The posterior cruciate ligament (PCL), located in the center of the knee, is the axis about which the knee flexes, extends, and rotates. Intactness of the PCL is thought to be key to classification of knee instability (McCluskey & Blackburn, 1980). Ruptures of the PCL may represent up to 20% of all knee ligament injuries (Miller & Harner, 1993). Predraft physical examination of NFL players has revealed a 2% incidence of PCL deficiency; in fact, players are often unaware that they have sustained the injury (Bergfield, 1993). The PCL is most commonly injured in sports or during a high-speed vehicle accident (Hughston et al., 1980).

Anatomy

The PCL is named according to its spatial orientation and insertion site. It originates from the lateral wall of the medial femoral condyle and runs posteriorly and laterally in relation to the ACL to insert on the intracondylar fossa on the posterior aspect of the tibial plateau. The ligament has a broad origin and insertion and is narrowest at its midportion (Decter & Robbins, 1994).

The PCL is composed of anterolateral and posteromedia}
knee flexion (Covey & Sapega, 1994). Secondarily, it provides restraint to varus and valgus stress and hyperextension as well as limiting internal rotation of the tibia on the femur (Decter & Robbins, 1994). In addition, in conjunction with the ACL, it regulates the screw home mechanism (Fukubayashi et al., 1982).

Skyhar et al. (1993) found significant increases in medial compartment and patellofemoral contact forces after isolated sectioning of the PCL and combined sectioning of the PCL and PLC. They reasoned that tibiofemoral contact forces were altered secondary to a medial shift in the joint's center of rotation.

In addition, they noted that posterior tibial translation results in greater patellofemoral contact forces secondary to a decrease in the angle between the quadriceps tendon and patellar tendon.

**Mechanism of Injury**

Mangine and Eifert-Mangine (1991) describe three mechanisms of injury to the PCL. The first and most common is that of a direct blow to the tibial crest with the knee flexed 70 to 90°. The second mechanism is that of extreme internal or external rotation. The third mechanism described is hyperextension (Hughston et al., 1980).

Fowler and Messieh (1987) reviewed 13 athletes who sustained isolated PCL injuries. The most common mechanism of injury was hyperflexion without pretibial trauma. Pretibial trauma to the hyperflexed knee, more commonly known as the “dashboard” injury, was the second most common mechanism. Forces leading to posterolateral injury may also cause injury to cruciate ligaments (Veltri & Warren, 1994).

**Symptoms**

Isolated PCL injuries often go unrecognized at first. The examiner should look carefully for lacerations and abrasions in the region of the anterior tibia. The symptoms of chronic instability may lead to diagnosis at a later date (Decter & Robbins, 1994).

Patients with chronic PCL injury may complain of anterior and/or medial knee pain secondary to the increased medial compartment and patellofemoral contact forces. Occasionally some instability associated with twisting and jumping is noted (Miller & Harner, 1993).

In the case of acute isolated posterolateral injury, the patient's chief complaint is that of pain in the posterolateral knee. Associated peroneal nerve injury is possible and may lead to dysesthesias and leg and foot weakness (Veltri & Warren, 1994). Once the initial pain and swelling resolve, the patient may experience instability in positions of extension and a tendency toward buckling of the knee into hyperextension.