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Mechanism of Injury and Risk Factors for Anterior Cruciate Ligament Injury

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Participation in recreational and competitive athletics continues to increase. Injury rates, including anterior cruciate ligament (ACL) tears, have increased concomitantly. As such there has been increasing interest and research related to ACL injury prevention. The first steps in injury prevention are gaining an understanding of the mechanism of injury and the risk factors predisposing to injury. The focus of this article is on the current understanding of ACL injury mechanism and risk factors. ACL injury mechanisms are stratified and discussed as noncontact and contact injuries. Additionally, risk factors, both modifiable and nonmodifiable, are reviewed.

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Introduction

Participation in recreational and competitive athletics continues to increase. Injury rates, including anterior cruciate ligament (ACL) tears, have increased concomitantly. As such there has been increasing interest and research related to ACL injury prevention. The first steps in injury prevention are gaining an understanding of the mechanism of injury and the risk factors predisposing to injury. The focus of this article is on the current understanding of ACL injury mechanism and risk factors.

Mechanism of ACL Injury

Injuries to the ACL may be debilitating in the short term owing to the loss of athletic participation, time away from work, and financial costs associated with treatment.[1] Additionally, ACL injuries can have long-term consequences on the affected knee, including alteration of knee kinematics, associated meniscal and cartilage damage, and ultimately knee osteoarthritis.[2] The incidence of such injuries has been reported to be greater than 200,000 per year in the United States alone.[3] Most ACL ruptures (approximately 75%) are sustained with minimal or no contact at the time of injury.[4] Recent research using multiple modalities, including video analysis, computer simulation, cadaveric studies, and epidemiologic data, have all aided in our understanding of the mechanism of injury for ACL tears. However, despite all the information gathered, the mechanism of ACL injury remains incompletely understood. The goal of heightened investigation has been to determine the common characteristics, forces, and body positions that are noted at the moment of ACL injury. Although a single sole mechanism has not been found, these studies have shed light on risk factors and activities associated with ACL injuries. The importance of understanding the mechanism and risk factors contributing to increased injury risk lies in helping to formulate preventive techniques, specifically for noncontact ACL injuries.

Noncontact ACL Injury

Sport-related activities at the moment of noncontact ACL injury could vary depending on the sport, but most often involve a change in velocity or the generation of multidirectional force across the knee joint while bearing weight.

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Specifically, rapid deceleration moments, including those that also involve planting the injured leg to cut and rapidly change direction, have been linked to ACL injuries. Landing from a jump, twisting, and pivoting have also been associated.[4]

A primary feature of these noncontact ACL injuries is the involvement of multiple vectors of force on the knee, or multiplanar loading.[5] Specifically, sagittal and coronal loads, in combination with imbalanced muscle contraction forces of the quadriceps and hamstrings muscle groups, can lead to significant stresses on the ACL.[6] Observational studies of ACL injury moments have shown that a combination of valgus and rotational moments at the knee are commonly noted. Studies have also shown that the addition of these rotational or transverse forces to the coronal forces in a hyperextended knee can multiply the tension on the ACL.[7,8] A difficulty in elucidating the mechanism of ACL injury is the complex multiplanar knee motion that is implicated in ACL damage. Multiple studies have shown that forces in the sagittal, coronal, and transverse planes may increase tensioning of the ACL and result in ACL injury. In a systematic review of the literature regarding mechanisms of ACL injury, Quatman et al[9] reported that 82% of direct ACL injury mechanism studies showed a multiplanar mechanism of injury. They relate their findings to the pivot shift clinical examination technique, where knee flexion and axial load combined with tibial rotation and valgus force may reproduce the original mechanism of injury.

The ACL is the primary restraint of the knee with anterior loading of the tibia. It has been postulated that anterior tensioning on the proximal tibia subjects the ACL to increased tensile load by muscular forces about the knee with the quadriceps and gastrocnemius typically countered by the hamstrings. Several studies have shown increased tensioning of the ACL with isolated quadriceps contraction in lower degrees of knee flexion.[10-14] Assessments of the role that the gastrocnemius has in anterior shear forces of the tibia have been contradictory and inconclusive.[15,16] Most recently, Pflum et al[17] showed with a computer model that the gastrocnemius produces minimal anterior shear forces of the tibia with drop landing. Evaluation of hamstrings contraction in addition to the quadriceps has shown decreased tensioning of the ACL; however, its protective effect is dissipated with relative extension of the knee.[18-21] In addition, the hip in a flexed position further shortens the hamstrings, decreasing their protective ability during jump landing. Boden et al[22] discussed the role of quadriceps and hamstrings forces across the tibia, noting that they both subjected more significant compressive load than anterior shear force in relative extension. They cite several sources in which axial compression may increase tension and result in ACL disruption.[22] In total, these series of studies show that with relative knee extension, there is increased anterior tibial shear and decreased protective hamstring contraction, leading to ACL tension and possible risk of failure.

In addition to muscle forces across the knee, osseous constraint in the form of tibial slope has been implicated in ACL injury. Meyer et al in 2004 and 2008 concluded that axial compression resulted in posterior translation of the distal femur relative to the tibia secondary to posterior tibial slope.[23,24] A study specifically showed that with increasing degree of posterior slope of the tibial plateau, the ACL deficient knee had increased anterior tibial translation.[25] Another study by Giffin et al demonstrated that following a 5-mm anterior wedge-opening osteotomy, compressive forces produced an increase in anterior translation of the tibia whereas anterior-posterior loading did not affect the resting tibial position.[26] In 1994, Torzilli et al[27] implicated anterior translation of the tibial plateau with either axial load or quadriceps contraction as a potential mechanism of ACL injury. These findings were corroborated with further cadaveric testing by Wall et al,[28] who found that with the knee in 15° of flexion, axial compression alone was capable of producing ACL injury whereas the addition of quadriceps loading reduced the necessary axial force to produce injury.

Studies of limb position at the time of noncontact injury have largely demonstrated consistent findings. The sagittal position of the involved knee often ranges from early flexion to hyperextension at the moment of injury.[4] This is likely to be the result of the ACL having higher tensile properties regarding resisting anterior forces and shear within this range of motion.[29] Quadriceps muscle contraction in this range of early flexion also was found to produce higher ACL tension than in deeper flexion, suggesting a higher susceptibility to injury with excessive contractions in this range of motion.[5] This can explain the association of ACL injuries with moments of rapid deceleration. At these times, the quadriceps forces required to stop the athlete are increased and thus contractions can place significant stresses on the ACL. Video analysis by Boden et al[30] found that, in the sagittal plane, hip and ankle landing positions were the most significant: specifically, flatfoot landing and a flexed hip posture. Evaluation of knee sagittal positioning found that there was no significant difference between injured athletes vs control; however, there was a trend toward more knee extension in the ACL-injured subjects. In the coronal plane, knee abduction (or valgus) was significantly greater for patients with ACL injury. A separate study with video analysis of 39 international soccer injuries also found that knee abduction was associated with ACL injury although valgus collapse was rare.[31]

Contact ACL Injury

Contact-type mechanisms are typically a result of higher energy mechanisms of injury. This could include traumatic knee dislocations or high-energy on-field injuries. Typically such an injury is by sudden deceleration or abrupt change in knee direction. Much like the noncontact type, there may be hyperextension, or collision, where varus or valgus stress is applied to the knee in the form of a translation and shearing motion.[4,31] O'Donoghue's Triad has been described specifically as a mechanism resulting in ACL, medial collateral ligament, and medial meniscus injuries. This was reevaluated by Shelbourne and Nitz,[32] who established that with combined ACL and medial collateral ligament injuries, the more