
Rose Excellence in Research Award Orthopaedic Section 1990



Richard L. Smith

Richard Smith, MS, PT is the recipient of the Rose Excellence in Research Award for his paper, "Shoulder Kinesthesia After Anterior Glenohumeral Joint Dislocation." This paper, written with John Brunolli, was published in *Physical Therapy* in February 1989.

Richard Smith received his BS in Physical Therapy at the University of Colorado in 1976. His studies of human anatomy and arthrology led to an MS in Anatomy at Colorado State University. In 1982, he moved to Montana to teach basic sciences in the University of Montana Physical Therapy Program in Missoula.

Richard met John Brunolli at U of M. Both shared an interest in joint instability and carried out the study of kinesthesia in dislocated shoulders. The results of their work indicate that recurrent dislocating shoulders have significant kinesthetic deficits. More importantly, therapists should focus some of their rehabilitation efforts on facilitating shoulder joint and muscle receptors to improve reflexive neuromuscular control of unstable shoulders.

Richard and his wife, Edie Smith, are owners of Missoula Physical Therapy Center. Both practice orthopaedic physical therapy, serve on the Montana Chapter APTA Board of Directors, and teach in the U of M Physical Therapy Program. Richard is the PT consultant to the Montana Division of Worker's Compensation and a Subject Matter Expert for the journal, *Physical Therapy*. Future professional endeavors include teaching and consulting with Isernhagen and Associates, promoting direct access and freedom of choice to physical therapy services, and developing more progressive rehabilitation programs for musculoskeletal injuries.

Shoulder Kinesthesia After Anterior Glenohumeral Joint Dislocation¹

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*The purpose of this study was to examine kinesthesia in normal (uninjured) shoulders and in shoulders with a history of glenohumeral joint dislocations. Both shoulders of 10 healthy subjects and 8 subjects with a history of unilateral anterior dislocation were tested for accuracy of angular reproduction, threshold to sensation of movement, and end-range reproduction using a motor-driven shoulder-wheel apparatus. An analysis of variance revealed significant differences ($p < 0.001$) between the injured and uninjured shoulders for all three tests. Post hoc analysis showed significant differences ($p < 0.02$) between the involved shoulders and all uninvolved shoulders. No significant difference was found among the uninvolved shoulders. The results of this study indicate that kinesthetic deficits occur after glenohumeral dislocation and may result in abnormal neuromuscular coordination and subsequent reinjury of the shoulder. Clinicians should consider rehabilitation of shoulder kinesthesia using therapeutic activation of the shoulder joint and muscle receptors when treating patients with previous dislocations. [Smith RL, Brunolli J: Shoulder kinesthesia after anterior glenohumeral joint dislocation. *Phys Ther* 69:106-112, 1989.]*

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Anterior glenohumeral joint (GHJ) dislocation is a disabling injury associated with ligament disruption, rotator cuff tears, fractures, and neurological involvement (1, 2). The consequences of GHJ dislocation, including pain, instability, and patient apprehension during certain shoulder movements, are particularly serious for individuals who use their shoulders during employment or sports activities.

In the general population, the recurrence rate of GHJ dislocation ranges from 33% to 50% and is reported to be as high as 66% to 92% in patients less than 20 years of age (3, 4). The rate of recurrence is much higher in athletes (83%)

than nonathletes (30%) (4). Clearly, this high recurrence rate is a major complication after dislocation. Protzman suggested that chronic GHJ instability, or recurrent subluxation, may be as incapacitating as recurrent dislocation (5). Simonet and Cofield noted that patients with past shoulder dislocation complain of decreased strength and do not trust their shoulder because it "goes dead" or "slips in and out" (4). Patients' fear of recurrent episodes of dislocation may limit their work capacity or sports participation.

Recurrent instability results from various causes, including capsular laxity and detachment of the glenoid labrum. Another underlying cause still to be investigated may involve loss of peripheral sensory reception and neuromuscular coordination. Kinesthesia, the complex perceived sensation of the position and movement of joints and muscles, plays an important role in coordination of muscular control of peripheral joints (6-8). This sensation may be compromised after GHJ dislocation. Freeman et al suggested that functional instability of the foot and ankle resulted from muscular incoordination consequent to rupture of afferent nerve fibers in damaged ankle joint ligaments and capsules (9). Increased laxity of joints may be related to below-normal protective reflexes (10). Muscle contraction may occur too late in sports situations to protect the joint (11, 12).

¹ This article was presented in poster format at the Sixty-Third Annual Conference of the American Physical Therapy Association, San Antonio, TX, June 28-July 2, 1987. The study was supported in part by the Montana Chapter of the American Physical Therapy Association and was submitted June 24, 1987; was with the authors for revision for 34 weeks; and was accepted June 9, 1988.

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Recurrent shoulder dislocation may occur when the muscles spanning the joint are overpowered or caught off guard (1). Thus, subtle changes in the sensory system, specifically deficits in shoulder joint and muscle kinesthetic sensibility, may predispose the GHJ to instability and, therefore, to reinjury.

Researchers have examined the extent of sensory deficits in subjects with damaged hip, knee, and ankle joints. The ability to detect hip joint position was affected minimally by total-joint replacement surgery (13, 14). Other investigators found a marked decline in joint position sense associated with degenerative joint disease in the knee (15) and in severe ankle sprains (16). Despite these studies, sensory disability after peripheral joint injury and surgery has not been established clearly.

Few data exist describing kinesthesia in the normal (uninjured) shoulder (17) with no reports of shoulder kinesthesia after dislocation. The purposes of this study were 1) to examine kinesthetic values for normal shoulder joints and 2) to determine whether kinesthetic deficits occur after shoulder dislocation. We hypothesized that kinesthetic impairment would exist after GHJ dislocation.

METHOD

Subjects

A total of 18 subjects volunteered to participate in this study. Eight subjects (6 men, 2 women) with a history of anterior GHJ dislocation were referred to us by colleagues. These subjects were diagnosed as having had a frank dislocation. They were unilaterally involved, had received no surgical joint repair, and were at least three months past the last dislocation. Ten subjects (5 men, 5 women) included in the study had normal shoulders bilaterally and no previous history of musculoskeletal problems or pathological conditions in their shoulders. Table 1 presents descriptive data on the subjects.

All subjects signed informed consent documents approved by the University of Montana Institutional Review Board for Human Research. All subjects were informed that they would be

tested for their ability to reproduce joint angles and to detect limb movement in both shoulders. They were not told the specific purposes of the study nor given details about the tests (eg, joint angles, rates of movement, or accuracy of shoulder alignment).

Instrumentation and Subject Positioning

A shoulder-wheel apparatus was designed to laterally (externally) rotate the shoulders. Subjects rested supine on a padded treatment table with their GHJ positioned at 90° of abduction and no horizontal adduction (the starting position for each test). An air splint was applied to the arm and forearm to be tested to stabilize the upper extremity and neutralize cutaneous sensation of the arm. The arm to be tested was placed in the splint with the elbow flexed 90° and the forearm fully pronated before inflation. The arm and splint were then fixed to the wheel (Fig. 1).

A motor-driven winch controlled the movement of the wheel by pulling a cable (Fig. 1). The motor was controlled by a rheostat and was geared to rotate the shoulder wheel at a speed of 1° to 2°/sec. Angular displacements were measured to the nearest degree using a pointer mounted on the shoulder wheel. Surface electrodes of a portable J-53 electromyographic biofeedback unit (Cyborg Corp, 1350 S Kostner, Chicago, IL 60623) set at the highest sensitivity were applied over the pectoralis major muscle to monitor any antagonistic activity of this muscle during the tests.

Procedure

Three tests were used to measure kinesthesia in both shoulders of all subjects during one testing session. The right and left shoulders of the uninjured subjects were tested randomly. The uninjured shoulder of the injured subjects was tested first to lessen apprehension when the involved shoulder was tested. Subjects kept their eyes closed to eliminate the potential influence of vision. Our testing time intervals and speeds of shoulder movement were arbitrarily chosen but were based on intervals and speeds used in other

TABLE 1
Physical characteristics of subjects (N = 18)

Variable	Subjects with Dislocations (N = 8)			Healthy Subjects (N = 10)		
	\bar{X}	s	Range	\bar{X}	s	Range
Age (years)	34.0	14.3	(18-64)	22.9	1.9	(21-27)
Height (cm)	178.0	7.0	(168-185)	167.0	8.0	(155-178)
Weight (kg)	74.1	10.4	(63.6-88.6)	62.4	9.5	(50.1-75.9)
Number of dislocations	3.5	2.0	(1-6)			
Time since last incident (months)	8.4	4.1	(3-14)			

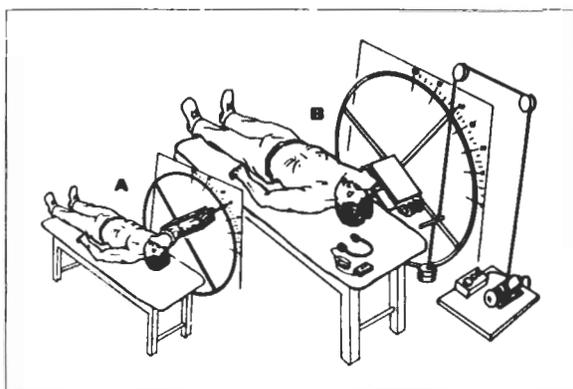


Figure 1. Shoulder-wheel apparatus used for testing. (A) Starting position of shoulder for angular reproduction test. Note that glenohumeral joint (GHJ) is at midrange of lateral rotation. (B) Starting position for threshold-to-sensation-of-movement and end-range reproduction tests. Note that GHJ is at end range of lateral rotation. The motor-driven winch (lower right) turns the weighted wheel. The headphones block auditory cues. Electromyographic biofeedback (not shown) was used to monitor pectoralis major muscle activity.

studies (14, 15, 17). The tests were performed in the following order.

Angular reproduction (AR)

This test was used to examine the subject's ability to reproduce an angle when the shoulder was placed in intermediate ranges of lateral rotation. Each subject was placed in the shoulder-wheel apparatus and instructed to relax. The midrange of lateral rotation was used as the starting position for the test (Fig. 1). This position was determined by subtracting the physiological end-range value from zero (neutral position between medial [internal] and lateral rotation) and dividing by two. For the test, one of the researchers (J. B.) manually rotated the shoulder laterally at a steady rate of approximately $20^\circ/\text{sec}$ through a range of 5 to 30° to a random angle. The angle of this "set" position was recorded. The shoulder was held in the set position for 30 sec, and the subject was instructed to concentrate on this position. The subject's arm was then returned to the starting position at the same speed. After a brief rest, the shoulder was passively rotated laterally back toward the set angle, and the subject was instructed to report when that set position was reached. The angular displacement of the reproduced angle was observed and recorded. Three trials were conducted on each arm, and the mean difference between the set angle and the reproduction angle was recorded as the AR position-sense deficit.

Threshold to sensation of movement (TSM)

This test was used to examine the threshold to the sensation of movement. The subjects were

placed in the apparatus with their tested shoulder positioned in lateral rotation. A standard $3.2 \text{ ft}\cdot\text{lb}$ ($1 \text{ ft}\cdot\text{lb} = 1.356 \text{ N}\cdot\text{m}$) torque was used to produce a slight stretch on the shoulder in this position. The starting position was recorded, and the subjects were instructed to respond when they detected movement of the shoulder. The subjects were given earphones to remove auditory cues, and the motor was started without the shaft engaged. At a random time interval between 5 and 30 seconds, the shaft of the motor was engaged to rotate the shoulder at a rate of $1.5^\circ/\text{sec}$. The angular displacement before the subjects perceived a change of position was recorded as the threshold to sensation. Three trials were conducted on each limb, and the mean value was recorded as the TSM deficit.

End-range reproduction (ERR)

This test, a continuation of the TSM test, was used to examine the subjects' ability to reproduce an angle at the end-range position of shoulder lateral rotation. The shoulder, with $3.2 \text{ ft}\cdot\text{lb}$ of torque applied at the starting position, was moved passively at $1.5^\circ/\text{sec}$ toward lateral rotation. The wheel and cable system caused a total of $4.8 \text{ ft}\cdot\text{lb}$ of torque to be placed on the GHJ. This torque was predetermined to be close to the maximum amount that subjects could comfortably tolerate in this position. The end-range position was recorded in degrees of angular displacement while the subjects concentrated on the position. After 30 sec, the shoulder was passively rotated medially by the motor to the starting position at the same angular velocity. After a brief rest in the starting position, the winch again passively rotated the shoulder laterally toward the end-range position. A faster angular velocity was used for this repositioning to prevent the subjects from duplicating a position based on time. The subjects were instructed to respond when they reached the end-range position of lateral rotation. At that point, the wheel was stopped and the angular displacement was recorded as the subjects' perceived ERR. The subjects' arm was briefly removed from the wheel between each of three ERR trials. The mean angular differences between the end-range position and the subjects' perceived end-range position was recorded as the deficit in ERR position sense.

Angular measurements for all three tests were measured to the nearest degree. A random selection of test-retest measurements ($N = 20$) in unaffected shoulders demonstrated that reliability was high for all three tests (Pearson $r > 0.98$). On the basis of this reliability and because similar tests have been used to study knee joints (15) we concluded that the instrument and procedure were valid for measuring kinesthetic sense.

Data Analysis

The four groups of shoulders tested were 1) dislocated shoulders of injured subjects (designated "involved"), 2) contralateral uninvolved shoulders of injured subjects (designated "uninvolved"), 3) dominant shoulders of uninjured subjects (designated "dominant"), and 4) contralateral nondominant shoulders of uninjured subjects (designated "nondominant"). Dominance was classified according to the handedness of the subject.

We used a one-way analysis of variance (ANOVA) to analyze the kinesthetic deficits among the four groups of shoulders for each of the AR, TSM, and ERR tests. When differences were found, a Scheffé post hoc comparison was performed to determine where the differences in kinesthesia occurred. The level of significance was set at 0.05.

RESULTS

The kinesthetic deficits for the AR, TSM, and ERR tests are shown in Tables 2, 3, and 4, respectively. The involved shoulders demonstrated greater average kinesthetic deficits in all three tests when compared with the uninvolved, dominant, and nondominant shoulders.

TABLE 2

Average kinesthetic deficits for angular reproduction test (N = 18)*

Shoulder Group	\bar{X}	s	Range
Dominant	1.50	0.63	(0.33–2.33)
Nondominant	1.40	0.60	(0.33–2.33)
Involved	2.75	1.22	(1.00–3.67)
Uninvolved	1.08	0.64	(0.33–2.33)

* Values recorded in degrees.

TABLE 3

Average kinesthetic deficits for threshold-to-sensation-of-movement Test (N = 18)*

Shoulder Group	\bar{X}	s	Range
Dominant	1.18	0.77	(0.33–2.33)
Nondominant	1.04	0.46	(0.50–2.00)
Involved	2.58	0.93	(1.00–3.67)
Uninvolved	0.91	0.65	(0.33–2.17)

* Values recorded in degrees.

TABLE 4

Average kinesthetic deficits for end-range reproduction test (N = 18)*

Shoulder Group	\bar{X}	s	Range
Dominant	1.40	0.64	(0.67–2.67)
Nondominant	1.05	0.58	(0.00–2.00)
Involved	3.00	0.81	(1.67–4.33)
Uninvolved	0.98	0.59	(0.00–2.00)

* Values recorded in degrees.

The one-way ANOVA revealed significant differences ($p < 0.001$) among the four shoulder groups for the AR, TSM, and ERR tests (Table 5). Scheffé post hoc comparisons revealed a significant difference ($p < 0.02$) between the involved shoulders and the other three groups of shoulders for all tests. We found no significant differences among the uninvolved, dominant, and nondominant shoulder for any test.

DISCUSSION

The results indicate that shoulder kinesthesia is significantly affected after anterior GHJ dislocation. Our data also demonstrate that the contralateral uninvolved shoulder of the injured subjects had normal kinesthesia as compared with the shoulders of the uninjured subjects. Extremity dominance has no apparent effect on shoulder kinesthesia.

We expect that variations of kinesthesia among healthy individuals exist. Nevertheless, clinicians should establish goals of improving kinesthetic deficits in patients with shoulder dislocations. Our results indicate that the uninvolved shoulder could be used as the standard.

Kinesthesia in Uninjured Shoulders

The average kinesthetic deficit in the shoulders of the uninjured subjects ranged from 0.91 to 1.50 degrees for the three tests (Tables 2–4). Barrack et al. reported average TSM and AR deficits to be approximately 3.8 and 3.6°, respectively, in normal knee joints (15). Grigg et al. measured TSM in normal hip joints and found slight deficits of 0.13–1.32° (13). Accurate detection of shoulder TSM with the GHJ moving 2°/sec has been documented (17). Kinesthetic deficits in normal should-

TABLE 5

Analysis of variance results for kinesthetic deficits in shoulders (N = 18)

Test	df	SS	MS	F	p
AR*					
Shoulder type	3	13.12	4.37	6.95	0.001
Error	32	20.14	0.63		
Total	35	33.27			
TSM†					
Shoulder type	3	14.82	4.94	9.69	0.0002
Error	32	16.32	0.51		
Total	35	31.44			
ERR‡					
Shoulder type	3	22.14	7.38	17.18	0.0001
Error	32	13.75	0.43		
Total	35	35.89			

* Angular reproduction

† Threshold to sensation of movement

‡ End-range reproduction.

ders may be too small to be measured with a routine clinical examination. Our results suggest, however, that kinesthesia in normal shoulders is very precise.

The function of joint receptors as detectors of kinesthesia is controversial. Newton (7) and Rowinski (8) provided extensive reviews of the contributions of joint mechanoreceptors and muscle spindles to joint position and movement sensation. Early studies of anesthetization of the joint capsule and pericapsular structures around the metatarsophalangeal joint of the great toe (18) and the index finger (19) resulted in severely impaired joint position and movement sensation. These results indicated that joint afferents were responsible for kinesthesia. On the other hand, based on findings that 85% AR accuracy was not altered by local anesthetization of knee joint tissues (20) and that total joint replacement did not significantly impair TSM (13–15), Burgess et al. concluded that no current valid evidence shows that joint receptors are important for the conscious awareness of joint positions (21). They suggested that joint receptors contribute to the feeling of deep pressure experienced near the limits of the joint's range of motion and that muscle spindle receptors are involved with conscious perception of joint position sense. The most likely source of position sense in rematching types of studies, therefore, seems to be muscle receptors with minor influences from joint and skin receptors (20).

Ruffini endings in the joint capsule and Golgi endings in the ligaments are slowly adapting receptors sensitive to joint capsule and ligament stretching, respectively (8). Pacinian capsule receptors are rapidly adapting receptors sensitive to high-velocity changes in joint position (8). We theorized that we could test the physiological function of the Ruffini receptors with the AR test, and both the Ruffini receptors and Golgi ligament endings with the TSM and ERR tests. For the TSM and ERR tests, we moved the shoulder at a very slow speed to decrease the discharge of rapidly adapting receptors. We attempted to decrease cutaneous input by using the air splint on the forearm and by preventing the shoulder from contacting the table. Muscle relaxation was required and monitored by EMG biofeedback. We were certain that we had reduced the sensory input from rapidly adapting joint receptors and skin, as well as the influence of muscle contraction on capsule deformation, and thus were testing primarily the integrity of slowly adapting capsular mechanoreceptors and muscle spindles.

Angular Reproduction in Midrange of Lateral Rotation

We observed a significant AR deficit in the involved shoulders of the injured subjects. This

result confirmed the work by Glencross and Thornton (16) which showed that errors in AR measurements between sprained and uninvolved ankle joints are caused by articular receptor damage resulting in distortion of kinesthesia. The error was greatest in the most severely injured ankles. Professional ballet dancers who met criteria for knee-joint laxity were significantly worse than control subjects in their ability to perform an AR test (10).

An interesting trend appeared in the results of AR testing of the involved shoulders. Larger deficits were detected in AR at a set angle of 30° than at other set angles of less than 30°. We believe that sensation of larger ROMs requires a greater number of activated receptors. Our findings of significant deficits in the involved shoulders during AR measurements, combined with the trend of larger AR errors with increased joint ROM, imply that the number of receptors available for activation may be reduced after GHJ dislocation.

Although our results showed significant AR deficits in the involved shoulders of the injured subjects, we do not know with certainty what type of receptor signaled these position changes. Burgess and Clark presented evidence that joint receptors are not activated in intermediate joint positions (22). If the muscle spindle signals intermediate joint position and movement, our results indicate that spindle function may be altered by GHJ dislocation.

Grigg and Greenspan showed that capsular tension and joint-receptor discharge resulted from passively stretching muscles that cross the joint, but the discharge required a heavy load (23). Based on these findings, Newton postulated that muscle contraction would activate a pattern of joint receptors signaling joint position in intermediate ranges (7). This theory does not explain our observations, which included relaxed muscles. Further examination of kinesthesia in intermediate positions is needed.

Threshold to Sensation of Movement at End Range of Lateral Rotation

The TSM test determined the subject's ability to perceive a slow angular change in shoulder position at the end range of lateral rotation. Our findings indicate that TSM significantly declines after dislocation.

In other studies, subjects lost passive-movement sensation in anesthetized metacarpophalangeal joints of the great toe (18) and index finger (19) when tested in intermediate joint positions. End-range positions were not tested in these studies. The threshold to detect the change in knee intermediate joint positions was shown to decline markedly with degenerative joint disease

but to remain unaffected by total-joint replacement (15). A TSM test of intermediate positions of the hip showed that impairment in the threshold to sensation occurred after total-hip arthroplasty in most patients, although differences were significant in only some individuals (13). A more recent study demonstrated that TSM was affected minimally by hip replacement and that the initial test position of the limb, whether placed in the midrange or end range, did not affect the results (14). Anterior GHJ dislocation may cause specific damage to capsular and muscle receptors resulting in impaired TSM. Because lateral rotation end range is the position in which many dislocations occur, the TSM deficit may be related to the specific mechanism of GHJ injury.

End-Range Reproduction of Lateral Rotation

Results of the ERR test showed a significant difference between the involved shoulders and the other shoulders. From observations that slow-adapting capsular afferents serve as "limit detectors" signaling proximity of the joint to its limit of rotation (24) and that Golgi ligament endings and free nerve endings help protect the joint from deformation beyond its anatomical limits (25, 26) we believe the deficit in ERR after GHJ dislocation may be explained by a change in the function of joint afferents. Increased laxity in joints as a result of trauma or stretching may result in either damage to the receptors and consequent impaired inability to detect joint position and movement and angle replication (10, 15, 16) or insufficient discharge or a reduced number of receptors available for discharge after GHJ dislocation. Joint afferent discharge may serve as a warning signal to induce motor reflexes that counteract excessive joint movement and prevent joint damage (26). Individuals with previously dislocated shoulders may have insufficient signals and motor reflexes, be subject to excessive movement, and, thus, be predisposed to recurrent dislocation.

Clinical Considerations

Kinesthetic acuity in the shoulder is critical for placement of the hand in upper limb function. Examples when shoulder position and movement sense are important include using the upper extremity in sports (e.g., throwing a ball), activities of daily living (e.g., grooming), and occupational tasks (e.g., manual labor). Motor control for executing complex activities depends on afferent inputs (6) and may be affected by extensive athletic training (27). Data suggest that risks of injury to a joint increase as the load shifts from the fatigued muscles onto the ligaments (11). Thus, prevention of GHJ injuries depends on the strength and endurance of shoulder musculature. Whether the

ligament protective reflexes and tendon-stretch reflexes (11) are fast enough to prevent dislocation of normal shoulders is unknown.

Our findings of kinesthetic deficits after GHJ dislocation indicate that clinicians should consider shoulder treatment programs that include kinesthetic rehabilitation. For example, clinicians could challenge a patient with an injured shoulder to match and rematch various positions of the involved joint. Exercises such as balancing on all fours with the involved limb on a freely moving platform will facilitate reflexive motor coordination (9). Proprioceptive neuromuscular rehabilitation methods are thought to activate joint and muscle afferents, which in turn elicit reflexive motoneuron activity (28). Therapeutic exercise must incorporate a relearning paradigm in addition to muscle restrengthening. Shoulder dislocation represents, in part, a peripheral neurological dysfunction, and, therefore, therapeutic recovery must include re-development and reestablishment of motor skills based on new and probably abnormal sensory input (8). Recovery of shoulder kinesthesia and reflexive muscular splinting after dislocation most likely requires extensive therapeutic training. The effects of this training require further study.

CONCLUSION

Significant shoulder kinesthetic deficits occur after anterior GHJ dislocation. Kinesthesia is normal in the uninvolved shoulders of subjects with shoulder dislocations and does not seem to be affected by extremity dominance in uninjured subjects. Recurrence of GHJ dislocation may occur as a result of deficits in joint and muscle receptor input and a related loss of neuromuscular coordination. Clinicians should consider rehabilitation of kinesthesia in the treatment of patients with shoulder dislocations. Therapeutic activation of shoulder joint and muscle receptors sensitive to joint motion and position may improve reflexive protection and neuromuscular control of the unstable GHJ.

We thank Richard Gajdosik, PT, Physical Therapy Program, University of Montana, and Kathleen Miller, PhD, Health and Physical Education Department, University of Montana, for their statistical advice and assistance with this manuscript.

REFERENCES

1. McLaughlin HL, MacLellan DI: Recurrent anterior dislocation of the shoulder: Part II. A comparative study. *J Trauma* 7:191-201, 1967
2. Matsen FA, Zuckerman JD: Anterior glenohumeral instability. *Clin Sports Med* 2:319-338, 1983
3. Rowe CR, Sakellandes HT: Factors related to recurrences of anterior dislocations of the shoulder. *Clin Orthop* 20:40-47, 1961
4. Simonet WT, Cofield RH: Prognosis in anterior shoulder dislocation. *Am J Sports Med* 12:19-24, 1984
5. Protzman RR: Anterior instability of the shoulder. *J Bone Joint Surg (Am)* 62:909-918, 1980
6. McCloskey DI: Kinesthetic sensibility. *Physiol Rev* 58:763-820, 1978
7. Newton RA: Joint receptor contributions to reflexive and kinesthetic responses. *Phys Ther* 62:22-29, 1982

8. Rowinski MJ: Afferent neurobiology of the joint. In Gould JA, Davies GJ (eds): Orthopaedic and Sports Physical Therapy Vol 2, pp 50-64 St Louis, MO: CV Mosby Co, 1985
9. Freeman MAR, Dean MRE, Hanhan IWF: The etiology and prevention of functional instability of the foot. *J Bone Joint Surg (Br)* 47:678-685, 1965
10. Barrack RL, Skinner HB, Brunet ME, et al: Joint laxity and proprioception in the knee. *The Physician and Sportsmedicine* 11(6):130-135, 1983
11. Pope MH, Johnson RJ, Brown DW, et al: The role of the musculature in injuries to the medial collateral ligament. *J Bone Joint Surg (Am)* 61:398-402, 1979
12. Kennedy JC, Alexander IJ, Hayes KC: Nerve supply of the human knee and its functional significance. *Am J Sports Med* 10:329-335, 1982
13. Grigg P, Finerman GA, Riley LH: Joint position sense after total hip replacement. *J Bone Joint Surg (Am)* 55:1016-1025, 1973
14. Karanjia PN, Ferguson JH: Passive joint position sense after total hip replacement surgery. *Ann Neurol* 13:654-657, 1983
15. Barrack RL, Skinner HB, Cook SD, et al: Effect of articular disease and total knee arthroplasty on knee joint position sense. *J Neurophysiol* 50:684-687, 1983
16. Glencross D, Thornton E: Position sense following joint injury. *J Sports Med Phys Fitness* 21(1):23-27, 1981
17. Hall LA, McCloskey DI: Detections of movements imposed on finger, elbow and shoulder joints. *J Physiol (Lond)* 335:519-533, 1983
18. Browne K, Lee J, Ring PA: The sensation of passive movement at the metatarso-phalangeal joint of the great toe in man. *J Physiol (Lond)* 126:448-458, 1954
19. Provins KA: The effect of peripheral nerve block on the appreciation and execution of finger movements. *J Physiol (Lond)* 143:55-67, 1958
20. Clark FJ, Horch KW, Bach SM, et al: Contributions of cutaneous and joint receptors to static knee position sense in man. *J Neurophysiol* 42:877-888, 1979
21. Burgess PR, Wei JY, Clark FJ, et al: Signaling of kinesthetic information by peripheral sensory receptors. *Annu Rev Neurosci* 5:171-187, 1982
22. Burgess PR, Clark EJ: Characteristics of knee joint receptors in the cat. *J Physiol (Lond)* 203:317-335, 1969
23. Grigg P, Greenspan BJ: Response of primate joint afferent neurons to mechanical stimulation of knee joint. *J Neurophysiol* 40:1-8, 1977
24. Rossi A, Grigg P: Characteristics of hip joint mechanoreceptors in the cat. *J Neurophysiol* 47:1029-1042, 1982
25. Schultz RA, Miller DC, Clare SK, et al: Mechanoreceptors in human cruciate ligaments. *J Bone Joint Surg (Am)* 66:1072-1076, 1984
26. Schiabel HG, Schmidt RF: Responses of fine medial articular nerve afferents to passive movement of knee joint. *J Neurophysiol* 49:1118-1126, 1983
27. Barrack RL, Skinner HB, Brunet ME, et al: Joint kinesthesia in the highly trained knee. *J Sports Med Phys Fitness* 24(1):18-20, 1984
28. Voss De, Ionta MK, Myers BJ: *Proprioceptive Neuromuscular Facilitation*, Ed 3. Philadelphia, JB Lippincott Co, 1985