaction time deficit represents 2 to 4 seconds, because the testing speed was 0.5°/sec. The normal contralateral shoulders in this group were found to have kinesthetic and joint position sensibility comparable to that of the healthy shoulders (group 1). TTDPM was significantly longer in the unstable shoulder at both test positions of neutral and 30° external rotation moving into external and internal rotation. RPP in the unstable shoulder was also abnormal when compared with that in the contralateral normal shoulder.

No previous studies have examined the pattern of proprioception in a group of individuals with chronic, traumatic, anterior shoulder instability. Electromyographic studies[8, 18, 21, 32] and other biomechanical work[7, 44, 46] have suggested that a complex, coordinated, synergistic function of the rotator cuff and biceps muscle is required for normal function of the shoulder.[31] We believe that in these individuals, injury to the capsule, labrum, ligaments, and surrounding muscles results in damage to neural mechanoreceptors that mediate normal proprioceptive sensation. The deficit in proprioception that results may contribute to ongoing instability and injury of the shoulder joint. These proprioceptive deficits may also contribute to disruption of the normal protective stabilization of coordinated contraction of the rotator cuff muscles, although this study cannot confirm this hypothesis.

Although our understanding of neural mechanisms in joint position sense in normal and uninjured points is growing, the effect of surgery on proprioception is less clear. Most of the work reported discusses proprioception after ACL reconstruction and after knee or hip arthroplasty. Several authors have observed that function is better when proprioception is normalized, and failure of a surgical procedure may be associated with poor proprioception.[3, 5, 13, 35] We ob-

served that a group of individuals (group 3) undergoing either arthroscopic or open stabilization of the unstable shoulder had proprioceptive sensibility that was not significantly different from that of individuals with normal shoulders. We hypothesize that clinical stability of the shoulder may be restored not only through return of static mechanical constraint to the joint but also through restoration of the normal proprioceptive feedback mechanism, which permits proper muscular control of joint stability. Retensioning of the capsule and ligaments may facilitate proprioceptive function by decreasing the afferent threshold. Because these mechanoreceptors need to be mechanically deformed or loaded to transmit impulses to the central nervous system, they may not be sufficiently stimulated in a lax or injured capsule, which cannot develop tension when the humerus is rotated or displaced on the glenoid. Tightening the capsule and ligaments during repair will restore proper tension for this mechanism to occur. In addition histologic studies have shown that after ACL reconstruction, a repopulation of mechanoreceptors occurs in ACL graft tissue[12, 19]; therefore shoulder capsule reconstruction may similarly affect the population of these receptors.

It is possible that another mechanism was responsible for normalization of proprioception in individuals after surgical stabilization of the shoulder. Rehabilitation may have led to enhanced proprioception after surgery. Although the instability group (group 2) underwent a similar rehabilitation program as the surgery group (group 3), surgical stabilization may have allowed the latter group to participate more effectively in their rehabilitation program. The rehabilitation program for these patients emphasized proprioceptive input to recognize joint position and the learning of correct movement patterns and techniques in addition to development of strength and endurance. These exercises included matching and rematching joint position, weight bearing through the upper extremity, and open kinetic chain exercises.[36, 38] The later stages of the rehabilitation focuses on activities that promoted proprioceptively mediated reflex joint stabilization. Although this reflex arc has not been demonstrated in the shoulder, similar neuromuscular mechanisms in the knee are believed to play a key role in joint arthrokinematics.[40, 45]

SUMMARY

This study provides insight into shoulder proprioception in normal, unstable, and surgically corrected shoulders. We observed that arm dominance in healthy individuals does not influence proprioceptive sensibility, that athletically active individuals who have chronic, traumatic anterior shoulder instability have significant deficits in their proprioception, and that surgical stabilization of such a shoulder normalizes proprioceptive sensibility. On the basis of our understanding of the role of the neurosensory mechanism or proprioception relative to function of the upper limb and stabilization of the shoulder joint, these data have significant implications for the management of shoulder instability. Future directions for proprioception research should consider the effects of upper extremity training on proprioception in both normal and unstable shoulders. The relationship between the proprioceptive characteristics assessed in the laboratory and the clinical function in both normal activities and sports should also be considered. Such clinical studies should be designed as randomized and prospective.

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