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The Sensorimotor System, Part II: The Role of Proprioception in Motor Control and Functional Joint Stability

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Objective: To discuss the role of proprioception in motor control and in activation of the dynamic restraints for functional joint stability.

Data Sources: Information was drawn from an extensive MEDLINE search of the scientific literature conducted in the areas of proprioception, motor control, neuromuscular control, and mechanisms of functional joint stability for the years 1970–1999.

Data Synthesis: Proprioception is conveyed to all levels of the central nervous system. It serves fundamental roles for optimal motor control and sensorimotor control over the dynamic restraints.

Conclusions/Applications: Although controversy remains over the precise contributions of specific mechanoreceptors, proprioception as a whole is an essential component to controlling activation of the dynamic restraints and motor control. Enhanced muscle stiffness, of which muscle spindles are a crucial element, is argued to be an important characteristic for dynamic joint stability. Articular mechanoreceptors are attributed instrumental influence over gamma motor neuron activation, and therefore, serve to indirectly influence muscle stiffness. In addition, articular mechanoreceptors appear to influence higher motor center control over the dynamic restraints. Further research conducted in these areas will continue to assist in providing a scientific basis to the selection and development of clinical procedures.

Key Words: neuromuscular, stability, motor control

This is Part II of a 2-part series discussing the current understanding surrounding peripheral afferent information acquisition, processing, and levels of motor control as they relate to functional joint stability. In Part I, the sensorimotor system and the mechanisms responsible for proprioception and neuromuscular control as they relate to functional joint stability were addressed. The purpose of Part II is to build upon and apply the concepts developed in the Part I. Specifically, we will address the contribution of proprioception in controlling the activation of the dynamic restraints and motor control.

The Role of Proprioception in Motor Control

Critical to effective motor control is accurate sensory information concerning both the external and internal environmental conditions of the body.[1–4] During goal-directed behavior, such as picking up a box while walking, provisions must be made to adapt the motor program for walking to changes occurring in the external environment (uneven ground) and internal environment (change in center of mass because of the additional load). These provisions are stimulated by sensory triggers occurring in both feedback (mechanoreceptor detection of altered support surface) and feedforward (anticipating center-of-mass change from previous experience) manners. Although some of the afferent information may be redundant across the 3 sensory sources (somatosensory, visual, vestibular), specific unique roles are associated with each source that may not be entirely compensated for by the other sensory sources. For example, proprioceptive information plays an integral role in the ability to modify internal models used with feedforward control[5,6] that has been demonstrated to be only partly compensated for by visual information.[7]

The role of proprioceptive information in motor control can be separated into 2 categories.[2] The first category involves the role of proprioception with respect to the external environment. Motor programs often have to be adjusted to accommodate unexpected perturbations or changes in the external environment. Although the source of this information is often largely associated with visual input, there are many circumstances in which proprioceptive input is the quickest or the most accurate, or both.[1] In the above example, modification of the motor program for walking was required in response to the uneven support surface. If the person's vision was fixed on the box to be picked up, he or she might not have visually noted the uneven support surface. In addition to alterations in the plantar cutaneous receptors, muscle and joint mechanoreceptors would have reported the degree of altered ankle joint position and stimulated the motor program modification required. The planning of movements also requires attention to environmental constraints.[8] This is especially true with respect to the selection of strategies for the maintenance of postural control.[9–11] For example, sensory detection of an unstable handrail from peripheral signals (kinesthesia, changing joint

positions) would alter the motor program used to avoid falling on a slippery staircase. During the planning stages of a movement, visual images are used to create a model of the environment in which the movement will occur. Proprioception has been described as essential during the movement execution to update the feedforward commands derived from the visual image.[5,6]

The second category of roles proprioceptive information plays in motor control is in the planning and modification of internally generated motor commands.[2] Before and during a motor command, the motor control system must consider the current and changing positions of the joints involved to account for the complex mechanical interactions within the components of the musculoskeletal system.[2] Proprioception best provides the needed segmental movement and position information to the motor control system.[1,2] In the situation of a single joint moving through a 10° arc of motion, the precise muscle force required to perform the task depends upon the joint angle. As one can surmise, the task of determining how much tension in a muscle is required for a movement becomes extremely complex and important with movements involving several joints.[2,4,12] Accompanying each angular change in joint position are changes in the mechanical advantages associated with all the muscles that traverse the joint. Many tasks involve a sequence of overlapping joint movements. The motor control system must consider the multiple motions occurring as both a direct function of muscle activation and indirectly from intersegmental dynamics (movement of one joint inducing movement of another). Proprioception provides much of the information required to solve all these movement problems.[2,4,7,12,13]

Role of Proprioception in Sensorimotor Control of Functional Joint Stability

Motor control for even simple tasks is a plastic process[3] that undergoes constant review and modification based upon the integration and analysis of sensory input, efferent motor commands, and resultant movements. Proprioceptive information stemming from joint and muscle receptors, as previously demonstrated, plays an integral role in this process. Underlying the execution of all motor tasks are particular events, often very subtle, that are aimed at preparing, maintaining, and restoring stability of both the entire body (postural stability) and the segments (joint stability). With respect to joint stability, these actions represent neuromuscular control. Proprioceptive information, first recognized and described by Sherrington[14] almost 100 years ago, is essential to maintaining both types of stability. Because articular mechanoreceptors are believed to become disrupted in conjunction with joint injury, this section will focus on the role articular mechanoreceptors serve in sensorimotor control over functional joint stability. A discussion of the contribution of articular receptors to postural control has recently been published.[15]

Since the work of Palmer,[16] one of the major tenets concerning the role of joint afferents in functional joint stability has been direct reflexive activation of alpha motor neurons (α MNs).[17–19] This belief, however, is not uncontested[20–22] and represents one of the biggest ongoing debates within the sensorimotor system. Direct evidence supporting the existence of ligament-muscle reflexes has largely arisen though direct electric and mechanical stimulation of the knee, ankle, and shoulder ligaments or capsule (or both).[19,23–26] Similar to the use of electric stimulation on afferent nerve fibers to document cortical projections, the applicability of these findings to normal physiologic function remains speculative and uncertain.[27] Specific to the mechanical stimuli, the loading required to elicit α-MN responses has been criticized as exceeding normal physiologic loads.[22,28,29] Even assuming that the ligament-muscle reflex exists, one must question its effectiveness in contributing to joint protection because of the latency times[22,29,30] and weak response magnitudes,[31] especially in comparison with reflexes stimulated from muscle spindles.[28] Despite the controversial basic science and empirical support, in vivo human studies involving ankle and knee joint perturbations in conjunction with electromyography have been conducted and have produced varying results.[17,18,32–40] For example, at the ankle, the number of investigations demonstrating increased latencies with mechanically or functionally unstable joints (or both)[32–35] is matched by just as many studies failing to elicit differences.[36–40] Several factors must be considered with respect to the conclusions that can be drawn from this experimental model. These are reviewed in a subsequent paper describing sensorimotor measurement techniques.[41]

In contrast with the seemingly controversial activation of α MNs, joint afferents are more unanimously credited with eliciting similar effects on gamma motor neurons (γ MNs).[21,22,29,42,43] Interestingly, and in opposition to what many have claimed, Freeman and Wyke[43] attributed increases in muscle activity in response to joint mechanoreceptor stimulation to activation of γ MNs, not α MNs. Since their study, many investigations have demonstrated reflexive action of joint afferents on γ MNs through electric stimulation[44] and tissue traction using force levels below those associated with tissue damage and nociception.[21,22,42,45,46] Increased γ-MN activation, which may occur from input arising from cutaneous or muscle sources as well as descending supraspinal commands, serves to heighten muscle spindle sensitivity. What does increased muscle spindle sensitivity have in connection with sensorimotor control of functional joint stability? The answer to this question will become evident in the following discussion of stiffness.

Muscle stiffness is defined as the ratio of change in force per change in length.[29,47,48] In contrast to muscle stiffness, which refers specifically to the stiffness properties exhibited by tenomuscular tissues, joint stiffness involves the contributions of all of the structures located within and over the joint (muscles, tendons, skin, subcutaneous tissue, fascia, ligaments, joint capsule, and cartilage).[49–51] Several studies have been conducted in attempts to quantify the contributions of each structure to joint stiffness. These studies generally indicate that the muscle and joint ligamentous and capsular structures traversing the joint contribute equally in passive modes.[50,51]

The constituents of muscle stiffness can be categorized into intrinsic and extrinsic (reflex) components.[52] Many of the elements comprising muscle tissue and connecting noncontractile tissues (tendon, fascia) contain high amounts of collagen and, therefore, exhibit the properties of elasticity and viscosity when stretched. In addition, the intrinsic component encompasses the number of actin-myosin cross-bridges (level of muscle activation) existing at an instant,[29,53] as well as the factors of both single muscle fibers (ie, sarcomere length-tension and force-velocity relationships) and whole muscles (ie, arrangement of muscle fibers within a muscle).[54] The levels of activation existing within a muscle at a given instant are a

function of both preceding reflexes and descending influences on the α-MN pool.[29]

The extrinsic contribution of muscle stiffness arises from the increased reflexive neural activation of the muscle. This is largely determined by the excitability of the motorneuron pool,[29] which in itself is largely dependent upon the sensitivity of primary muscle spindle afferents eliciting autogenetic and heterogenetic reflexes, as well as descending neural commands. Superimposed on these constituents are a number of interacting factors involving the whole muscle-joint complex, such as joint kinematics (ie, angle, velocity), attachment sites (ie, exact location of muscle insertions), and tissue transitions (ie, muscle, tendon, bone).[54]

From a theoretic perspective, increased muscle stiffness and, therefore, enhanced joint stiffness, appears to be a beneficial characteristic for augmented functional joint stability. First, stiffer muscles should potentially resist sudden joint displacements more effectively.[29,47,55,56] Although not all destabilizing forces may be entirely countered, many could potentially be lessened in magnitude, thereby reducing the incidence of joint subluxation and injury. This may be essential in maintaining functional stability when mechanical stability is deficient and may assist in explaining the moderate correlation between hamstring muscle stiffness and functional ability in anterior cruciate ligament (ACL)-deficient individuals found by McNair et al.[47] Directly, voluntary muscle contraction of a muscle group has been demonstrated to increase joint stiffness.[56,57] Cocontraction of antagonistic muscles is believed to further enhance joint stiffness by increasing the compression between the articular surfaces.[29,56,57]

Second, intrinsically stiffer muscles enhance the potential capacities of the extrinsic component. Stiffer muscles as a result of increased activation are also believed to transmit loads to muscle spindles more readily, thereby reducing some of the lag time associated with initiation of reflexive activity.[58,59] Some of the physical events contributing to electromechanical delay, such as the time interval between muscle activation and onset of segmental acceleration,[60] are reduced in muscles with higher activation levels. Thus, not only is the initial resistance to joint displacement superior through heightened intrinsic stiffness, but the ability to recruit an improved reflexive response is also enhanced.

Higher motor control centers have been credited with compensating for static stabilizer deficiency losses through altered movement and muscle activation patterns.[61–63] Similar to the spinal reflexes discussed, both direct and indirect evidence suggests that joint and ligmentous mechanoreceptors are important for supraspinal sensorimotor control over dynamic joint stability. In humans, the difficulty surrounding this aspect of the sensorimotor system arises from the inability to easily induce isolated experimental manipulations to one or more target structures without eliciting numerous confounding factors. Thus, researchers most often attempt to retrospectively measure patients with different conditions and speculate concerning whether elicited changes or adaptations result from damage to static stabilizers, neural elements, or both. Direct evidence supporting the role of articular receptors in sensorimotor control of dynamic joint stability can only be obtained from animal studies after experimentally induced deafferentation. An exorbitant amount of retrospective human research has documented alterations in movement and muscle activation patterns in mechanically and functionally unstable joints, so we will only review several of the common themes supporting the role of articular receptors to higher motor control centers.

With knee injuries, for example, persons sustaining an ACL rupture develop an adaptive motor pattern that involves increased hamstrings activation before joint loading[64–67] and maintaining the knee in a more flexed position during the acceptance of the load.[61,68,69] Both of these alterations are believed to prevent anterior tibial translation in the absence of the ACL. The increase in hamstrings activity occurs before joint loading in a feedforward control manner. This suggests that the motor program for the activity was changed and indirectly supports the idea of motor control change above a reflexive level.

The alterations in muscle activation sequences appear to occur not only at the involved joint but also at distal and proximal joints, further supporting the idea of higher motor changes. With respect to alterations in proximal joint positioning and activation, evidence has been found in subjects sustaining ACL rupture[61] and ankle injury.[38,70] Increased activation of musculature acting on the ankle and lower leg (anterior tibialis and soleus) has been demonstrated in ACL-deficient subjects.[65] After ankle injury, several investigators[71–73] have reported use of postural control strategies that rely more heavily on proximal joint (hip) muscle activation. Collectively, all of these investigations support the premise of higher center motor control changes after orthopaedic injury. Again, the stimulus for these changes remains debatable: afferent changes from the articular receptors, loss of mechanical stability, or both.

Freeman and Wyke[74] pioneered direct evidence supporting the importance of articular receptors in sensorimotor control over joint stability by surgically resecting the posterior or medial articular nerves of cats. Since both of these nerves convey afferent information predominantly from the knee joint, the surgical procedure caused the deafferentation of the joint without disrupting mechanical stability. After the surgery, in addition to spinal-level motor alterations, the animals displayed changes in supraspinal motor programs controlling voluntary movements. Further, postural control adjustments that were initiated from visual and vestibular sources were also altered. The authors hypothesized that the alterations developed secondary to the loss of local input concerning stresses on the knee joint capsule. When accompanied by mechanical stability disruptions, the adaptations in movement programs developed after injury may help prevent damage to secondary restraints and arthropathy.[28] O'Connor et al,[75] using dogs, reported that although joint deafferentation alone was not enough to induce joint degeneration, when combined with ACL transection, severe degenerative changes became more quickly evident than after ligament transection alone.

Thus, it appears that proprioception is fundamental for sensorimotor control over joint stability, with articular receptors providing unique, subtle roles. With respect to stiffness, muscle spindles with higher γ-MN drive enhance both the feedforward and feedback controls of the dynamic restraint mechanism through direct regulation of muscle activation levels. Since γ-MN activation is largely influenced by peripheral afferent input, the adequacy and accuracy of the input become important considerations. Given the sensitivity of joint and ligament receptors through ranges of joint motion and their potent influences on γ-MN activity, it becomes quite likely that this indirect mechanism may outweigh the importance of the controversial direct α-MN reflexes. At higher motor levels, joint receptors may play essential roles in the development of

motor program adaptations to compensate for losses in mechanical stability. Figure 4 in Part I summarizes the role of articular receptors in sensorimotor control of functional stability. Further research is needed in all of these areas to fully elucidate the precise mechanisms by which joint receptors contribute.

CONCLUSIONS

Proprioception is conveyed to all levels of the central nervous system, where it provides a unique sensory component to optimize motor control. Additionally, proprioceptive information is necessary for neuromuscular control of the dynamic restraints. Joint receptors, which are often damaged to some degree during articular injury, appear to be an important component to proprioception. While their role in eliciting direct muscular reflexes remains controversial, their role in influencing the γ MNs and supraspinal motor programs appears to be more substantial. Further research concerning the role of articular mechanoreceptors in promoting γ-MN activation and supraspinal motor control is needed. Supraspinal control over the dynamic restaints may be the area that has the most relevance to the development of preventive and rehabilitative strategies. Intervening at supraspinal levels may provide the key to promoting increased dynamic stability from a preparatory perspective, rather than the debatable reactive perspective.

REFERENCES

1. Ghez C. The control of movement. In: Kandel ER, Schwartz JH, Jessell TM, eds. *Principles of Neural Science.* 3rd ed. New York, NY: Elsevier Science; 1991:533–547.
2. Hasan Z, Stuart DG. Animal solutions to problems of movement control: the role of proprioceptors. *Annu Rev Neurosci.* 1988;11:199–223.
3. Leonard CT. *The Neuroscience of Human Movement.* St Louis, MO: Mosby-Year Book Inc; 1998.
4. Matthews PB. The 1989 James A.F. Stevenson memorial lecture. The knee jerk: still an enigma? *Can J Physiol Pharmacol.* 1990;68:347–354.
5. Sainburg RL, Ghilardi MF, Poizner H, Chez C. Control of limb dynamics in normal subjects and patients without proprioception. *J Neurophysiol.* 1995;73:820–835.
6. Bard C, Fleury M, Teasdale N, Paillard J, Nougier V. Contribution of proprioception for calibrating and updating the motor space. *Can J Physiol Pharmacol.* 1995;73:246–254.
7. Rothwell JC, Traub MM, Day BL, Obeso JA, Thomas PK, Marsden CD. Manual motor performance in a deafferented man. *Brain.* 1982;105:515–542.
8. Enoka RM. *Neuromechanical Basis of Kinesiology.* 2nd ed. Champaign, IL: Human Kinetics; 1994.
9. Horak FB, Nashner LM, Diener HC. Postural strategies associated with somatosensory and vestibular loss. *Exp Brain Res.* 1990;82:167–177.
10. Diener H, Dichgans J, Guschlbauer B, Mau H. The significance of proprioception on postural stabilization as assessed by ischemia. *Brain Res.* 1984;296:103–109.
11. Inglis JT, Horak FB, Shupert CL, Jones-Rycewicz C. The importance of somatosensory information in triggering and scaling automatic postural responses in humans. *Exp Brain Res.* 1994;101:159–164.
12. Sainburg RL, Poizner J, Ghez C. Loss of proprioception produces deficits in interjoint coordination. *J Neurophysiol.* 1993;70:2136–2147.
13. Cordo P, Bevan L, Gurfinkel V, Carlton L, Carlton M, Kerr G. Proprioceptive coordination of discrete movement sequences: mechanism and generality. *Can J Physiol Pharmacol.* 1995;73:305–315.
14. Sherrington CS. *The Integrative Action of the Nervous System.* New York, NY: C Scribner's Sons; 1906.
15. Riemann BL, Guskiewicz KM. Contibution of peripheral somatosensory system to balance and postural equilibrium. In: Lephart SM, Fu FH, eds. *Proprioception and Neuromuscular Control in Joint Stability.* Champaign, IL: Human Kinetics; 2000.
16. Palmer I. On injuries to ligaments of knee joint: clinical study. *Acta Chir Scand.* 1938;5:3.
17. Beard DJ, Kyberd PJ, Fergusson CM, Dodd CA. Proprioception after rupture of the anterior cruciate ligament: an objective indication of the need for surgery? *J Bone Joint Surg Br.* 1993;75:311–315.
18. Wojtys E, Huston L. Neuromuscular performance in normal and anterior cruciate ligament-deficient lower extremities: an objective indication of the need for surgery? *Am J Sports Med.* 1994;22:89–104.
19. Kim AW, Rosen AM, Brander VA, Buchanan TS. Selective muscle activation following electrical stimulation of the collateral ligaments of the human knee joint. *Arch Phys Med Rehabil.* 1995;76:750–757.
20. Jennings AG, Seedhom BB. Proprioception in the knee and reflex hamstring contraction latency. *J Bone Joint Surg Br.* 1994;76:491–494.
21. Miyatsu M, Atsuta Y, Watakabe M. The physiology of mechanoreceptors in the anterior cruciate ligament: an experimental study in decerebrate-spinalised animals. *J Bone Joint Surg Br.* 1993;75:653–657.
22. Raunest J, Sager M, Burgener E. Proprioceptive mechanisms in the cruciate ligaments: an electromyographic study on reflex activity in the thigh muscles. *J Trauma.* 1996;41:488–493.
23. Knatt T, Guanche CA, Solomonow M, Lu Y, Baratta RM, Zhou BH. The glenohumeral-biceps reflex in the feline. *Clin Orthop.* 1995;314:247–252.
24. Guanche CA, Knatt T, Solomonow M, Lu Y, Baratta RV. The synergistic action of the capsule and the shoulder muscles. *Am J Sports Med.* 1995; 23:301–306.
25. Ekholm J, Eklund G, Skoglund S. On the reflex effects from the knee joint of the cat. *Acta Physiol Scand.* 1960;50:167–174.
26. Solmonow M, Baratta R, Zhou BH, D'Ambrosia RD. The synergistic action of the anterior crutiate ligament and thigh muscles in maintaining joint stability. *Am J Sports Med.* 1987;15:207–213.
27. Wand P, Prochazka A, Sontag KH. Neuromuscular responses to gait perturbations in freely moving cats. *Exp Brain Res.* 1980;38:109–114.
28. Hogervorst T, Brand RA. Mechanoreceptors in joint function. *J Bone Joint Surg Am.* 1998;80:1365–1378.
29. Johansson H, Sjolander P. The neurophysiology of joints. In: Wright V, Radin EL, eds. *Mechanics of Joints: Physiology, Pathophysiology and Treatment.* New York, NY: Marcel Dekker Inc; 1993:243–290.
30. Pope MH, Johnson RJ, Brown DW, Tighe C. The role of musculature in injuries to the medial collateral ligament. *J Bone Joint Surg Am.* 1979; 61:398–402.
31. Barrack RL, Lund PJ, Skinner HB. Knee joint proprioception revisited. *J Sport Rehabil.* 1994;3:18–42.
32. Karlsson J, Peterson L, Andreasson G, Hogfors C. The unstable ankle: a combined EMG and biomechanical modeling study. *Int J Sport Biomech.* 1992;8:129–144.
33. Konradsen L, Ravn JB. Prolonged peroneal reaction time in ankle instability. *Int J Sports Med.* 1991;12:290–292.
34. Konradsen L, Ravn JB. Ankle instability caused by prolonged peroneal reaction time. *Acta Orthop Scand.* 1990;61:388–390.
35. Lofvenberg R, Karrholm J, Sundelin G, Ahlgren O. Prolonged reaction time in patients with chronic lateral instability of the ankle. *Am J Sports Med.* 1995;23:414–417.
36. Ebig M, Lephart SM, Burdett RG, Miller MC, Pincivero DM. The effect of sudden inversion stress on EMG activity of the peroneal and tibialis anterior muscles in the chronically unstable ankle. *J Orthop Sports Phys Ther.* 1997;26:73–77.
37. Johnson MB, Johnson CL. Electromyographic response of peroneal muscles in surgical and nonsurgical injured ankles during sudden inversion. *J Orthop Sports Phys Ther.* 1993;18:497–501.
38. Beckman SM, Buchanan TS. Ankle inversion injury and hypermobility: effect on hip and ankle muscle electromyography onset latency. *Arch Phys Med Rehabil.* 1995;76:1138–1143.
39. Nawoczenski DA, Owen MG, Ecker ML, Altman B, Epler M. Objective evaluation of peroneal response to sudden inversion stress. *J Orthop Sports Phys Ther.* 1985;7:107–109.
40. Isakov E, Mizrahi J, Solzi P, Susak Z, Lotem M. Response of the peroneal muscles to sudden inversion of the ankle during standing. *Int J Sport Biomech.* 1986;2:100–109.

41. Riemann BL, Lephart SM. Sensorimotor system measurement techniques. *J Athl Train.* 2002;37:85–98.
42. Johansson J, Sjolander P, Sojka P. A sensory role for the cruciate ligaments. *Clin Orthop.* 1991;268:161–178.
43. Freeman MA, Wyke B. Articular reflexes at the ankle joint: an electromyographic study of normal and abnormal influences of ankle joint mechanoreceptors upon reflex activity in the leg muscles. *Br J Surg.* 1967;54: 990–1001.
44. Johansson H, Sjolander P, Sojka P. Actions on gamma-motoneurones elicited by electrical stimulation of joint afferent fibres in the hind limb of the cat. *J Physiol.* 1986;375:137–152.
45. Sojka P, Johansson H, Sjolander P, Lorentzon R, Djupsjobacka M. Fusimotor neurons can be reflexly influenced by activity in receptor afferents from the posterior cruciate ligament. *Brain Res.* 1989;483:177–183.
46. Johansson H, Sjolander P, Sojka P, Wadell I. Reflex actions on the γ-muscle spindle systems of muscles acting at the knee joint elicited by stretch of the posterior cruciate ligament. *Neuro-Orthopedics.* 1989;8:9–21.
47. McNair PJ, Wood GA, Marshall RN. Stiffness of the hamstring muscles and its relationship to function in anterior cruciate ligament deficient individuals. *Clin Biomech.* 1991;7:131–137.
48. Nichols TR. The organization of heterogenic reflexes among muscles crossing the ankle joint in the decerebrate cat. *J Physiol.* 1989;410:463–477.
49. Wright V. Stiffness: a review of its measurement and physiological importance. *Physiotherapy.* 1973;59:107–111.
50. Helliwell PS. Joint stiffness. In: Wright V, Radin EL, eds. *Mechanics of Joints: Physiology, Pathophysiology and Treatment.* New York, NY: Marcel Dekker Inc; 1993:203–218.
51. Johns RJ, Wright V. Relative importance of various tissues in joint stiffness. *J Appl Physiol.* 1962;17:824–828.
52. Sinkjaer T, Toft E, Andreassen S, Hornemann BC. Muscle stiffness in human ankle dorsiflexors: instrinsic and reflex components. *J Neurophysiol.* 1988;60:1110–1121.
53. Morgan DL. Separation of active and passive components of short-range stiffness of muscle. *Am J Physiol.* 1977;232:C45–C49.
54. Lieber RL, Friden J. Neuromuscular stabilization of the shoulder girdle. In: Matsen FA, Fu FH, Hawkins R, eds. *The Shoulder: A Balance of Mobility and Stability.* Rosemont, IL: American Academy of Orthopaedic Surgeons; 1993:91–105.
55. Grillner S. The role of muscle stiffness in meeting the changing postural and locomotor requirements for force development by the ankle extensors. *Acta Physiol Scand.* 1972;86:92–108.
56. Louie JK, Mote CD Jr. Contribution of the musculature to rotatory laxity and torsional stiffness at the knee. *J Biomech.* 1987;20:281–300.
57. Olmstead TG, Wevers HW, Bryant JT, Gouw GJ. Effect of muscular activity on valgus/varus laxity and stiffness of the knee. *J Biomech.* 1986; 19:565–577.
58. Fellows SJ, Thilmann AF. The role of joint biomechanics in determining stretch reflex latency at the normal ankle. *Exp Brain Res.* 1989;77:135–139.
59. Rack PM, Ross HF, Thilmann AF, Walters DK. Reflex responses at the human ankle: the importance of tendon compliance. *J Physiol.* 1983;344: 503–524.
60. Grabiner MD. Bioelectric characteristics of the electromechanical delay preceding concentric contraction. *Med Sci Sports Exerc.* 1986;18:37–43.
61. Gauffin H, Tropp H. Altered movement and muscular-activation patterns during the one-legged jump in patients with an old anterior cruciate ligament rupture. *Am J Sports Med.* 1992;20:182–192.
62. McNair PJ, Marshall RN. Landing characteristics in subjects with normal and anterior cruciate deficient knee joints. *Arch Phys Med Rehabil.* 1994; 75:584–589.
63. DeMont RG, Lephart SM, Giraldo JL, Swanik CB, Fu FH. Muscle preactivity of anterior cruciate ligament-deficient and reconstructed females during various lower extremity activities. *J Athl Train.* 1999;34:115–120.
64. Kalund S, Sinkjaer R, Arendt-Nielsen L, Simonsen O. Altered timing of hamstring muscule action in anterior cruciate ligment deficient patients. *Am J Sports Med.* 1990;18:245–248.
65. Ciccotti MG, Kerlan RK, Perry J, Pink M. An electromyographic analysis of the knee during functional activities, II: the anterior cruciate ligament-deficient knee and reconstructed profiles. *Am J Sports Med.* 1994;22:651–658.
66. Branch TP, Hunter R, Donath M. Dynamic EMG analysis of anterior cruciate deficient legs with and without bracing during cutting. *Am J Sports Med.* 1989;17:35–41.
67. Sinkjaer T, Arendt-Nielsen L. Knee stability and muscle coordination in patients with anterior cruciate ligament injuries: an electromyographic approach. *J Electromyogr Kinesiol.* 1991;1:209–217.
68. Berchuck M, Andriacchi TP, Bach BR, Reider BR. Gait adaptations by patients who have a deficient anterior cruciate ligament. *J Bone Joint Surg Am.* 1990;72:871–877.
69. Andriacchi TP. Dynamics of pathological motion: applied to the anterior cruciate deficient knee. *J Biomech.* 1990;23(suppl 1):99–105.
70. Bullock-Saxton JE, Janda V, Bullock MI. The influence of ankle sprain injury on muscle activation during hip extension. *Int J Sports Med.* 1994; 15:330–334.
71. Tropp H, Odenrick P. Postural control in single-limb stance. *J Orthop Res.* 1988;6:833–839.
72. Brunt D, Andersen JC, Huntsman B, Reinhert LB, Thorell AC, Sterling JC. Postural responses to lateral perturbation in healthy subjects and ankle sprain patients. *Med Sci Sports Exerc.* 1992;24:171–176.
73. Pintsaar A, Brynhildsen J, Tropp H. Postural corrections after standardized perturbations of single limb stance: effect of training and orthotic devices in patients with ankle instability. *Br J Sports Med.* 1996;30:151–155.
74. Freeman MA, Wyke B. Articular contributions to limb muscle reflexes: the effects of partial neurectomy of the knee joint on postural reflexes. *Br J Surg.* 1966;53:61–68.
75. O'Connor BL, Visco DM, Brandt KD, Meyers SL, Kalasinki LA. Neurogenic acceleration of osteoarthrosis: the effects of previous neurectomy of the articular nerves on the development of osteoarthrosis after transection of the anterior cruciate ligament in dogs. *J Bone Joint Surg Am.* 1992;74:367–376.