Peroneal Nerve Palsy I: Evaluation and Diagnosis

Tyler R Morris, MD
Keith D Baldwin, MD, MPH, MSPT

Department of Orthopaedic Surgery
School of Medicine
University of Pennsylvania
Philadelphia, Pennsylvania, USA

Background

Palsy of the peroneal, or fibular nerve, can be a devastating injury and is the most common mononeuropathy of the lower extremity. The clinical manifestations of peroneal neuropathy range from mild cutaneous symptoms to widespread sensory loss, pain and foot drop leading to ambulatory dysfunction. Injury to the nerve can be acute or chronic, and may result from intrinsic dysfunction or extrinsic manifestations. While most commonly injured at the knee, dysfunction may occur due to compression or injury of nerve fibers anywhere from the spine to the hip or ankle. This article reviews the anatomy of the peroneal nerve, the clinical findings, etiology and evaluation of suspected peroneal nerve injury, and treatment options.

Anatomy

The peroneal nerve contains fibers from L4-S1 spinal roots from the lumbosacral nerve plexus and is a direct branch of the sciatic nerve. In the distal posterior thigh, the sciatic nerve branches into the tibial nerve and the common peroneal nerve; the latter courses proximally between the biceps femoris muscle and the lateral head of the gastrocnemius muscle. Exiting the popliteal fossa posterior to the knee joint, the nerve then wraps around the head of the proximal fibula deep to the peroneus longus muscle, which is a frequent site of compression or injury.

The proximal nerve supplies three articular sensory branches to the knee, as well as the lateral sural cutaneous nerve (LSCN). The LSCN supplies sensation to a portion of the posterolateral leg as well as anastomosing with the medial sural cutaneous nerve (a branch of the tibial nerve) to form the sural nerve.

Below the level of the head of the fibula, the common peroneal nerve then branches into superficial and deep branches; the superficial peroneal nerve (SPN) innervates the two muscles of the lateral leg compartment, the peroneus longus and brevis, which aid in eversion and plantar flexion of the foot. The SPN supplies cutaneous sensory innervation to the majority of the dorsal foot and the lateral ankle.

The deep peroneal nerve (DPN) travels deep to the peroneus longus muscle and innervates the anterior leg compartment, responsible for toe extension and foot dorsiflexion, namely the tibialis anterior, extensor hallicus longus, extensor digitorum longus, and peroneus tertius muscles. At the ankle the nerve supplies an articular branch to the joint itself, and then splits into a lateral and medial branch. The lateral branch innervates intrinsic foot extensors, namely the EDB and EHB, while the medial branch supplies cutaneous sensory innervation to the first web space of the dorsum of the foot.

Etiology

There are numerous ways in which the peroneal nerve can be injured, either directly or indirectly. Compression of the common peroneal nerve at the level of the fibular head is the most common modalities of injury to the nerve. This occurs due to the position over the bony prominence, combined with its relatively superficial position and frequent tethering of the nerve by the origin of the Peroneus Longus tendon. Tight clothing, thin body habitus, compressive casts/splints/pneumatic devices have all been reported to contribute to direct compression of the peroneal nerve at the level of the fibular head with resulting nerve palsy. Positioning during surgical intervention, as well as in cases of prolonged bedrest, has been shown to cause peroneal neuropathy secondary to prolonged direct compression. In addition, direct compressive injury due to rare mass lesions, varus malalignment of the knee (causing repetitive stretch) and lateral osteophytes (causing direct traction injury) has been reported in the literature.

Direct injury to the peroneal nerve, as well as its antecedent and descending branches, can occur due to penetrating wounds, iatrogenically during surgery, compartment syndrome of the leg, or musculoskeletal injuries with displaced osseous fragments. A concomitant injury rate to the peroneal nerve in tibial plateau fractures has been reported in as high as 1% of cases, with nerve injury occurring with both initial trauma and iatrogenically during surgical fixation. Injuries to antecedent nerve fibers in the sciatic nerve can occur in the setting of acetabular and hip fractures, as well as posterior hip dislocation.

Furthermore, peroneal nerve injury has been reported in the setting of acute ligamentous injury to the knee, most commonly when seen

with rupture of the anterior cruciate ligament. A recent study has shown that posterolateral corner injuries of the knee with concomitant bicep avulsion or fibular head fractures have a 90% incidence of peroneal nerve displacement, which could potentially predispose to injury during surgical exploration.

The peroneal nerve can also experience dysfunction in the setting of acute stretching or traction injuries. This can occur with dislocations of the knee as the nerve is stretched over the posterior condyle of the femur, and can frequently coincide with vascular compromise. Additionally, total knee arthroplasty does have a rare complication of peroneal nerve palsy due to increased traction of the nerve, either during surgery or with post-operative positioning. While rare, release of flexion contractures or correction of a valgus deformity in total knee arthroplasty places new demands on the excursion distance of the peroneal nerve fibers and may cause clinical symptoms. A similar concept is seen in the sciatic nerve with total hip arthroplasty procedures that produce increased leg length and offset.

Ankle sprains have also been shown to cause direct injury to the peroneal nerve, proportional to the grade of the sprain itself. The typical injury pattern of plantar flexion and inversion of the ankle places stretch not only on the everting muscles of the lateral compartment but also on the superficial peroneal nerve, due to its proximal tethering at the level of the fibula. While the use of potentially compressing splints and casts is a further cause of peroneal neuropathy, a study of 66 patients with ankle sprain showed that over eighty percent of patients with a grade III ankle sprain had electrodiagnostic evidence of peroneal nerve injury 2 weeks later.

Clinical findings

Depending on the level of injury, different clinical manifestations can be seen in peroneal nerve injury. Injuries to the L5 nerve root itself can frequently be seen with disc herniation, and may be accompanied by low back pain as well as peripheral symptoms to the nerve. In addition, patients may complain of weakness of ankle inversion, controlled primarily by the tibialis posterior and the tibial nerve.

Injuries to the proximal nerve fibers, either as they exit the spinal column or in the sciatic nerve, will cause a global injury that affects the entire peroneal nerve and its descending branches, causing widespread cutaneous and muscular deficits, and may vary depending on the location of the injury and the specific fibers affected.

A proximal injury to the common peroneal nerve itself will cause foot drop and an inability to extend the toes, due to the lack of innervation of the anterior compartment by the DPN. The SPN will also be affected, causing a deficit in foot eversion due to palsy of the SPN, which innervates the lateral compartment of the leg. Patients with foot drop will frequently complain of a "slapping gait," and that their toes drag the ground while walking. In addition, cutaneous sensation will be lost over the dorsum of the foot and the lateral ankle.

As many common peroneal nerve injuries occur at or below the level of the fibular head, it is important to differentiate if the the lateral sural cutaneous nerve (LSCN) is still intact. Absense of LSCN will cause a loss of cutaneous sensation to the posterolateral proximal calf, as well as affect fibers in the Sural Nerve.

Injuries to SPN and DPN will cause discrete clinical manifestations that correspond to their muscular compartments and sensory maps. Superficial peroneal neuropathy will cause cutaneous symptoms of the lateral ankle and dorsal foot, and will result in an inability to evert the foot. Conversely, DPN palsy will cause sensory deficits of the first dorsal webspace, as well as a lack of dorsiflexion and toe extension resulting in the characteristic "foot drop." An isolated lesion to the distal DPN may cause a deficit in toe extension only.

Testing modalities

When a peroneal nerve injury is suspected, a detailed history and physical exam is paramount to proper diagnosis. Cutaneous manifestations such as burning, tingling, and numbness may be seen upon provocative testing such as Tinel's sign, while gait and strength analysis may show muscular deficits when compared to the contralateral side. Reflexes of the lower legs should be tested and compared, as the presence of a pathological reflex (i.e. a positive Babinski reflex) may indicate a central nerve lesion. However, several modalities exist to elucidate the precise cause and level of possible injury.

Imaging studies such as X-rays, CT's and MRI's are particularly useful in the diagnosis of nerve injuries. Spinal conditions such as herniated disks as well as mass lesions may require an MRI, while arthritis, fractures, or dislocations of the knee and surroundings structures can cause direct injury to the Common Peroneal Nerve and can be elucidated on Xray or CT-scan.

In the absence of extrinsic injuries to the nerve, it may be necessary to consider intrinsic factors involved in nerve palsy. Systemic abnormalities such as diabetes, polyarteritis nodosa, Hereditary Neuropathy with Liability to Pressure Palsy (HNPP), and Charcot-Marie-Tooth can cause demyelination and dysfunction of the nerve at any point along its length as well as predispose to increased susceptibility to pressure palsy. Electrical studies such as EMG (electromyography) or NCS (nerve conduction study) can test the ability of electrical impulses to travel along individual muscles and nerves, respectively. Nerve conduction studies are particularly useful in determining if neuropathy is due to demyelination (which shows slowing of action potential propagation) or axon loss (illustrated by decreased amplitude of action potentials).

Biopsy of a muscle or nerve itself can be considered definitive proof of instrinsic dysfunction; however, the above testing modalities have all but obviated the need for invasive biopsy.

Conclusion

Peroneal nerve palsy is a common lower extremity injury after trauma. It requires a thoughtful approach by the treating physician, and in **Part II** we will discuss treatment options.

For a full list of references, please contact the primary author.