ELSEVIER

Operative Techniques in

Sports Medicine

Peroneal Nerve Injuries: Repair, Grafting, and Nerve Transfers

Matthew R. Prince, DO,[*,†] Alexander H. King, BS,[*,†] Alexander Y. Shin, MD,[*,†] Allen T. Bishop, MD,[*,†] Michael J. Stuart, MD,[*,†] and Bruce A. Levy, MD[*,†]

Knee dislocations are severe musculoskeletal injuries that are often associated with neurovascular injury. The most common nerve injured with a knee dislocation is the peroneal nerve. Nerve injury can result in severe functional consequences to the injured patient. The purpose of this article is to review the most current literature on treatment options and outcomes when addressing these injuries.

Oper Tech Sports Med 23:357-361

KEYWORDS peroneal nerve, knee dislocation, injury

Introduction

Knee dislocations are rare but frequently devastating injuries. A knee dislocation is the result of a high-energy mechanism, which causes severe soft tissue damage about the knee and often includes neurovascular compromise. Injury to the popliteal artery and peroneal nerve are well-recognized complications. The physician managing these injuries must maintain a heightened index of suspicion when assessing these patients and their neurovascular status.

Due to the recognized diversity of patient characteristics and injury patterns, it is difficult to estimate the true incidence of knee dislocations in the general population.[1] Previous literature has cited the prevalence of knee dislocation as ranging from 0.28%-5.3% and 0.02%-2% of all orthopedic injuries per year.[2-5] However, these estimates likely underrepresent the true incidence, due to the frequent nature of spontaneous relocations and the increasing recognition of "low-velocity" knee dislocations in the obese population.[6-8] Regardless of whether a knee dislocation reduces spontaneously or manually, the risk of neurovascular injury is present. Consequently, any patient with a bicruciate injury or a 3 or 4-ligament injury should be managed as though a dislocation has occurred.

This article reviews the current treatment options for peroneal nerve injury in patients who sustain a knee dislocation. The best available evidence is presented.

*Department of Orthopedic Surgery, Mayo Clinic, Rochester, MN.
†Sports Medicine, Mayo Clinic, Rochester, MN.
Address reprint requests to Bruce A. Levy, MD, Department of Orthopedic Surgery, Mayo Clinic, 200 First St SW, Rochester, MN 55905. E-mail: Stuart.Michael@Mayo.edu, Levy.Bruce@mayo.edu

Mechanism of Peroneal Nerve Injury

The vast majority of knee dislocations cause a bicruciate ligament injury along with one or both collaterals leading to multidirectional instability. Although both the tibial and peroneal nerves are at risk with a knee dislocation, the peroneal nerve accounts for the vast majority of nerve injuries reported. The rate of peroneal nerve injury following knee dislocation has been documented between 25% and 36%.[9,10]

The most common mechanism of peroneal nerve injury is a stretch injury as the result of a varus moment to the knee (Fig. 1). As a result, knee dislocations with associated posterolateral corner injuries are more prone to peroneal nerve injury.[11] The peroneal nerve is especially susceptible to injury due to several anatomic features. The peroneal nerve has a relatively poor vascular supply at the posterolateral knee, a thin layer of eprineural connective tissue, and limited mobility as it wraps around the fibular head (Fig. 2).[12]

Pathology and Prognosis

Peripheral nerve injuries exist on a spectrum from stretching to frank transection. Classically, peripheral nerve injuries are separated into the following 3 classes: neuropraxia, axonotmesis, and neurotmesis. The mildest of these injuries, neuropraxia, involves solely demyelination without Wallerian degeneration or axonal injury. Neurotmesis is the most severe injury type and represents complete disruption of the nerve. Axonotmesis is between these 2 types and does involve both demyelination and axonal injury with maintenance of the axonal sheaths.

http://dx.doi.org/10.1053/j.otsm.2015.06.006
1060-1872//

Neurotmesis and axonotmesis neural injuries would lead to distal Wallerian degeneration past the zone of injury. In contrast, a neuropraxia is transient in nature due to local ischemia and demyelination. Neuronal axons would begin regenerating 4-6 weeks after injury and would continue at a rate of 1 mm/day or 1 in/mo.[13] Permanent muscle fibrosis and motor endplate death occurs after 9-12 months denervation. In order to prevent these irreversible changes it is optimal that surgical intervention to restore nerve integrity occurs within 6 weeks to 6 months.[14-16] Historically, complete peroneal nerve injuries carry a dismal prognosis and require diligent patient follow-up. Although more advanced treatment strategies are continuously being developed, only 14%-40% of peroneal nerve injuries following knee dislocation would achieve functional recovery.[9] If the nerve injury is incomplete, observation is the treatment of choice and holds a significantly more optimistic prognosis. Recently, Krych et al[17] compared the patient outcome scores between multiligament knee injury patients with and without peroneal nerve palsy. The authors reported equivalent International Knee Documentation Committee and Lysholm scores between the 2 groups at late term follow-up. These results may highlight the fact that our current outcome measures for multiligamentous knee injuries fail to capture the true disability associated with peroneal nerve palsy.

Electromyogram (EMG) and nerve conduction studies are helpful in predicting prognosis but are not informative until 4-6 weeks after injury, due to the delayed nature of Wallerian degeneration.[14] Sensation loss is reliably much less severe with neuropraxia than that seen with neurotmesis and axonotmesis. EMG demonstrates distal motor conduction with neuropraxia but not be present with neurotmesis and axonotmesis.[18]

Treatment Options

Initial management of peroneal nerve palsy associated with knee dislocation typically consists of observation. Ultimately both surgical and nonsurgical options are employed in

Figure 1 Typical injury mechanism with varus moment at the knee resulting in peroneal nerve injury. (Color version of figure is available online.)

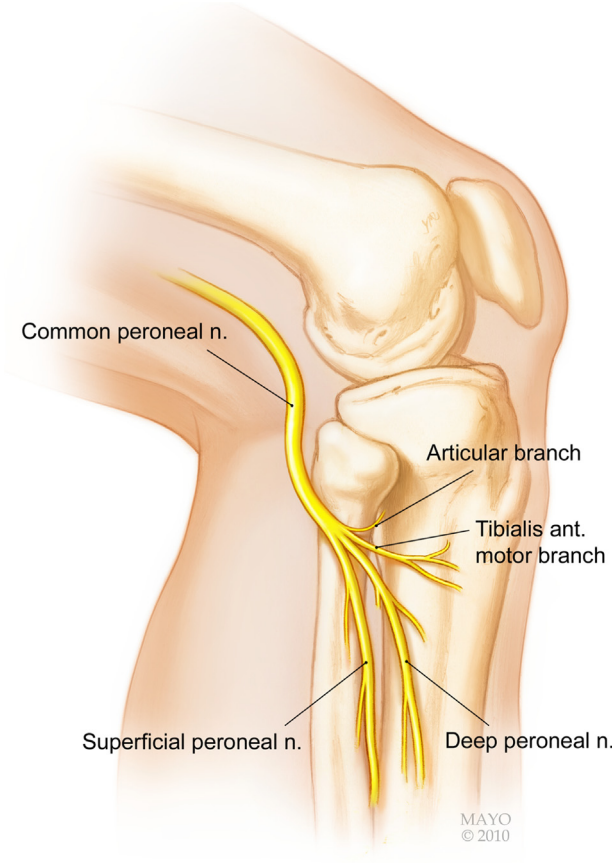

Figure 2 Peroneal nerve anatomy. (Color version of figure is available online.)

peroneal nerve palsy treatment. Often, the initial surgical management of lateral ligamentous and associated soft tissue injury allows direct visualization and decompression of the nerve. The goal of early management is to maintain mobility and prevent distal contractures. This is achieved by a combination of physical therapy and an ankle-foot orthosis (AFO). Physical therapy consists of strengthening of any anterior musculature that is still functioning and stretching of the posterior gastrosolieus complex and posterior capsule. The AFO prevents plantarflexion during ambulation and additionally stretches posterior to prevent equinovarus contracture.[9]

Timing of surgical intervention is critical. For lesions that do not recover by 6 months, surgical intervention should be considered. An irreversible, time-dependent degeneration of the motor endplates occurs and if the nerve is not reconstructed. Given that it would take approximately 3-6 months for the reconstructed nerve to regenerate to the motor endplates, ideal timing for nerve surgery is by 6 months after injury. Classic surgical options include neurolysis, primary nerve repair, nerve grafting, and tendon transfer. Recently, interest in local nerve transfer has also been described.[19]

Neurolysis and Nerve Grafting

It is common that the index surgical procedure to address the lateral ligamentous injury allows early exploration along with proximal and distal neurolysis of the peroneal nerve (Fig. 3).