Proprioception in Sports Medicine

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Ligaments play a major role in normal joint kinematics, providing mechanical restraint to abnormal joint movement when a stress is placed on the joint. The primary concern of the orthopaedic surgeon has been the biomechanical restoration of these ligaments following injury, in an attempt to restore the joint’s stability and therefore its kinematics. In restoring normal joint kinematics it is speculated that sport activities will be resumed, recurrent injury will be minimized, and progressive joint degeneration can be avoided.

Kennedy, however, observed that in addition to their mechanical restraining function, articular ligaments provide important neurological feedback that directly mediates muscular reflex stabilization about the joint. The neuromuscular controlling mechanism is mediated by articular mechanoreceptors and provides the individual with the proprioceptive sensations of kinesthesia and joint position sense. The neurological feedback for the control of muscular actions serves to protect against excessive strain on passive joint restraints and provides a prophylactic mechanism to recurrent injury. Following joint injury, disruption to these articular mechanoreceptors inhibits normal neuromuscular reflex joint stabilization and contributes to repetitive injuries and the progressive decline of the joint. This neuromuscular reflex is essential for joint stabilization, especially activities requiring running and cutting.

HISTORY OF SENSATION

Awareness of the body and its relationship with the surrounding environment is mediated by the phenomenon of sensation. The history of sensation dates to the Greek philosopher Aristotle, who was the first to...
describe the five senses. Sir Charles Bell described sensation as it relates to limb position and motion as the "sixth sense." This chapter will address articular sensation in its entirety as it mediates the perception of joint position and joint motion that regulates muscle contraction for movement and joint stabilization.

The relevance of articular sensation has been observed by a select few scientists over the past 2 centuries. The French neurologists Duchenne and Charcot drew attention to articular sensations in 1865, while Sherrington and Adrian received the Nobel prize in 1932 for their work on the mechanisms of sensation and were the first to describe proprioception. Abbott, in 1944, was the first to suggest that knee articular sensations were the first step in a kinetic chain that accounted for dynamic joint stabilization. The role of proprioceptive input from knee ligaments in mediating the reflex contraction of the hamstrings and resultant weakening of the reflex following ligament trauma has been demonstrated. Also, current investigators, including Barrack et al., Barrett, and Lephart et al., have demonstrated that joint position perception is altered following articular pathology.

TERMINOLOGY OF JOINT SENSATION

The terminology related to joint sensation is often misunderstood and used inappropriately, which has led to confusion and a lack of appreciation for these mechanisms during rehabilitation. Articular sensations are described as proprioception and kinesthesia. There is considerable discrepancy in the definitions of these two terms as related to their physiological functions. Mountcastle and Willis define proprioception as the conscious awareness of limb position, while they define kinesthesia as the awareness of joint motion. On the other hand, Bastian defines the kinesthetic mechanism as a complex of sensations, including those in which movement is not featured, while Sherrington describes the proprioceptive sense as including vestibular sensations and inputs from muscles and joints that are not necessarily perceived. For the purpose of this chapter, we will define proprioception as a specialized variation of the sensory modality of touch that encompasses the sensations of joint movement (kinesthesia) and joint position (joint position sense).

Conscious proprioception is essential for proper joint function in sports, activities of daily living, and occupational tasks. Unconscious proprioception modulates muscle function and initiates reflex stabilization. Much effort has been dedicated to elucidate the mechanical function of articular structures and the corresponding mechanical deficits that occur secondary to disruption of these structures. Articular structures also have a significant sensory function, which plays a role in dynamic joint stability, acute and chronic injury, pathologic wearing, and rehabilitation training.

JOINT AND MUSCLE NEURAL RECEPTORS

ANATOMY OF NEURAL RECEPTORS

The extrinsic innervation of joints follows Hilton's law, which states that joints are innervated by articular branches of the nerves supplying...
the muscles that cross that joint. The afferent innervation of joints is based on peripheral receptors located in articular, muscular, and cutaneous structures. Articular receptors include nociceptive free nerve endings and proprioceptive mechanoreceptors.

Several authors have identified mechanoreceptors in the cruciate ligaments (CL) of the cat knee joint. Schultz and coworkers were the first to identify mechanoreceptors in the human CL. Using gold chloride, Bielschowsky, and Bodian staining techniques, these investigators histologically examined anterior cruciate ligaments (ACL) obtained from cadaver at the time of total knee arthroplasty and demonstrated the presence of mechanoreceptors in the CL. Using a modified gold chloride technique, Schultz and Zimny further characterized these ACL receptors into three morphologic types of mechanoreceptors (Ruffini endings, Ruffini corpuscles or Golgi tendon-like organs, and Pacinian corpuscles), and free-nerve endings. Others have demonstrated similar mechanoreceptors in cat knee menisci. In their extensive study of the innervation of joints, Freeman and Wyke histologically identified the three types of mechanoreceptors in the capsular and ligamentous structures of cat ankle joints. The presence of mechanoreceptors in human ankle joints, however, has not yet been demonstrated. Finally, the three distinct mechanoreceptors have recently been histologically identified by Vangsness in the glenoid labrum and the glenohumeral ligaments of the shoulder, suggesting that these structures possess the anatomic basis for perceiving joint position and joint motion.

PHYSIOLOGY OF NEURAL RECEPTORS

Mechanoreceptors transduce some function of mechanical deformation into a frequency-modulated neural signal that is transmitted via cortical and reflex pathways. An increased stimulus of deformation is coded by an increased afferent discharge rate or a rise in the population of activated receptors. Grigg and Hoffman have correlated mechanoreceptor afferent discharge with strain energy density and have calibrated mechanoreceptors as in vivo load cells in the posterior capsule of the feline knee. Receptors demonstrate different adaptive properties based on their response to a continuous stimulus. Quick-adapting (QA) mechanoreceptors, such as the Pacinian corpuscle, decrease their discharge rate to extinction within milliseconds of the onset of a continuous stimulus. Slow-adapting (SA) mechanoreceptors, such as the Ruffini ending, Ruffini corpuscles, and the Golgi tendon-like organ, continue their discharge in response to a continuous stimulus. QA mechanoreceptors are very sensitive to changes in stimulation and are therefore thought to mediate the sensation of joint motion. Different populations of SA mechanoreceptors are maximally stimulated at specific joint angles, and thus a continuum of SA receptors is thought to mediate the sensation of joint position and change in joint position. In animal models, these mechanoreceptors respond to active or passive motion with maximal stimulation occurring at the extremes of knee motion. Stimulation of these receptors results in reflex muscle contraction about the joint. In addition to the joint receptors, the muscle spindle receptors are a complex, fusiform, SA receptors found within skeletal muscle. Via afferents and
efferents to intrafusal muscle fibers, the muscle spindle receptor can measure muscle tension over a large range of extrafusal muscle length.

There is considerable debate over the relative contribution to proprioception of muscle receptors vs. joint receptors, with traditional views emphasizing joint mechanoreceptors and more contemporary views emphasizing muscle receptors. Recent work suggests that joint receptors and muscle receptors are probably complementary components of an intricate afferent system in which each receptor modifies the function of the other. With the identification of these receptor types in most joints, and the knowledge of their function, it appears that the ligamentous, cartilaginous, and muscular structures of joints contain the neural components necessary for the sensation of motion (rapidly adapting receptors, e.g., Pacinian corpuscles), joint position and acceleration (slowly adapting receptors, e.g., Ruffini endings and Ruffini corpuscles), and pain (free-nerve endings). This would therefore support the contemporary view that both joint and muscle receptors contribute to the sensory appreciation of joint position.

FUNCTIONAL PROPRIOEPTION STUDIES

Functionally, kinesthesia is assessed by measuring threshold to detection of passive motion (TTDPM), whereas joint position sense is assessed by measuring reproduction of passive positioning (RPP) and reproduction of active positioning (RAP). TTDPM, when tested at slow angular velocity (0.5 degrees/sec–2.5 degrees/sec), is thought to selectively stimulate Ruffini or Golgi-type mechanoreceptors, and because the test is performed passively, it is believed to maximally stimulate joint receptors while minimally stimulating muscle receptors. In shutting down muscle activity, TTDPM is often chosen to assess afferent activity following ligament pathology. RAP, although usually performed at slow speed, stimulates both joint and muscle receptors and provides a more functional assessment of the afferent pathways. Neither TTDPM, RPP, nor RAP provides an assessment of the unconscious reflex arc believed to provide dynamic joint stability. The assessment of reflex capabilities is usually performed using electromyographic (EMG) interpretation of firing patterns of those muscles crossing the respective joint. In patients with unilateral joint involvement, the contralateral uninvolved extremity serves as an internal control, whereas uninjured joints in a normative population serve as external controls.

We have designed a Proprioception Testing Device (PTD) (Fig 1), similar to that described by Barrack and Skinner, to assess TTDPM and RPP of the knee and shoulder. Proprioception is measured using a PTD, which is designed to assess kinesthetic awareness and joint position sense (JPS). The PTD measures the angular displacement of the joint being tested prior to detection of movement by the subject and measures the patient's accuracy in reproducing selected joint angles. The PTD moves the joint at a constant angular velocity ranging from 0.5 degrees/
FIGURE 1.
Proprioceptive Testing Device for the knee: a = rotational transducer; b = motor; c = moving arm; d = stationary arm; e = control panel; f = digital microprocessor; g = hand-held disengage switch; h = pneumatic compression boot; i = pneumatic compression device.

sec to 2.5 degrees/sec. A rotational transducer interfaced with a digital microprocessor counter provides angular displacement values.

KNEE PROPRIOCEPTION TESTING

With the demonstration of the neural framework necessary for joint sensation, investigators have just recently begun to perform functional studies of knee joint proprioception. Barrack and Skinner experimentally assessed proprioception by measuring TTDPM and RPP. They found decreased kinesthesia with increasing age and ACL disruption. Barrack and Skinner also found enhanced kinesthesia in trained dancers. Barrett and coworkers further demonstrated a decline in JPS with osteoarthritis. It is hypothesized that ACL disruption, meniscal injury, and osteoarthritis damage articular structures containing mechanoreceptors and, therefore, result in deficits of kinesthesia and JPS. Skinner further demonstrated that deficits in proprioception are related to increased age, fatigue, and degenerative joint pathology.

Our laboratory has more recently revealed enhanced kinesthesia in intercollegiate gymnasts, as well as some enlightening kinesthetic findings in individuals following ACL reconstruction. Our studies on athletically active patients following either arthroscopically assisted patellar-tendon autograft or allograft ACL reconstruction revealed the following:

1. Kinesthetic deficits were present in the ACL-reconstructed knee compared to the uninvolved knee when tested at 15 degrees of knee flex-
ion (near terminal extension), while moving into both flexion and extension (Fig 2);

2. There was no difference in TTDPM between the ACL-reconstructed knee and the contralateral uninvolved knee from a starting position of 45 degrees of knee flexion (mid-range), while moving into either flexion or extension;

3. Kinesthetic awareness was more sensitive from a starting position of 15 degrees of knee flexion than from a starting position of 45 degrees of knee flexion for both the ACL-reconstructed knee and the uninvolved knee while moving into both flexion and extension (Fig 3); and

4. Kinesthetic awareness in the ACL reconstructed knee was significantly enhanced with the use of a neoprene sleeve (Fig 4).

Although we were primarily focusing on joint receptors in knee injuries, muscle receptors are an integral component of a complex afferent system and may also play a role in kinesthetic awareness of slow, passive motion. In addition to reflex pathways, both joint and muscle mechanoreceptors have been shown to have cortical pathways that account for conscious appreciation of joint movement and position. Our finding of enhanced kinesthetic awareness in the near-terminal range of motion is commensurate with neurophysiological studies that have shown maximal response of joint mechanoreceptors at the extremes of motion.31, 32, 50

As previously stated, Barrack and coworkers9 showed that a proprioceptive deficit exists after ACL disruption, and we have found that a deficit continues after reconstruction.11 Knee articular structures, including the ACL, possess mechanoreceptors, and damage to these structures can result in partial deafferentation. Barrack and coworkers found a longer TTDPM in the ACL-disrupted knee compared to the contralateral uninvolved knee when tested at 30 degrees to 40 degrees of knee flexion.9 In

FIGURE 2.
Mean TTDPM for reconstructed vs. uninvolved knee from a starting position of 15 degrees flexion moving into flexion and extension (± SE, *P < 0.05).
our study we found a longer TTDPM in the ACL-reconstructed knee compared to the contralateral uninvolved knee when tested at 15 degrees knee flexion and no significant difference when tested at 45 degrees knee flexion.11 Thus, kinesthesia in the midrange of motion may have returned following ACL reconstruction. However, kinesthesia is more sensitive in the near-terminal range of motion, hence any difference between the involved and uninvolved knee would be more apparent.

FIGURE 3.
Mean TTDPM for reconstructed vs. uninvolved knee from starting positions of 15 degrees and 45 degrees moving into extension (± SE, *P < 0.05).

FIGURE 4.
Mean TTDPM for reconstructed knee and reconstructed knee with neoprene sleeve from a starting position of 45 degree flexion moving into flexion and extension (± SE, *P < 0.05).
For years, knee surgeons have postulated that the sensory loss associated with ACL injury may affect the results of ACL repair and reconstruction. Du Toit, Insall, and Noyes have all advocated certain reconstructive techniques due, in part, to increased afferent preservation. Theoretically, operative techniques can restore proprioception directly through reinnervation of damaged structures or indirectly through restoration of appropriate tension in capsuloligamentous structures. Acute ACL repair may facilitate regeneration along with maintaining anatomic relationships. The extent of reinnervation in the reconstructed ligament and its relationship to revascularization needs to be addressed. Prosthetic grafts, vascularized grafts, free grafts, and allografts all may have different reinnervation potential.

Bracing and wrapping have been thought to serve a sensory function in addition to a mechanical function. Barrett and coworkers found that an elastic bandage enhanced joint position sense in patients with osteoarthritic knees as well as in patients after total knee arthroplasty. We found enhancement of kinesthesia with the use of a commercially available neoprene sleeve (Pro Orthopedic Devices, Inc, Tucson, Arizona). Proprioception is mediated by afferent input from articular, muscular, and cutaneous structures. The neoprene sleeve could have augmented afferent input by providing increased cutaneous stimulation.

Proprioception may play a protective role in acute knee injury through reflex muscular splinting. The protective reflex arc initiated by mechanoreceptors and muscle spindle receptors occurs much more quickly than the reflex arc initiated by nociceptors (70–100 m/sec vs. 1 m/sec). Thus, proprioception may play a more significant role than pain sensation in preventing injury in the acute setting. Proprioceptive deficits, however, probably play more of a role in the etiology of chronic injuries and reinjury. Initial knee injury results in partial deafferentation and sensory deficits that can predispose to further injury. Proprioceptive deficits may also contribute to the etiology of degenerative joint disease through pathologic wearing of a joint with poor sensation. It is unclear whether the proprioceptive deficits that accompany degenerative joint disease are a result of the underlying pathologic process or contribute to the etiology of the pathologic process.

It is clear that joint effusion, particularly in the knee, contributes to a decreased mechanoreceptor afference, resulting in the inhibition of muscular contractions. In the knee, this inhibition is mediated by slowly adapting mechanoreceptors and appears to provide long-term quadriceps shutdown, particularly in the vastus medialis. A 30% to 50% inhibition of the reflex-evoked quadriceps contraction can be observed with 60 cc of intraarticular effusion. This muscular inhibition severely disrupts neuromuscular training during rehabilitation and provides a fundamental basis for relief of joint effusions, from a neurological basis. Any proprioceptive deficits resulting from chronic joint effusion may contribute to the inability to provide neuromuscular joint control and therefore result in joint degeneration.

A proprioceptive deficit may detract from the functional result of knee surgery, inhibit complete rehabilitation, and predispose the athlete to reinjury. Thus it is clear, based on the results of these studies, that any com-
prehensive rehabilitation program designed to return athletes to preinjury levels of activity following knee injuries should include an extensive proprioception element.

ANKLE PROPRIOCEPTION TESTING

Freeman et al.23 were the first to postulate that chronic ankle instability was due, in part, to partial deafferentation of articular mechanoreceptors with joint injury. They subjectively observed decreased stability in one-legged stance in the sprained ankle vs. the contralateral uninjured ankle. Konradsen et al.53 studied the reaction of subjects with chronic ankle instability to sudden inversion using EMG and joint motion analysis. They found a prolonged peroneal reaction time in these patients vs. age-matched controls, suggestive of a partial deafferentation of reflex stabilization. Garn and Newton54 studied the ability of a subject to properly sense a passive movement or no movement state in the dorsiflexion-plantarflexion plane and found decreased kinesthetic awareness in the involved ankle of subjects with unilateral ankle sprains. Glenncross and Thornton55 reported deficits in active replication of passive ankle/foot positioning in the dorsiflexion-plantarflexion plane while testing the sprained ankle vs. the contralateral uninjured ankle.

Subsequent to these early studies on ankle proprioception, many other studies have demonstrated that proprioceptive deficits play a role in functional stability of the ankle joint.55, 57, 58 Gross59 most recently reported that decrease in sensory input from joint receptors can lead to abnormal body positioning and diminished postural reflex responses leading to an increased probability of reinjury.

The results of studies using stabilometric techniques (force plate, optoelectronic joint analysis) to assess postural sway and balance in patients with chronic ankle instability have been equivocal. Tropp et al.60 found no increase in postural sway when comparing a group of soccer players with histories of ankle sprains to a control group of uninjured soccer players. Tropp et al.60 also compared the involved ankle to the uninjured ankle in a group of soccer players with a history of unilateral, recurrent ankle sprains and found no differences in postural sway. Cornwall and Murrell,51 however, found a significant increase in postural sway when comparing individuals with an acute ankle sprain to uninjured controls as much as two years following the injury.

“Proprioceptive training” techniques following acute and chronic ankle sprain injuries are the most widely used compared with other injuries, yet these techniques have only empirical evidence of effectiveness and remain untested. Ankle wrapping/bracing has also been suggested to carry a proprioceptive benefit; however, this also remains unproven.

SHOULDER PROPRIOCEPTION TESTING

Although recent investigations involving the knee and ankle have drawn attention to the sensory role of articular structures and proprioception deficits following injury,9, 11 proprioceptive sensation of the shoulder, in contrast, has not been well studied. The perception of joint position and joint movement in the shoulder is essential for placement of the hand in
upper limb function. In addition, proprioception may play an important role in dynamic shoulder stability and modulation of muscle function.

Symptoms of instability in the shoulder are commonly attributed to the loss of static and dynamic mechanical restraint provided by intact muscular and capsuloligamentous structures. Shoulder capsuloligamentous structures may contain receptors that, along with muscular and cutaneous receptors, provide the basis for a more active mechanism of protective joint restraint and joint position sense. With injury to these structures, partial deafferentation may occur with resultant proprioceptive deficits. This, in turn, could lead to reinjury. The contribution of proprioceptive deficits to the vicious cycle of insidious microtrauma involved in impingement syndrome and recurrent instability is unclear (Fig 5). In addition, the protective role of proprioceptive-initiated reflex muscular splinting in acute traumatic instability is unknown.

It is unclear whether proprioceptive deficits occur after shoulder injury and how these deficits affect joint function and symptoms of instability. The role of proprioception in the pathogenesis of instability and impingement syndrome remains to be elucidated. In addition, the effect of surgical intervention on shoulder proprioception is unknown. Recent work by Smith and Brunolli has suggested that a sensory deficit occurs in patients with recurrent, atraumatic, anterior instability.45

The PTD discussed previously also permits us to assess both TTDPM and RPP of the shoulder (Fig 6). Testing is performed with the subject positioned supine with the shoulder at 90 degrees abduction and the elbow in 90 degrees of flexion. TTDPM of internal and external rotatory movements are measured from starting positions of neutral rotation and 30 degrees to 75 degrees external rotation. In a population of college-age individuals without any history of shoulder injury, we found minimal variation in kinesthesia and no differences between dominant and non-dominant shoulders (Fig 7).44

In a group of male subjects with unilateral, traumatic, recurrent, anterior shoulder instability, we demonstrated both TTDPM and RPP deficits,12 similar to the findings of Smith and Brunolli (see Fig 7).47 These two studies elucidate a pattern of proprioceptive deficits in unstable shoulders. The uninvolved shoulders in our study demonstrated proprioceptive measurements similar to those of the normative population with-
out shoulder dysfunction. Although we have not yet had the opportunity to study it, one can hypothesize that altered proprioception in unstable shoulders may influence the dynamic mechanisms of joint restraint. This would therefore indicate the necessity of integrating shoulder kinesthetic and joint position sense exercises as a part of shoulder rehabilitation. It is logical to assume that methods to improve proprioception in patients with shoulder disorders could improve shoulder function and decrease the risk of reinjury.

Reconstruction of the capsuloligamentous structures about various joints has proven successful for the restoration of the structural mechanism responsible for joint stability. Yet few investigations have elucidated the effects surgery has on the sensory mechanism that provides proprioceptively mediated dynamic stabilization and joint position sensibility for skilled movements.

While our understanding of neural mechanisms in joint position sense is growing, consideration of proprioception in relation to surgery is still in its infancy. Most of the work reported discusses proprioception following ACL reconstruction and following knee or hip arthroplasty. As stated previously in this chapter, several authors have revealed that function is better with enhanced proprioception, and failure of this procedure may be due to poor proprioception. We recently studied proprioception following capsulolabral reconstruction for shoulder instability and revealed smaller deficiencies between the surgical and normal contralateral shoulder than in the group of instability patients (see Fig 7).

There are a number of surgical factors that may preserve and restore joint proprioception. The first is the degree of surgical trauma to intact

FIGURE 6. Proprioception Testing Device for shoulder testing: a = rotational transducer; b = motor; c = moving arm; d = control panel; e = digital microprocessor; f = pneumatic compression device; g = hand-held disengage switch; h = pneumatic compression sleeve.
structures. Surgery around the joint should be done in such a way to preserve the integrity of its mechanoreceptors and afferent nerves of the joint structures. With minimal disruption to intake nervous tissue there will be preservation of uninjured afferents.

The second consideration related to proprioception during surgery is the facilitation of regrowth, or regeneration, of sensory afferents. Recent histology studies have demonstrated repopulation of mechanoreceptors in canine, sheep, and goat models after ACL reconstruction. Repair, rather than excision, of traumatized tissue theoretically maintains tension, and thus input, to the mechanoreceptors while promoting regeneration of damaged neural structures.

The most likely explanation for enhanced proprioception in these patients undergoing shoulder capsulolabral reconstruction centers around
the procedural technique used that promoted modification of joint sensation. As previously mentioned, because the procedure minimized soft-tissue dissection there was a minimal loss of intact mechanoreceptors and a promotion of repopulation. In addition, the use of the capsular shift in these shoulder instability cases, which tightens the capsule, “retensions” the soft tissue and most likely facilitates proprioception function.

Finally, rehabilitation considerations may be related to enhanced proprioception following shoulder capsulolabral reconstruction. Although in our studies the instability group underwent similar rehabilitation activities as the surgery group, mechanical stabilization following surgery could have provided for more effective neuromuscular retension and hence promoted enhanced function of this mechanism. The rehabilitation program for these patients emphasized proprioceptive input to recognize joint position as well as learning correct movement patterns and techniques apart from development of strength and endurance. These exercises included matching and rematching joint position, weight bearing through the upper extremity, and open kinetic chain exercises. The later stages of the rehabilitation focused 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 have been have been identified reproducibly and believed to play a key role in joint arthrokinematics.67, 68

**NEUROMUSCULAR/PROPRIOCEPTION REHABILITATION**

Developing a sports rehabilitation program that incorporates proprioceptively mediated muscular control of joints necessitates an appreciation for the influence of the central nervous system (CNS) on motor activities. Joint afferents contribute to CNS function at three distinct levels of motor control.54 At the spinal level, reflexes subserve movement patterns that are received from higher levels of the nervous system. This provides for reflex splinting during conditions of abnormal stress about the joint and has significant implications for rehabilitation.11, 59 The muscle spindles play a major role in the control of muscular movement by adjusting activity in the lower motor neurons.54 Partial deafferentation of joint afferent receptors has also been shown to alter the musculature’s ability to provide co-contraction joint stabilization by antagonistic and synergistic muscles, thus resulting in the potential for reinjury.1

The second level of motor control is at the brain stem, where joint afference is relayed to maintain posture and balance of the body. The input to the brain stem about this information emanates from the joint proprioceptors, the vestibular centers in the ears, and from the eyes.70

The final aspect of motor control includes the highest level of CNS function (motor cortex, basal ganglia, and cerebellum) and is mediated by cognitive awareness of body position and movement. These higher centers initiate and program motor commands for voluntary movements. Movements that are repeated can be stored as central commands and can be performed without continuous reference to consciousness.70 Although considerable controversy still exists relative to the role of joint and muscle receptors in the perception of joint movement, deep pressure, at the very least, can be consciously appreciated during joint motion.71
FIGURE 8.
Lower extremity kinesthetic training on an unstable platform to retrain reflex muscular stabilization.

With these three levels of motor control in mind, mediated in part by joint and muscle afferents, one can begin to develop rehabilitation activities to address proprioceptive deficiencies. The objectives must be to stimulate the joint and muscle receptors in order to encourage maximum afferent discharge to the respective CNS level. At the spinal level, activi-

FIGURE 9.
Shoulder joint repositioning exercise at end ranges of motion.
ties that encourage reflex joint stabilization should be addressed. Such activities include sudden alterations in joint positioning that necessitate reflex muscular stabilization (Fig 8). Balance and postural activities, both with and without visual input, will enhance motor function at the level of the brain stem. While consciously performed, joint positioning activities, especially at joint end ranges, will maximally stimulate the conversion of conscious to unconscious motor programming (Fig 9).

**SUMMARY**

The recent appreciation of the sensory mechanism of articular structures has shed light on the role of proprioception following joint injury. This is especially relevant following sport-related injuries due to the role proprioception plays in performance of skilled movements and with reflex joint stabilization during running and throwing. Our research and that of others has provided a basis for substantiating proprioception deficits with joint injury and the necessity for integrating specific activities into the rehabilitation program for the athlete. Additionally, this work provides exciting information relative to the potential effect surgery has on enhancing this neurosensory mechanism. Although this information provides us with a basis for understanding proprioception, there is much still not understood relative to this complex mechanism.

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