Shoulder Kinesthesia in Healthy Unilateral Athletes Participating in Upper Extremity Sports

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Joint proprioception and kinesthesia have often been referred to as the sixth sense of bodily function. Proprioception is the ability to determine the location of a joint in space, whereas kinesthesia is the ability to detect movement (7,10). Joint position sense is mediated by joint and muscle receptors as well as visual, vestibular, and cutaneous input (7,10,13,16,17,19,22,32). These five inputs can be used on a conscious or unconscious (reflexive pathways) level so that motor tasks are performed smoothly (13,25,29,32,42).

Early research suggested that the joint receptors had the predominant role in proprioception and kinesthesia (7,8,10,13-16,17,21,22). Joint receptors have been identified in joint capsules, ligaments, menisci, labrum, and fat pads (22,23,33,36,38,39,41,43-46). Recent research has identified Ruffini-like endings in the glenohumeral joint capsule; Pacinian corpuscles in glenohumeral ligaments, and free nerve endings in the glenoid labrum of human cadavers (41,43).

Physiologic properties of joint receptors include direction and range-specific patterns of firing. Joint receptors fire predominantly at the end ranges of motion (8,10,20,23,39). Contraction of muscles surrounding a joint can excite joint receptors, but stimulation can be induced only when the receptors are in proximity to the tendinous insertion of a given muscle (13,20,22). Joint receptors are responsive to mechanical deformation of the inert structure in which they are embedded (7,10,14,17,21,22,35).

Kinesthetic deficits have been measured by threshold to detection of passive motion and reproduction of passive positioning. Threshold to detection of passive motion appears to be a more sensitive measure for kinesthetic deficits than reproduction of passive positioning (21,30). The ability to detect movement also appears to be velocity dependent, with detection being more precise at slower speeds (26).

Kinesthetic deficits have been identified in anterior cruciate liga-
ment (ACL) deficient knees (3,11, 28), ACL-reconstructed knees (both allograft and autograft) (4,30), osteoarthritis knees (5), joint replacements (5,21,26), and ankle instability (12,27,38). Kinesthetic deficit patterns identified in ACL-reconstructed knees exemplify the properties of joint receptor's response to mechanical stimulation. The ACL-reconstructed knee exhibits no kinesthetic deficits at 45°, but does exhibit kinesthetic deficits in terminal extension where the ACL is normally taut (30).

A limited number of studies have examined shoulder kinesthesia. Smith and Brunolli (40) reported kinesthetic deficits of both threshold to detection of passive motion and reproduction of passive positioning in shoulder's status postdislocation.

This study evaluated shoulder kinesthesia (threshold to detection of passive motion) in healthy upper extremity athletes who participated in unilateral overhead sports, including baseball, tennis, and football (quarterback). Typical range of motion (ROM) patterns for upper extremity athletes include excessive external rotation and compromised internal rotation (6,9,24,34). The notion of kinesthetic deficits in subjects with nonpathological laxity is controversial (1,2,30). The purposes of this study were to determine if: 1) there were differences in threshold to detection of passive motion between the dominant and nondominant shoulders of healthy overhead athletes; 2) there was a difference in threshold to detection of passive motion at 0° and 75° of external rotation in dominant and nondominant shoulders of unilateral upper extremity athletes; 3) there was an interaction between the two independent variables (position and dominance) on threshold to detection of passive motion; and 4) there was a relationship between external rotation and internal rotation ROM measures and the threshold to detection of passive motion values.

METHODS

Subjects

Twenty collegiate male overhead athletes between the ages of 18 and 21 years old (X = 18.75 ± 1.33) participated in this study. The subjects included 11 baseball players, five quarterbacks, and four tennis players who were intercollegiate athletes with a mean of 10.9 (± 2.83) years experience and a range of 5-15 years of experience in their sport. All subjects practiced in their sport at least four times a week and competed regularly during the competitive season.

Exclusion criteria for subjects included any individual that competitively or consistently participated in any bilateral upper extremity sport, such as swimming. Subjects were also eliminated if they had a history of shoulder injuries, including dislocation, prior surgery, or any other shoulder problem that prevented the athlete from participating in their sport activity for greater than 2 weeks in length during the past year. Any cervical lesion that limited function of the athlete during the past year also excluded them from participating in the study.

All testing was performed in the Proprioception Laboratory at the University of Pittsburgh on a single day for each subject. Prior to participation in the study, all subjects signed an informed consent that had been approved by the University of Pittsburgh Institutional Review Board for Biomedical Research.

Procedure

Passive shoulder internal rotation and external rotation ROM were measured with a standard goniometer at 90° of shoulder abduction. The subject was positioned supine with the scapula stabilized by the underlying table. Goniometric measurements were performed prior to proprioceptive testing. Intrarater reliability for assessing internal rotation and external rotation was established prior to proprioceptive testing (external rotation, ICC = .98; internal rotation, ICC = .99) on 10 subjects.

Kinesthetic awareness of the shoulder was assessed using a proprioception testing device, which consisted of a motor-driven goniometer that passively moved the shoulder at a speed of 5°/second through an arc of internal and external rotation (Figure 1). The device was used to measure threshold to detection of passive movement for both the dominant and nondominant shoulders. The dominant shoulder was the arm that the athlete used to throw or play tennis. A rotational transducer was interfaced with a digital microprocessor which measured angular displacement values. The angular displace-
Subjects were instructed to relax and to prevent visual input during testing. The forearm was placed in a pneumatic compression splint remaining in the neutral position during testing. The forearm was placed in a pneumatic compression splint which was attached to the motor-driven device that moved the shoulder through passive range of motion. Two starting positions were used: neutral and 75° of external rotation. The test position at 75° of external rotation had a component of horizontal abduction due to the fact that there was no distal support under the elbow in this testing device. However, the component of horizontal abduction was present in all subjects in this position, and it did not appear to affect the results of the reliability testing.

Subjects were instructed to push a hand-held switch when they detected movement. Threshold to detection of passive motion was measured by recording angular rotation between the starting position and the position where movement was detected. Prior to beginning testing in each position, the subject was given two practice trials to become familiar with the testing procedure. There was no other warm-up or restrictions to activity on the day of testing.

The dominant and nondominant shoulders were tested three times in each direction (internal and external rotation) for both testing positions (0° and 75°). A total of 12 trials was performed for each shoulder. Randomization was used for determining the order of the testing sequence for dominance, position (0° or 75°), and the direction of movement. To eliminate bias from the sound of the motor starting, testing was randomly initiated 5-30 seconds after the motor was turned on. Subjects wore headphones with white noise at a consistent volume to further eliminate auditory input and were blindfolded to prevent visual input during testing.

Test-retest reliability of the proprioceptive testing device was determined prior to data collection by testing a single shoulder twice according to the methodology described above. Nine normal subjects that were not included in this study were used for reliability testing. A short break was given to subjects between test sessions. Intraclass correlation coefficients (37) were calculated using the fixed effects model (external rotation at 0°, ICC = .82; external rotation at 75°, ICC = .87; internal rotation at 0°, ICC = .86; internal rotation at 75°, ICC = .92).

Data Analysis

This study was a two-way factorial design. The mean of the three trials for both ROM and threshold to detection of passive motion were used for calculations. Dominance (dominant and nondominant shoulder) was one factor, and position (neutral and 75° of external rotation) was the other factor.

Range of motion was reported as a mean and standard deviation for both the dominant and nondominant shoulders. A t test was used to determine if there was a significant difference between dominant and nondominant shoulder internal and external rotation. Range of motion and threshold to detection of passive motion were correlated with the Pearson product correlation coefficient. Threshold to detection of passive motion was analyzed with a two-way analysis of variance (ANOVA) with repeated measures for both factors (shoulder and position) for internal rotation and external rotation. The alpha level for all statistical tests was set at $p < .05$.

RESULTS

Range of motion measurements revealed significantly greater external rotation range of motion ($\bar{X} = 119.04 \pm 9.13$) on the dominant shoulder than the nondominant shoulder ($\bar{X} = 106.91 \pm 9.13$). The range of internal rotation was less on
the dominant shoulder ($\bar{X} = 58.83 \pm 8.49$) than the nondominant shoulder ($\bar{X} = 59.21 \pm 9.35$). A $t$ test revealed that differences in the range of external rotation and internal rotation between dominant and nondominant shoulders was significant ($t = 5.43, p < .001; t = -2.73, p < .05$, respectively).

The mean values for threshold to detection of passive motion are reported in Table 1. The nondominant shoulder exhibited a significantly enhanced ability to detect motion for both internal and external rotation (Figures 2 and 3). The data exhibited a normal distribution with the exception of one outlier who tended to skew the data. However, when the data were analyzed without this outlier, no change in the results was noted. This outlier is responsible for the large standard deviation seen in Figure 2 for nondominant external rotation.

Analysis of variance (ANOVA) was significant ($p < .05$) for the effect of position and the effect of dominance for both internal and external rotation (Tables 2 and 3). Subjects demonstrated significantly better kinesthetic acuity (lower threshold to detection of passive motion scores) at $75^\circ$ of external rotation compared with $0^\circ$ for both internal and external rotation. The subjects also exhibited enhanced ability to detect motion on the nondominant shoulder compared with the dominant shoulder.

A post hoc analysis for simple main effect was calculated to determine if there was a difference between dominant and nondominant shoulder threshold to detection of passive motion moving into internal rotation at the position of $75^\circ$ of external rotation. No significant difference was found between the dominant and nondominant data points at $75^\circ$ of external rotation when moving into internal rotation. This suggests that the difference between dominant and nondominant shoulders is more pronounced at $0^\circ$ than at $75^\circ$.

A Pearson product correlation coefficient was used to correlate range of motion and threshold to detection of passive motion values. A negative relationship was seen between the range of dominant shoul-
The dominant shoulder exhibited decreased kinesthetic awareness.

The dominant shoulder exhibited decreased kinesthetic awareness (higher threshold to detection of passive motion values) compared with the nondominant shoulder for both 0° and 75° of external rotation. This suggests that excessive range of motion may result in decreased ability to detect passive motion. Decreased kinesthesia was noted into the direction of external rotation, the same direction that the athletes exhibited increased motion. However, no significant differences were noted between the dominant and nondominant shoulder when moving into internal rotation at 75° of external rotation. Limited internal rotation on the dominant shoulder was noted, suggesting that the posterior capsule may be tight. The difference in tautness of the joint capsule at 0° and 75° may affect joint kinesthesia when moving into internal rotation. At 0°, the posterior capsule is relatively taut. However, at 75° of external rotation, the posterior capsule should be relaxed. Therefore, joint kinesthesia should be enhanced where the joint capsule is relatively taut at 0°. This may explain why there was a significant difference between the nondominant and dominant shoulder when moving into internal rotation at neutral rotation but no significant difference between the dominant and nondominant shoulders at 75° when moving into internal rotation.

A negative correlation was found between dominant shoulder internal rotation and detection to passive motion in internal rotation. This suggests that as the range of internal rotation values increased, the threshold to detection of passive motion decreased. This finding is not consistent with the other findings of this study. However, the correlation between the range of internal rotation and threshold to detection of passive motion was only moderate ($r = -0.50$).

Another issue to consider is the tissue tension placed on the joint capsule in the position of 75° of ex-

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TABLE 3. Two-way analysis of variance of internal rotation threshold to detection of passive motion for position, dominance, and the interaction of position and dominance.

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* Indicates significant for $p < .05$. 

\[ r = -0.50, \] \[ p < .05 \]
ternal rotation. The tissue tension may vary from the dominant to non-dominant shoulder due to the greater amount of external rotation in the dominant shoulder. The test position of 75° of external rotation may be further from the end range of motion for the dominant shoulder. In fact, the dominant shoulder may have enhanced kinesthesia if it was tested closer to the end range.

Whether the kinesthetic deficits found in the dominant throwing shoulder have pertinent clinical implications is uncertain. The differences between the dominant and nondominant shoulder’s ability to detect passive motion were found to be relatively small (<2°). However, the demands placed on the shoulders of overhead athletes are extremely high. Speeds encountered at the shoulder are reported to exceed 6,000/second during pitching (34). Large repetitive forces at the gleno-humeral joint may result in microtrauma and attenuation of the joint capsule and ligaments (24). The results of this progressive microtrauma may cause proprioceptive changes, which, in turn, may lead to further damage due to poor biofeedback from joint receptors. Subtle changes in joint mobility and resultant joint kinesthesia may be a mechanism for the occurrence of instability.

The natural history of instability in throwing athletes has not been observed. Will longer periods of participation for throwing athletes create greater laxity and further diminish kinesthetic abilities? At what point does this deficiency become clinically significant? Repetitive microtrauma to the joint capsule and joint receptors may unavoidably result in alterations of joint laxity and diminished kinesthesia. However, the shoulders may not become symptomatic until the dynamic stabilizers fail to compensate for traumatized static restraints. In the case of actual instability, the dynamic stabilizers may not fire quickly enough to prevent humeral head translation. A protective reflex may be present in the shoulder, similar to reflexes that have been identified in the knee (40). Glousman et al identified a decrease in EMG firing of the shoulder internal rotators during the throwing motion in shoulders with anterior instability (18). This may be indicative of a protective reflex to minimize subsequent injury or subluxation. Further research is needed to determine if this is the case.

Enhanced kinesthetic senses identified in athletes may or may not be a result of training (2). Individuals who are born with enhanced proprioceptive abilities may develop into competitive athletes. The effect of training and rehabilitation on proprioception and kinesthesia deserves further study before this conclusion is made.

Further research needs to determine the natural history of instability in the throwing athlete. The relationship between shoulder joint laxity and kinesthesia needs to be evaluated more closely. At the present time, there is no quantitative and reliable method for measuring shoulder joint laxity. This makes determining the relationship between laxity and kinesthesia in the shoulder difficult. Correlating joint kinesthesia with an objective measure of shoulder laxity is an area that needs to be further explored. Generalized joint laxity has been previously studied in conjunction with joint kinesthesia. Normal shoulders that demonstrated generalized ligament laxity (thumb to forearm and metacarpal phalangeal hyperextension) exhibited a trend toward higher threshold to detection of passive motion values or decreased kinesthesia (32).

Overall, this design tested a small subset of the whole proprioceptive process (42). This was a passive test, which mainly tested joint receptors. Input from the muscle receptors and the visual system may be able to compensate for deficits in ability to detect passive motion. Whether this system can compensate under all conditions is unknown. Further research is needed to determine the relative contributions from joint and muscle receptors to shoulder kinesthesia. The kinesthetic mechanism of the shoulder may vary somewhat from the model constructed at the knee due to differences in joint and cutaneous receptors.

Another limitation of this study was the fact that only the ability to detect motion was tested but the direction of this motion was not measured. Adding detection of direction should increase the time it takes for the subject to determine movement and add to the complexity of this task.

Joint kinesthesia is a complex process, involving both conscious and unconscious mechanisms. Research involving shoulder proprioception and kinesthesia is in its infancy. Further research needs to be done in the area of basic science on the physiologic mechanisms of joint and muscle receptors of the shoulder. Further clinical research needs to be pursued in determining the natural history of shoulder problems in throwing athletes and determining the effect of training on shoulder kinesthesia.

CONCLUSION

Kinesthetic abilities were studied in unilateral upper extremity athletes. The dominant arm of throwing athletes had significantly diminished ability to detect passive motion at both 0° and 75° of external rotation when compared with the nondominant shoulder. The differences in the dominant vs. nondominant shoulders may be related to differences in ROM between the shoulders. However, the dominant and nondominant shoulders of overhand athletes appeared to have enhanced ability to detect passive motion compared with that of normal subjects. The kinesthetic deficits identified in the dominant shoulder of throwing athletes may be a mechanism for instability of the shoulder.