Knee dislocation involves loss of the tibiofemoral continuity after disruption of two to three knee ligaments.1,2 The structures most commonly torn are the anterior cruciate ligament (ACL) and posterior cruciate ligament (PCL), and anterior tibial translation is the most common displacement mechanism. Such an injury is typically caused by high-energy trauma (i.e., motor vehicle accident), which is rare in sports.3,4 A dislocated knee will spontaneously reduce in most cases, so the true incidence of the injury is unknown.3,4 The principal concern is the possibility of neurovascular compromise. Disruption of neurovascular structures can lead to permanent damage, which could ultimately require leg amputation. Because spontaneous reduction often occurs,2,3,5 neurovascular function should be evaluated after the occurrence of any acute knee ligament injury, especially when rupture of two or more ligaments is suspected.

Background

A basic understanding of knee anatomy is essential for evaluation of a knee dislocation.6,7 In addition to the ACL and PCL, the anatomic structures of the posterolateral corner (i.e., iliobibial band, biceps femoris tendon, popliteus muscle, popliteus tendon, popliteus ligament, and posterior capsule) are important knee stabilizers.8,9 Neurovascular structures that cross the knee joint include the popliteal artery, popliteal vein, tibial nerve, and common peroneal nerve (CPN).6 The tibial nerve is a branch of the sciatic nerve that runs along the fibular head. The CPN innervates the extensor digitorum brevis, extensor hallucis brevis, tibialis anterior, peroneus longus, peroneus tertius, and peroneus brevis muscles, and it conveys afferent sensory signals associated with the L4, L5, S1, and S2 dermatomes.6 The CPN injury is vulnerable to injury from a direct blow to the lateral aspect of the knee in the area of the fibular head,10 and varus displacement of the knee can subject the CPN to traction.3,11

Senes et al.12 collected data on 523 patients with CPN lesions over a 32-year period. They reported that 318 patients required surgical procedures to repair the CPN, 12% of which had some type of laceration. The injuries resulted from broken glass, chain saws, gunshot wounds, or lawn mower blades, and none of the cases involved complete disruption of the CPN. The literature contains several reports of knee dislocations that have involved CPN injury.3,8,12 Niall et al.8 suggested that complete disruption of the ACL, PCL, and posterolateral corner might be associated with CPN rupture. Stretch-induced CPN palsy associated with knee dislocation has been reported to occur in 25%–30% of cases.8,12 None of the cases reported in the literature involved a complete CPN rupture that resulted from a sport-related knee injury.3,8,12

Case Report

A 14-year-old male, junior varsity wrestler, who had no previous history of knee injury, was carried by teammates to the athletic training room after having sustained a right knee injury. The athlete had been
participating in a wrestling practice drill with another wrestler. The athlete’s right knee was apparently locked in a varus position when he fell backward, and the second wrestler fell on top of him. The athlete reported having felt a “pop” and the immediate onset of moderate pain.

The athletic trainer found that the patient was too apprehensive to allow a thorough evaluation. An inability to actively tolerate dorsiflexion of the foot and a lack of sensation along the L5 and S1 dermatomes was noted. Active plantar flexion, inversion, and eversion were demonstrated, but strength was assessed as 2/5 by manual muscle test (MMT) for each of the motions. Vascular supply was found to be normal. Throughout the evaluation, the patient was very calm; he did not appear to be in much pain (except during evaluation of knee ligament integrity), and he was able to communicate clearly. Ice, compression, and elevation treatment was administered and crutches were provided for ambulation. The patient’s parents were notified, and he was taken to a local emergency room.

On the day after injury occurrence, the patient reported that he was diagnosed as having a “knee sprain” and was discharged with a physician’s permission to participate as tolerated. The patient was subsequently referred to an orthopaedic surgeon. A magnetic resonance imaging (MRI) examination revealed extensive soft tissue damage that included ACL rupture, PCL rupture, partial tear of the iliotibial band, biceps femoris tendon avulsion fracture, LCL rupture, and popliteal ligament rupture. This MRI did not reveal any injury to the CPN, which could not be identified on the images.

The patient underwent three surgeries to repair the damaged knee structures. The first surgery was performed at one month postinjury, which consisted of a posterolateral corner reconstruction that used a tibialis anterior tendon allograft and anchoring of the biceps femoris to the fibular head. During surgical inspection of the posterolateral corner, the distal end of the ruptured CPN was found near the joint line. The proximal end could not be located. Postsurgical management involved very light weight-bearing (i.e., \( \leq 10 \) lbs), ankle range of motion (ROM) exercises, active-assisted knee ROM exercises within \( 0^\circ - 90^\circ \), and continuous utilization of a foot orthosis to maintain the ankle in a neutral position. Full weight-bearing was permitted at four weeks postsurgery.

The second surgery was performed eight weeks after the posterolateral corner reconstruction (2 months and 17 days postinjury). An Achilles tendon contracture had developed, which restricted ankle dorsiflexion by \( 10^\circ \) with the knee extended. Surgical exploration of the ruptured CPN identified a proximal neuroma extending to the division of the sciatic nerve, and a distal neuroma extending to the bifurcation of the deep and superficial peroneal nerves. Both neuromas were resected until healthy nerve fascicles were visible, leaving a retraction of 15 centimeters in the CPN. To reconstruct the CPN, the entirety of the sural nerve was harvested from both extremities to serve as a graft (Figure 1). Five interfascicular nerve cable grafts, each of which was 16 cm in length, were used to bridge the CPN defect. During this surgery, the Achilles tendon was lengthened sufficiently to allow passive ankle dorsiflexion of \( 20^\circ \) with the knee extended. The posterior tibialis tendon was translocated through the interosseous membrane to the dorsum of the third cuneiform to balance muscle forces around the ankle, thereby facilitating nerve regeneration. The patient was placed in a long cast, with the ankle in \( 90^\circ \) dorsiflexion and the knee in \( 30^\circ \) of flexion, for a duration of six weeks. The patient was subsequently allowed to begin weight-bearing in a walking boot as tolerated.

At 10 months and 22 days postinjury, surgical reconstruction of the ACL (patellar bone-tendon autograft) and PCL (Achilles tendon allograft) was performed. Prognosis was for 90% overall function returning to the lower extremity, and for activities of daily living returning to normal, but patient was unlikely to return to a 5/5 MMT for dorsiflexion or

![Figure 1](image-url) Surgical reconstruction of the common peroneal nerve with 5 sural nerve cable grafts of 16 cm. The distal portion of the extremity is to the left. The division of the sciatic nerve into the tibial nerve and common peroneal nerve can be seen at the more proximal end of the incision.