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Reestablishing Proprioception and Neuromuscular Control in the ACL-Injured Athlete

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Anterior cruciate ligament (ACL) injury disrupts static and dynamic knee restraints, compromising functional stability. Deafferentation of ACL mechanoreceptors alters the spinal reflex pathways to motor nerves and muscle spindles in addition to the cortical pathways for conscious and unconscious appreciation of proprioception and kinesthesia. These pathways are required by the feed-forward and feedback neuromuscular control systems to dynamically stabilize joints. Feed-forward motor control is responsible for preparatory muscle activity, while feedback motor control regulates reactive muscle activity. The level of muscle activation, preparatory or reactive, influences muscular stiffness, thereby providing dynamic restraint for the ACL-deficient athlete. Rehabilitation protocols should incorporate activities that enhance muscle stiffness while encouraging adaptations to peripheral afferents, spinal reflexes, and cortical motor patterns. Four elements crucial for reestablishing neuromuscular control and functional stability are proprioceptive and kinesthetic awareness, dynamic stability, preparatory and reactive muscle characteristics, and conscious and unconscious functional motor patterns.

The role of capsuloligamentous sensory receptors in the dynamic restraint system of the knee is well established (17, 27, 44, 45, 77, 87). Sensory feedback from anterior cruciate ligament (ACL) receptors projects directly to cortical and reflex pathways, mediating reactive muscle activity for dynamic restraint (27, 53). This sensory feedback mechanism is defined as proprioception and kinesthesia (74). Recently, the interaction between ACL receptors and muscle spindles has received even greater attention for its contribution to dynamic restraint (18, 27, 44). Muscle spindles, with both sensory and motor nerve fibers, have been implicated in the regulation of muscle tone (stiffness) (4, 43, 44, 75). Increased muscle stiffness prior to joint loading is another mechanism utilized for dynamic restraint of the knee (24). Preactivation of muscles may have a greater impact on knee stability than previously realized.

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Disruption of the ACL results in not only a mechanical disturbance but also a loss of joint sensation, due to deafferentation of peripheral sensory receptors (mechanoreceptors) (45, 81, 87). This partial deafferentation alters reflex pathways to skeletal muscle, muscle spindles, and higher motor centers. There is substantial evidence that aberrations in muscle activity subsequent to ACL injury result from disrupted reflex pathways (11, 15, 45, 61, 92, 93). Therefore, ACL injury not only reduces the joint's mechanical stability but also often diminishes the capability of the dynamic restraint system, rendering the knee functionally unstable.

Approximately one-third of ACL-deficient individuals have sophisticated, possibly innate compensatory mechanisms that may prevent serious pathological sequelae (76, 88, 96). The functionally stable ACL-deficient individual must possess sensory characteristics and movement strategies that maximize the dynamic support system, counteracting mechanical insufficiencies. This process preserves joint integrity and prevents functional instability.

Many researchers and clinicians seek to identify the neuromuscular control characteristics that compensate for mechanical insufficiencies in the ACL-deficient knee so that similar adaptations can be encouraged to restore functional stability. Four elements crucial for reestablishing neuromuscular control and functional stability are joint proprioception and kinesthesia, dynamic stability, preparatory and reactive muscle characteristics, and conscious and unconscious functional motor patterns (65).

The following text will discuss the sensory receptors and neural pathways that contribute to normal joint stabilization. With respect to ACL-injured athletes, deficits in joint sensation and muscle activity will be presented as well as the mechanisms whereby diminished neuromuscular control may compromise functional stability. The theoretical framework for reestablishing neuromuscular control will be presented, followed by specific activities designed to encourage the peripheral, spinal, and cortical adaptations crucial for increasing functional stability.

Terminology

Proprioception refers to conscious and unconscious appreciation of joint position, while *kinesthesia* is the sensation of joint motion or acceleration (74). These signals are transmitted to the spinal cord via afferent (sensory) pathways. The efferent (motor) response to sensory information is termed *neuromuscular control* (48). Two motor control mechanisms are involved with interpreting afferent information and coordinating efferent responses (23, 50). *Feed-forward* neuromuscular control involves planning movements based on sensory information from past experiences (23, 59). The *feedback* process continuously regulates motor control through reflex pathways. Feed-forward mechanisms are responsible for preparatory muscle activity, while feedback processes are associated with reactive muscle activity. The level of muscle activation, whether it is preparatory or reactive, largely determines muscular stiffness properties (75, 81). From a mechanical perspective, muscle stiffness is the ratio of the change of force to the change in length (4, 22, 24). In essence, stiff muscles resist stretching episodes more effectively, have greater tone, and provide more effective dynamic restraint to joint displacement (4, 69).

Joint Stabilization

Static and Dynamic Systems

In the healthy knee, static support is provided by structures such as the joint capsule, cruciate and collateral ligaments, and meniscal and bony geometry. The primary roles of these structure, stabilizing and guiding skeletal segments, are mechanical (96). This requires all of the elements to possess complex biomechanical characteristics contingent upon their responsibilities as primary and secondary restraints (88). Capsuloligamentous tissue also has a sensory role; it detects joint motion and position, thereby mediating motor control for dynamic stability (27, 51, 83).

Dynamic support for the knee is achieved through preparatory and reflexive neuromuscular control (22, 23, 31, 35, 40). In the knee, the primary dynamic stabilizers are the quadriceps, hamstrings, and gastrocnemius. Because of the orientation and contractile properties (concentric, eccentric, and isometric) of skeletal muscle, a diverse array of movements can be achieved while excessive joint motion is restricted. Muscle contractions can increase knee joint stiffness 10-fold; therefore, the level of muscle stiffness greatly enhances dynamic joint stability (56, 67, 69).

Clinical studies addressing the role of muscle stiffness in the dynamic restraint system have been limited. McNair et al. (69) demonstrated that increased hamstring muscle activation also significantly increased hamstring stiffness, and that there is a moderate correlation between the degree of muscle stiffness in ACL-deficient athletes and their functional ability. Therefore, McNair et al. concluded that ACL-deficient athletes with greater hamstring stiffness were more functional. Efficient regulation of muscle stiffness may embody all of the components in the dynamic restraint system and thus may be the most vital element related to neuromuscular control.

Articular and Tenomuscular Mechanoreceptors

Articular

The dynamic restraint system is mediated by specialized cells called mechanoreceptors (34). A mechanoreceptor functions by transducing mechanical deformation of tissue into frequency-modulated neural signals (34). As a stimulus (deformation) increases, so does the frequency of discharge and number of activated mechanoreceptors (34, 36). These signals provide sensory information concerning internal and external joint forces. Three morphological types of mechanoreceptors have been identified in the knee. Pacinian corpuscles, found in the joint capsule, are classified as quick adapting (QA) because they cease discharging shortly after the onset of a stimulus (27, 34, 82). Meissner corpuscles and free nerve endings are located in ligaments, menisci, and capsular tissue (2, 27, 34, 51, 82). Both adapt slowly to stimuli and are therefore classified as slow adapting (SA) mechanoreceptors (17, 53, 82). In healthy knees, QA mechanoreceptors are believed to provide conscious and unconscious kinesthetic sensations in response to flexion, extension, rotation, and tibial translation (28, 34, 85). Conversely, SA mechanoreceptors provide continuous feedback and thus proprioceptive information relative to joint position (17).

Debate exists over the relative contribution of ACL afferents in the dynamic restraint system. Solomonow et al. (89) found it necessary to use high load levels (near failure) in order to evoke reflex hamstring activity in cats. Mechanoreceptors in the ACL, it would appear, contribute to the dynamic restraint system when the knee is "overloaded" (89). Athletes who are ACL deficient but functionally stable must compensate for the absence of ACL sensory information with secondary structures, thereby maintaining dynamic joint stability (10, 61, 86).

Tenomuscular Mechanoreceptors

In addition to capsuloligamentous receptors, two types of mechanoreceptors have been identified in muscle and tendinous structures. The first, muscle spindles, are imbedded within skeletal muscle and detect muscle length and rate of length changes (5, 18, 36). Muscle spindles are innervated by small motor fibers called gamma efferents, as opposed to the large motor fibers that innervate the contractile elements of skeletal muscle (5, 36, 60). This independent arrangement of motor fibers permits the muscle spindle to continuously transmit afferent signals concerning muscle length and rate of length changes (5, 36). Muscle spindle afferents project directly on skeletal motoneurons through very fast monosynaptic reflexes (94). When muscle spindles are stimulated, by length changes, they elicit a reflex contraction in the agonist muscle. Increased signals from the gamma motor fibers heighten the stretch sensitivity of muscle spindles (43, 44). This is the mechanism (stretch reflex) whereby muscle spindles have the capacity to mediate muscle activity (43, 71, 94).

Changes in muscle length also alter muscle tension. The second type of mechanoreceptor, the Golgi tendon organ (GTO), is responsible for monitoring muscle tension (36). Located near the musculotendinous junction, GTOs protect the tenomuscular unit by reflexively activating agonist and inhibiting antagonist muscles (41). Golgi tendon organs, therefore, have the opposite effect of muscle spindles. When stimulated by high muscle tension, GTOs cause reflex inhibition (relaxation) in the muscle being loaded (36, 41).

Neural Pathways of Peripheral Afferents

Determining the extent to which articular and tenomuscular sensory information is utilized requires analysis of the reflexive and cortical pathways employed by peripheral afferents. Sherrington (83) first outlined the conceptual role of peripheral receptors as a source of signals for joint sensations, mediating reflex systems of the locomotor apparatus. Encoded signals concerning joint motion and position are transmitted from peripheral receptors, via afferent pathways, to the central nervous system (CNS) (25, 27). Ascending pathways to the cerebral cortex provide conscious appreciation of proprioception and kinesthesia, whereas balance and postural control are processed at the brain stem (18, 28, 29, 36, 50). Balance is influenced by the same peripheral afferent mechanism that mediates joint proprioception and is partially dependent upon the individual's inherent ability to integrate joint position sense with neuromuscular control. Balance, therefore, is frequently used to measure functional joint stability, and balance deficits may result from aberrations in the afferent feedback loop of the lower extremity. There are also synapses at the spinal level linking afferent fibers with efferent motor nerves. These

pathways constitute the reflex loops between sensory information and motor responses.

This reflexive neuromotor link between ligaments and muscles was first demonstrated by Palmar (77). By loading the medial collateral ligament, Palmar elicited reflexive electromyographic (EMG) activity in the adductor muscles (77). In the case of anterior tibial translation, loading the ACL excites the hamstring and gastrocnemius muscles (12, 49, 89, 93). There also appears to be an inhibitory pathway that represses quadriceps activity (16, 45, 65). These reflex pathways contribute to dynamic stability by using the feedback process for reactive muscular activation (12, 89).

However, Johansson et al. (44) contended that articular afferent pathways do not exert as much influence directly on skeletal motoneurons as previously reported but rather have more frequent and potent effects on muscle spindles, which in turn regulate muscle activation through the stretch reflex. ACL afferents, therefore, have some influence on the large skeletal motor nerves to the hamstrings as well as the tenomuscular receptors, via small gamma motor nerves (43–45). The significance of the muscle spindle system is beginning to receive attention for its role in regulating sensory information and muscle stiffness (45, 56, 60, 69, 75).

This sophisticated articular–tenomuscular link has been described as the "final common input" (3, 45). The final common input suggests that muscle spindles integrate peripheral afferent information and transmit a final modified signal to the CNS (3, 45). This feedback loop is responsible for continuously modifying muscle activity during locomotion via the muscle spindle's stretch reflex arc (39, 75). Through coordination of reflexive and descending motor commands, muscle stiffness is modified and dynamic knee stability is maintained (45, 56).

Preparatory and Reactive Muscle Activity

The efferent response of muscles "transforming neural information into physical energy" is termed *neuromuscular control* (48). Neuromuscular control is influenced by proprioceptive, kinesthetic, visual, and vestibular information as well as cortical and spinal motor commands (50). Traditional beliefs on the processing of afferent signals into efferent responses for dynamic stabilization were based on reactive or feedback neuromuscular control pathways (60). More contemporary theories emphasize the significance of preactivated muscle tension in anticipation of movements and joint loads. The preactivation theory suggests that prior sensory feedback (experience) concerning the task is used to preprogram muscle activation patterns. This process whereby sensory feedback from the past is "fed forward" to preprogram muscle activity is described as feed-forward neuromuscular control (22, 24, 33, 60, 71, 90). Preactivated muscles can provide quick compensation for external loads and are critical for dynamic joint stability (22, 35).

Feed-forward motor control uses advance information about a task, usually from experience, to preprogram muscle activity (23, 59). This requires building an internal model depicting the expected conditions with all of the known task parameters (23, 47, 50). These centrally generated motor commands are responsible for preparatory muscle activity and high-velocity movements (50). The feed-forward mechanism does not depend on reflex pathways and once initiated executes very quick motor commands (50). Sensory information about the task is then used to evaluate the results and help program future muscle patterns. Unexpected events that occur during these movements may be identified by joint and muscle receptors but may be used only for future planning, not for reflex stabilization.

The feedback mechanism of motor control is characterized by numerous reflex pathways continuously adjusting ongoing muscle activity (13, 23, 60, 71). Information from joint and muscle receptors is used to reflexively coordinate muscle activity toward the completion of a task. This feedback process, however, results in long conduction delays and is best used to maintain posture and regulate slow movements (50). Therefore, the efficacy of reflex-mediated dynamic stabilization is related to the speed and magnitude of joint perturbations. The relative contribution that feedback-mediated muscle reflexes provide when in vivo loads are placed on joints is unclear.

Both feed-forward and feedback motor control can enhance dynamic stability if the sensory and motor pathways are frequently stimulated. Each time a particular signal passes through a sequence of synapses, the synapses become more capable of transmitting the same signal the next time (36, 38). If these pathways are "facilitated" enough, memory of that signal is created and can be recalled to program future movements (36). Frequent facilitation, therefore, enhances both the memory about tasks for preprogrammed motor control and reflex pathways for reactive neuromuscular control. This mechanism has significant implications for developing sport-specific movement repertoires as well as preparatory and reactive actions for dynamic restraint.

Clinical research assessing muscle activation patterns of the knee frequently employs landing and hopping models. These movements require both preparatory and reactive muscle activity along with related changes in muscle stiffness. Muscle activity prior to landing reveals that activation occurs in a pattern from distal to proximal, beginning approximately 132–200 ms before touchdown (90). This preparatory muscle activation prior to landing is considered to be preprogrammed, while activation after ground contact is considered reactive. The preparatory muscle activity serves several functions that contribute to the dynamic restraint system. An increase in muscle activation levels increases the stiffness properties of the entire tenomuscular unit (72). This increased muscle stiffness provides dynamic support for functional stability (56, 69). Increased stiffness also heightens muscle spindle and GTO sensitivity to stretch and tension while reducing electromechanical delay (19, 35, 45, 69, 72, 81).

Muscle stiffness can modulate quickly depending upon task requirements (24). For example, during hopping, energy must be stored and then released in a manner similar to a spring; therefore, preparatory muscle activity increases muscle stiffness. During landing or deceleration tasks, energy must be absorbed; hence, muscle stiffness remains low (24, 69). Assessing preparatory EMG activity and muscle stiffness provides valuable information about feed-forward neuromuscular control and its capacity for dynamic restraint.

Pathoetiology

The mechanisms predisposing athletes to ACL ruptures have been well documented. Unlike traumatic ACL injuries involving large external forces, nontraumatic ACL injuries usually involve a failure of some aspect of the dynamic restraint system which exposes static structures to threshold loads that jeopardize their integrity.

There are several mechanisms by which neuromuscular control system deficiency actually exposes the ACL to failure.

At the peripheral level, well-conditioned individuals appear to have enhanced joint motion and position sense (7, 8, 62, 85, 86). Deconditioned individuals may lack sufficient somatosensory awareness to coordinate muscle activity and dynamic restraint, thus predisposing them to joint pathology. Solomonow suggested that substantial increases in joint translation occur with muscles that are deconditioned or fatigued or have low tone, compared to muscles with high baseline tone (89). Moreover, Wojtys and Huston (93) correlated increased anterior tibial translation with hamstring reaction times after fatiguing exercise. Aberrations in joint motion and position sense will impact feed-forward and feedback neuromuscular control mechanisms and diminish the capability of the dynamic restraint system.

The speed and complexity of movements in athletic competition require rapid integration of sensory information by feed-forward and feedback neuromuscular control systems. Errors within the CNS in anticipating or reacting to joint loads can result in uncoordinated muscle activity, compromising dynamic stability. The brain, at any given moment, discards 99% of all sensory information as irrelevant (36). This may account for the inconsistencies that develop during feed-forward programming between anticipated and real-life conditions (36, 50). Such is the case when an experienced athlete initiates a cutting maneuver, something that the athlete performs routinely, and "catches" his or her foot. Greenwood and Hopkins (33) suggested that this "jarring" sensation is similar to descending the final stair in the dark when another step is expected. Because of safety concerns, only a limited number of researchers have attempted these types of perturbations. Thompson (90) induced sensory conflicts by having subjects randomize visual input and height while landing from a jump. It was found that preparatory muscle activity was attenuated until landing, suggesting that preactivation of the hamstrings may be nullified if sensory information is confusing or misinterpreted (90). Furthermore, the preprogramming of movements can occur several hundred milliseconds before the movements are initiated, while cognitive appreciation of an event may take up to 120–150 ms (21). Unexpected perturbations, such as high joint loads, can occur during this period. Without adequate preparatory activity, the static structures may be exposed to insult unless reactive muscle activity can be initiated to contribute to dynamic restraint. There is some concern whether the CNS can negotiate the speed and magnitude of muscle activity required to prevent tibial subluxation during ballistic movements, such as those in athletics (89).

While many peripheral, spinal, and cortical elements contribute to the neuromuscular control system, dynamic joint stabilization is contingent upon both cortically programmed preactivation and reflex-mediated muscle activation. Critical errors in timing, coordination, conditioning, and concentration may disrupt the dynamic restraint system.

Neuromuscular Characteristics of ACL-Deficient Athletes

Characteristic patterns of proprioceptive, kinesthetic, and neuromuscular deficits have been demonstrated in ACL-injured athletes (6, 12, 61, 64 84, 85, 93). Although identifying these abnormalities may be difficult in a clinical setting, a thorough appreciation of the pathoetiology of the conditions is necessary to guide

clinicians who are attempting to reestablish joint afferents and neuromuscular characteristics in the ACL-injured athlete.

Most researchers believe that ACL disruption results in some level of deafferentation to ligamentous and probably capsular mechanoreceptors (20, 26, 61, 85). In the acute phase of healing, joint inflammation and pain compound sensory deficits (9, 53). However, this cannot account for the chronic proprioceptive and kinesthetic deficits associated with ACL-deficient knees. Research has demonstrated that ACL-deficient athletes have diminished capability for detecting joint motion or position (7). These proprioceptive and kinesthetic characteristics, coupled with mechanical instability, lead to functional instability (Figure 1) (63, 64). Developing or reestablishing proprioception and kinesthesia in ACL-injured athletes will minimize the risk of reinjury. ACL reconstruction, coupled with traditional rehabilitation, appears to restore some kinesthetic awareness, although not equal to that of noninvolved limbs (10, 20, 63).

Assessment of the reactive characteristics of dynamic stabilization focuses on the feedback process of motor control. Beard et al. (11) reported reflex latencies in ACL-injured athletes by simulating episodes of anterior tibial translation, eliciting hamstring activation via feedback from the ACL, capsular, and probably tenomuscular receptors (94). This work revealed that reflex hamstring latency in the involved leg was nearly twice that of uninvolved and control limbs, and Beard et al. concluded that the increased reflex latency was due to proprioceptive deficits that may impair joint function (12). Wojtys and Huston (93), with a similar model, substantiated differences in the timing and sequence of muscle firing in the ACL-deficient limb. They found that increased reflex latency will diminish the reactive muscle characteristics and must be reestablished through joint perturbation techniques under controlled intensities.

Preprogrammed muscle stiffness, prior to the onset of anterior tibial translation, can enhance reflex muscle activity. Although if preparatory muscle stiffness

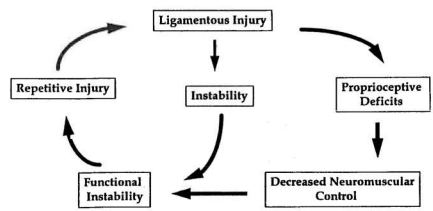

Figure 1 — Functional stability paradigm depicting the influence of mechanical instability and proprioceptive deficits on functional stability, which predisposes the knee to repetitive injury. Reprinted by permission from S.M. Lephart & T.J. Henry, 1996, "The Physiological Basis for Open and Closed Kinetic Chain Rehabilitation for the Upper Extremity," *Journal of Sport Rehabilitation*, 5(1): 78.

is inadequate, the ACL may be jeopardized at high joint loads. McNair et al. (69) found that hamstring stiffness was not significantly different in the involved and uninvolved limbs of ACL-deficient athletes. However, there was a positive correlation between hamstring stiffness and functional ability, implying that high muscle stiffness prior to injury provides additional dynamic restraint and greater functional ability in athletes subsequent to ACL injury (69).

Because tibial translation also lengthens the hamstrings muscles, increased preparatory muscle stiffness reduces the electromechanical delay required to develop muscle tension (22, 89). Moreover, increased muscle activation can drastically improve the stretch sensitivity of the muscle spindle system (19, 40, 56). Enhancing stretch sensitivity and stiffness could improve the reactive capabilities of the hamstrings by providing additional sensory feedback and superimposing stretch reflexes onto descending motor commands (22, 46, 70). Research has shown that the stretch reflex can increase muscle stiffness one to three times (37, 66). Whether muscle stiffness increases stretch sensitivity or decreases electromechanical delay, it appears to be crucial for dynamic restraint and functional ability in ACL-deficient athletes.

ACL-deficient athletes exhibit characteristic EMG patterns while walking and running. The feed-forward process of motor control is responsible for compensatory antagonist preactivation and agonist inhibition during ambulation. Several profiles have demonstrated increased lateral hamstring activity during the stance phase of walking and cutting maneuvers (12, 16, 49, 61, 84). Increased hamstring activation compensates for ACL insufficiency by inhibiting anterior tibial translation and rotation (16, 69). It has been shown that during the "swing to strike" transition phase of gait, quadriceps activity decreases (61). Preprogrammed quadriceps inhibition, prior to heel strike, prevents excessive anterior shearing forces at ground contact. Hamstring activation and quadriceps inhibition reflect preparatory mechanisms to anticipated joint loads that preserve joint equilibrium and stability (89).

Reestablishing Proprioception, Kinesthesia, and Neuromuscular Control

Activities designed to restore neuromuscular control following ACL injury are intended to complement traditional rehabilitation protocols that focus on modulating pain and inflammation and restoring flexibility, voluntary strength, and endurance. The objective of neuromuscular control activities is to integrate peripheral sensations relative to joint position and process these signals into efferent motor responses. In the ACL-injured knee, these dynamic mechanisms compensate for a lack of static restraints and can produce a functionally stable joint. In order to restore and induce dynamic muscle activation necessary for functional stability, one must employ simulated positions of vulnerability that necessitate muscle reactive stabilization. Although there are inherent risks in placing the knee in positions of vulnerability, if it is done in a controlled and progressive fashion, neuromuscular adaptations will occur and subsequently permit the athlete to return to competition with confidence that the dynamic mechanisms about the knee will protect the joint from subluxation and reinjury.

Four elements are crucial to reestablishing neuromuscular control and functional stability: proprioceptive and kinesthetic sensation, dynamic joint stabilization, reactive neuromuscular control, and functional motor patterns (62). Several

afferent and efferent characteristics contribute to efficient regulation of these elements for maintenance of neuromuscular control and functional stability. These characteristics include the sensitivity of peripheral receptors and facilitation of afferent pathways, muscle stiffness, the onset rate and magnitude of muscle activity, agonist/antagonist co-activation, reflex muscle activation, and discriminatory muscle activation. Specific rehabilitation techniques produce adaptations that enhance the efficiency of these characteristics. These techniques include closed kinetic chain activities, balance training, eccentric and high-repetition/low-load exercises, reflex facilitation through reactive training, stretch-shortening activities, and biofeedback training.

Modifying Afferent/Efferent Characteristics

The objective of neuromuscular rehabilitation is to develop and reestablish afferent and efferent characteristics that enhance dynamic restraint capabilities to in vivo loads. The plasticity of the neuromuscular system permits rapid adaptations during rehabilitation that enhance preparatory and reactive muscle activity (38, 41). Although clinical research continues, several exercise techniques show promise for inducing beneficial adaptations in the neuromuscular system.

Reliable kinesthetic and proprioceptive information is the foundation on which feedback and feed-forward neuromuscular control is based. Altered peripheral afferent information may disrupt preparatory and reactive muscle activity, affecting motor control and functional stability. Closed kinetic chain exercises create axial loads that maximally stimulate articular receptors, while length/tension changes excite tenomuscular receptors (17, 34, 45). Chronic athletic participation may enhance proprioceptive and kinesthetic acuity by frequently facilitating afferent pathways from peripheral receptors. Highly conditioned athletes demonstrate greater appreciation of joint kinesthesia than sedentary controls (62, 66). Whether this a congenital anomaly or a training adaptation, greater awareness of joint motion and position may improve feed-forward and feedback neuromuscular control (62).

It is evident that muscle stiffness has a significant role in preparatory and reactive dynamic joint restraint; therefore, exercise modes that increase muscle stiffness should be encouraged. Research by Bulbulian and Bowles (14) and Pousson et al. (80) has established that eccentric loading increases muscle tone and stiffness. Chronic overloading to muscle and tendon may result in connective tissue proliferation, thus desensitizing GTOs and increasing muscle spindle activity (41). Such evolutions impact the neuromuscular and tendinous components of stiffness (14, 32, 72, 80). Eccentric loading is accomplished by activities such as forward and backward stair climbing and backward downhill walking. Stretch-shortening activities, emphasizing the landing phase, also impose eccentric loads on the hamstring group.

Training techniques that emphasize low loads and high repetitions cause similar connective tissue adaptations as those found with eccentric training. However, increased muscle stiffness resulting from this rehabilitation technique may be attributed to fiber type transition (32, 41, 57, 58). Slow-twitch fibers have longer cross-bridge cycle times and can maintain the prolonged, low-intensity contractions necessary for postural control (58). Goubel and Marini (32) found that in animal models, low-load/high-repetition training resulted in higher muscle stiffness compared to strength training. However, Kyrolainen and Komi (58), in a retrospective analysis of power- and endurance-trained athletes, inferred that muscle

stiffness was greater in power-trained individuals because the onset of muscle preactivation (EMG) was faster and higher prior to joint loading. It appears that endurance training may increase stiffness by increasing the baseline motor tone and cross-bridge formation time, while power training enhances the rate and magnitude of muscle tension during preactivation. Both of these adaptations readily adhere to existing principles of ACL rehabilitation, where early strength exercises focus on low loads with high repetitions, progressing to shorter, more explosive, sport-specific activities. Research assessing the efficacy of low-load/high-repetition training versus high-load/low-repetition training would be beneficial for optimizing muscle stiffness and functional progression in ACL-deficient athletes.

Training adaptations may account for the vigorous reflex responses (tendon–tap) in sprint- and power-trained subjects relative to sedentary and endurance-trained subjects (54, 55, 91). McComas (68) suggested that strength training increases descending (cortical) drive to the large motor nerves of skeletal muscle and the small efferent fibers to muscle spindles. As discussed earlier, increasing muscle spindle activity increases their sensitivity to stretch, resulting in shorter reflex latencies (41). Melvill-Jones and Watt (70) suggested that stretch reflexes are superimposed on preprogrammed muscle activity from higher centers, illustrating the concomitant use of feed-forward and feedback processing for regulating preparatory and reactive muscle stiffness. Walla et al. (92) found that ACL-deficient athletes' reflex hamstring control correlated well with functional rating scores. Therefore, reactive neuromuscular control in ACL-deficient athletes may improve function if muscle stiffness is enhanced.

A limited number of clinical training studies have been directed at improving reaction times (42). Ihara and Nakayama (42) significantly reduced the latency of muscle reactions over a 3-week period by inducing perturbations to athletes on unstable platforms. Similar modifications to the spinal stretch reflex have been investigated for their role in motor control (95). Although it was once believed that the speed of stretch reflexes could not be directly enhanced, efforts to do so have been successful in animal and human studies (94, 95). This has significant implications for reestablishing the reactive capability of the dynamic restraint system. Reducing the electromechanical delay between joint loading and protective muscle activation can increase dynamic stability and function.

Biofeedback training provides instantaneous sensory feedback concerning muscle activation patterns and may help athletes correct errors by consciously altering muscle activity (10, 30). The objective of biofeedback training is to reacquire voluntary muscle control and promote functionally specific motor patterns, eventually converting these patterns from conscious to unconscious control (10). Using biofeedback for discriminative hamstring control may help the ACL-injured athlete reestablish preparatory and reactive muscle activity for dynamic joint stability (23, 30).

Elements for Neuromuscular Control and Functional Stability

Proprioception and Kinesthesia. The objective of kinesthetic and proprioceptive training is to restore the neurosensory properties of injured ligaments and enhance the sensitivity of secondary peripheral afferents (65). It is unknown to what degree this occurs in conservatively managed athletes; however, ACL reconstruction with extensive rehabilitation does appear to normalize joint motion and position sense (8, 63).

In the lower extremity, joint compression is believed to maximally stimulate articular receptors and can be accomplished with closed chain exercises throughout the available ROM (17, 34, 45). Early joint repositioning tasks enhance conscious proprioceptive and kinesthetic awareness, eventually leading to unconscious appreciation of joint motion and position. Applying a neoprene sleeve may provide additional proprioceptive and kinesthetic information by stimulating cutaneous receptors (8, 63). Partial weight bearing, in pools or with unloading devices, simulates the closed chain environment without subjecting the knee to excessive loads (52). A multitude of closed kinetic chain exercises emphasizing hamstring activation increase hamstring stiffness and muscle spindle stretch sensitivity. The closed kinetic nature of these exercises causes joint compression, thus enhancing joint congruency and proprioceptive feedback while minimizing joint shear forces (78). To induce adaptations in muscle stiffness, exercises should be done with high repetitions and low rest intervals, focusing on the eccentric phase. Increased muscle stiffness will increase the stretch sensitivity of tenomuscular receptors, providing additional sensory information concerning joint motion and position. Techniques believed to increase muscle stiffness include stiff-leg deadlifts and lunges, progressing from no weight to weight assisted. Early kinesthetic training may begin to reestablish reflex pathways from articular afferents to skeletal motor nerves, the muscle spindle system, and cortical motor control centers, while enhancing muscle stiffness increases the stretch sensitivity of tenomuscular receptors.

Dynamic Stabilization. The objective of dynamic joint stabilization exercises is to encourage preparatory agonist/antagonist coactivation. Efficient coactivation restores the force couples necessary to balance joint forces and increase joint congruency, thereby reducing the loads imparted onto static structures. Dynamic stabilization from muscles requires anticipating and reacting to joint loads. This includes placing the joint in positions of vulnerability where dynamic support is established under controlled conditions. Balance and stretch-shortening exercises both require preparatory and reactive muscle activity through feed-forward and feedback motor control systems, while closed kinetic chain exercises are excellent for inducing coactivation and compression.

Early dynamic joint stabilization exercises begin with balance training consisting of partial weight bearing on stable surfaces progressing to partial weight bearing on unstable surfaces, ultimately balancing with full weight bearing on unstable surfaces. Exercises such as kickers also require balance and can begin on stable surfaces, progressing to unstable platforms (Figure 2).

Slide board training and basic strength exercises may be used to stimulate hamstring/quadriceps coactivation while increasing muscular endurance. Strength exercises focus on eccentric and endurance-type activities in a closed kinetic orientation, further enhancing dynamic stability through increases in preparatory muscle stiffness and reactive characteristics. Strength and balance exercises can be combined and executed with light external forces to increase the level of difficulty (Figure 3).

Biofeedback may also assist athletes trying to develop quadriceps and hamstring coactivation during strength exercises. Biofeedback provides information concerning muscle activation and encourages voluntary muscle activation by facilitating efferent pathways. Reeducating selective muscle activation is necessary for dynamic stabilization and neuromuscular control.

Stretch-shortening exercises, which are necessary for conditioning the neuromuscular apparatus to respond more quickly and forcefully, develop explosive

Figure 2 — Kickers: The athlete stands on either the involved or uninvolved limb, with an elastic band fixed to the distal aspect of the contralateral leg. While attempting to balance, the athlete executes short kicks with either knee extension or hip flexion.

Figure 3 — One-legged squats: The athlete squats while an elastic band, fixed distal to the knee, applies an external load.

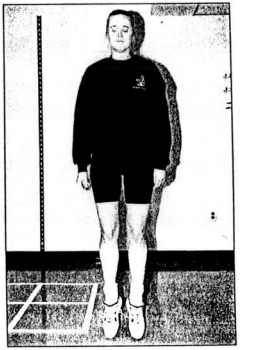

Figure 4 — Calf pushes: With the knees slightly bent, the athlete initiates ankle plantar flexion without knee flexion. As the calf pushes are repeated, emphasis should be placed on absorbing energy in the calf.

concentric contractile properties while permitting eccentric deceleration of the knee joint (1). Stretch-shortening exercises need not be withheld until the late stages of rehabilitation. There are multiple plyometric exercises with varying degrees of intensity. Low-impact plyometric activities may commence once weight bearing is achieved. Examples of these low-impact exercises are stiff-legged bunny hops and calf pushes (Figure 4).

Double-leg bounding is an effective intermediate stretch–shortening exercise because the uninvolved leg can be used for assistance. Stretch-shortening activities are made more difficult with alternate-leg bounding, then single-leg hopping. Activities such as hopping with rotation, lateral hopping, and hopping onto various surfaces are instituted, using a mirror if possible (Figure 5). The use of a mirror will enable the athlete to internalize proper kinematics, further facilitating specific movement patterns. One exercise that bridges the gap between lateral movements and plyometrics is the lateral box jumps (Figure 6). The athlete performs the lateral box jump standing lateral to the box and jumping onto the box from both sides. Plyometric training requires preparatory muscle activation and may facilitate reflexive pathways for reactive neuromuscular control.

Reactive Neuromuscular Control. Reactive neuromuscular training focuses on stimulating the reflex pathways from articular and tenomuscular receptors to skeletal muscle. Although preprogrammed muscle stiffness can enhance reflex latency, the objective is to induce unanticipated joint perturbations, stimulating reflex

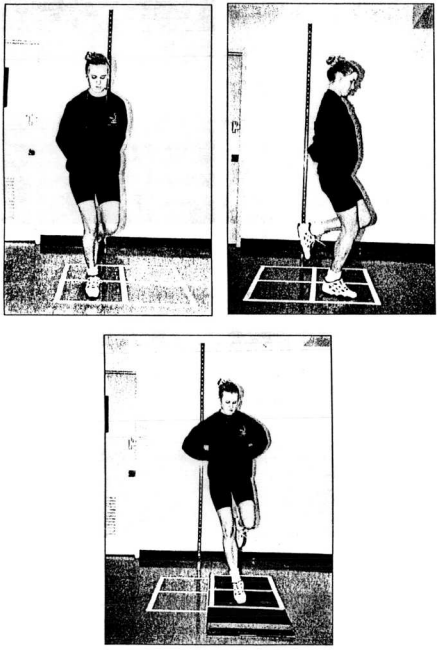

Figure 5 — Hopping progression: The athlete is positioned on a surface divided into four squares. Hopping between squares becomes progressively more difficult by including lateral and diagonal hops, eventually progressing to hops with rotation and landings on various surfaces.

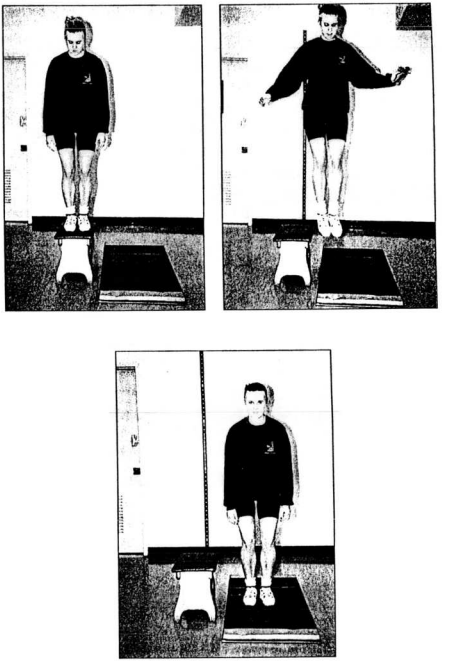

Figure 6 — Lateral box jumps: The athlete stands on a box and hops off laterally. In more advanced stages, the athlete lands only on the involved leg or bounds up from the landing back onto the box.

igure 7 — Multidirectional leg reaction drills: The athlete is seated in a comfortable osition of knee flexion. A clinician randomly applies multidirectional forces to the ower extremity while the athlete attempts to maintain the position of the leg.

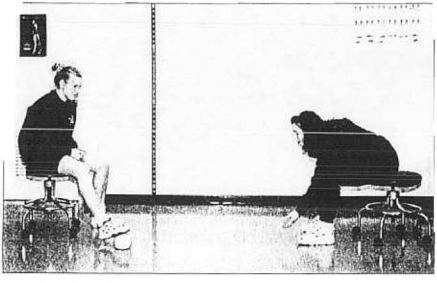

Figure 8 — Foot push pass: While the athlete is seated, a clinician rolls a ball toward the athlete, varying the speed and direction. The athlete's goal is to receive the ball with the foot and then pass it back.

stabilization. The efficacy of reactive neuromuscular exercises was demonstrated over a decade ago (42). Persistent use of these reflex pathways may decrease response time and develop reactive strategies to unexpected events (36).

During early rehabilitation, clinicians can include rhythmic stabilization exercises (Figure 7) and foot pass drills (Figure 8). These exercises are designed to enhance lower extremity neuromuscular coordination and reaction to light joint loads. The intensity of rhythmic stabilization is increased by applying greater joint loads. Foot pass drills can begin with large balls and progress to smaller balls, or light balls progressing to heavier balls.

Unstable platforms are used to induce perturbations to the joint and alter the athlete's center of gravity manually while he or she attempts to maintain balance. These exercises can facilitate adaptations to reflex pathways mediated by peripheral afferents and resulting in quadriceps and hamstring muscle activation. Ball tossing may be incorporated in conjunction with balance exercises to disrupt concentration and induce unconscious, reactive adaptations. Walking and running in sand also require similar reactive muscle activity and may enhance reflexive joint stabilization.

During the later stages of rehabilitation, reactive neuromuscular activity incorporates trampoline hopping. The athlete begins by hopping and landing on both feet, progressing to hopping on one foot, and hopping with rotation (Figure 9). The most difficult reactive tasks include hopping while catching a ball (Figure 10) and hopping off a trampoline onto various landing surfaces such as artificial turf, grass, and dirt. All reactive exercises should induce joint perturbations in order to facilitate reflex muscle activation. Reflex-mediated muscle activity is a crucial element of the functionally stable joint and should complement preprogrammed muscle activity.

Functional Activities. The last element requires adaptations to preprogrammed, functionally specific motor patterns. These activities incorporate all of the available resources for stimulating peripheral afferents, muscle coactivation, and reflex and preprogrammed motor control. Emphasis should be placed on sport-specific techniques, including positions and maneuvers where the ACL is vulnerable. With repetition and controlled intensity, muscle activity (preparatory and reactive) gradually progresses from conscious to unconscious motor control (50). Reestablishing functionally specific movement repertoires within a controlled setting can decrease the risk of injury upon completion of rehabilitation.

Functional activities begin with restoring normal gait. Verbal instruction can assist athletes during gait training (partial weight bearing). Emphasis is placed on normal kinematics and quadriceps/hamstring coactivation. This can include backward (retro) walking, which further facilitates hamstring activation and balance (73). If a pool or unloading device is available, crossover walking and figure eights can begin, progressing to jogging and hopping as tolerated. Functional activities with partial weight bearing help restore motor patterns without compromising static restraints. Weight-bearing activities are continued on land with the incorporation of acceleration and deceleration and pivot maneuvers. Drills including jogging, cutting, and cariocas are initiated, with the speed of maneuvers gradually increased.

The most difficult functional activities are designed to simulate the demands of the individual's sport and position. Activities such as shuttle runs, carioca crossovers, retro sprinting, and forward sprinting are implemented with sport-specific drills such as fielding a ball, receiving a pass, and dribbling a soccer ball. It is necessary to understand the afferent and efferent characteristics that contribute to

Figure 9 — Trampoline hopping: The athlete first bounces on both feet, then progresses to hopping off of two feet and landing on one foot. Finally, the athlete hops with rotation.

Figure 10 — Trampoline and ball tossing: To intentionally disrupt concentration, the clinician tosses a ball to the athlete while she is bouncing on a trampoline.

joint sensation, dynamic stabilization, reflex activity, and functional motor pattern in order to reestablish neuromuscular control and functional stability (see Table 1).

Summary

Disruption of the ACL can compromise both the static and dynamic restraining mechanisms of the knee. Static support depends upon the biomechanical properties of articular structures, while dynamic restraint is mediated by articular and tenomuscular mechanoreceptors, via cortical and reflex pathways. Muscle spindles have received special consideration for their capacity to integrate peripheral afferent information and reflexively modify muscle activity. The efferent response to peripheral afferent information is termed *neuromuscular control*. Feed-forward and feedback neuromuscular control both utilize sensory information for preparatory and reactive muscle activity. The degree of muscle activation greatly determines the stiffness of the muscle, which in turn assists dynamic restraint by resisting changes in muscle length. To reestablish neuromuscular control and functional stability, clinicians may use rehabilitation techniques including closed kinetic chain activities, balance training, eccentric and high-repetition/low-load exercises, reflex facilitation through reactive training, stretch-shortening activities, and biofeedback training. These techniques produce adaptations in the sensitivity of peripheral receptors and facilitate afferent pathways, agonist/antagonist coactivation, muscle stiffness, the onset rate and magnitude of muscle activity, reflex muscle activation, and discriminatory muscle activation.

Table 1 The Elements, Rehabilitation Techniques, and Afferent/Efferent Characteristics Necessary for Restoring Proprioception and Neuromuscular Control in the ACL-Injured Athlete

Elements	Rehabilitation techniques	Afferent/efferent characteristics
Proprioception and kinesthesia	Joint repositioning Functional range of motion Axial loading Closed kinetic chain exercises	Peripheral receptor sensitivity Facilitate afferent pathways
Dynamic stability	Closed kinetic chain exercises and translatory forces High repetition/low resistance Eccentric loading Stretch-shortening exercises Balance training	Agonist/antagonist coactivation Muscle activation rate and amplitude Peripheral receptor sensitivity Muscle stiffness
Reactive neuromuscular control	Reaction to joint perturbation Stretch-shortening, plyometrics Balance reacquisition	Reflex facilitation Muscle activation rate and amplitude
Functional motor patterns	Biofeedback Sport-specific drills Control-progressive participation	Discriminatory muscle activation Arthrokinematics Coordinated locomotion

These afferent and efferent characteristics regulate the following four elements, which are critical to neuromuscular control and functional stability: proprioception and kinesthesia, dynamic stabilization, reflex muscle activation, and functional motor control. Each phase of traditional rehabilitation protocols can incorporate some aspect of all four elements, depending upon each individual's tolerance to functional progression. By integrating these elements into the rehabilitation of an ACL-injured athlete, clinicians can maximize the contributions of the dynamic restraint mechanisms to functional stability.

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