



Investigating the Involvement of the Anterior Cruciate Ligament in Knee Proprioception

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The anterior cruciate ligament (ACL) is an internal structure of the joint that is of paramount importance to the normal motion of the knee as it provides both static and dynamic stability [1]. Dynamic stability is favored by the presence of specific ligament mechanoreceptors that prove to be an essential element of proprioception as revealed by several histological and anatomical studies. The existence of mechanoreceptors and their potential role in knee function has been discussed for more than a century. However, it was only as recently as 1984 that their existence was demonstrated on the human ACL after the identification of type III receptors in the ligaments (according to Freeman and Wyke) and with free ends. Later, more detailed studies identified three types of mechanoreceptors according to different morphological features: two types of Ruffini terminal organelles, Paccinian corpuscles and a small number of free nerve endings [2]. In this study, it was reported that nerve elements occupy about 1% of the surface of the ligament. Although the definition of ownership has been attempted many years ago, there is still no widely accepted definition. Sherrington has included the term in the literature describing the results of afferent nerve impulses from muscles, tendons, joints, and related tissues. Input processing leads to reflex control and muscle control.

This description defines sensation as primarily a sensory process. Recently, the meaning of the term proprioception has been extended to include the interaction of the afferent and efferent pathways of the somatosensory system [3]. Most authors refer to two types of somatic sensations for clinical reasons. Static proprioception is defined as a sense of common place, that is, the direction of the limbs in space, and the perception of their reciprocal relationship. Dynamic or kinesthetic proprioception includes sensation of movement of the extremities and changes in speed, acceleration, or deceleration.

These two elements of somatic perception must be accompanied by the last, but no less important, element of the somatic sensation of force/tension, which occurs during muscle contraction. Injury to joint structures such as the ACL, meniscus, and subsequent osteoarticular changes are associated with damage to mechanical receptors. The following damage to the afferent pathways and transmission of information to the CNS results in disturbances in sensation of joint position and movement [4,5]. Decreased somatic sensitivity is noted outside of ACL injury, due to aging whereas impaired joint position sensation is noted after osteoarticular changes in the knee. Sensory ownership after tearing of the ACL has been studied in detail. Preset angle reconstruction (JPS) and passive motion detection threshold (TTDPM) are the most used sensor system measurement techniques.

Two other methods for assessing neuromuscular control are reflex hamstring contraction latency (RHCL) and assessment of postural control. Most studies report posttraumatic somatosensory reduction and ACL failure whereas the opposite view receives limited support. During the acute phase of ligament damage, it is not clear whether arthritis and edema contribute to the deficiency state. However, these parameters are thought to be unrelated to the reduction in static and dynamic ownership, which is evident in chronic ACL failure. Since sensory receptors are present not only in the anterior cruciate ligament but also in the skin, muscles, tendons, and other tissues surrounding

the knee, damage to the motor sensory system due to the ligament injury can also cause damage to the sensory system due to damaged ligaments afferent pathways lead to reduced neuromuscular control and dynamic joint stabilization.

In this case, damage to the afferent pathways or reflex action on the muscle spindles results in decreased neuromuscular control and dynamic joint instability. Thus, the lack of knee sensing mechanism with ruptured or nonfunctional ACL is probably not only a result of loss of ligamentous mechanism and subsequent loss of feedback but also a consequence of abnormal neural output of the joint capsule and the remaining ligaments.

Conclusion

The anterior cruciate ligament is not only a major obstacle to anterior displacement of the tibia but also an important dynamic stabilizer of the knee joint. However, the contribution of the ACL to knee proprioception has not been fully elucidated. More clinical and laboratory studies are needed to better understand this complex mechanism.

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