Dynamic Knee Stability: Current Theory and Implications for Clinicians and Scientists

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We will discuss the mechanisms by which dynamic knee stability may be achieved and relate this to issues that interest clinicians and scientists concerned with dynamic knee stability. Emphasis is placed on the neurophysiologic evidence and theory related to neuromuscular control. Specific topics discussed include the ensemble firing of peripheral mechanoreceptors, the potential for muscle stiffness modulation via force and length feedback, postural control synergies, motor programs, and the neural control of gait. Factors related to answering the difficult question of whether or not knee ligament injuries can be prevented during athletic activities are discussed. Prevention programs that train athletes to perform their sport skills in a safe fashion are put forth as the most promising prospect for injury prevention. Methods of assessing neuromuscular function are reviewed critically and the need for future research in this area is emphasized. We conclude with a brief review of the literature regarding neuromuscular training programs. *J Orthop Sports Phys Ther 2001;31:546–566.*

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thletic success depends on the ability to run, jump, and change direction at high rates of speed in a rapidly changing environment. The knee joint is subjected to extremely high forces and moments during these activities because it lies between the 2 longest lever arms in the body and is surrounded by its most powerful muscles. The ability of the knee joint to remain stable when subjected to the rapidly changing loads it withstands during activity is referred to as *dynamic knee stability*. The purpose of this paper is to discuss the anatomic and physiologic bases for dynamic knee stability and to relate these concepts to current clinical and research concepts. This information may assist clinicians in developing more effective knee injury treatment programs and provide scientists a framework for answering important questions related to these injuries and their prevention and treatment.

Dynamic knee stability is the result of the integration of articular geometry, soft tissue restraints, and the loads applied to the joint from weight-bearing and muscle action. The bony architecture of the knee provides little stability to the joint due to the incongruity of the tibial and femoral condyles. Although the shape, orientation, and functional properties of the menisci improve joint congruity and may provide some stability via a mechanism similar to a chop block, the stability provided is minimal considering the large loads transferred through the joint.2,50,114,153 Conversely, the orientations and material properties of the ligaments, capsule, and musculotendinous soft tissues of the knee contribute significantly to its stability.^{25,26,92,123} The knee ligaments guide adjacent skeletal segments during joint motion and are the primary restraints to knee joint translations during passive loading.51,177,178 Although the knee has many ligaments, the 4 primary contributors to its stability are the anterior cruciate ligament (ACL), posterior cruciate ligament (PCL),

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FIGURE 1. Path diagram describing feedback and feed-forward control systems. Adapted from Houk JC, Rymer WZ. Neural control of muscle length and tension. In: Brooks VB, ed. Handbook of Physiology: Section 1: The Nervous System. Vol. 2. Motor Control. Bethesda, Md: American Physiological Society; 1981:260. Reprinted with permission from the American Physiological Society.

medial collateral ligament (MCL), and lateral collateral ligament (LCL).^{26,141,172} The fiber recruitment of each of these ligaments varies depending on joint angle and the plane in which the knee is loaded.^{24,52,60,85,168,170,173} Consequently, in most instances there are several ligaments synergistically contributing to knee stability although one of them usually bears a significant portion of the load. While the combined efforts of ligaments and other soft tissues provide the knee with good stability in conditions when the loads applied to the joint are moderate, the strain applied to these tissues during aggressive activities (eg, stopping or changing direction quickly in sports) often exceeds their material strength.^{14,30,55,107} For this reason, additional stabilizing forces are required to keep the knee in a position where the strains in the ligaments remain within a safe range. Joint compressive forces, resulting from weight-bearing and loads applied to the joint segments by muscle activity, provide these stabilizing forces.^{35,92,115,121,164,169} The stabilizing forces provided by the nervous system's control of muscle activity are of particular interest because this is the only component of dynamic knee stability that can be addressed with therapeutic interventions. Our remaining discussion will focus on the neuromuscular mechanisms associated with dynamic knee stability and the application of this knowledge to clinical and scientific practice.

THE NEUROMUSCULAR CONTROL SYSTEM

The ability to produce controlled movement through coordinated muscle activity is commonly referred to as *neuromuscular control*. Neuromuscular control results from a complex interaction between the nervous system and the musculoskeletal system. In a very basic model, the neuromuscular system can be reduced to 3 components: sensory organs, neural pathways, and muscles. The control theory concepts of feedback and feed-forward control are typically used to model the function of this system (Figure 1).^{18,57,88,90} In a system that uses feedback control, sensors continually measure a specific parameter (the regulated parameter) and send data to a controller that compares the sensor's measurement to a reference value (set point). If the sensor's measurement is different than the reference value, an error signal is generated. The error signal functions to trigger a compensatory response that forces the regulated parameter back toward homeostasis with the reference value. The classic example of this type of system is a heating system that regulates temperature via a thermostat. Conversely, a system that uses feed-forward control has sensors situated to detect potential disturbances in the environment that would alter the status of the regulated variable. When a sensor in this system detects a potential disturbance, it sends an impulse to the controller indicating an impending change in the regulated parameter. In response to this signal, the controller institutes commands to counteract the anticipated effects of the disturbance. The commands instituted are chosen based upon past experience with similar disturbances. Pavlov's experiment demonstrating conditioned reflexes (the dogs salivating when a bell was rung) provides a physiologic example of feed-forward control. Although many of the details of the neuromuscular system's control strategy have yet to be precisely defined, this system is thought to use a complex control strategy that incorporates both feedback and feed-forward mechanisms.^{18,90,111} In our opinion, an understanding of the neuromuscular control system is fundamental to designing effective treatment programs and meaningful research studies related to dynamic knee stability.

Mechanoreceptors

The sensors in the neuromuscular control system are referred to as mechanoreceptors. These small

Receptor type	Location	Sensitive to	Active when the joint is	Activation threshold	Response to persistent stimuli
Ruffini endings	Capsule and ligaments	Joint position intra-articular pressure amplitude of movement velocity of movement	Static or dynamic	Low	Slowing adapting
Pacinian corpuscles	Capsule, ligaments, menis- ci, and fat pads	Acceleration or decelera- tion	Dynamic only	Low	Rapidly adapting
Golgi tendon organ-like	Ligaments and menisci	Tension in ligaments, es- pecially at end-range of motion	Dynamic only	High	Slowly adapting
Free nerve endings	Widely distributed in cap- sule, ligaments, and fat pads, fewer in menisci	Pain from mechanical or chemical origin	Inactive, except in the pres- ence of noxious stimuli; then static or dynamic	High	Slowly adapting

sensory organs are located in soft connective tissues and respond to various forms of mechanical deformation (eg, tension, compression, or the rate of loading), as their name implies. Mechanoreceptors are generally classified into the following 3 groups based upon the tissues in which they are found: joint receptors, cutaneous (skin) receptors, and muscle receptors. When stimulated at a sufficient intensity, these receptors generate impulses referred to as afferent (sensory) signals that are subsequently propagated to the central nervous system (CNS). Afferent signals are mediated at 3 levels of the CNS: the spinal cord, the brain stem and cerebellum, and the cognitive centers (cortex).¹² The CNS processes the afferent signals and generates motor response signals (efferent signals), which modify conditions at the local level by modulating muscle activity. While it is common to discuss the function of the individual classes of mechanoreceptors separately, neurophysiologic evidence suggests that the nervous system processes ensembles of sensory signals from many receptors, rather than processing each individual signal separately.^{53,54,95,145,146,151,175} In fact, the neurophysiologic evidence suggests that the CNS processes hundreds of thousands of impulses each second, with input coming from thousands of receptors.145,146 This allows the nervous system to obtain a more complete picture of the conditions at the periphery and maintains a level of redundancy within the system at all times. This redundancy of sensory information may allow the nervous system to maintain normal or near normal function despite the presence of errors or a lack of feedback that may occur during unexpected circumstances, such as an injury.

Joint receptors Four types of joint receptors have been identified in the soft tissues of the knee: Ruffini endings, Pacinian corpuscles, Golgi tendon organlike receptors, and free nerve endings.^{16,46,71,79–81,104} Joint receptors are generally described by the stimuli they respond to and by the following characteristics: (1) the joint state in which they are active (static, dynamic, or both), (2) the stimulus intensity at which they reach their threshold for activation (low-threshold versus high-threshold), and (3) whether they remain active with persistent stimuli (slowly adapting) or respond quickly and then become quiet (rapidly adapting) (Table 1).

The literature is contradictory regarding the functional role of joint receptors.29,41,70,73,100 The role of these sensory organs has been studied in both animal (primarily the cat) and human models.^{20,73,96,106,150,160} Although researchers have analyzed each of the soft tissues of the knee, most studies have focused on the ligaments because of their important role in knee stability.^{81,106,152,163,181} Each type of joint receptor has been observed in both the ligaments and the joint capsule.13,80,81,103,104,152,181 Researchers have described the capsule as richly innervated, while the ligaments appear to be best described as sparsely innervated.^{70,72,82,152} Due to the high incidence of ACL injuries, the ACL has been the object of study in a large number of the papers devoted to knee joint receptors.62,81,98,105,108,152,181 Most of the mechanoreceptors located in the ACL are found in the superficial tissue of the ligament and at its terminal ends.^{104,147,152,181} Several theories have been put forth that suggest that joint receptors located in the ACL and other tissues of the knee play a significant role in maintaining knee joint stability.34,99,100,104,164 The basis for these theories comes from studies that have reported that reflex pathways exist between the receptors of the knee joint and the musculature of the thigh. These pathways have been established with electromyographic recordings of hamstrings and quadriceps activity in response to electrical stimulation or mechanical loads applied to the knee ligaments.4,8,96,98,106,162-164 While several studies have confirmed the presence of reflex pathways that originate with joint receptor signals from the collateral ligaments and joint capsule,38,97,106,163 the results of studies evaluating the existence of reflexes between the ACL and hamstrings have been equivocal.66,67,98,142,164 The mechanoreceptors and reflex pathways described provide evidence for a protective

mechanism at the knee; however, the degree to which this mechanism contributes to dynamic knee stability remains unclear.

Perhaps the most convincing evidence for a potential joint receptor contribution to dynamic knee stability is provided by a series of studies from Johansson and coauthors.^{96-98,101,162,163} These authors have reported that both electrical96,97 and mechanical stimulation^{97,98,162,163} of joint receptors in the knee of the cat resulted in reflex responses involving the γ -motor system. These responses could be measured both from muscle spindles and at the spinal level. The fact that the reflex responses observed were elicited with relatively low loads (5-40 N) and could be observed with as little as 5-10 N makes it unlikely that these authors were observing a nociceptive response.98,162,163 Based on their findings, the authors suggested that joint receptors contribute to joint stability by modulating the stiffness (change in force per unit change in length) of muscles in a continuous fashion.95,99,100 Although this theory is intriguing and promising, it must be noted that researchers have yet to demonstrate the presence of these reflex pathways in humans or during functional activities. As a result, it is currently unknown whether the observed findings are simply the result of well-designed laboratory experiments or if they are physiologic processes that are present during normal joint function in humans. If further research determines that joint receptors modulate muscle stiffness through the γ motor system during the normal activity of humans, it would still need to be determined whether the stiffness provided by the joint receptor γ -motor system pathway is sufficient to make a meaningful contribution to joint stability.

Nearly half a century ago, researchers suggested that joint receptors were responsible for joint position sense.^{17,19,160} Researchers have also reported that these mechanoreceptors are active throughout the range of motion and facilitate kinesthetic sense (sensation of joint movement).41,42 Yet, Burke et al23 and Gandevia et al54 reported that although joint receptors do provide kinesthetic information, their contribution is minor in comparison to that of muscle receptors. The fact that most researchers have reported that joint receptors become active as the joint approaches the end-range of motion (especially in extension), but are minimally active in the mid-range of motion, supports this theory.21,29,69,126 For this reason, it is believed that although joint receptors may contribute to joint position sense and kinesthesia to some degree, the primary roles of these mechanoreceptors are signaling that the end-range of joint motion is approaching and facilitating protective reflexes that prevent the joint from being moved beyond its limits of motion.

Cutaneous receptors The role of cutaneous receptors in initiating reflexive responses (eg, the flexion



FIGURE 2. The Golgi tendon organ. Adapted from Gordon J, Ghez C. Muscle receptors and spinal reflexes: the stretch reflex. In: Kandel ER, Schwartz JH, Jessell TM, eds. *Principles of Neural Science*. 3rd ed. New York, NY: Elsevier; 1991:565. Reprinted with permission from McGraw-Hill.

withdrawal reflex) in response to potential harmful mechanical or thermal stimuli is well established.⁶⁴ Although feedback from these receptors is included within the variety of ensembles of information that the nervous system processes, there is no evidence that supports the idea that cutaneous receptors contribute significantly to dynamic knee stability.²² Some evidence suggests that these receptors may signal information regarding joint position and kinesthesia when the skin is stretched;^{37,94,120} however, there is also evidence that indicates that they make no appreciable contribution to these sensations.144 As with the joint receptors, the contribution of the cutaneous receptors to joint position sense is believed to be less substantial than the contribution from muscle receptors.22,23,54

Muscle receptors There are 2 primary types of muscle receptors, the Golgi tendon organ (GTO) and the muscle spindle.⁶⁵ The GTO is typically located at the musculotendinous junction where the collagen fibers of the tendon attach to the extrafusal muscle fibers (in series). A single axon enters the GTO capsule and then branches into many unmyelinated endings that are interwoven through and between the collagen fibers (Figure 2). When a muscle contracts, the slack in the collagen fibers of the



FIGURE 3. The (A) gross structure and (B) structure and innervation of the intrafusal fibers of the muscle spindle. Adapted from Gordon J, Ghez C. Muscle receptors and spinal reflexes: the stretch reflex. In: Kandel ER, Schwartz JH, Jessell TM, eds. *Principles of Neural Science*. 3rd ed. New York, NY: Elsevier; 1991:564. Reprinted with permission from McGraw-Hill.

GTO is taken up and the nerve endings interwoven between them are stimulated. Golgi tendon organs are primarily sensitive to changes in muscle tension (force).^{89,90,134,165} The current understanding of these sensory organs suggests that each organ is connected to a small number of muscle fibers (~ 3–25), instead of being attached to many fibers, as was once thought. The number of motor units represented in this small group of muscle fibers is also small (\leq 15).^{89,165} Furthermore, evidence suggests that these receptors can respond to forces of less than 0.1 grams.⁸⁹ Golgi tendon organs are therefore very sensitive to changes in force and able to provide the nervous system with very specific force feedback.^{89,90,134}

The second type of muscle receptor, called muscle spindles, are encapsulated structures that range from 4–10 mm in length and lie parallel with muscle fibers (Figure 3).65 These specialized sensory organs are sensitive to changes in muscle length (stretch) and velocity.65,124,125 Each spindle has 3 main components: (1) intrafusal muscle fibers, (2) sensory axons that wrap around the intrafusal fibers and project afferent information to the CNS when stimulated, and (3) motor axons which innervate the intrafusal fibers and regulate the sensitivity of the muscle spindle (γ motoneurons).65,93,125 The primary sensory axons (Ia afferents) from the spindle make monosynaptic connections with α -motoneurons in the ventral roots of the spinal cord that, in turn, innervate the muscle within which the spindles are found. This feedback loop is known as the muscle stretch reflex.⁶⁵ In addition, the primary sensory axons from muscle spindles make di- and tri-synaptic connections with α -motoneurons that impact not only the homonymous muscle, but other muscles as well.^{132,133} The primary function of muscle spindles is to modulate muscle length.65.90,134 When an external load is applied to the muscle, the intrafusal fibers in the spindle increase the firing rate in the afferent axon projecting to the spinal cord. This afferent discharge causes the α -motoneuron supplying that muscle to fire, resulting in a contraction of the homonymous muscle. When the load decreases, the spindle's firing rate decreases, reducing the firing rate of the α -motoneuron. Because the sensitivity of the muscle spindles is continually modulated by the γ -motor system (fusimotor system), the spindles allow for joint position and velocity to be sensed throughout the range of motion.65,93,125

Muscle receptors may function to reflexively modulate muscle stiffness.^{90,91,133,134} Muscle stiffness modulation is the result of 3 factors: (1) the intrinsic stiffness properties unique to the muscle involved (as described by its length-tension curve), (2) force feedback, which is mediated by the GTO pathway and inhibits motor activity, and (3) length feedback, which is mediated by the muscle spindle pathway and facilitates motor activity.90,135,155,156 The intrinsic stiffness of the muscle is also known as nonreflex stiffness, while the ratio of GTO activity (the numerator of the stiffness equation) to muscle spindle activity (the denominator of the stiffness equation) is known as reflex stiffness.90 Perturbations usually result in loads that alter both muscle length and muscle force.⁹⁰ Whether a change in motor output occurs depends on the balance between force and length feedback. If the resultant GTO activity is greater than the muscle spindle activity, a decrease in motor activity and muscle stiffness will occur; however, if the opposite is true, there will be an increase in muscle activity and stiffness.90 The total magnitude of muscle stiffness is a result of the intrinsic stiffness of the muscle represented by the slope of its length-tension curve and the increase or decrease that occurs when GTO activity and muscle spindle activity are added (Figure 4).90 This basic control system described for muscle stiffness modulation has been referred to as the "motor servo."87,90,91

The motor servo is believed to be active in all movements and has been reported to be a final common processor of motor commands.⁹⁰ Although there is considerable evidence to support the concept of stiffness modulation, many of the details regarding this process have yet to be defined or are currently debated among neuroscientists. Nevertheless, the feedback provided by muscle receptors is undoubtedly of vital importance in the maintenance of dynamic knee stability. In theory, loads that may challenge the stability of the knee would disrupt the



Muscle Length

FIGURE 4. Component diagram of muscle stiffness modulation demonstrating the interaction of force feedback and length feedback (reflex stiffness; defined by the slope of the line segment a-d) and the component arising from the length-tension properties of the muscle (nonreflex stiffness; represented by the dashed curve and labeled muscular component). Adapted from Houk JC, Rymer WZ. Neural control of muscle length and tension. In: Brooks VB, ed. Handbook of Physiology: Section 1: The Nervous System. Vol. 2. Motor Control. Bethesda, Md: American Physiological Society; 1981:267. Reprinted with permission from the American Physiological Society.

balance between the ensembles of feedback from GTOs and muscle spindles (force feedback:length feedback) and produce a neuromuscular response in the muscles surrounding the knee that results in a more stable joint.¹³² There is no evidence to suggest that the neuromuscular system directly regulates joint stiffness (resistance to displacement); rather, changes in joint stiffness are an indirect result of changes in muscle activity.¹³²

Muscle contraction and co-contraction can increase joint stiffness and unload the ligaments of the knee.^{3,61,115,117,122,136} Markolf et al¹²² demonstrated, in a study performed in vivo, that knee joint stiffness could be increased by 2- to 4-fold, and knee joint laxity decreased by 25 to 50 percent when subjects voluntarily co-contracted their quadriceps and hamstrings muscles. Similarly, Louie and Mote¹¹⁷ demonstrated a 400% increase in knee joint stiffness with voluntary co-contraction. Although the increases in knee joint stiffness demonstrated in these studies are the result of forceful voluntary co-contractions and may not be representative of what typically occurs during activity, the results of these studies suggest that coordinated muscle activity can increase knee joint stability and decrease the strain in its ligaments.

Motor Response Pathways

The sensory signals provided by the mechanoreceptors are mediated at 3 levels in the nervous system: the segmental level of the spinal cord, the brain stem and cerebellum, and the cerebral cortex.¹² Each of these centers makes unique contributions to the neuromuscular control system. At the segmental level of the spinal cord, spinal reflexes are produced. Spinal reflexes provide the nervous system with elementary patterns of coordination that can be initiated in response to signals from sensory inputs or descending signals from the brain.⁶⁴ The output of segmental spinal reflexes is generally stereotypical and modified by the intensity of the afferent signals.65 The most basic spinal reflexes are monosynaptic reflexes in which the afferent pathway from the peripheral receptors synapses directly with the α -motoneuron in the ventral horn of the spinal cord.65 These are the quickest neuromuscular responses with latencies of 30-50 ms (Table 2).18.151 Most spinal reflexes, however, have more complex circuits that include additional synapses with interneurons and result in the coordinated activity of groups of muscles rather than affecting a single muscle.^{18,65,131,133} As seen in our discussion of muscle receptors, some segmental spinal reflexes are excitatory, while others are inhibitory. Spinal reflexes are part of a distributed neural network in the spinal cord that is thought to provide the framework for rapid postural responses and the regulation of limb mechanics during movement.133

The motor responses resulting from sensory input mediated in the brain stem and cerebellum are typically referred to as long-loop reflexes.^{18,110,151} As a result of the increased length of these pathways, the latencies of their responses (50–80 ms) are longer than those for segmental spinal reflexes.^{18,110,151} Yet, because these reflexes are processed at a higher level of the CNS, they are more flexible than the segmental spinal reflexes.^{18,40,151} Research has demonstrated that these reflexes can adapt when prior instructions are provided to the system.^{40,128} As a result of this adaptability and the relative quickness with which they occur, these pathways are thought to be impor-

TABLE 2. Motor response pathways.

Motor response type	Level of mediation	Typical latency (ms)	Able to be modified	Impacted by degrees of joint freedom
Spinal reflexes	Segmental level of the spinal cord	30-50	No	No
Long-loop reflexes	Brain stem and cerebellum	50-80	No	No
Triggered reactions	Cortical centers	80-120	Yes	Yes
Reaction time	Cortical centers	120-180	Yes	Yes

tant in the maintenance of dynamic knee stability.³⁴ The extent to which therapeutic interventions can affect these reflexes remains unclear.

Voluntary reactions to disturbances in the environment (reaction time movements) are processed in the cortical centers of the brain. The latency of these responses is usually greater than 120 ms.^{18,59,151} These motor responses are highly flexible due to the complex processing that occurs in the cerebral cortex, but are also impacted by the number of degrees of joint freedom that are present at the time of processing.^{18,59,151} The greater the number of variables that need to be processed, the longer the latency for the response.^{18,59,151}

Crago, Houk, and Hasan³² have described a fourth type of motor responses referred to as triggered reactions. The latency of these responses (80–120 ms) falls between that of long-loop reflexes and reaction time movements.32,151 They have been described as preprogrammed, coordinated reactions that occur in response to afferent stimuli that trigger them into action.¹⁵¹ Although their characteristics are similar to reaction time responses, triggered reactions occur more quickly because their preprogrammed organization allows them to bypass some of the typical processing stages.^{32,151} The gains in response rate from the preprogrammed organization are, however, offset to some degree by greater difficulty in adapting to additional degrees of joint freedom.32,151 Therefore, in typical situations, triggered reactions expedite motor responses, but in atypical situations, they may be unable to accommodate to the circumstances and subsequently produce movement errors.

Control Strategies in Stance and Gait

Postural control involves controlling the body's position in space for the dual purposes of stability and orientation.154 Postural orientation refers to the ability to maintain an appropriate relationship between body segments and also between those body segments and the environment when performing a task. Postural stability is defined as the ability to maintain the body's center of mass within specific boundaries (stability limits).¹⁵⁴ The nervous system uses sensory information from 3 sources to produce postural control: (1) somatosensory feedback from peripheral receptors, (2) vision, and (3) the vestibular system.^{58,154} Each of these sensory systems makes unique contributions to postural control. The function of the mechanoreceptors has already been discussed in relative detail earlier in this paper. As might be expected, the visual system's contribution to postural control comes from the detection of motion in the visual field.58 The vestibular system uses the semicircular canals of the ear to enable the nervous system to differentiate whether the head or the external environment is in motion.58,151 The combined effort of these

sensory modalities lays the framework for dynamic balance (stability). If feedback from any one of these modalities is impaired, then postural stability suffers.

Investigators have identified several postural control strategies that result from different types of perturbations in stance. These strategies are composed of characteristic muscle recruitment patterns, often referred to as muscle synergies. In situations where the perturbation to equilibrium is small and the support surface is firm, a postural control strategy referred to as the ankle strategy is usually employed.¹³⁰ This strategy restores the body's center of mass to stability through body movement centered primarily around the ankle joints.¹³⁰ Another strategy called the hip strategy is used when large perturbations to stance are experienced or when the individual is unable to generate enough force with the ankle strategy.86 A third strategy called the stepping strategy is used in instances where the perturbation is strong enough to displace the center of mass outside the person's base of support.¹⁵⁴ In this strategy, the person either takes a step or hops in order to regain his or her balance.18,154 Unique muscle synergies accompany each of these postural control strategies.^{18,86,129,130,154} In general, if forward sway is induced as a result of a posterior horizontal perturbation, then muscles on the posterior aspect of the body are recruited (eg, gastrocnemius, hamstrings, erector spinae).129,130 Conversely, if backward sway is induced from an anterior horizontal perturbation, muscles on the anterior aspect of the body are recruited (eg, tibialis anterior, quadriceps, rectus abdominus).129,130 These postural control strategies can be modified and are adaptive to the circumstances of the moment; however, in the absence of other instructions, they are predictable.¹⁵⁴ As a result, they are often called automatic postural adjustments.154 Evidence suggests that a person's expectations of impending perturbations and training can have a significant impact on the magnitude and variability of the responses.^{1,154} These postural control strategies provide stability in stance and, therefore, are applicable to the maintenance of knee stability in stance. Stability during locomotion, however, is more complex and involves additional mechanisms.

Scientists believe that the neuromuscular system uses *motor programs* to achieve movement because of the many degrees of joint freedom that would need to be processed during movement and the temporal issues associated with feedback loops mediated in the higher brain centers.^{18,151} Motor programs are sequences of commands within the nervous system that produce coordinated movement when they are initiated.¹⁵¹ These programs are under central control and are generally not dependent on feedback from the periphery.¹⁵¹ Feedback, however, is used extensively to select the appropriate motor program, monitor whether the movement is in keeping with the

program, and reflexively modulate the movement when necessary.^{18,151} While most motor programs are coded in the higher centers of the brain, the general program for the gait pattern is not.36,74-77,137 Animal studies, including those with spinalized cat models, have demonstrated that the rhythmic pattern of gait can continue in the absence of feedback from the limbs or descending control from the brain.^{36,74–77,137} This is possible through complex neural circuits in the spinal cord called central pattern generators or limb controllers.^{36,74-77,137} Each limb has its own controller.36,75,137 These neural circuits can be turned on and off by various stimuli but are generally initiated or terminated by signals originating in the brain stem.36,74-76,137 While the basic pattern of gait produced by the limb controllers (whether walking or running) is programmed, descending input from the higher centers of the brain and reflexive feedback from mechanoreceptors in the periphery can modify the gait pattern.74-76,137,171 Reflexive input from the periphery (ie, mechanoreceptors) is, however, primarily directed at ensuring that the patterns specified by the limb controllers are maintained effectively even when unexpected changes in the environment are encountered (eg, changes in terrain or perturbations).75,137,139,151,171 Although reflexes are generally stereotypical, Forssberg and coauthors⁴⁵ have demonstrated that during the gait cycle, a stimulus applied to the foot results in different reflexive responses depending on whether it is applied during the swing or stance phase of gait. This effect has been referred to as the reflex reversal phenomenon.^{45,64,151} This variation of the triggered reaction results from feed-forward control signals that modify reflexive activity at the segmental level of the spinal cord in order to maintain appropriate movement patterns.¹⁵¹ Thus, the control of gait is achieved by a complex interaction between limb controllers, descending input from higher brain centers, and feedback from peripheral receptors. It is through this complex interaction and similar processes that occur with other motor programs that the neuromuscular system acts to maintain knee stability during dynamic situations. Attempts to extrapolate data from static or simplified conditions to dynamic athletic situations, without taking into account the ongoing neural control of locomotion, warrant caution because this practice may result in an incomplete or inaccurate description of dynamic neuromuscular function.

Summary

The neuromuscular system uses a complex motor control system that consists of prestructured motor programs and a distributed network of reflex pathways mediated throughout the CNS to produce movement that is defined by coordinated muscle activity (neuromuscular control). This neuromuscular control system is believed to use both feedback and feed-forward control mechanisms to accommodate for unexpected circumstances, such as a perturbation that challenges dynamic knee joint stability. Evidence suggests that the nervous system simultaneously processes sensory feedback from ensembles of joint, cutaneous, and muscle receptors rather than dealing with signals separately. This method of information processing provides the nervous system with a more complete instantaneous representation of the conditions at the periphery, maintains a level of sensory redundancy in the system, and allows the processing to occur more rapidly. The redundancy within the system is important because it may allow the neuromuscular system to maintain near normal function in the presence of injuries or other events that may produce decrements in feedback or an increase in errors. Descending control signals from the brain (eg, motor programs and responses to visual and vestibular feedback), ensemble feedback from muscle, joint and cutaneous receptors, and the ongoing neural control process for locomotion are involved in a complex interaction through which the neuromuscular system produces coordinated movement. Muscle stiffness modulation appears to be an important mechanism in the maintenance of dynamic knee stability. Muscle stiffness is modulated as a result of force and length feedback provided by the GTOs and muscle spindles, respectively. When GTO activity is greater than muscle spindle activity, motor output and stiffness are reduced; the opposite is true when muscle spindle activity is greater than GTO activity. The total magnitude of stiffness is determined by the reflexive stiffness provided by the ratio of GTO to muscle spindle activity and the nonreflex stiffness provided by the length-tension properties of the involved musculature. Most challenges to knee joint stability alter the force and length properties of several muscles in the lower limb. As a result, the balance between force and length feedback in the involved muscles is disrupted and the activity in the muscles changes. While there is significant evidence to support the presence of a motor control strategy like the one we have described, many of the details of this neuromuscular control system have yet to be precisely defined or are a matter of controversy among neuroscientists.

IMPLICATIONS FOR CLINICIANS AND SCIENTISTS

In this section, we will apply our understanding of the neuromuscular control system to the following issues that may be important to clinicians and scientists who are interested in dynamic knee stability: (1) whether or not knee ligament injuries can be prevented, (2) the assessment of neuromuscular function, and (3) training programs related to dynamic knee stability. Because other papers in this special issue discuss the theory and research related to some of these issues in detail, our discussion of these issues will be relatively brief and focus instead on how they relate to our understanding of the neuromuscular control system.

Can Knee Ligament Injuries Be Prevented?

The answer to this question is quite complex. Any load that is greater in magnitude than the composite stability provided by the knee's structural strength, stabilizing muscle forces, and joint compressive forces from weight-bearing will result in an injury. For this reason, it is unlikely that injury can be prevented when, for example, a 300-pound lineman diving for a tackle makes full contact with the fixed knee of another player. The complexity in answering the prevention question arises in more subtle scenarios when the load conditions are such that the muscles are strong enough to protect the knee as long as they are activated appropriately. In these scenarios, the answer depends on: (1) the magnitudes and directions of the destabilizing forces, (2) the rate at which the loads are applied to the ligaments, (3) the amount of muscle activity present as the event unfolds, (4) the knee joint position and activity being performed, and (5) whether the ensuing injury mechanism is anticipated.

The magnitudes and directions of destabilizing force dictate the magnitude of the required compensatory response and the muscles that can contribute to a stabilizing force. The joint angle at which the knee is being loaded is important because the length-tension properties (nonreflex stiffness) of the muscles involved change with joint position. The amount of muscle activity present in the involved muscles as the event unfolds will determine the amount of active force present. The combination of the force associated with muscle activity and the passive force from intrinsic muscle stiffness results in the total neuromuscular stabilizing forces present as the event unfolds. The difference between these forces and the destabilizing loads being applied to the knee determines the magnitude of the response required to maintain knee stability. The activity being performed will determine the rate at which the ligaments are loaded, unless the loads being applied are from an external source (ie, contact). The magnitude of the required stabilizing forces and the rate of ligament loading are of key importance because they determine whether it is possible for the stabilizing forces to be generated prior to the ligament being damaged. If the stabilizing forces can be generated quickly enough, the injury will be prevented. If not, however, the ligament is ruptured or sprained because loading occurs too quickly to allow sufficient compensations to be produced.

Most injuries in sports occur very rapidly. Pope et

al¹⁴³ simulated an MCL injury during a ski accident and estimated that ligament loading begins approximately 39 ms after the event is initiated. Pain was estimated to be first perceived at 51.9 ms and ligament rupture was estimated to occur at 73 ms. The average reflex response measured in this study was not observed until 128 ms after the stimulus was given, and a forceful sustained contraction was not recorded until 215 ms had passed. The latencies observed in this experiment led the authors to suggest that muscular response is far too slow to prevent such an injury. There are limitations in the design of this study, however, that compromise our ability to extrapolate them to a realistic injury scenario. The latencies for muscle activation were measured from the point in time when a mechanical stimulus was provided on the medial aspect of the foot of a subject lying on the testing device.143 As a result, there was little to no muscle activity present at time zero-a scenario that is unlikely to be found during dynamic activity. The stimulus method suggests that the "reflexive" motor response observed at 128 ms was most likely initiated in response to feedback from cutaneous receptors of the foot, which is quite different than the response that would be expected in the proposed injury scenario.148 The expected initial reflex response would occur in response to the ensembles of feedback propagated from the muscle receptors in the thigh and the joint receptors in the knee, and this would most likely occur in less than 128 ms because it would be mediated at the segmental level of the spinal cord rather than in the brain (subjects in this study voluntarily contracted their muscles when they felt a tap on their foot). The likelihood of pre-existing muscle activity and ongoing muscle stiffness modulation in a real injury scenario suggests that the time required to achieve a significant motor response (215 ms) would also be reduced. Despite these weaknesses, we agree with the authors' conclusion that in most athletic injury scenarios, the rate of ligament loading and the forces involved are likely to be too great to allow prevention of these injuries via a feedback mechanism.143

But, if the athlete could anticipate that an injury was about to occur (by conscious or subconscious means), the coordinated muscular response could begin prior to the onset of the injury mechanism. As a result, preparatory actions could be taken to reduce the impact of the impending injury mechanism, and substantially greater forces could be produced in an effort to prevent the injury. The feedforward mechanisms in the neuromuscular control system may enable such a prevention strategy to be employed. Yet, unless the warning occurred well in advance of the injury event or the required forces to stabilize the knee were minimal, the latencies associated with the motor response would still make sports injury prevention unlikely. In theory, the odds for injury prevention could be improved by the presence of preprogrammed movement strategies that could be triggered when receptors detected an impending injury. Although the idea of preprogrammed reactions has been discussed in the joint stability literature, research has yet to substantiate that such mechanisms contribute to dynamic knee stability.^{111,113,166}

Our current understanding of the neuromuscular control system and injury mechanisms leads us to believe that unless the injury mechanism occurs at a relatively slow rate or substantial stiffness is present in the system when the event ensues, injuries are unlikely to be prevented with reflexive control strategies. In our opinion, the best prospect for injury prevention is to reduce the likelihood that individuals will have injury-producing loads applied to their knee ligaments in the first place. Several training programs have been described that are specifically directed at training athletes to reduce knee ligament loading by having better control of their center of mass by producing more coordinated and consistent movement patterns, and by appropriately positioning their knees during athletic participation.^{27,39,68,83,84} By developing motor programs that are characterized by coordinated muscle activity and training athletes to perform skills in a more biomechanically safe fashion, it is likely that we can reduce the incidence of serious knee ligament injuries to some degree. Continued research in this area is of great importance.

Assessment of Neuromuscular Function

Valid and reliable methods for assessing neuromuscular function enable us to describe: (1) the effects of an injury on neuromuscular function, (2) a patient's level of neuromuscular function, (3) the progress he or she makes with treatment, (4) the contributions the neuromuscular system makes to dynamic knee stability, and (5) the effectiveness of training programs related to neuromuscular function. The selection of an appropriate assessment technique depends on the purpose for assessing neuromuscular function and what aspects of neuromuscular function are being evaluated. Some methods of testing can be effectively used in the clinical setting, while others require equipment and lengthy study sessions that make them inappropriate for most clinical settings. The assessment techniques that may be suited for the clinical environment include threshold to detection of passive motion (TTDPM) testing, joint position sense (JPS) testing, stabilometry, functional tests (such as hopping or perturbations in stance), and force platform tests. Those which may be better suited for the laboratory include kinematic and kinetic analysis with motion analysis, force plates, and electromyography (EMG); assessment of responses to perturbation of support surfaces; and the assessment of motor responses to loads or stimuli applied to the

limb or soft tissues of the knee. Some clinical tests focus primarily on the neurosensory component (eg, TTDPM and JPS tests), while others measure general neuromuscular function (eg, stabilometry and functional testing). In addition, some tests evaluate an individual's active neuromuscular control (eg, stabilometry and hop tests), while others assess an individual's reactive control (eg, perturbations or loads applied to the knee). Because of the variety of testing methods and strategies, it is important that clinicians and scientists carefully consider the question they are trying to answer when selecting neuromuscular assessment methods.

It is not uncommon for clinical scientists to attribute the results of general neuromuscular assessments performed on individuals with ACL injuries to abnormal joint receptor function.7,15 While it may be possible to make this claim with complex assessment techniques that involve directly stimulating or loading the receptors and associated tissues, more general assessment techniques, such as JPS and TTDPM testing, are not specific enough to allow such inferences. As previously stated, the neurophysiologic evidence suggests that although signals from the joint receptors are undoubtedly included in the information that is fed back to the CNS, these receptors may not be the primary contributors to joint position sense or kinesthesia.^{22,23,53,54,70,72,151,175} The fact that few mechanoreceptors have been observed in the ACL adds further support to this statement.^{70,72,152,181} A more accurate explanation may be that the deficits observed result from the impact of ACL injury, not only on joint receptors, but also on muscle function (including muscle receptor activity and other factors that could be involved).22,23,124,125 Despite their limited specificity, clinically feasible neuromuscular tests may be useful benchmarks of neuromuscular function and be helpful in the treatment planning process.

When performing assessments of neuromuscular function or interpreting the results of studies that use these tests, there are several factors that should be considered. One of these considerations is the precision of the test equipment and the test methods. Not only does the equipment used need to be accurate and precise enough to measure the expected differences, but the precision of the measurement process also needs to be evaluated. For example, in TTDPM testing, the knee joint is passively rotated at a slow rate (~ $0.5^{\circ}/s$).^{5,15,157} When the patients first sense the movement, they respond by pushing a button (Figure 5). In this situation, the accuracy and precision of the device rotating the joint and the time involved with pushing the button will contribute to the measured response, along with the sensory feedback from the peripheral receptors. Because reaction times associated with button pushing are somewhat variable, several repetitions and averaging



FIGURE 5. Example of a device used to test the threshold to detection of passive motion. From Borsa PA, Lephart SM, Irrgang JJ, Safran MR, Fu FH. The effects of joint position and direction of joint motion on proprioceptive sensibility in anterior cruciate ligament-deficient athletes. *Am J Sports Med.* 1997;25:337. Reprinted with permission from the American Journal of Sports Medicine.

of response times for each individual are necessary. If you are attempting to measure the neurosensory function of the knee, then visual, auditory, and other sensory feedback should be addressed. In addition, the translation of degrees of rotation to milliseconds of response time (in our opinion, a more meaningful measurement) may not be as straightforward as it would seem and can be limited by the precision of the equipment (these devices commonly measure tenths of degrees and have some inherent variability). Because the differences measured in TTDPM tests are generally small (≤ 0.5 degrees),^{11,15,119,159} these factors may be problematic when clinicians attempt to attribute test results to altered sensory function. This is just one example of the difficulty associated with making accurate neuromuscular assessments and drawing conclusions from test results.

Subject age, sex, limb-dominance, and past athletic experience may affect some neuromuscular factors and, therefore, also may limit the ability to compare results among subjects.^{78,112,140,149} Assessment methods can also be specific to the joint position or the format in which testing is performed.^{15,47,48,148} Therefore, tests that are performed in stance, such as stabilometry and perturbation of support surfaces, may only be applicable to stance. The degree to which the results of tests performed in stance can be generalized to neuromuscular function in dynamic activities is unknown; therefore, any extrapolation of the results of such tests to dynamic sports should be recognized as being speculative. Most important is the

consideration of whether or not the results are clinically meaningful. Because research related to dynamic knee stability is still in its infancy, it is unclear which neuromuscular assessment techniques and test values are clinically meaningful. The lack of consistency in the methods described by authors who have published on this topic makes determining what is clinically meaningful even more difficult. For this reason, research is needed to identify the factors that are predictive of neuromuscular function in activity and how these factors can best be assessed. Additional research is required that either supports the clinical meaningfulness of the tests currently available or refutes them. Is a difference in the TTDPM of 0.2 degrees (a typical result) meaningful considering the rapid rates of joint motion typically present in sports? Can the results of studies that evaluate reflex latencies associated with loads applied to the knee in stance be generalized to dynamic scenarios? The clinical meaningfulness of several neuromuscular assessment methods will remain in question until researchers provide answers to questions like these.

Effect of Injury on Neuromuscular Function

Many studies have evaluated the effect of injury on the neuromuscular system.^{5,6,15,49,118} The effect of ACL injury has been the primary topic of papers related to this issue in the knee joint.^{7,11,15,28,43,48} The studies on this topic can be divided into 2 general categories: (1) studies evaluating joint position sense and kinesthesia and (2) studies evaluating the muscle firing patterns of individuals with ACL deficiency.

Barrack et al5 produced one of the first studies that evaluated the effect of ACL injury on neuromuscular function in 1989. These authors reported that the TTDPM measurements from the ACL-deficient limbs of their subjects were 25% greater (0.96 degrees) than the measurements from their uninjured limbs. They also reported that TTDPM results were not correlated with strength or thigh girth, but were weakly correlated (r = 0.46) with KT1000 measurements of ligamentous laxity. Other reports by Barrack and Skinner^{6,157,158} demonstrated that TTDPM and JPS deficits were observed in the involved limbs of patients with other pathological knee conditions. Several other groups have substantiated Barrack and Skinner's work by demonstrating either JPS or TTDPM deficits in individuals with ACL-deficient limbs;^{15,31} however, Good et al⁶³ failed to demonstrate significant differences in the JPS of the ACL-deficient and uninjured limbs of their subjects. It should be noted that in several of the studies, the standard deviations of the TTDPM and JPS tests were greater than the observed differences between groups or were 50% of the total measured value (eg, 1.33 \pm 0.76 degrees).^{5,63,159} Consequently, the results of some

of these studies may need to be interpreted with caution.

The second category of studies has described abnormal patterns of muscle activity in individuals with ligament injuries. Some researchers have demonstrated these results when loads were applied to the knee,10,138,176 while others have demonstrated these alterations in functional tasks such as walking, jumping, or a series of functional activities.33,56,102,109,116,127,167 Common findings include alterations of timing and magnitude of activity of the muscle that affect knee motion. The results of these studies provide evidence that knee ligament injuries have an effect on neuromuscular function. The altered muscle firing patterns are believed to be a compensatory strategy that accounts for decrements in knee stability. While deficiencies in joint receptor function may exist after injury, it is unlikely that the observed neuromuscular impairments are primarily due to the abnormal function of receptors in the injured ligaments because of the number and location of these receptors. Based on the physiologic evidence discussed above, altered firing from receptors present in other joint tissues would only be expected to be significant enough to produce notable alterations in muscle activity in situations when abnormal joint translations or rotations might occur (eg, pivoting, cutting, or stumbling). Instead, it is more consistent with current neurophysiologic theory to suggest that it is the combined input of joint, cutaneous, and predominantly muscle receptors that causes the observed alterations in motor activity. According to this thinking, alterations in muscle activity (stiffness) occur in response to instability patterns that disrupt the homeostasis between force and length feedback in the muscles of the limb.

It should be acknowledged that deficits observed with TTDPM tests, JPS tests, or more complex analyses with EMG (eg, reflex latencies and dynamic muscle patterns) and biomechanical testing may be affected by factors such as pain, effusion, muscle atrophy, reduced physical activity levels, or reduced patient confidence in the limb. It is important that researchers control and discuss these factors in their studies and manuscripts. Furthermore, clinicians should be conscious of these factors as they critically read articles related to neuromuscular testing or employ these techniques in the clinical setting.

Neuromuscular Training Programs

Like the injury research, most of the studies published related to the effect of neuromuscular training have had individuals with ACL injuries as their subjects.^{7,9,28,43,179,180} Several methods of neuromuscular training have been discussed in the literature, including training on wobble boards, stabilometry, functional training (jumping and landing), agility training, and perturbation training.^{9,27,43,68,84,161,174,179} The basic concept behind these training methods is that repetitively challenging an individual's ability to maintain static or dynamic control of his or her knee joint results in improved neuromuscular control and, subsequently, improved joint stability.

A few studies have evaluated the impact of neuromuscular training programs on ACL injury incidence.^{27,39,84} Caraffa et al²⁷ studied the effects of adding a wobble board training program to the traditional training program of competitive soccer players. In their study, Caraffa et al instructed one group of 300 soccer players to perform their team's traditional training program, while another 300 players were instructed to perform their team's traditional training program plus a progressive wobble board training program (5 phases) for 20 minutes per day, 3 times per week during the soccer season. Over a period of 3 soccer seasons, the athletes in the group that performed wobble board training had an incidence of ACL injury that was one-seventh of that observed in the group that performed traditional training alone. More recently, Hewett et al⁸⁴ reported that female athletes who performed a neuromuscular training program related to landing techniques had significantly fewer serious knee injuries than a group of female athletes that did not perform the training and a group of male athletes. The untrained group, however, had a significantly higher number of individuals playing basketball and soccer than the trained group, which were primarily volleyball players.84 This fact, and the small number of injuries that occurred in the study (a total of 14 in the 3 groups, including contact injuries), may warrant caution in drawing conclusions until further data is available. Finally, Ettlinger and authors³⁹ have instituted a highly successful injury awareness program that instructs skiers on how to avoid knee ligament injuries. These researchers demonstrated that during the 1993-1994 ski scason, "awareness-trained" skiers had a 62% lower incidence of serious knee injuries than an untrained control group of skiers.³⁹ The results of these studies are very promising, but additional research is needed. Assuming that the training programs are effective in reducing the incidence of ACL injuries, the means by which the training programs affect ACL incidence (eg, coordination activity, endurance, and increased awareness of knee position) will need to be defined.

Two prospective studies have assessed the effect of balance board training programs on the prevention of a more broad range of injuries in female athletes.^{161,174} Wedderkopp et al¹⁷⁴ evaluated the impact of ankle disk training in 237 female European team handball players over a 10-month season. The athletes in this study were randomized into an intervention group and a control group. The intervention group performed 10–15 minutes of ankle disk training at each practice over the season, while those in



FIGURE 6. Algorithm used to identify individuals with anterior cruciate ligament (ACL) deficiency who may be nonoperative rehabilitation candidates. UI indicates uninvolved; KOS, knee outcome survey; ADLS, activities of daily living scale.

*Compared to maximum score possible.

Adapted from Fitzgerald GK, Axe MJ, Snyder-Mackler L. Proposed practice guidelines for nonoperative anterior cruciate ligament rehabilitation of physically active individuals. *J Orthop Sports Phys Ther.* 2000;30:196. Reprinted with permission from the Journal of Orthopaedic and Sports Physical Therapy.

the control group performed only their normal practice routine. Results indicated that athletes in the control group were 5.9 times as likely to be injured than those who performed the ankle disk training.174 The majority of the injuries that occurred in this study were ankle injuries. Soderman et al¹⁶¹ evaluated the impact of balance board training on lower extremity injury prevention in 221 female soccer players over a 7-month outdoor season. These athletes were also randomly assigned to an intervention and a control group. The intervention group performed a progression of 5 balance board training exercises daily at home for the first month and then at a frequency of 3 times per week; the control group performed only standard practice activities. The results of this study indicated that there was no significant difference in the number of injuries between groups. More surprising is the fact that 4 of the 5 ACL injuries that occurred in this study were in the intervention group.¹⁶¹ Therefore, it is not clear if the addition of a balance board training program to standard athletic practice is an effective means of preventing musculoskeletal injuries.

Several studies have evaluated the effects of neuromuscular training on the outcomes of patients with ACL-deficient or ACL-reconstructed knees. Zatterstrom and coauthors¹⁷⁹ demonstrated that patients with chronic ACL deficiency who performed 12 months of stabilometry training in single leg stance along with conventional rehabilitation had significantly better postural stability in single leg stance than patients who performed the conventional therapy alone. Beard et al9 reported that in a randomized clinical trial, patients with ACL deficiency who performed a progressive neuromuscular training program had significantly shorter reflex hamstring contraction latencies than a matched group of patients with ACL deficiency that performed strengthening exercises alone. Fitzgerald et al43 also conducted a clinical trial in which subjects with ACL deficiency were randomized into 1 of 2 treatment groups: (1) traditional rehabilitation, and (2) traditional rehabilitation and perturbation training. The traditional training program performed by both groups included strengthening exercises in both open and closed kinetic chain formats and agility drills. The perturbation training program performed by the second treatment group consisted of a series of progressively more challenging exercises that were performed on rocker boards and roller boards. The results of this study indicated that the group receiving the perturbation training had significantly greater success in returning to high-level activities without experiencing symptoms of giving way.

The evidence from the described studies suggests that neuromuscular training can have a significant impact of the functional level of individuals who have had ligament injuries. The exact reasons for



FIGURE 7. An example of sport-specific perturbation training.

these improvements have yet to be defined. Our educated guess is that the improvements that occur from this training are due to a multifactorial effect that may include: alterations in muscle firing patterns; increased awareness of the body's position in space from "tuning" the neuromuscular system and increased attention; increased confidence; more biomechanically sound skill performance; and alterations in strength and endurance.

At the University of Delaware, active patients with ACL deficiency who pass a screening examination that identifies them as rehabilitation candidates (Figure 6) receive perturbation training based on the protocol described by Fitzgerald et al.⁴⁴ The training program consists of 10 treatment sessions that are generally administered 2-3 times per week. The frequency of treatment and the program progression are determined by the patient's proficiency in performing the techniques, the response of the patient's knee joint to the training (decreased frequency and slower progression if knee effusion arises or increases), and the time constraints unique to the situation (including the amount of time left in the competitive season). Five variables of the applied perturbations are altered in order to progress the perturbation

Treatment goals: Expose the patient to perturbations in all directions

Elicit an appropriate muscular response to applied perturbations (no rigid co-contraction) Minimize verbal cues

Overview: Perturbations are initially applied slowly and predictably. Verbal cues will be necessary for the onset and direction of the perturbation. Progress to moving in off-plane directions. Then, randomize the direction of the perturbations and decrease verbal cues. As the patient's ability to elicit an appropriate response improves, increase the challenge of the perturbations by performing them with more force, larger magnitude, and increased speed.

Technique	Direction of Board Movement	
Rocker Board	Anterior/Posterior, Medial/Lateral	
Roller Board/Platform	Initial: Anterior/Posterior, Medial/Lateral Progression: Diagonal, Rotation	
Roller Board	Initial: Anterior/Posterior, Medial/Lateral Progression: Diagonal, Rotation	

Middle Phase (Treatments 5-7):

Criteria to enter phase: The patient must be able to elicit an appropriate muscular response to perturbations and demonstrate few or no falls during the rocker board and roller board techniques.

Treatment goals: Add light sport-specific activity during perturbation techniques Improve accuracy in matching the muscular response to the force, direction, and speed of the applied perturbation

Overview: Initially apply the perturbations as in the Early Phase (slowly, predictably, planes as noted) until the patient elicits an appropriate muscular response while performing the sport-specific activity, then progress according to Early Phase guidelines.

Late Phase (Treatments 8-10):

Criteria to enter the phase: While performing light sport-specific activity, the patient must be able to elicit an appropriate muscular response to perturbations and demonstrate few or no falls during the rocker board and roller board techniques.

Goals: Increase the difficulty of the perturbations by using sport-specific stances or performing more difficult sport-specific activity (eg, on-command drills)

Elicit accurate, selective muscular responses to perturbations in any direction and of any magnitude and speed.

Technique	Direction of Board Movement	
Rocker Board	Diagonal with respect to the position of the foot	
Roller Board/Platform	All directions	
	Stance: Vary stance (eg, staggered stance)	
Roller Board	All directions	

FIGURE 8. University of Delaware perturbation training treatment guidelines.

training: predictability, direction, speed, amplitude, and intensity (force). These perturbation training techniques are discussed in detail in Fitzgerald et al.⁴⁴

Agility training is performed in conjunction with perturbation training to allow for carry-over of improvements in postural and dynamic knee stability into more sport-specific movement. Examples of the agility drills that our patients perform include side shuffles, cariocas, shuttle running, 45° and 90° cutting, and changing direction on command. The agility training begins with drills performed in straight paths and progresses to activities involving cutting and pivoting. Similarly, the intensity of the drills begins at 50% effort and progresses to 100% effort. Patients at our institution are required to wear a functional brace when performing these tasks.

When the patient demonstrates little difficulty with each of the perturbation and agility drills, an attempt is made to make the activities more sport-specific. For instance, when standing on a rocker board, the patient may perform chest passes with a basketball (Figure 7), passing with a lacrosse stick, or kick a soccer ball with the contralateral leg while perturbations are being applied. In the platform and roller board activities, the patient may simultaneously receive upper body perturbations to simulate checking or blocking type contact that commonly occurs in sports like football, basketball, and soccer. We also attempt to make the agility drills more sport-specific by adding skills like dribbling a basketball while performing a 45° cutting drill or catching fly balls while performing forward and backward stop-start drills. In our experience, athletes with ACL deficiency are often able to progress to sport-specific activities in as few as 5 perturbation training sessions.

Patients are usually able to begin a partial returnto-sport by the eighth perturbation training session. A partial return-to-sport is defined as the ability to participate in practice-type drills, but not competition. Patients are generally discharged to full competition by the 10th treatment as long as they successfully pass a post-treatment ACL screening by scoring \geq 90% on the screening criteria (timed hop test, KOS-ADL scale, and global rating) and demonstrate \geq 90% contralateral quadriceps maximum voluntary isometric contraction strength.

More detailed guidelines for progressing each perturbation training technique can be found in Figure 8. In this Figure, the 10 perturbation training sessions are divided into early, middle, and late phases, with goals listed for each phase. This framework gives clinicians assistance with making treatment decisions concerning the introduction of perturbations in a new direction, sport-specific activity, and sportspecific stance. Clinicians should be aware that patients rarely progress to the middle and late phases at the same time for each perturbation technique. Patients should only be allowed to progress to a new phase for a particular technique when they meet the criteria for entering the phase.

CONCLUSION

Dynamic knee stability is the result of several factors, including articular geometry, soft tissue restraints, and the loads applied to the joint from weight-bearing and muscle action. In this paper we have focused on the contribution made by the neuromuscular system because this is the only component of dynamic knee stability that can be addressed with therapeutic interventions. The neuromuscular system utilizes a complex motor control system that consists of prestructured motor programs and a distributed network of reflex pathways mediated throughout the CNS to produce movement that is defined by coordinated muscle activity (neuromuscular control). The neuromuscular control system is believed to utilize both feedback and feed-forward control mechanisms. Descending control signals from

the brain (eg, motor programs and responses to visual and vestibular feedback), ensemble feedback from muscle, joint and cutaneous receptors, and the ongoing neural control process for locomotion are involved in a complex interaction through which the neuromuscular system produces coordinated movement. The concept of muscle stiffness modulation is put forth as a key mechanism by which dynamic knee stability may be maintained. Muscle stiffness is the result of 3 factors: (1) the intrinsic properties of the muscle (nonreflex stiffness), (2) force feedback provided by ensembles of GTOs, and (3) length feedback from ensembles of muscle spindles. Most challenges to knee joint stability alter the force and length feedback of several muscles in the lower limb. As a result, muscle activity patterns are altered in an attempt to maintain stiffness and, indirectly, joint stability. Our understanding of the neuromuscular control system, however, remains somewhat theoretical.

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