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Review Article

Exploring Complexity: ACL Hidden in a Complex Sensomotor System

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ABSTRACT

Our therapies are unable to adequately restore knee joint function, prevent subsequent injuries, and prevent post-traumatic osteoarthritis. Therefore, largely pre-planned rehabilitation techniques and test batteries are of dubious effectiveness when it comes to preparing for the chaos of sports (dynamic systems theory). As trainers or physiotherapists, the sooner we accept these facts and recognize the complexity of this problem, the sooner we can solve it. Nothing can fill this gap. Instead, we must consider the many intertwined fields of health and sport.

Keywords: Knee joint; Motor control; Neurocognition; Rehabilitation; Sensomotor system

INTRODUCTION

More than biomechanics, Anterior Cruciate Ligament (ACL) injuries arise from biomechanical deficiencies (e.g., excessive knee valgus, etc.), right? Well, sort of as previously discussed by Dr. Chaput and Harjiv Singh in science, these injuries are the result of complex nonlinear system failures [1]. Biomechanical errors are necessary but insufficient to cause injuries alone. Sport is a complex system. The final result is determined by the relative skills and achievements of individual players, the way each player and coach interacts with teammates, the ever-evolving game dynamics (e.g., shifts, crowd noise, referee, etc.), and the elimination of time on the game clock [2,3]. Two cooperating teams are rarely "perfect," but each adapts to mistakes to score points, maximize possession, and win. This adaptive trait applies to all complex systems despite apparent deficiencies, complexity allows for variability in system operation. In this respect, biomechanical deficiencies, "minor flaw" in human movement and do not always lead to injury [4,5]. Can natural biomechanical variability be the scapegoat for broader system failures?

Sports navigation in sport, two cooperative adaptive systems (i.e., teams) disrupt each other's effectiveness in scoring points [6,7]. This contributes to the evolution of the larger system (i.e., the game) over time. Ultimately, the motor options available to individual athletes depend on the environment, task, and organism dynamics (ecological psychology); all of which affect how well a task can be performed and initiated [5]. Suggesting that the presence of a biomechanical defect can singly determine an athlete's injury risk is unlikely because it does not account for interpersonal dynamics [8]. So how can we take this into account? An individual's behavior depends on the environment. Interpersonal dynamics must be considered in ACL injury and recovery [9].

Interpersonal dynamics and nonlinear pedagogy. More than two-thirds of ACL injuries result from non-contact mechanisms, many of which involve avoiding collisions such as changing direction to avoid a defender [10]. Anecdotally, many of us believe that the interpersonal dynamics of the sport contribute to ACL injuries; which tend to occur more frequently in games than training. Do people exposed to (or subsequent to) ACL injuries fare worse in reactive interpersonal coordination tasks? Can this be optimized through training? If so, how? The awareness that an opponent's actions disrupt movement patterns threatens the validity of traditional rehabilitation [11-13]. Are we effectively developing sports readiness? A deeper understanding of our patients' ability to adapt to environmental disturbances (and how to optimize them) is an area of much-needed research [14,15].

Brain and sprains, the brain is the director of sensomotor control. Research over the past two decades has popularized the fact that sensomotor control determines behaviors and biomechanical flaws contributing to Anterior Cruciate Ligament (ACL) injury or resulting from its pathophysiology [16-18]. The sensomotor system is embodied in a rapidly changing

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environment. A closer look at the complex mechanisms affecting musculoskeletal rehabilitation will help determine the scope of the problem.

THE SENSOMOTOR SYSTEM IS A FEEDBACK LOOP IN DYNAMIC SYSTEMS

Our Central Nervous System (CNS) continually integrates sensory information from multiple modalities (visual, vestibular, somatosensory and auditory, etc.) to create a representation of the environment. Subsequent motor actions change sensory stimuli, and the cycle continues [19]. Without feedback, successful motor behavior is not possible. Beneath the surface of movement, our sensomotor system must first distinguish between stimuli generated by the environment and expected feedback generated by our own behaviors. Impairments in sensory stimuli or sensory weighting (e.g., visual dependency) can impact the accuracy of this distinction between the environment and the auto-afferent system and are the basis for claims that sensory prediction and motor errors contribute to ACL injury [20-22]. Considering how individual variability and impairments (movement errors) may affect sensomotor feedback loops, intrapersonal coordination (i.e., the ability to control our body in space) becomes as complex as the dynamics of the sport [23].

Intrapersonal dynamics, consider a basketball player attempting to make an effective lay-up. As they approach the hoop, the sensomotor system controls infinite degrees of freedom while perceiving the evolving environment [24]. Such sports activities require the coordination of distributed muscle and joint groups. Several studies support this concept in individuals with ACL reconstruction, who have reduced adaptive joint coordination during single-leg balance tasks, with greater stiffness indicating a higher risk of re-injury [25,26]. The same pattern of less variable joint coordination applies to gait. Unfortunately, these impairments are less clinically tangible than muscle weakness or range of motion limitations. What modifiable factors contribute to these impairments and movement errors? How can we be certain we are addressing them? Sensory feedback disruption in the knee joint, differences in perceptual and cognitive processing, and subsequent changes in muscle recruitment are believed to impact intrapersonal coordination. As we travel through the sensomotor system, we remember sensory stimuli and sensory integration to form motor output and effective movement patterns [27,28].

Somatosensory afferents, what happens to ACL Mechano-receptors after injury? Proprioception is generated by receptors in ligament tissues, joint capsules, and muscle-tendon units throughout the body [29]. As we know, these signals enable the perception of body position, movements, and muscle effort. The integration of this diverse and dispersed range of somatosensory afferents is incredibly complex, involving spinal cord centers, the cerebellum, and higher-order central nervous system (supraspinal level) [30]. The ACL and surrounding knee joint structures make up the largest sensory organ in the human body. Neuroscience simply does not yet have techniques to describe what is encoded in afferent signals, but the presence (or lack) of a signal provides information on function post-ACL injury. People without an ACL increase stiffness and hamstring activity, thereby creating active stability [31]. Nonetheless, given that all individuals who have undergone ACL reconstruction once had an ACL deficiency, the state of this pathway still has functional and clinical implications "up the chain (sensomotor)" but is largely unknown or unmeasured in the early postoperative stages. Are there clinical symptoms that can help us?

Disinhibition at the spinal level, effective therapies inhibiting quadriceps joint injury and surgery cause capsular swelling, causing Arthrogenic Muscle Inhibition (AMI). This spinal-level dysfunction underlies bilateral quadriceps weakness following ACL injury and reconstruction [32]. (That is why patients present with rapid muscle atrophy despite the lack of muscle damage). Uninhibiting interventions targeting afferent sensory activity (such as TENS and focal joint cooling) have proven incredibly promising addressing the neuronal mechanisms causing AMI [33]. Masking inhibitory sensory stimuli with TENS or ice create a therapeutic "treatment window" in which quadriceps motor unit excitability and strength are temporarily restored. Every clinician should consider their use to maximize quadriceps strengthening in the acute stages of recovery (until a "quiet knee" is achieved). To be clear, science is uncertain whether these treatments address the development of brain changes over chronic periods of time [34].

Role of the integrative cortex and neurocognition "Periods of deafferentation following ACL injury appear sufficient to catalyze long-term neuroplastic changes in the brain, and functional differences in brain activity exist pre-injury."- Dave Sherman [35].

After initial integration in the spinal cord, sensory signals are transmitted to the brain, where sensory integration helps anticipate the emerging environment. An athlete must use perceived information to make hundreds of motor decisions during the game [36]. The key to this success is the athlete's ability to intentionally seek, interpret, and anticipate relevant information concerning the current and future task and environment dynamics. In other words, performance is constrained by the situation, the athletes' physical capabilities, and their perceptual-cognitive control. For example, those who continue to suffer from ACL injury have slower neuropcognitive processing speed and visual-motor reaction time preceding the injury [37]. These impairments likely persist (and worsen) post-injury. Can we train this? If so, how?

Neuronal efficiency in athlete's neuronal efficiency is one person's ability to integrate more perceptual-cognitive information than another, assuming a ceiling of neuronal capacity. This means among other things, more processed information, more efficient motor actions, and more activity in the sensomotor areas of the brain. Neuronal efficiency is demonstrated with higher intelligence, musical abilities, and isolated motor skills. In all fields, experts require less neuronal activity to perform a standardized task. However, in complex environments such as sports, experts use more neuronal activity, not less. For example, highly skilled athletes show greater activation of the mirror motor neuron system than less skilled

athletes when predicting or analyzing an opponent's movements. These results suggest that gradually reducing cortical demand for single tasks allows experts to process more information and better navigate the complexity and chaos of sports. For now, findings of increased fMRI activity in visual and attention networks during simple rhythmic tasks indicate neuronal inefficiency post-ACLR [38]. This suggests potential intervention practices targeting neuromodulatory abilities and integrative networks focusing on neurocognition (Figure 1).

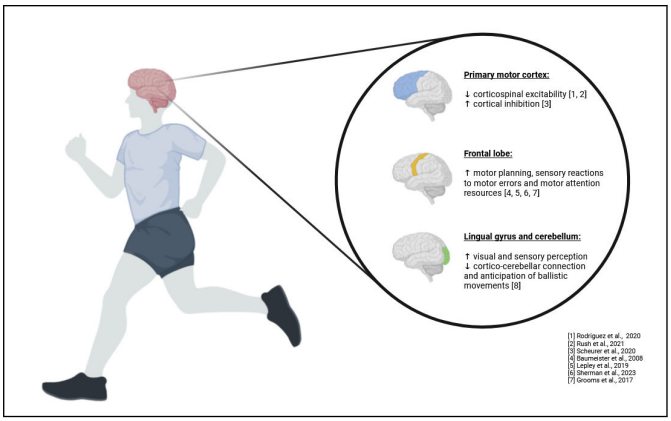

Figure 1: Illustration of the effects of exercise on different brain regions. The highlighted areas show the primary motor cortex, frontal lobe, and lingual gyrus and cerebellum with their respective changes due to exercise.

DISCUSSION

Attention in general, selective attention prepares the cognitive system to distinguish relevant and irrelevant features of the environment. A growing body of research suggests that goal-directed attention (called external focus) results in better performance than self-directed attention (called internal focus), with implications for ACL rehabilitation discussed elsewhere. Results suggest that external attention focus facilitates the ability to plan, select, and perform actions with better environmental perception, while internal attention focus detaches perception from the surrounding environment. Following ACLR, fMRI results suggest greater cognitive demands during rhythmic motor tasks, such as infinity walk [39]. Moreover, healthy individuals at high risk of ACLR injury exhibit less variable cortical activity and cortical symptoms suggesting less adaptive motor coordination. Although the relationship is still theoretical, neuromuscular training strategies based on attention target these suboptimal cognitive-motor strategies. But does all this solve neurocognitive training? What implications does the pre-movement quiet period have? Neuronal processes of the limbic system (emotions and memory) are complex intertwined with motor behavior [40]. Neuroplasticity in this system is theoretically linked to negative behavioral change in lower back pain and chronic pain models and has been extended to the ACLR population. This means we should consider the impact of motivation, fear, anxiety, pain, memory, etc., on the motor control of our athletes. Several recent papers highlight the broad range of psychological, social, and contextual factors that influence our patients' recovery following knee injury. Critically important for mental and social well-being, psychological factors have also been directly linked to quadriceps function and re-injury rates post-ACLR. Psychological, social, and contextual factors evolve with recovery stages and should be prioritized in the management of people following ACLR [41].

The venerated 3 sets of 10 reps is heresy the basal ganglia and associated motor cortex are involved in action selection, initiation, and motor task switching. As trainers, we focus on largely pre-planned and intentional motor activities, making patients very proficient at overusing this "3 sets of 10 repetitions" motor system (i.e., supplementary motor area) [42]. However, differences in neuronal activation between the control group and people with ACLR suggest decreased propensity for reactive motor control. Reactive movement is likely more important in sports and utilizes a different motor system (i.e., premotor areas). This suggests the need for therapeutic exercises (and research paradigms) that focus on reactive motor planning in complex/changing environments and the ongoing compensation of new movement assumptions and learning from prediction errors (Table 1).

Category	Key Points
Therapies and rehabilitation	Insufficient therapies, dubious pre-planned techniques, acceptance of complexity needed.
Biomechanics	ACL injuries from complex nonlinear failures, not just biomechanical deficiencies.
Sports dynamics	Sport involves complex interplay of skills, dynamics, biomechanical defects not sole cause.
Interpersonal dynamics	Non-contact mechanisms, higher injury frequency in games, need to optimize adaptation.
Sensomotor system	Brain directs sensomotor control, feedback important for motor behavior, sensory weighting impacts.

Intrapersonal dynamics	Coordination of muscle/joint groups, reduced joint coordination post-ACL reconstruction.
Proprioception and mechanoreceptors	Proprioception involves signals from ligaments, joint capsules, ACL structures major sensory organs.
Spinal level disinhibition	Capsular swelling leads to muscle inhibition, TENS and cooling can restore strength temporarily.
Neurocognition and integration	Sensory signals processed in brain for motor decisions, neurocognitive impairments affect performance.
Neuronal efficiency	Efficient perceptual-cognitive integration, skilled athletes show greater brain activation.
Attention focus	External attention focus improves performance, neuromuscular training can address cognitive-motor strategies.
Emotional and psychological factors	Emotions, motivation, psychological factors crucial for recovery, linked to function and re-injury rates.
Reactive motor control	Reactive control vital for sports; traditional rehab overemphasizes pre-planned activities.
Corticospinal pathway	Reduced excitability post-reconstruction, strategies needed to increase excitability for recovery.
Muscle atrophy	Quadriceps atrophy post-ACLR, treatments like blood flow restriction promote recovery.
Future directions	Focus on cortical neuroplasticity, comprehensive understanding and diverse assessments required.

Table 1: Key points in understanding and addressing Anterior Cruciate Ligament (ACL) injuries through various categories.

"All or Nothing" is a little harder to achieve. The descending corticospinal pathway is responsible for initiating voluntary muscle contractions and regulating descending motor control. Like any neuronal pathway, the balance between excitatory and inhibitory potential affects transmission and activation of "all or nothing" motor neurons. Reduced corticospinal pathway excitability following ACL reconstruction means more activation is required before movement initiation. Despite treatment, this impairment worsens over time, and the pathway itself appears to atrophy. Additionally, corticospinal excitability is strongly correlated with key features of quadriceps function (e.g., rate of torque development) and thus motor function recovery [43]. To this end, we must develop and adopt treatment strategies that increase corticospinal synaptic excitability. EMG biofeedback, motor imagery, ballistic and eccentric exercises are promising possibilities.

What's happening in the muscle? A primary clinical feature of people with ACLR is quadriceps atrophy. Clinicians struggle with persistent inhibition, atrophy, and weakness of the quadriceps muscle. Detaching nerve tissue from muscles (known as denervation) severely limits the ability to voluntarily contract muscles, increases intramuscular fat deposits, catalyzes fiber type transformations, increases circulating atrophy mediators, and lowers satellite cell numbers [44]. Treatments, such as blood flow restriction and eccentric exercises, are mechanistically adapted to these myological disorders.

CONCLUSION

What's Next? As trainers, we cannot continue doing the same thing and expect a different outcome. Given a single change in the sensory-motor system, such as altered sensory afferentation post-ACLR, the CNS must change incrementally throughout its distributed network in a way that maintains key behavioral features (i.e., balance, gait, etc.). The complexity of the CNS makes it extremely difficult to know where to intervene. But we cannot be afraid to try. Changes in the CNS are not set in stone, and the potential to induce long-term neuroplastic changes has been demonstrated in populations with much greater denervation (i.e., stroke, spinal cord injury). The future of ACL injury rehabilitation must consider interventions that direct beneficial neuroplasticity through neuromodulation. Moving forward, it will be necessary to globally appreciate the embodied sensory-motor system to test these theories scientifically and systematically. This will require diverse neurophysiological and neurochemical assessments, most importantly including clinical knowledge, considering patient experience, and utilizing available methodologies. Areas where we need deeper understanding include interpersonal dynamics, changes in neuronal networks, reactive motor control, and guidelines for psychological, social, and contextual factors. For now,

interventions that induce beneficial plasticity should address reflex spinal excitability, sensory motor re-experience, visuomotor dependence, corticospinal excitability, and local muscle growth factors.

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