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Neuroplasticity Following Anterior Cruciate Ligament Injury: A Framework for Visual-Motor Training Approaches in Rehabilitation

Anterior cruciate ligament (ACL) rupture is a common activity-related knee injury that usually requires surgical reconstruction to restore knee stability and function.¹⁴⁰ The lifetime burden of ACL injury ranges from \$7.6 to \$17.7 billion per year in the United States.⁹⁶ Despite surgical reconstruction and physical rehabilitation, injury of the ACL dramatically increases the risk for costly and long-term disabling osteoarthritis, associated decreased lifelong physical activity, and decreased work productivity.^{4,14,93,96} Importantly, reconstruction and rehabilitation that rely primarily on traditional neuromuscular interventions have a failure rate of up to 30% for rerupture after return to sport.^{74,120,121,154} This high failure

rate is further compounded by the inability of a majority of individuals to return to preinjury levels of activity.¹⁰

Although evidence supports neuromuscular training for effective injury prevention and rehabilitation, many of these approaches primarily target biomechanical

factors, such as muscle strength, balance, and plyometric function, and give less consideration to cognitive or neurological components.^{60,107,109,156} While rectifying the biomechanical profile and restoring muscle strength are vital components of the rehabilitation process, there may be potential to improve function and decrease reinjury risk.^{58,99} Recent reports have found unresolved neuroplastic alterations after injury, reconstruction, and rehabilitation that may limit function and return to sports participation.^{9,17,87,94} By targeting these neurologic factors and integrating neurocognition during neuromuscular rehabilitation progressions, it may be possible to improve the transfer of sensorimotor adaptations from the clinic to activity, and ultimately to improve patient outcomes.^{16,61}

The training, and even restoration, of primarily biomechanical factors relative to ACL-injury risk^{67,132} may not address all the physiologic consequences of injury, as patient-reported dysfunction and poor movement control may persist for years.^{8,108,119,129,131,158} The impaired physical performance and patient-reported dysfunction might, in part, have a neurologic origin.^{79,84,123} The capacity for neuroplasticity after injury and during therapy may present an opportunity to

● **SYNOPSIS:** The neuroplastic effects of anterior cruciate ligament injury have recently become more evident, demonstrating underlying nervous system changes in addition to the expected mechanical alterations associated with injury. Interventions to mitigate these detrimental neuroplastic effects, along with the established biomechanical changes, need to be considered in the rehabilitation process and return-to-play progressions. This commentary establishes a link between dynamic movement mechanics, neurocognition, and visual processing regarding anterior cruciate ligament injury adaptations and injury risk. The proposed framework incorporates evidence from the disciplines of neuroscience, biomechanics, motor control, and psychology to support integrating

neurocognitive and visual-motor approaches with traditional neuromuscular interventions during anterior cruciate ligament injury rehabilitation. Physical therapists, athletic trainers, strength coaches, and other health care and performance professionals can capitalize on this integration of sciences to utilize visual-training technologies and techniques to improve on already-established neuromuscular training methods.

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● **KEY WORDS:** ACL, motor control, neuroscience, return to sports

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close the gap between rehabilitation and activity by targeting a broader spectrum of sensorimotor function during neuromuscular training.^{58,64,108,109} Alternative approaches and adjunct therapies may help to address the neurological system functions associated with the faulty movement patterns underlying ACL reinjury risk.^{18,87,94,121}

As an example, typical rehabilitative exercises are completed with an internal focus of control, meaning that full attention is directed to the internal aspects of the movement only (eg, avoidance of excessive knee valgus or increasing knee flexion).^{20,155,156} Internal focus can offer positive benefits early in rehabilitation, when the need to develop or restore a motor pattern or muscle contraction ability is vital. But, function in the athletic environment, or even in activities of daily living, requires constant interactions with the dynamic and constantly changing visual environment. Sport and activities of daily living therefore require an external focus of control, where attention is directed to the environment and the body relies on automatic motor control to maintain joint-to-joint integrity.^{11,41,128}

The need to challenge a broad spectrum of sensorimotor control is demonstrated by the noncontact ACL-injury scenario: a failure to maintain knee neuromuscular control while attending to an external focus of attention, involving highly complex dynamic visual stimuli, variable surfaces, movement planning, rapid decision making, variable player positions and environment interactions, and unanticipated perturbations.^{26,68,82,88} The need to bridge the intense neurocognitive and motor control demands of sport during rehabilitation may therefore benefit from specific interventions that target these neurocognitive factors in addition to the biomechanical techniques that are already widely addressed.

The transition from rehabilitation to sport activity is challenged by complex environmental interactions that

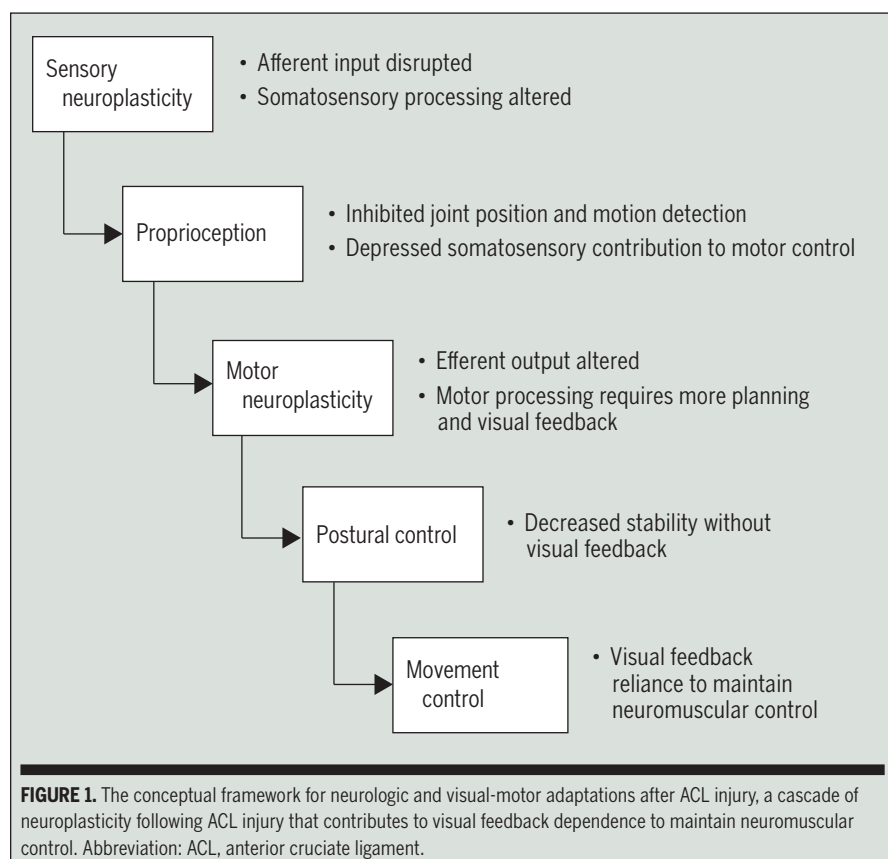
place high demand on cognitive and sensorimotor processes and, in turn, increase ACL reinjury risk.^{26,68,82,88} In a constantly changing environment, the sensory system's 3 primary afferent pathways (vestibular, visual, and somatosensory) provide the complex and integrated information necessary for the efferent neuromuscular control system to maintain adequate stability and control.^{95,153} One area of sensorimotor function that may uniquely be affected by ACL injury is motor control requiring visual feedback.⁹⁵ The visual system provides a fundamental mechanism for coordination, regulation, and control of movement while managing environmental interactions (external focus).^{122,139,150} Visual feedback is especially needed in executing movement sequences^{5,111} and increasing task complexity and variability.^{40,89,128,150} The interplay between vision and somatosensation is particularly vital to provide sufficient afferent input to the central nervous system (CNS) to regulate motor control and to maintain neuromuscular integrity during action and environmental interaction.^{133,134,143,149,153} In this sensory-to-motor feedback loop, changes to visual or sensory feedback lead to subsequent alterations in neuromuscular control during movement (closed-loop processing).^{23,95,133,143,150,153} Trauma to the ACL has been shown to modify how the nervous system processes these interactions between vision and somatosensation.^{2,3,55,79,115} Targeting injury-induced sensory-motor plasticity presents a unique opportunity to improve the translation of neuromuscular system enhancements from the rehabilitation environment to the return-to-sport environment.^{29,76,100} Thus, our purpose in this commentary is to highlight the contributions of nervous system function and reorganization in the ACL-injury rehabilitation process, and specifically how adding visual-motor approaches during neuromuscular training may mitigate potentially limiting factors during return to high-demand physical activities.

ACL Injury–Induced Sensory Visual–Motor Processing Compensations

To better understand the rationale for how visual-motor training may enhance ACL-injury rehabilitation, a thorough understanding of the current evidence on neuroplastic changes associated with ACL injury is required. The overarching concept is that the CNS afferent input is disrupted due to the lost somatosensory signals from the ruptured ligament and increased nociceptor activity associated with pain, swelling, and inflammation. The disrupted sensory input and injury-associated joint instability, muscle atrophy, and movement compensations combine to facilitate motor control adaptations. The reconstruction process leads to further deafferentation of the joint, causing continued neuroplastic modifications that result in maladapted efferent neuromuscular output (**FIGURE 1**).

CNS Adaptations

In animal models, the ACL mechanoreceptor and afferent connections can be traced within the nervous system to the spinal cord, brain stem, and cerebral regions, contributing to proprioceptive, nociceptive, and reflex function.^{59,118} The initial sensorimotor neuroplasticity after ACL injury is likely caused by the abrupt loss of this connection, which once provided the nervous system with continuous feedback. In human studies, the afferent loss is demonstrated by altered or absent somatosensory-evoked potentials with stimulation of the common peroneal nerve^{37,39,147,148} or the ACL directly.¹²⁵ The loss of primary afferent information combined with the pain and inflammatory response contribute to fundamentally alter the somatosensory feedback.^{32,75,79,91} The disrupted input, combined with mechanical changes and compensations^{110,131} (contralateral loading,^{15,119} hip or ankle strategies^{48,56}), facilitates the adaptations for motor control.^{68,128,129} On a foundational level, altered motor output manifests in disrupted gamma motor neuron function^{83,85,86} and perturbation reflexes,^{38,44} which play a key role in the abil-



ity to maintain neuromuscular integrity in a changing environment that requires rapid and precise muscle stiffness or activation strategies.^{30,81,145} The lost ability to rely on reflex and gamma motor neuron drive to prepare alpha motor neuron function requires the CNS to engage in supplementary mechanisms, such as increased utilization of visual feedback, to maintain the required sensory input for motor control. As such, neuromuscular control after ACL injury may require enhanced visual feedback, depriving the CNS of resources once used for managing environmental interaction to maintain knee joint stability.

These deficits in neural function are not rectified with ACL reconstruction and may become even more pronounced and/or present bilaterally.^{24,83,84,87,94,130,148} The bilateral motor control, reflex, and proprioceptive changes are theorized to be due to both spinal^{59,118} and supraspinal^{39,123} mechanisms.¹²⁴ This ongoing neu-

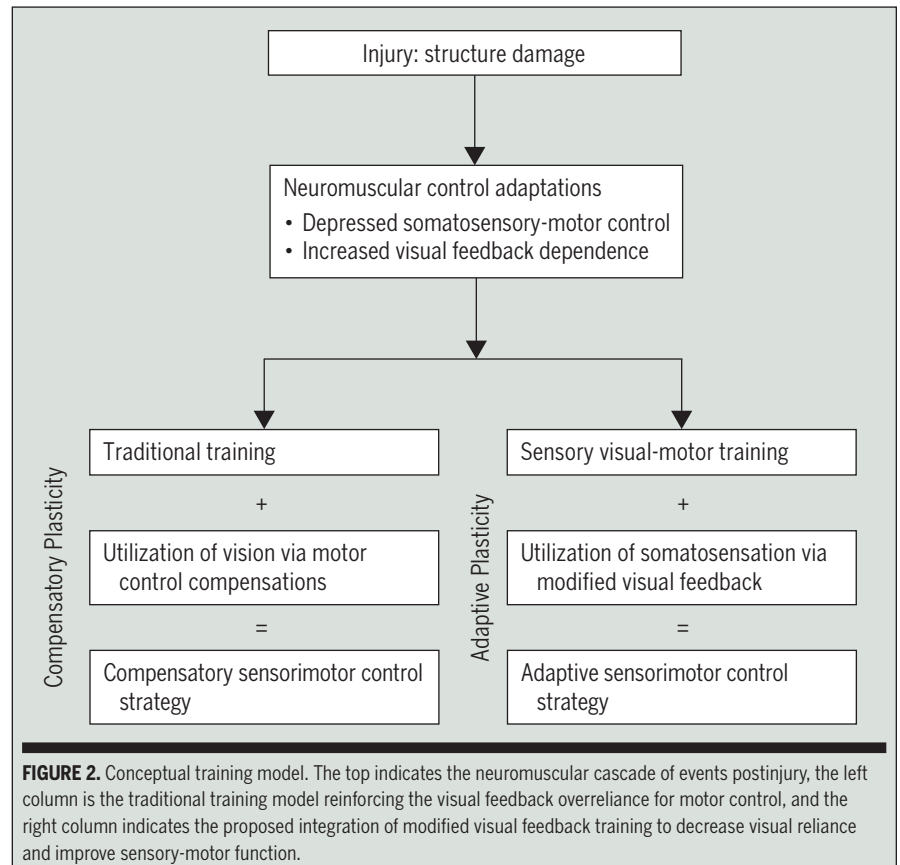
roplasticity and altered mechanical and biological function of the joint combine to reduce proprioception acuity as measured by joint position sense,^{27,92} movement detection,^{27,54} and force sense.⁶⁶ To investigate the neurologic adaptations of functional sensory loss, Baumeister et al^{17,18} used electroencephalography during force- and joint-sense tasks and found that those with ACL reconstructions had greater brain activation in attentional and sensory areas. The increased activation may be attributed to less neural efficiency or increased neural load to complete the same task; interestingly, despite increased cortical activation, proprioceptive performance was still worse in those with ACL reconstruction as compared to controls.^{17,18} These results indicate that the loss of the native ACL not only constitutes a mechanical instability but also a degree of nervous system deafferentation that is not rectified with reconstructive surgery and rehabilitation.⁷⁸

This partial deafferentation is further illustrated by investigations utilizing transcranial magnetic stimulation to assess the CNS efferent pathway between the quadriceps and the brain.^{65,90,113,123} Heroux and Tremblay⁶⁵ reported enhanced resting corticomotor excitability in those with ACL injury. A potential mechanism for increased resting motor cortex excitability may be due to the affected sensory feedback, as the brain attempts to maintain motor output with attenuated sensory input. This increase in excitability may increase potential feedforward mechanisms by decreasing the threshold for connections with motor planning areas, or allow for increased input from other sensory sources (vision, vestibular).^{62,116,144,159} A recent neuroimaging investigation by Kapreli et al⁷⁹ provides further evidence of the neuroplastic effects of ACL injury. They performed functional magnetic resonance imaging of the brain during knee flexion and extension, and found that those with an ACL injury had increased activation of the presupplementary motor area, posterior secondary somatosensory area, and posterior inferior temporal gyrus compared to matched controls.⁷⁹ The presupplementary motor area is highly involved in complex motor planning,^{12,111} and, despite the relative simplicity of the movement task (single-joint movement of 40° of knee flexion/extension while lying supine), those with an ACL injury needed to engage higher-level motor control areas to a greater degree to execute the movement. This increased activation may indicate that, on a neural control level, simple movements are more taxing to those with a previous ACL injury.¹⁰⁴ The increase in posterior secondary somatosensory area provides further evidence of sensory-based neuroplasticity after injury, as this area is involved in regulating painful stimuli but is highly interconnected with the anterior secondary somatosensory area that integrates somatosensory inputs.^{33,49,145} Interestingly, the participants in the study did not report pain during the movement, conceivably indicating

a sensory processing adaptation from the initial increase in nociceptive input from the traumatic nature of the injury. Alternatively, the prolonged nature of the rehabilitation, chronic pain, or joint instability may continue to disrupt typical somatosensory-system afferent integration. The posterior inferior temporal gyrus plays a role during many cerebral functions,^{25,28} but may primarily be involved with visual processing of movement.¹²² As such, an increase in posterior inferior temporal gyrus activation during movement may indicate that, in response to ACL injury, there is an increased utilization of visual processing and motor planning resources for movement concurrent with depression of somatosensory function.^{37,39,55,79,147,148}

Biomechanical Adaptations

These neuroplastic observations following ACL injury are further supported by biomechanical evidence suggesting that with increased task complexity, neuromuscular control is deteriorated in individuals with an ACL injury or reconstruction to a greater extent than in controls, possibly due to overload of motor planning resources.^{73,112} The specific neuroplastic visual-motor control adaptation is observed during static balance, as those with ACL injury have significantly diminished postural control when vision is obstructed (blindfold or eyes closed),^{114,115} but limited to no degradation in postural control with eyes open, as they are able to use vision to compensate and maintain balance.^{69,97} A more pronounced effect on neuromuscular control is observed when disrupting visual-motor processing during complex landing and cutting maneuvers that play an even greater role in injury risk.^{102,103,142} The simple addition of a target during a jump-landing task increased injury risk mechanics⁵² and altered muscle activation, decreasing postural stability.¹⁵² The effects of forcing visual focus on the environment during more complex cutting or direction-change tasks further degrade neuromuscular control capability



in healthy athletes with the addition of a defender,¹⁰² a virtual soccer interface,³⁶ or a level of unanticipated decision making during the task (selecting direction).^{101,126} The effect of occupying the visual system with environmental cues during landing or change of direction is even greater in those with a history of ACL injury.^{72,73}

These findings, taken together, suggest that ACL injury may lead to a cascade of neuroplastic and neuromuscular alterations that increase reliance on visual feedback and cortical motor planning for the control of knee movement. The postinjury disrupted sensory feedback, combined with the observed motor compensations, contributes to fundamentally alter the CNS mechanisms for motor control.^{1,17,18,80,94,147,148,160} In attempting to regulate neuromuscular control in the presence of decreased somatosensory input, the nervous system supplements with increased motor planning, conscious cortical involvement,

and greater reliance on visual feedback. This ACL injury-induced neuroplasticity can have consequences for function and further injury risk, as the visual feedback and motor planning neural mechanisms become overloaded in the athletic environment. Specific additions to current neuromuscular interventions targeting these neuroplastic imbalances may play a significant role to induce sensorimotor adaptations to decrease dependence on visual feedback when transitioning to more demanding activities.

Visual-Motor Training as a Rehabilitation Tool

Typically, neuromuscular interventions (eg, plyometrics, balance training, strengthening exercises) allow full focus of attention on the movement, whereas in sporting situations this is rarely the case.^{88,98} Traumatic injuries (ACL ruptures) tend to occur during complex game situations when the player must



FIGURE 3. Strobe glasses. The bottom picture on the left is the opaque condition and on the right is the clear condition. These conditions continuously alternate, with potential for 8 settings, with the clear condition always lasting 100 milliseconds and the opaque conditions varying from 25 milliseconds to 900 milliseconds. The top picture provides an illustrative example of a first-person perspective with the glasses at a moderate level of occlusion (approximately level 5 or 6, with a 150- to 233-millisecond opaque interval).

manage multiple variables (eg, ball, players, field position, game strategy) requiring full visual attention to the environment, theoretically leaving less cognitive processing resources for neuromuscular control.^{26,68,71,82,88,98} These environmental demands and the increased need for visual feedback for knee control in individuals after ACL injury^{55,79,115} combine to create a higher-risk state for the athlete. This framework indicates a CNS alteration of afferent processing, to compensate for the lost somatosensory contribution by increasing utilization of visual resources for neuromuscular control, which may be an adaptation of the disrupted proprioceptive afferent input from the damaged ACL and associated noxious stimuli.^{1,17,18,50,117} It is possible that after extensive time and/or training, a measure of motor function may be restored, but at the expense of compensations that allow the sensory deficits to remain.^{2,53,57} Motor function may normalize with basic tasks in the clinic, such as hop or strength tests, but may not transfer to the demanding athletic environment, where the proprioceptive sensory loss may result in impaired motor function as the task and environmental complexities increase.^{19,46,76,77} Currently used

rehabilitative methods may even be further contributing to the neuromuscular control compensations and facilitating possible compensatory neuroplasticity (FIGURE 2).^{17,18,65,79,83,87,129} Recognizing and addressing the specific postinjury neuroplasticity during neuromuscular training may provide an avenue for the clinician to address both the physical and neurocognitive demands of return to sport.^{13,121,157}

This framework highlights 3 related sensorimotor adaptations occurring in the athlete with an ACL injury: (1) depressed or disrupted somatosensory input and altered sensorimotor processing, which induce (2) increased visual processing to plan movement and maintain neuromuscular control and (3) increased cortical top-down motor control strategies.

Modifying Visual Feedback

The need to transfer neuromuscular control strategies from the stable training environment to the chaotic athletic field requires that interventions integrate complex sensory inputs (environmental stimulus, visual and proprioceptive acuity) in conjunction with the motor outputs (strength, movement quality).^{2,17,18,20,55,61} Disrupting visual feedback

as an adjunct to traditional rehabilitation may more closely mimic actual activity demands via escalated load on the neurocognitive system^{36,52,98,102,152} and smooth the transition back to activity by providing a closer analog to the inherent environmental challenges of sport.^{95,98} Historically, the primary method to disrupt the visual feedback system has been to use complete visual obstruction (eyes closed or blindfold). This kind of visual deprivation presents a motor control challenge in a healthy population¹³³ and has a more pronounced effect in individuals with an ACL injury.^{55,115} However, rehabilitation using eyes-closed or blindfolded conditions⁵⁵ has been restricted to static balance,¹¹⁵ proprioceptive,⁵⁵ or simplified single-movement^{34,133} tasks. The influence of modifying the visual input by any means (ball, defender, blindfold, target, visual signals) during more challenging dynamic tasks, such as rapid direction change or jump landing, has an even greater effect on neuromuscular control.^{34,36,52,133,152}

Direct Visual Disruption

Ideally, inhibiting visual input during these dynamic, more athletic maneuvers would provide a means to directly address the compensatory neuroplastic sequelae after ACL injury and train the neuromuscular system in a functional manner. A recent technological innovation has made this possible by decreasing visual input without fully removing it.^{21,22} This tool, stroboscopic eyewear (eg, PLATO Visual Occlusion Spectacles [Translucent Technologies Inc, Toronto, Canada], Nike SPARQ Vapor Strobes [Nike Inc, Beaverton, OR], PRIMARY Strobe Glasses [Appreciate Co, Ltd, Kyoto, Japan]) (FIGURE 3), has the ability to partially obstruct vision by intermittently switching from clear to opaque, allowing highly complex, dynamic athletic maneuvers to be performed under degraded visual input (FIGURE 4).^{6,21,22,106} Practice with a stroboscopic vision system has already been shown to enhance aspects of basic visual cognition, such as tran-

Exercise

Single-leg hop with air target, unstable landing and reaction ball

Stand on 1 leg, jump forward, contact air target in flight with opposite hand of jump-landing leg; on landing, catch ball. Shown with Vertec target at 50% of maximum jump height



Broad jump with reaction ball and unanticipated cut

Stand on both feet, jump out, land on both feet, and track the ball to determine cut direction. Progress to decrease anticipation time from landing to ball release and cut direction



Single leg, air target, and reaction ball

Stand on both feet with knees slightly bent, then jump and rotate 180° while in the air. The opposite hand of the landing foot reaches out to contact a target. When landing, catch the ball. Shown with Vertec target at 50% of maximum jump height



FIGURE 4. Examples of higher-level, dynamic neuromuscular training exercises incorporating visual target acquisition, environmental interaction, anticipatory ability, unstable surfaces, and stroboscopic visual interference, using stroboscopic glasses.

sient attention,⁷ anticipatory trajectory estimation,¹³⁸ and short-term memory.⁶ These abilities may play a role in mitigating or avoiding injurious collisions or situations via improved anticipation and processing speed,⁶³ which in turn may modify ACL-injury risk.¹⁴¹ Training with intermittent visual input also offers a simple, easy-to-implement, and novel stress to the neural control system that is compatible with current neuromuscular training exercises. The disrupted visual feedback may more closely simulate the neurocognitive demands of activity in the safety of a controlled clinic or field environment under the supervision of a qualified professional. Such stroboscopic visual training can also be tailored to fit a desired difficulty level by altering the rate of stroboscopic interruption (ratio of opaque versus transparent status).

This adjustability is an important feature because postinjury, visual interference could increase reinjury risk during rehabilitation, particularly if the athlete has not yet adapted to the depressed visual feedback. The ability to scale the level of interference up or down provides a means for the clinician to progress the patient, based on clinical judgment. Also, a warm-up period is recommended to allow patients to familiarize themselves with the visual effect by doing less aggressive movements, such as single-leg balance or upper extremity exercises (ball toss), before advancing to jump-landing or direction-change tasks. The eyewear is also wireless and portable, making for flexible implementation in a wide assortment of clinics or on-field progressions of already-established neuromuscular training exercises.¹⁰⁷

After injury, the CNS experiences a compensatory overutilization of visual feedback to maintain neuromuscular control. The suggested intermittent visual training can decrease the available visual feedback to the CNS. This may force the CNS to engage in an adaptive strategy by increased weighting of the remaining proprioceptive inputs, as opposed to continuing to compensate with visual feedback (FIGURE 2). The neural mechanisms underlying this sensory visual-motor interaction are theoretical at this point, but may include increased utilization or efficiency of the remaining proprioceptive or vestibular inputs, and/or improved visual-motor processing efficiency to make up for the increased demand. Alternatively, intermittent visual training could lead to increased attentional focus⁷⁰ and/or changes in the rate of memory consoli-

dation⁶ that, in turn, improve the use of afferent information for guiding motor control. Regardless of the mechanism, this training may improve the transition to athletic activity by decreasing dependence on vision to maintain dynamic motor control, allowing its use for environmental interaction on return to the complex athletic environment.

Indirect Visual Distraction

Other techniques to modify visual feedback outside of stroboscopic eyewear may be more accessible and still provide a means to encourage adaptive neuroplasticity. Progressively increasing the difficulty of the sensorimotor challenge can not only facilitate neuroplasticity for motor control, but also improve sensory integration and address the visual processing bias. The key considerations to completing the latter are the focus of attention, task complexity, visual input, and cognitive load during rehabilitation.^{29,126} Many mechanisms, such as incorporating reaction-time components,¹²⁶ ball tracking, engaging other players,¹⁰² adding decision-making²⁹ or anticipatory aspects,¹²⁶ and having the patient dual task¹¹² by engaging the upper extremity while performing lower extremity exercises or occupying the mind with memory or related tasks, can increase the neural demand of neuromuscular training strategies. Recently, Negahban et al¹¹² used a classic dual-task paradigm by having individuals post-ACL reconstruction maintain single-leg postural control while performing a cognitive task (holding a string of numbers in mind). This additional demand degraded postural stability in the ACL cohort but had little effect in control participants. This builds on previous work that has established that adding environmental interactions, such as a target, another player, a ball, or decision making, has greater influence in those with ACL injury.^{42,73,151} Consequently, strategies to address these performance deficits should be incorporated in neuromuscular training targeting the transition from clinic to activity.

Visual-Motor Training

Stroboscopic training, dual tasking using environmental interaction or adding visual obstruction, and facilitating motor learning are methods to decrease visual feedback during established exercises to make vision a less salient form of information for motor programming. These strategies may stimulate the CNS to re-weight information from the somatosensory and vestibular inputs to decrease excessive reliance on visual feedback. An alternative to reducing visual feedback dependence is to make the visual processing system more efficient and able to handle the increased demand. The injury may only allow so much sensory adaptation, and a degree of increased visual feedback may have to be regulated to maintain neuromuscular integrity during action, regardless of how much we attempt to force proprioceptor or vestibular upregulation. In this case, visual training in isolation or in combination with neuromuscular training methods may provide a means to further address the compensatory neuroplasticity following injury. Visual training has been shown to enhance sport performance^{35,106} and improve reaction time and visual processing ability.^{6,7,138} Simply increasing these fundamental neurocognitive attributes may allow the athlete to handle the dual task of maintaining knee control while interacting with the environment and responding to potentially injurious situations.

Many methods for training the visual system exist, and each one tends to focus on a different visual construct. These constructs include oculomotor control, multiple-object tracking, visual sensitivity, spatial attention, visual memory, reaction time, and processing speed. Several commercial tools exist that target 1 or more of these visual processing attributes. These tools range in cost and approach, from high-level computer-based systems, such as CogniSens (CogniSens Inc, Montreal, Canada), Dynavision (Dynavision International LLC, West Chester, OH), and the SPARQ Sensory Training Station

(Nike, Inc), to web-based applications, to simple paper or object manipulation.^{35,47} Software or full electronic station setups are ideal and the literature supports their ability to increase performance metrics in athletes, but the clinician may not have access to this type of technology. Alternative mechanisms to train the visual system with minimal equipment include the use of a tachistoscope (flash cards of objects/numbers/letters of increasing value that must be attended to and recalled) to improve object recognition in the visual field, a Brock string (string of colored balls at different distances held to the face; the participant must focus on each one in sequence) to improve oculomotor muscle capacity to focus on targets rapidly, and saccades (charts of random letters on a wall; the player must focus on each one and call out letters in sequence) to improve rapid visual processing.

Motor Learning Applications

The increased level of cortical drive during movement, seen in individuals with an ACL injury, provides a neurological mechanism^{18,65,79} to explain the greater amount of cocontraction and muscle-guarding strategies seen after injury.¹⁴⁶ Such a neuromuscular control strategy is consistent with an increase in internal focus of control, likely due to the increased conscious awareness of the injured joint and its movement as opposed to attention to the external environment.^{58,155} Rehabilitation guidelines that focus on explicit feedback (eg, contract quadriceps or keep knees over your toes) might be further promoting the top-down cortical and visual feedback control of the movement, as opposed to facilitating a return to a more autonomic somatosensory feedback control and visual feedback on the environment. As discussed earlier, the increased activation of the presupplementary motor area to perform a simple knee joint movement⁷⁹ further demonstrates the increased need for cortical motor planning of movement after ACL injury. The implications of these findings are concerning for return to sport, as the

demands of complex, more dynamic motion may exceed the capability of the region to program optimal movement and may contribute to injury risk.

The classic mechanism to rectify increased reliance on cortical mechanisms for lower extremity control is to advance patients to the autonomous stage of motor learning.⁹⁵ In early-stage rehabilitation, explicit focus is needed to restore muscle function, and internally focused feedback such as “contract your quad,” “knees over the toes,” or “bend your knee” is commonly used. Advancing rehabilitative feedback to an external focus, such as “land on the markers” or “touch the target as you land,” will facilitate transfer of motor control to subcortical regions and free cortical resources for programming more complex motor actions.^{58,127,137} Such motor learning principles applied to neuromuscular training may assist in transferring knee control strategies to the athletic field when conscious attention is being paid to the environment and not knee position.^{127,136} The additions to neuromuscular training previously discussed can also help speed the process of acquiring the ability to transfer motor skills to the field.

Limitations

The framework described above provides an opportunity to develop hypothesis-driven clinical and research constructs for further exploration. Prior to steady clinical recommendations, rigorous longitudinal and controlled trials should be undertaken; however, exploration of novel neuromuscular re-education techniques may provide immediate enhancement to current rehabilitation and prevention methods. We have suggested some methods to address the postinjury neuroplasticity during the rehabilitation process, and, undoubtedly, clinicians and researchers will develop more novel and applicable methods in the near future. There is insufficient evidence to recommend one method or system over any other at this time, but we encourage clinicians to consider visual-motor function on any level as a part of ACL rehabilita-

tion and injury prevention programs. Implementation can be as basic as adding an eyes-closed or a cognitive dual task during quadriceps contraction sets as the patient progresses toward the autonomous stage in that exercise during the first few clinic visits, or including environmental stimuli (other players, unanticipated direction changes, target acquisition, or reaction ball) during functional tasks later in rehabilitation, and can advance to highly complex virtual reality simulations that are increasing in quality and accessibility.^{43,51} Collectively, these vision-based interventions are gaining widespread use in a number of clinical (eg, concussion, cognitive disorders) and nonclinical (eg, entertainment, performance enhancement, military) applications, and future integration with musculoskeletal injury rehabilitation may create an entirely new avenue for improving neuromuscular function to prevent and treat orthopaedic injuries.

Visual-Motor Training in Primary ACL Injury Prevention

The presented evidence suggests that ACL injury can alter the nervous system utilization of somatosensory input, afferent integration, and motor output. These neuroplastic effects induce a neuromuscular control strategy that increases dependence on visual feedback to regulate dynamic stability of the system. However, some of the described postinjury adaptations may actually be present prior to injury, potentially playing a role in primary ACL risk. Swanik et al¹⁴¹ reported initial findings of decreased visual processing capabilities in individuals prior to ACL injury. Using neurocognitive testing, they found that a decrease in visual processing speed and reaction time was predictive of subsequent ACL injury.¹⁴¹ A theorized mechanism for visual function influencing injury risk is in the ability to prepare the neuromuscular system in anticipation of high-risk situations, maneuvers, or incoming players.^{63,105} If visual processing resources are taxed to maintain the afferent input for knee mo-

tor control, this may decrease the ability to compensate for environmental stimuli and attenuate unanticipated maneuvers, such as cutting or landing, that depend on quick visual processing.^{31,72,102} Future studies should investigate the potential association of varying visual processing capabilities and sensorimotor neural integration with musculoskeletal risk, in addition to investigating visual feedback modification or visual-motor additions to neuromuscular training in relation to injury prevention.

CONCLUSION

THIS REVIEW HIGHLIGHTS A CONCEPTUAL framework for integrating a variety of visual-motor constructs during neuromuscular rehabilitation as a future avenue of research to optimize musculoskeletal therapy interventions. A strength of these recommendations is that they act as adjunct strategies to foundational neuromuscular techniques for optimizing strength, multiplanar knee and trunk control, and movement asymmetries.⁴⁵ These suggestions provide an opportunity to supplement more traditional interventions by further targeting neuroplastic, cognitive, and visual-motor capabilities. The clinician can approximate the neurocognitive demands of higher-intensity athletic activity in a safe, controlled, and—most importantly—feedback-rich environment under the supervision of a well-trained professional before reintegration into sport. Recognition of the visual-motor implications of neuromuscular control and injury recovery and prevention, combined with new technologies and approaches, may help to mitigate postinjury movement dysfunction and decrease injury risk when returning to activity. ●

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REFERENCES

- Adachi N, Ochi M, Uchio Y, Iwasa J, Ryoike K, Kuriwaka M. Mechanoreceptors in the anterior cruciate ligament contribute to the joint position sense. *Acta Orthop Scand*. 2002;73:330-334. <http://dx.doi.org/10.1080/000164702320155356>
- Ageberg E, Fridén T. Normalized motor function but impaired sensory function after unilateral non-reconstructed ACL injury: patients compared with uninjured controls. *Knee Surg Sports Traumatol Arthrosc*. 2008;16:449-456. <http://dx.doi.org/10.1007/s00167-008-0499-9>
- Ageberg E, Roberts D, Holmström E, Fridén T. Balance in single-limb stance in patients with anterior cruciate ligament injury: relation to knee laxity, proprioception, muscle strength, and subjective function. *Am J Sports Med*. 2005;33:1527-1535. <http://dx.doi.org/10.1177/0363546505274934>
- Ajuied A, Wong F, Smith C, et al. Anterior cruciate ligament injury and radiologic progression of knee osteoarthritis: a systematic review and meta-analysis. *Am J Sports Med*. 2014;42:2242-2252. <http://dx.doi.org/10.1177/0363546513508376>
- Amador N, Fried I. Single-neuron activity in the human supplementary motor area underlying preparation for action. *J Neurosurg*. 2004;100:250-259. <http://dx.doi.org/10.3171/jns.2004.100.2.0250>
- Appelbaum LG, Cain MS, Schroeder JE, Darling EF, Mitroff SR. Stroboscopic visual training improves information encoding in short-term memory. *Atten Percept Psychophys*. 2012;74:1681-1691. <http://dx.doi.org/10.3758/s13414-012-0344-6>
- Appelbaum LG, Schroeder JE, Cain MS, Mitroff SR. Improved visual cognition through stroboscopic training. *Front Psychol*. 2011;2:276. <http://dx.doi.org/10.3389/fpsyg.2011.00276>
- Ardern CL, Taylor NF, Feller JA, Webster KE. Return-to-sport outcomes at 2 to 7 years after anterior cruciate ligament reconstruction surgery. *Am J Sports Med*. 2012;40:41-48. <http://dx.doi.org/10.1177/0363546511422999>
- Ardern CL, Taylor NF, Feller JA, Whitehead TS, Webster KE. Psychological responses matter in returning to preinjury level of sport after anterior cruciate ligament reconstruction surgery. *Am J Sports Med*. 2013;41:1549-1558. <http://dx.doi.org/10.1177/0363546513489284>
- Ardern CL, Webster KE, Taylor NF, Feller JA. Return to the preinjury level of competitive sport after anterior cruciate ligament reconstruction surgery: two-thirds of patients have not returned by 12 months after surgery. *Am J Sports Med*. 2011;39:538-543. <http://dx.doi.org/10.1177/0363546510384798>
- Babiloni C, Marzano N, Infarinato F, et al. "Neural efficiency" of experts' brain during judgment of actions: a high-resolution EEG study in elite and amateur karate athletes. *Behav Brain Res*. 2010;207:466-475. <http://dx.doi.org/10.1016/j.bbr.2009.10.034>
- Ball T, Schreiber A, Feige B, Wagner M, Lucking CH, Kristeva-Feige R. The role of higher-order motor areas in voluntary movement as revealed by high-resolution EEG and fMRI. *Neuroimage*. 1999;10:682-694. <http://dx.doi.org/10.1006/nimg.1999.0507>
- Baltaci G, Harput G, Haksever B, Ulusoy B, Ozer H. Comparison between Nintendo Wii Fit and conventional rehabilitation on functional performance outcomes after hamstring anterior cruciate ligament reconstruction: prospective, randomized, controlled, double-blind clinical trial. *Knee Surg Sports Traumatol Arthrosc*. 2013;21:880-887. <http://dx.doi.org/10.1007/s00167-012-2034-2>
- Barenius B, Ponzer S, Shalabi A, Bujak R, Norlén L, Eriksson K. Increased risk of osteoarthritis after ACL reconstruction – a 14-year follow-up study of a randomized controlled trial. *Arthroscopy*. 2013;29:e80-e81. <http://dx.doi.org/10.1016/j.arthro.2013.07.080>
- Bates NA, Ford KR, Myer GD, Hewett TE. Impact differences in ground reaction force and center of mass between the first and second landing phases of a drop vertical jump and their implications for injury risk assessment. *J Biomech*. 2013;46:1237-1241. <http://dx.doi.org/10.1016/j.jbiomech.2013.02.024>
- Baumeister J, Reinecke K, Liesen H, Weiss M. Cortical activity of skilled performance in a complex sports related motor task. *Eur J Appl Physiol*. 2008;104:625-631. <http://dx.doi.org/10.1007/s00421-008-0811-x>
- Baumeister J, Reinecke K, Schubert M, Weiss M. Altered electrocortical brain activity after ACL reconstruction during force control. *J Orthop Res*. 2011;29:1383-1389. <http://dx.doi.org/10.1002/jor.21380>
- Baumeister J, Reinecke K, Weiss M. Changed cortical activity after anterior cruciate ligament reconstruction in a joint position paradigm: an EEG study. *Scand J Med Sci Sports*. 2008;18:473-484. <http://dx.doi.org/10.1111/j.1600-0838.2007.00702.x>
- Beck S, Taube W, Gruber M, Amtage F, Gollhofer A, Schubert M. Task-specific changes in motor evoked potentials of lower limb muscles after different training interventions. *Brain Res*. 2007;1179:51-60. <http://dx.doi.org/10.1016/j.brainres.2007.08.048>
- Benjaminse A, Otten E. ACL injury prevention, more effective with a different way of motor learning? *Knee Surg Sports Traumatol Arthrosc*. 2011;19:622-627. <http://dx.doi.org/10.1007/s00167-010-1313-z>
- Bennett S, Ashford D, Rioja N, Elliott D. Intermittent vision and one-handed catching: the effect of general and specific task experience. *J Mot Behav*. 2004;36:442-449. <http://dx.doi.org/10.3200/JMBR.36.4.442-449>
- Bennett SJ, Elliott D, Weeks DJ, Keil D. The effects of intermittent vision on prehension under binocular and monocular viewing. *Motor Control*. 2003;7:46-56.
- Berthoz A, Lacour M, Soechting JF, Vidal PP. The role of vision in the control of posture during linear motion. *Prog Brain Res*. 1979;50:197-209. [http://dx.doi.org/10.1016/S0079-6123\(08\)60820-1](http://dx.doi.org/10.1016/S0079-6123(08)60820-1)
- Biedert RM, Zwick EB. Ligament-muscle reflex arc after anterior cruciate ligament reconstruction: electromyographic evaluation. *Arch Orthop Trauma Surg*. 1998;118:81-84.
- Binder JR, Desai RH. The neurobiology of semantic memory. *Trends Cogn Sci*. 2011;15:527-536. <http://dx.doi.org/10.1016/j.tics.2011.10.001>
- Boden BP, Torg JS, Knowles SB, Hewett TE. Video analysis of anterior cruciate ligament injury: abnormalities in hip and ankle kinematics. *Am J Sports Med*. 2009;37:252-259. <http://dx.doi.org/10.1177/0363546508328107>
- Bonfim TR, Jansen Paccola CA, Barela JA. Proprioceptive and behavior impairments in individuals with anterior cruciate ligament reconstructed knees. *Arch Phys Med Rehabil*. 2003;84:1217-1223.
- Bonner MF, Price AR. Where is the anterior temporal lobe and what does it do? *J Neurosci*. 2013;33:4213-4215. <http://dx.doi.org/10.1523/JNEUROSCI.0041-13.2013>
- Borotikar BS, Newcomer R, Koppes R, McLean SG. Combined effects of fatigue and decision making on female lower limb landing postures: central and peripheral contributions to ACL injury risk. *Clin Biomech (Bristol, Avon)*. 2008;23:81-92. <http://dx.doi.org/10.1016/j.clinbiomech.2007.08.008>
- Boudreau SA, Farina D, Falla D. The role of motor learning and neuroplasticity in designing rehabilitation approaches for musculoskeletal pain disorders. *Man Ther*. 2010;15:410-414. <http://dx.doi.org/10.1016/j.math.2010.05.008>
- Brown TN, Palmieri-Smith RM, McLean SG. Sex and limb differences in hip and knee kinematics and kinetics during anticipated and unanticipated jump landings: implications for anterior cruciate ligament injury. *Br J Sports Med*. 2009;43:1049-1056. <http://dx.doi.org/10.1136/bjsm.2008.055954>
- Brumagne S, Cordo P, Verschueren S. Proprioceptive weighting changes in persons with low back pain and elderly persons during upright standing. *Neurosci Lett*. 2004;366:63-66. <http://dx.doi.org/10.1016/j.neulet.2004.05.013>
- Chen TL, Babiloni C, Ferretti A, et al. Human secondary somatosensory cortex is involved in the processing of somatosensory rare stimuli: an fMRI study. *Neuroimage*. 2008;40:1765-1771. <http://dx.doi.org/10.1016/j.neuroimage.2008.01.020>
- Chu Y, Sell TC, Abt JP, et al. Air assault soldiers demonstrate more dangerous landing biomechanics when visual input is removed. *Mil Med*. 2012;177:41-47.
- Clark JF, Ellis JK, Bench J, Khoury J, Graman P.

- High-performance vision training improves batting statistics for University of Cincinnati baseball players. *PLoS One*. 2012;7:e29109. <http://dx.doi.org/10.1371/journal.pone.0029109>
36. Cortes N, Blount E, Ringleb S, Onate JA. Soccer-specific video simulation for improving movement assessment. *Sports Biomech*. 2011;10:22-34. <http://dx.doi.org/10.1080/14763141.2010.547591>
 37. Courtney C, Rine RM, Kroll P. Central somatosensory changes and altered muscle synergies in subjects with anterior cruciate ligament deficiency. *Gait Posture*. 2005;22:69-74. <http://dx.doi.org/10.1016/j.gaitpost.2004.07.002>
 38. Courtney CA, Durr RK, Emerson-Kavchak AJ, Witte EO, Santos MJ. Heightened flexor withdrawal responses following ACL rupture are enhanced by passive tibial translation. *Clin Neurophysiol*. 2011;122:1005-1010. <http://dx.doi.org/10.1016/j.clinph.2010.07.029>
 39. Courtney CA, Rine RM. Central somatosensory changes associated with improved dynamic balance in subjects with anterior cruciate ligament deficiency. *Gait Posture*. 2006;24:190-195. <http://dx.doi.org/10.1016/j.gaitpost.2005.08.006>
 40. Dault MC, Frank JS, Allard F. Influence of a visuo-spatial, verbal and central executive working memory task on postural control. *Gait Posture*. 2001;14:110-116.
 41. Del Percio C, Babiloni C, Marzano N, et al. "Neural efficiency" of athletes' brain for upright standing: a high-resolution EEG study. *Brain Res Bull*. 2009;79:193-200. <http://dx.doi.org/10.1016/j.brainresbull.2009.02.001>
 42. Demont RG, Lephart SM, Giraldo JL, Swanik CB, Fu FH. Muscle preactivity of anterior cruciate ligament-deficient and -reconstructed females during functional activities. *J Athl Train*. 1999;34:115-120.
 43. Deutsch JE. Virtual reality and gaming systems to improve walking and mobility for people with musculoskeletal and neuromuscular conditions. *Stud Health Technol Inform*. 2009;145:84-93.
 44. Di Fabio RP, Graf B, Badke MB, Breunig A, Jensen K. Effect of knee joint laxity on long-loop postural reflexes: evidence for a human capsular-hamstring reflex. *Exp Brain Res*. 1992;90:189-200.
 45. Di Stasi S, Myer GD, Hewett TE. Neuromuscular training to target deficits associated with second anterior cruciate ligament injury. *J Orthop Sports Phys Ther*. 2013;43:777-792. <http://dx.doi.org/10.2519/jospt.2013.4693>
 46. Emanuel M, Jarus T, Bart O. Effect of focus of attention and age on motor acquisition, retention, and transfer: a randomized trial. *Phys Ther*. 2008;88:251-260. <http://dx.doi.org/10.2522/ptj.20060174>
 47. Erickson GB, Citek K, Cove M, et al. Reliability of a computer-based system for measuring visual performance skills. *Optometry*. 2011;82:528-542. <http://dx.doi.org/10.1016/j.optm.2011.01.012>
 48. Ernst GP, Saliba E, Diduch DR, Hurwitz SR, Ball DW. Lower extremity compensations following anterior cruciate ligament reconstruction. *Phys Ther*. 2000;80:251-260.
 49. Ferretti A, Babiloni C, Gratta CD, et al. Functional topography of the secondary somatosensory cortex for nonpainful and painful stimuli: an fMRI study. *Neuroimage*. 2003;20:1625-1638.
 50. Flor H, Braun C, Elbert T, Birbaumer N. Extensive reorganization of primary somatosensory cortex in chronic back pain patients. *Neurosci Lett*. 1997;224:5-8.
 51. Fluet GG, Deutsch JE. Virtual reality for sensorimotor rehabilitation post-stroke: the promise and current state of the field. *Curr Phys Med Rehabil Rep*. 2013;1:9-20. <http://dx.doi.org/10.1007/s40141-013-0005-2>
 52. Ford KR, Myer GD, Smith RL, Byrnes RN, Dopirak SE, Hewett TE. Use of an overhead goal alters vertical jump performance and biomechanics. *J Strength Cond Res*. 2005;19:394-399. <http://dx.doi.org/10.1519/15834.1>
 53. Fremerey RW, Lobenhoffer P, Zeichen J, Skutek M, Bosch U, Tscherner H. Proprioception after rehabilitation and reconstruction in knees with deficiency of the anterior cruciate ligament: a prospective, longitudinal study. *J Bone Joint Surg Br*. 2000;82:801-806.
 54. Fridén T, Roberts D, Ageberg E, Waldén M, Zätterström R. Review of knee proprioception and the relation to extremity function after an anterior cruciate ligament rupture. *J Orthop Sports Phys Ther*. 2001;31:567-576. <http://dx.doi.org/10.2519/jospt.2001.31.10.567>
 55. Fridén T, Roberts D, Movin T, Wredmark T. Function after anterior cruciate ligament injuries. Influence of visual control and proprioception. *Acta Orthop Scand*. 1998;69:590-594.
 56. Goerger BM, Marshall SW, Beutler AI, Blackburn JT, Wilckens JH, Padua DA. ACL injury alters pre-injury coordination of the hip and knee: the JUMP-ACL study [abstract]. *Med Sci Sports Exerc*. 2013;45:221.
 57. Gokeler A, Benjaminse A, Hewett TE, et al. Proprioceptive deficits after ACL injury: are they clinically relevant? *Br J Sports Med*. 2012;46:180-192. <http://dx.doi.org/10.1136/bjsm.2010.082578>
 58. Gokeler A, Benjaminse A, Hewett TE, et al. Feedback techniques to target functional deficits following anterior cruciate ligament reconstruction: implications for motor control and reduction of second injury risk. *Sports Med*. 2013;43:1065-1074. <http://dx.doi.org/10.1007/s40279-013-0095-0>
 59. Gómez-Barrena E, Martínez-Moreno E, Munuera L. Segmental sensory innervation of the anterior cruciate ligament and the patellar tendon of the cat's knee. *Acta Orthop Scand*. 1996;67:545-552.
 60. Grindstaff TL, Hammill RR, Tuzson AE, Hertel J. Neuromuscular control training programs and noncontact anterior cruciate ligament injury rates in female athletes: a numbers-needed-to-treat analysis. *J Athl Train*. 2006;41:450-456.
 61. Hall KG, Magill RA. Variability of practice and contextual interference in motor skill learning. *J Mot Behav*. 1995;27:299-309. <http://dx.doi.org/10.1080/00222895.1995.9941719>
 62. Hamzei F, Liepert J, Dettmers C, Weiller C, Rijntjes M. Two different reorganization patterns after rehabilitative therapy: an exploratory study with fMRI and TMS. *Neuroimage*. 2006;31:710-720. <http://dx.doi.org/10.1016/j.neuroimage.2005.12.035>
 63. Harpham JA, Mihalik JP, Littleton AC, Frank BS, Guskiewicz KM. The effect of visual and sensory performance on head impact biomechanics in college football players. *Ann Biomed Eng*. 2014;42:1-10. <http://dx.doi.org/10.1007/s10439-013-0881-8>
 64. Herman DC, Weinhold PS, Guskiewicz KM, Garrett WE, Yu B, Padua DA. The effects of strength training on the lower extremity biomechanics of female recreational athletes during a stop-jump task. *Am J Sports Med*. 2008;36:733-740. <http://dx.doi.org/10.1177/0363546507311602>
 65. Heroux ME, Tremblay F. Corticomotor excitability associated with unilateral knee dysfunction secondary to anterior cruciate ligament injury. *Knee Surg Sports Traumatol Arthrosc*. 2006;14:823-833. <http://dx.doi.org/10.1007/s00167-006-0063-4>
 66. Heroux ME, Tremblay F. Weight discrimination after anterior cruciate ligament injury: a pilot study. *Arch Phys Med Rehabil*. 2005;86:1362-1368.
 67. Hewett TE, Myer GD, Ford KR, et al. Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes: a prospective study. *Am J Sports Med*. 2005;33:492-501. <http://dx.doi.org/10.1177/0363546504269591>
 68. Hewett TE, Torg JS, Boden BP. Video analysis of trunk and knee motion during non-contact anterior cruciate ligament injury in female athletes: lateral trunk and knee abduction motion are combined components of the injury mechanism. *Br J Sports Med*. 2009;43:417-422. <http://dx.doi.org/10.1136/bjsm.2009.059162>
 69. Hoffman M, Schrader J, Koceja D. An investigation of postural control in postoperative anterior cruciate ligament reconstruction patients. *J Athl Train*. 1999;34:130-136.
 70. Holliday J. Effect of Stroboscopic Vision Training on Dynamic Visual Acuity Scores: Nike Vapor Strobe® Eyewear. Logan, UT: Utah State University; 2013.
 71. Hootman JM, Dick R, Agel J. Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. *J Athl Train*. 2007;42:311-319.
 72. Houck JR, De Haven KE, Maloney M. Influence of anticipation on movement patterns in subjects with ACL deficiency classified as noncopers. *J Orthop Sports Phys Ther*. 2007;37:56-64. <http://dx.doi.org/10.2519/jospt.2007.2292>

73. Houck JR, Wilding GE, Gupta R, De Haven KE, Maloney M. Analysis of EMG patterns of control subjects and subjects with ACL deficiency during an unanticipated walking cut task. *Gait Posture*. 2007;25:628-638. <http://dx.doi.org/10.1016/j.gaitpost.2006.07.001>
74. Hui C, Salmon LJ, Kok A, Maeno S, Linklater J, Pinczewski LA. Fifteen-year outcome of endoscopic anterior cruciate ligament reconstruction with patellar tendon autograft for "isolated" anterior cruciate ligament tear. *Am J Sports Med*. 2011;39:89-98. <http://dx.doi.org/10.1177/0363546510379975>
75. Im HJ, Kim JS, Li X, et al. Alteration of sensory neurons and spinal response to an experimental osteoarthritis pain model. *Arthritis Rheum*. 2010;62:2995-3005. <http://dx.doi.org/10.1002/art.27608>
76. Jarus T, Gutman T. Effects of cognitive processes and task complexity on acquisition, retention, and transfer of motor skills. *Can J Occup Ther*. 2001;68:280-289.
77. Jensen JL, Marstrand PC, Nielsen JB. Motor skill training and strength training are associated with different plastic changes in the central nervous system. *J Appl Physiol* (1985). 2005;99:1558-1568. <http://dx.doi.org/10.1152/jappphysiol.01408.2004>
78. Kapreli E, Athanasopoulos S. The anterior cruciate ligament deficiency as a model of brain plasticity. *Med Hypotheses*. 2006;67:645-650. <http://dx.doi.org/10.1016/j.mehy.2006.01.063>
79. Kapreli E, Athanasopoulos S, Gliatis J, et al. Anterior cruciate ligament deficiency causes brain plasticity: a functional MRI study. *Am J Sports Med*. 2009;37:2419-2426. <http://dx.doi.org/10.1177/0363546509343201>
80. Kennedy JC, Alexander IJ, Hayes KC. Nerve supply of the human knee and its functional importance. *Am J Sports Med*. 1982;10:329-335.
81. Knecht S, Henningsen H, Elbert T, et al. Cortical reorganization in human amputees and mislocalization of painful stimuli to the phantom limb. *Neurosci Lett*. 1995;201:262-264.
82. Koga H, Nakamae A, Shima Y, et al. Mechanisms for noncontact anterior cruciate ligament injuries: knee joint kinematics in 10 injury situations from female team handball and basketball. *Am J Sports Med*. 2010;38:2218-2225. <http://dx.doi.org/10.1177/0363546510373570>
83. Konishi Y, Aihara Y, Sakai M, Ogawa G, Fukubayashi T. Gamma loop dysfunction in the quadriceps femoris of patients who underwent anterior cruciate ligament reconstruction remains bilaterally. *Scand J Med Sci Sports*. 2007;17:393-399. <http://dx.doi.org/10.1111/j.1600-0838.2006.00573.x>
84. Konishi Y, Fukubayashi T, Takeshita D. Mechanism of quadriceps femoris muscle weakness in patients with anterior cruciate ligament reconstruction. *Scand J Med Sci Sports*. 2002;12:371-375.
85. Konishi Y, Fukubayashi T, Takeshita D. Possible mechanism of quadriceps femoris weakness in patients with ruptured anterior cruciate ligament. *Med Sci Sports Exerc*. 2002;34:1414-1418.
86. Konishi Y, Konishi H, Fukubayashi T. Gamma loop dysfunction in quadriceps on the contralateral side in patients with ruptured ACL. *Med Sci Sports Exerc*. 2003;35:897-900. <http://dx.doi.org/10.1249/01.MSS.0000069754.07541.D2>
87. Konishi YU. ACL repair might induce further abnormality of gamma loop in the intact side of the quadriceps femoris. *Int J Sports Med*. 2011;32:292-296. <http://dx.doi.org/10.1055/s-0030-1270488>
88. Krosshaug T, Nakamae A, Boden BP, et al. Mechanisms of anterior cruciate ligament injury in basketball: video analysis of 39 cases. *Am J Sports Med*. 2007;35:359-367. <http://dx.doi.org/10.1177/0363546506293899>
89. Lajoie Y, Teasdale N, Bard C, Fleury M. Attentional demands for static and dynamic equilibrium. *Exp Brain Res*. 1993;97:139-144.
90. Lepley AS, Bahhur NO, Murray AM, Pietro-simone BG. Quadriceps corticomotor excitability following an experimental knee joint effusion. *Knee Surg Sports Traumatol Arthrosc*. 2015;23:1010-1017. <http://dx.doi.org/10.1007/s00167-013-2816-1>
91. Levine JD, Dardick SJ, Basbaum AI, Scipio E. Reflex neurogenic inflammation. I. Contribution of the peripheral nervous system to spatially remote inflammatory responses that follow injury. *J Neurosci*. 1985;5:1380-1386.
92. Littmann AE, Iguchi M, Madhavan S, Kolarik JL, Shields RK. Dynamic-position-sense impairment's independence of perceived knee function in women with ACL reconstruction. *J Sport Rehabil*. 2012;21:44-53.
93. Lohmander LS, Englund PM, Dahl LL, Roos EM. The long-term consequence of anterior cruciate ligament and meniscus injuries: osteoarthritis. *Am J Sports Med*. 2007;35:1756-1769. <http://dx.doi.org/10.1177/0363546507307396>
94. Madhavan S, Shields RK. Neuromuscular responses in individuals with anterior cruciate ligament repair. *Clin Neurophysiol*. 2011;122:997-1004. <http://dx.doi.org/10.1016/j.clinph.2010.09.002>
95. Magill R. *Motor Learning and Control: Concepts and Applications*. 8th ed. Boston, MA: WCB/McGraw-Hill; 2007.
96. Mather RC, 3rd, Koenig L, Kocher MS, et al. Societal and economic impact of anterior cruciate ligament tears. *J Bone Joint Surg Am*. 2013;95:1751-1759. <http://dx.doi.org/10.2106/JBJS.L.01705>
97. Mattacola CG, Perrin DH, Gansnedner BM, Gieck JH, Saliba EN, McCue FC, 3rd. Strength, functional outcome, and postural stability after anterior cruciate ligament reconstruction. *J Athl Train*. 2002;37:262-268.
98. McCormick BT. Task complexity and jump landings in injury prevention for basketball players. *Strength Cond J*. 2012;34:89-92. <http://dx.doi.org/10.1519/SSC.0b013e31823ee08e>
99. McLean SG. The ACL injury enigma: we can't prevent what we don't understand. *J Athl Train*. 2008;43:538-540. <http://dx.doi.org/10.4085/1062-6050-43.5.538>
100. McLean SG, Borotikar B, Lucey SM. Lower limb muscle pre-motor time measures during a choice reaction task associate with knee abduction loads during dynamic single leg landings. *Clin Biomech (Bristol, Avon)*. 2010;25:563-569. <http://dx.doi.org/10.1016/j.clinbiomech.2010.02.013>
101. McLean SG, Huang X, van den Bogert AJ. Association between lower extremity posture at contact and peak knee valgus moment during sidestepping: implications for ACL injury. *Clin Biomech (Bristol, Avon)*. 2005;20:863-870. <http://dx.doi.org/10.1016/j.clinbiomech.2005.05.007>
102. McLean SG, Lipfert SW, van den Bogert AJ. Effect of gender and defensive opponent on the biomechanics of sidestep cutting. *Med Sci Sports Exerc*. 2004;36:1008-1016.
103. McLean SG, Neal RJ, Myers PT, Walters MR. Knee joint kinematics during the sidestep cutting maneuver: potential for injury in women. *Med Sci Sports Exerc*. 1999;31:959-968.
104. Meister I, Krings T, Foltys H, et al. Effects of long-term practice and task complexity in musicians and nonmusicians performing simple and complex motor tasks: implications for cortical motor organization. *Hum Brain Mapp*. 2005;25:345-352. <http://dx.doi.org/10.1002/hbm.20112>
105. Mihalik JP, Blackburn JT, Greenwald RM, Cantu RC, Marshall SW, Guskiewicz KM. Collision type and player anticipation affect head impact severity among youth ice hockey players. *Pediatrics*. 2010;125:e1394-e1401. <http://dx.doi.org/10.1542/peds.2009-2849>
106. Mitroff SR, Friesen P, Bennett D, Yoo H, Reichow AW. Enhancing ice hockey skills through stroboscopic visual training: a pilot study. *Athl Train Sports Health Care*. 2013;5:261-264. <http://dx.doi.org/10.3928/19425864-20131030-02>
107. Myer GD, Ford KR, Brent JL, Hewett TE. An integrated approach to change the outcome part II: targeted neuromuscular training techniques to reduce identified ACL injury risk factors. *J Strength Cond Res*. 2012;26:2272-2292. <http://dx.doi.org/10.1519/JSC.0b013e31825c2c7d>
108. Myer GD, Martin L, Jr, Ford KR, et al. No association of time from surgery with functional deficits in athletes after anterior cruciate ligament reconstruction: evidence for objective return-to-sport criteria. *Am J Sports Med*. 2012;40:2256-2263. <http://dx.doi.org/10.1177/0363546512454656>
109. Myer GD, Paterno MV, Ford KR, Hewett TE. Neuromuscular training techniques to target deficits before return to sport after anterior cruciate ligament reconstruction. *J Strength Cond Res*. 2008;22:987-1014. <http://dx.doi.org/10.1519/JSC.0b013e31816a86cd>
110. Myer GD, Schmitt LC, Brent JL, et al. Utilization of modified NFL combine testing to identify

- functional deficits in athletes following ACL reconstruction. *J Orthop Sports Phys Ther*. 2011;41:377-387. <http://dx.doi.org/10.2519/jospt.2011.3547>
111. Nachev P, Wydel H, O'Neill K, Husain M, Kennard C. The role of the pre-supplementary motor area in the control of action. *Neuroimage*. 2007;36 suppl 2:T155-T163. <http://dx.doi.org/10.1016/j.neuroimage.2007.03.034>
112. Negahban H, Ahmadi P, Salehi R, Mehravar M, Goharpey S. Attentional demands of postural control during single leg stance in patients with anterior cruciate ligament reconstruction. *Neurosci Lett*. 2013;556:118-123. <http://dx.doi.org/10.1016/j.neulet.2013.10.022>
113. Norte GE, Pietrosimone BG, Hart JM, Hertel J, Ingersoll CD. Relationship between transcranial magnetic stimulation and percutaneous electrical stimulation in determining the quadriceps central activation ratio. *Am J Phys Med Rehabil*. 2010;89:986-996. <http://dx.doi.org/10.1097/PHM.0b013e3181f1c00e>
114. O'Connell M, George K, Stock D. Postural sway and balance testing: a comparison of normal and anterior cruciate ligament deficient knees. *Gait Posture*. 1998;8:136-142.
115. Okuda K, Abe N, Katayama Y, Senda M, Kuroda T, Inoue H. Effect of vision on postural sway in anterior cruciate ligament injured knees. *J Orthop Sci*. 2005;10:277-283. <http://dx.doi.org/10.1007/s00776-005-0893-9>
116. On AY, Uluda B, Taskiran E, Ertekin C. Differential corticomotor control of a muscle adjacent to a painful joint. *Neurorehabil Neural Repair*. 2004;18:127-133. <http://dx.doi.org/10.1177/0888439004269030>
117. Palmieri-Smith RM, Villwock M, Downie B, Hecht G, Zernicke R. Pain and effusion and quadriceps activation and strength. *J Athl Train*. 2013;48:186-191. <http://dx.doi.org/10.4085/1062-6050-48.2.10>
118. Park HB, Koh M, Cho SH, Hutchinson B, Lee B. Mapping the rat somatosensory pathway from the anterior cruciate ligament nerve endings to the cerebrum. *J Orthop Res*. 2005;23:1419-1424. <http://dx.doi.org/10.1016/j.orthres.2005.03.017>
119. Paterno MV, Ford KR, Myer GD, Heyl R, Hewett TE. Limb asymmetries in landing and jumping 2 years following anterior cruciate ligament reconstruction. *Clin J Sport Med*. 2007;17:258-262. <http://dx.doi.org/10.1097/JSM.0b013e31804c77ea>
120. Paterno MV, Rauh MJ, Schmitt LC, Ford KR, Hewett TE. Incidence of contralateral and ipsilateral anterior cruciate ligament (ACL) injury after primary ACL reconstruction and return to sport. *Clin J Sport Med*. 2012;22:116-121. <http://dx.doi.org/10.1097/JSM.0b013e318246ef9e>
121. Paterno MV, Schmitt LC, Ford KR, et al. Biomechanical measures during landing and postural stability predict second anterior cruciate ligament injury after anterior cruciate ligament reconstruction and return to sport. *Am J Sports Med*. 2010;38:1968-1978. <http://dx.doi.org/10.1177/0363546510376053>
122. Peuskens H, Vanrie J, Verfaillie K, Orban GA. Specificity of regions processing biological motion. *Eur J Neurosci*. 2005;21:2864-2875. <http://dx.doi.org/10.1111/j.1460-9568.2005.04106.x>
123. Pietrosimone BG, Lepley AS, Erickson HM, Gribble PA, Levine J. Quadriceps strength and corticospinal excitability as predictors of disability after anterior cruciate ligament reconstruction. *J Sport Rehabil*. 2013;22:1-6.
124. Pietrosimone BG, McLeod MM, Lepley AS. A theoretical framework for understanding neuromuscular response to lower extremity joint injury. *Sports Health*. 2012;4:31-35. <http://dx.doi.org/10.1177/1941738111428251>
125. Pitman MI, Nainzadeh N, Menche D, Gasalberti R, Song EK. The intraoperative evaluation of the neurosensory function of the anterior cruciate ligament in humans using somatosensory evoked potentials. *Arthroscopy*. 1992;8:442-447.
126. Pollard CD, Heiderscheit BC, van Emmerik RE, Hamill J. Gender differences in lower extremity coupling variability during an unanticipated cutting maneuver. *J Appl Biomech*. 2005;21:143-152.
127. Powers CM, Fisher B. Mechanisms underlying ACL injury-prevention training: the brain-behavior relationship. *J Athl Train*. 2010;45:513-515. <http://dx.doi.org/10.4085/1062-6050-45.5.513>
128. Remaud A, Boyas S, Lajoie Y, Bilodeau M. Attentional focus influences postural control and reaction time performances only during challenging dual-task conditions in healthy young adults. *Exp Brain Res*. 2013;231:219-229. <http://dx.doi.org/10.1007/s00221-013-3684-0>
129. Roberts D, Ageberg E, Andersson G, Fridén T. Clinical measurements of proprioception, muscle strength and laxity in relation to function in the ACL-injured knee. *Knee Surg Sports Traumatol Arthrosc*. 2007;15:9-16. <http://dx.doi.org/10.1007/s00167-006-0128-4>
130. Roberts D, Fridén T, Stomberg A, Lindstrand A, Moritz U. Bilateral proprioceptive defects in patients with a unilateral anterior cruciate ligament reconstruction: a comparison between patients and healthy individuals. *J Orthop Res*. 2000;18:565-571. <http://dx.doi.org/10.1002/jor.1100180408>
131. Roewer BD, Di Stasi SL, Snyder-Mackler L. Quadriceps strength and weight acceptance strategies continue to improve two years after anterior cruciate ligament reconstruction. *J Biomech*. 2011;44:1948-1953. <http://dx.doi.org/10.1016/j.jbiomech.2011.04.037>
132. Sadoghi P, von Keudell A, Vavken P. Effectiveness of anterior cruciate ligament injury prevention training programs. *J Bone Joint Surg Am*. 2012;94:769-776. <http://dx.doi.org/10.2106/JBJS.K.00467>
133. Santello M, McDonagh MJ, Challis JH. Visual and non-visual control of landing movements in humans. *J Physiol*. 2001;537:313-327.
134. Scheidt RA, Condit MA, Secco EL, Mussa-Ivaldi FA. Interaction of visual and proprioceptive feedback during adaptation of human reaching movements. *J Neurophysiol*. 2005;93:3200-3213. <http://dx.doi.org/10.1152/jn.00947.2004>
135. Schutte MJ, Dabezies EJ, Zimny ML, Happel LT. Neural anatomy of the human anterior cruciate ligament. *J Bone Joint Surg Am*. 1987;69:243-247.
136. Seidler RD. Neural correlates of motor learning, transfer of learning, and learning to learn. *Exerc Sport Sci Rev*. 2010;38:3-9. <http://dx.doi.org/10.1097/JES.0b013e3181c5cce7>
137. Seidler RD, Noll DC. Neuroanatomical correlates of motor acquisition and motor transfer. *J Neurophysiol*. 2008;99:1836-1845. <http://dx.doi.org/10.1152/jn.01187.2007>
138. Smith TQ, Mitroff SR. Stroboscopic training enhances anticipatory timing. *Int J Exerc Sci*. 2012;5:344-353.
139. Smith WM, Bowen KF. The effects of delayed and displaced visual feedback on motor control. *J Mot Behav*. 1980;12:91-101.
140. Spindler KP, Wright RW. Clinical practice. Anterior cruciate ligament tear. *N Engl J Med*. 2008;359:2135-2142. <http://dx.doi.org/10.1056/NEJMcP0804745>
141. Swanik CB, Covassin T, Stearne DJ, Schatz P. The relationship between neurocognitive function and noncontact anterior cruciate ligament injuries. *Am J Sports Med*. 2007;35:943-948. <http://dx.doi.org/10.1177/0363546507299532>
142. Swanik CB, Lephart SM, Giraldo JL, Demont RG, Fu FH. Reactive muscle firing of anterior cruciate ligament-injured females during functional activities. *J Athl Train*. 1999;34:121-129.
143. Thompson HW, McKinley PA. Landing from a jump: the role of vision when landing from known and unknown heights. *Neuroreport*. 1995;6:581-584.
144. Tinazzi M, Rosso T, Zanette G, Fiaschi A, Aglioti SM. Rapid modulation of cortical proprioceptive activity induced by transient cutaneous deafferentation: neurophysiological evidence of short-term plasticity across different somatosensory modalities in humans. *Eur J Neurosci*. 2003;18:3053-3060.
145. Torquati K, Pizzella V, Babiloni C, et al. Nociceptive and non-nociceptive sub-regions in the human secondary somatosensory cortex: an MEG study using fMRI constraints. *Neuroimage*. 2005;26:48-56. <http://dx.doi.org/10.1016/j.neuroimage.2005.01.012>
146. Vairo GL, Myers JB, Sell TC, Fu FH, Harner CD, Lephart SM. Neuromuscular and biomechanical landing performance subsequent to ipsilateral semitendinosus and gracilis autograft anterior cruciate ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc*. 2008;16:2-14. <http://dx.doi.org/10.1007/s00167-007-0427-4>
147. Valeriani M, Restuccia D, Di Lazzaro V, Franceschi F, Fabbriani C, Tonali P. Central nervous system modifications in patients with lesion of the anterior cruciate ligament of the knee. *Brain*. 1996;119 pt 5:1751-1762.

148. Valeriani M, Restuccia D, Di Lazzaro V, Franceschi F, Fabbriani C, Tonali P. Clinical and neurophysiological abnormalities before and after reconstruction of the anterior cruciate ligament of the knee. *Acta Neurol Scand.* 1999;99:303-307.
149. Wade MG, Jones G. The role of vision and spatial orientation in the maintenance of posture. *Phys Ther.* 1997;77:619-628.
150. Warren WH, Jr, Kay BA, Zosh WD, Duchon AP, Sahuc S. Optic flow is used to control human walking. *Nat Neurosci.* 2001;4:213-216. <http://dx.doi.org/10.1038/84054>
151. Webster KA, Gribble PA. Time to stabilization of anterior cruciate ligament-reconstructed versus healthy knees in National Collegiate Athletic Association Division I female athletes. *J Athl Train.* 2010;45:580-585. <http://dx.doi.org/10.4085/1062-6050-45.6.580>
152. Wikstrom EA, Tillman MD, Schenker S, Borsa PA. Failed jump landing trials: deficits in neuromuscular control. *Scand J Med Sci Sports.* 2008;18:55-61. <http://dx.doi.org/10.1111/j.1600-0838.2006.00629.x>
153. Winter DA. *Biomechanics and Motor Control of Human Movement.* 4th ed. Hoboken, NJ: Wiley; 2009.
154. Wright RW, Dunn WR, Amendola A, et al. Risk of tearing the intact anterior cruciate ligament in the contralateral knee and rupturing the anterior cruciate ligament graft during the first 2 years after anterior cruciate ligament reconstruction: a prospective MOON cohort study. *Am J Sports Med.* 2007;35:1131-1134. <http://dx.doi.org/10.1177/0363546507301318>
155. Wulf G. Attentional focus and motor learning: a review of 15 years. *Int Rev Sport Exerc Psychol.* 2013;6:77-104. <http://dx.doi.org/10.1080/1750984X.2012.723728>
156. Yoo JH, Lim BO, Ha M, et al. A meta-analysis of the effect of neuromuscular training on the prevention of the anterior cruciate ligament injury in female athletes. *Knee Surg Sports Traumatol Arthrosc.* 2010;18:824-830. <http://dx.doi.org/10.1007/s00167-009-0901-2>
157. Yosmaoglu HB, Baltaci G, Kaya D, Ozer H. Tracking ability, motor coordination, and functional determinants after anterior cruciate ligament reconstruction. *J Sport Rehabil.* 2011;20:207-218.
158. Zabala ME, Favre J, Scanlan SF, Donahue J, Andriacchi TP. Three-dimensional knee moments of ACL reconstructed and control subjects during gait, stair ascent, and stair descent. *J Biomech.* 2013;46:515-520. <http://dx.doi.org/10.1016/j.jbiomech.2012.10.010>
159. Zanette G, Manganotti P, Fiaschi A, Tamburin S. Modulation of motor cortex excitability after upper limb immobilization. *Clin Neurophysiol.* 2004;115:1264-1275. <http://dx.doi.org/10.1016/j.clinph.2003.12.033>
160. Zimny ML, Schutte M, Dabezies E. Mechanoreceptors in the human anterior cruciate ligament. *Anat Rec.* 1986;214:204-209. <http://dx.doi.org/10.1002/ar.1092140216>



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