**Neuroplasticity in Rehabbing Sports Injuries**

As clinicians, we are experts in treating the body’s hardware…but what about the body’s software?

Neuroplastic alterations secondary to orthopedic injury have become widely assayed in recent research. Cortical adaptations have been demonstrated amongst individuals living with low back pain, chronic ankle instability, and soft tissue rupture/repair.1-6 What may be most convincing in this realm of study are the long-lasting neurological changes demonstrated in response to anterior cruciate ligament injury.

Anterior cruciate ligament (ACL) rupture is a common orthopedic injury that usually requires surgical reconstruction and physical rehabilitation to restore mechanical stability and function.1 Aside from it’s mechanical necessity, the ACL also serves a crucial role in the proprioceptive function of the lower extremity. The ACL is densely innervated by mechanoreceptors responsible for detecting and communicating functional joint position, anatomical force production, and biomechanical forces which may lead to lower extremity injury.7 Upon rupture, these nerve cells become damaged with poor potential for regeneration or neuroplastic rerouting, thus creating a disconnect between the peripheral and cortical nervous systems.8 These mechanical disruptions ignite a cascade of complex physical and neurocognitive adaptations which may negatively impair long-term recovery.7

Statistically, we know that traditional recovery methods following ACL rupture have poor long-term outcomes. Even with extensive rehabilitation, individuals who sustain an ACL injury have a 30% failure rate on the ipsilateral structure, a significantly increased risk-of-injury to the contralateral structure, decreased health-related quality of life, an increased risk for osteoarthritis, and a self-reported “inability” to perform at pre-injury status.5-7 So, what does the brain have to do with this? And how can we help mitigate these effects?

fMRI studies have found evidence of hyperactivity in the motor, lingual, somatosensory cortexes amongst individuals who’ve undergone ACL reconstruction and rehabilitation (ACL-R).4,6 These effects can be largely attributed to altered sensory feedback, causing the nervous system to rely on other sources (i.e. visual stimuli, external feedback) to produce functional movement.6 Current rehabilitation paradigms do not place specific importance on neurocognitive or visual-motor training. Incorporating these ideals into our everyday clinical practice could be as simple as performing eyes-closed quad sets, providing external feedback cues, or adding quick cognitive tasks to basic strengthening exercises.7

By understanding the ways in which our brains react to orthopedic injury, we can better prepare our patients for full and successful recoveries. It is key to remember: there is no injury without deafferentation; there is no deafferentation without neuroplastic compensation. Everything that we do as rehabilitative clinicians has the ability to effect the minds and bodies of our patients. It is a responsibility we must handle with both intellect and care.

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