Peroneal Nerve Palsy: Evaluation and Management

Abstract

Peroneal nerve palsy is the most common entrapment neuropathy of the lower extremity. Numerous etiologies have been identified; however, compression remains the most common cause. Although injury to the nerve may occur anywhere along its course from the sciatic origin to the terminal branches in the foot and ankle, the most common site of compressive pathology is at the level of the fibular head. The most common presentation is acute complete or partial foot drop. Associated numbness in the foot or leg may be present, as well. Neurodiagnostic studies may be helpful for identifying the site of a lesion and determining the appropriate treatment and prognosis. Management varies based on the etiology or site of compression. Many patients benefit from nonsurgical measures, including activity modification, bracing, physical therapy, and medication. Surgical decompression should be considered for refractory cases and those with compressive masses, acute lacerations, or severe conduction changes. Results of surgical decompression are typically favorable. Tendon and nerve transfers can be used in the setting of failed decompression or for patients with a poor prognosis for nerve recovery.

Common peroneal nerve (CPN) palsy is associated with the onset of acute and progressive foot drop and is the most common compressive neuropathy of the lower extremity. When onset is atraumatic, compressive pathology may be identified anywhere along the course of the nerve, from the peroneal division of the sciatic nerve to the terminal branches in the foot and ankle. The most common site of compression is located at the bony prominence of the fibular neck. As with all compressive neuropathies, the key to a successful outcome is early identification and treatment.

Peripheral Nerve Structure and Pathophysiology

When evaluating patients with disorders of the peripheral nerves, the clinician must take into account the structural composition of the nerve and the pathophysiology of nerve injury. Individual myelinated fibers and groups of unmyelinated fibers are ensheathed in a layer of endoneurium. These fibers are grouped into fascicles covered by perineurium and combine to form peripheral nerves that are covered with two layers of epineurium. In 1942, Seddon described a classification system for peripheral nerve injuries that was based on the degree of structural disruption and the sequelae that follow (Figure 1). In the Seddon classification, the least severe injury is neurapraxia, which involves myelin damage with delayed conduction and intact components (Table 1). Axonotmesis is characterized by axon discontinuity, with the development of Wallerian degeneration being the key
component. Neurotmesis, the most severe nerve injury, is complete disruption of the nerve, including separation of the axons and the epineurium. Although full or partial spontaneous recovery is expected with neurapraxia and axonotmesis, minimal recovery is expected with neurotmesis. In 1951, Sunderland modified this classification by categorizing nerve injury by five degrees and incorporating clinical ramifications as well as potential nerve healing (Table 1).

With acute nerve compression, dysfunction results from ischemic changes that may cause a conduction block. Varying degrees of intraneural edema are seen in acute cases, but this typically resolves over time, after the causative factor is addressed. With chronic compression or compression that lasts >28 days, slowing of nerve conduction is seen along with an endoneurial inflammatory response. This response results in the formation of fibroblast-moderated scar tissue that may further exacerbate the compression. Axonal degeneration is seen with compression lasting 4 weeks.
Anatomy

The CPN is derived from the nerve roots of L4, L5, S1, and S2 as a part of the sciatic nerve. In the thigh, the peroneal division of the sciatic nerve is located in the lateral and posterior portion of the nerve. This anatomic position leaves the peroneal division more vulnerable than its tibial counterpart to stretch or direct injury during the exposure for hip arthroplasty.10

The peroneal division of the sciatic nerve travels through the posterior compartment of the thigh and supplies the short head of the biceps femoris muscle before separating from the tibial nerve in the upper popliteal fossa. Here, it becomes the CPN and crosses posterior to the lateral head of the gastrocnemius muscle through the posterior intermuscular septum and becomes subcutaneous while it curves around the head of the fibula deep to the peroneus longus muscle (Figure 2). The lateral cutaneous nerve of the calf branches off here, and the nerve subsequently divides into the superficial peroneal nerve (SPN), deep peroneal nerve (DPN), and an articular branch. The DPN lies directly on the bony surface of the fibula and wraps around it, thus predisposing it to compression in this area.11

The SPN travels distally in the lateral compartment of the leg, providing motor innervation to the peroneus longus and peroneus brevis muscles and sensory innervation to the lateral lower leg and the dorsum of the foot. The nerve lies on the cortex of the fibular shaft in the mid leg and then pierces the fascia anterior to the bone 10 to 12 cm above the tip of the fibula. On the anterior aspect of the distal fibula, the nerve is vulnerable to injury during fixation of an ankle fracture. Finally, the SPN terminates distally as the intermediate and medial dorsal cutaneous nerves.

The DPN travels for 3 to 4 cm along the anterior cortex of the fibula and then courses distally. It then courses just anterior and medial to the intermuscular septum between the anterior and lateral compartments as it travels with the anterior tibial artery. This is a potential source of DPN entrapment when the nerve is stretched. The DPN supplies motor innervation to the foot and toe dorsiflexors, including the tibialis anterior (TA), extensor digitorum longus (EDL), extensor hallucis longus, peroneus tertius, and extensor digitorum brevis (EDB) muscles. The nerve passes under the extensor retinaculum at the ankle and then terminates as a sensory branch in the dorsal first web space.

Etiology

The etiologies for peroneal nerve palsy are numerous. Although compressive etiology remains the most common cause, many other factors contribute to injury. Traumatic causes include knee dislocation, severe ankle inversion injuries, lacerations, and direct blunt trauma. These traumatic injuries are typically associated with poorer outcomes.12,13 The link between diabetes mellitus and lower extremity neuropathies (eg, polyneuropathy, mononeuropathy) has been well established.14 Iatrogenic injury is common as well, with acute foot drop often seen as a result of surgery about the hip, knee, and ankle; positioning during anesthesia; prolonged bed rest; casting; bracing; compression wrapping; and the use of pneumatic compression devices.15-18

Several risk factors for compressive peroneal neuropathy have been described. A recent history of significant weight loss has been shown to be related to nerve compression at the fibular head and may be associated with the loss of subcutaneous fat at this level.19 Habitual leg crossing and prolonged squatting are also associated with an increased risk of peroneal palsy and acute foot drop.1 Compressive masses, including both intraneural and extraneural lesions, may manifest with acute or progressive onset of symptoms (Figure 3). Finally, similar to other compressive peripheral neuropathies, entrapment of the CPN within the fibular tunnel by a fibrous band at the origin of the peroneus longus muscle is a common finding and must not be overlooked during nerve decompression.20
In patients with an injury to the peroneal nerve, clinical presentation varies based on the location and severity of the injury and the presence of anatomic variations. Most commonly, patients report the classic symptoms of foot drop or catching the toes while ambulating. Foot drop can develop acutely or over a period of days to weeks, depending on the etiology, and can be complete or partial in severity. Numbness or dysesthesia may also be present along the lateral leg, dorsal foot, and/or the first toe web space (Figure 4). Pain may be present in some cases (eg, traumatic wounds, compressive lesions), but it is not a common complaint.

Presentation

In patients with an injury to the peroneal nerve, clinical presentation varies based on the location and severity of the injury and the presence of anatomic variations. Most commonly, patients report the classic symptoms of foot drop or catching the toes while ambulating. Foot drop can develop acutely or over a period of days to weeks, depending on the etiology, and can be complete or partial in severity. Numbness or dysesthesia may also be present along the lateral leg, dorsal foot, and/or the first toe web space (Figure 4). Pain may be present in some cases (eg, traumatic wounds, compressive lesions), but it is not a common complaint.

Physical Examination

The clinical examination is directed by the symptoms reported by the patient and requires a thorough understanding of the relevant anatomy. Gait assessment may provide important clues to the etiology of the symptoms. A patient with weakened or paralyzed ankle dorsiflexors may ambulate with a steppage gait in which the ipsilateral knee is lifted higher than normal during the swing phase to avoid dragging the toes on the ground, followed by slapping the forefoot on the ground after heel strike. If injury to the peroneal nerve is suspected, a thorough examination should be performed, focusing on the elements of each component of the nerve’s innervation. Numbness or dysesthesia in the upper lateral leg indicates a lesion proximal to the fibular head, which may represent involvement of the sciatic nerve or lumbosacral nerve root. Tibial nerve involvement should be ruled out in this scenario by testing foot inversion with the foot passively dorsiflexed because inversion is normally weak in a plantarflexed foot. If examination reveals no compromise to the long head of the biceps femoris muscle, knee flexion strength will likely test normal despite possible weakness of the short head. Palpation may reveal decreased muscle contraction over the short head, although this may be difficult to appreciate by physical examination alone. Involvement of both the peroneal and tibial nerve divisions of the sciatic nerve may result in decreased knee flexion, ankle plantar flexion, and toe flexion in addition to weakness of the anterior and lateral compartments of the leg. Muscle stretch reflex will typically be normal unless severe damage to the sciatic nerve is present, and abductor strength testing may help the clinician to identify a peroneal nerve injury resulting from L5 radiculopathy.

Decreased or abnormal sensation in the lower lateral leg and the dorsum of the foot suggests involvement of the SPN or the portion of the sciatic nerve in those areas. Similarly, altered sensation in the dorsal aspect of the first web space of the foot suggests involvement of the DPN fibers. Weakness of foot eversion (SPN involvement) or foot/toe dorsiflexion (DPN involvement) may be present in isolation; however, appreciation of both of these findings suggests a lesion involving the CPN fibers. When injury about the fibular head occurs, the DPN may be severely affected. Tenderness or a Tinel sign near the fibular head may be present, but reflexes are typically preserved.

Diagnostic Studies

Imaging

When clinical examination indicates a potential injury to the peroneal nerve, plain radiography should be considered as part of the initial workup. The close proximity of the CPN to the fibular neck as well as its superficial location makes it susceptible to injury secondary to direct trauma and impingement from both soft-tissue and bony sources. CT may be used to further evaluate bony abnormalities.
MRI and ultrasonography should be considered to evaluate for potential soft-tissue sources of impingement or masses.\(^1\)\(^2\) (Figure 5).

**Electrodiagnostic Studies**

Nerve conduction velocity (NCV) studies and electromyography (EMG) are valuable tools for diagnosing suspected peroneal nerve palsy. These studies help the clinician evaluate the motor and sensory axons of the peroneal nerve and its branches. They also are useful for localizing the site of injury, determining the severity of a lesion, and monitoring recovery after a nerve injury has been identified.\(^2\)\(^2\) An electrophysiology study should be performed to obtain a baseline in all patients who present with new-onset foot drop; the study may be repeated every 3 months to monitor for improvement or deterioration. In the setting of traumatic injury or postoperative palsy, immediate neurodiagnostic tests are not warranted and should not be performed for 2 to 6 weeks.

Motor nerve conduction studies should be performed to evaluate the EDB and TA muscles, with stimulation applied above and below the fibular head. These results should be compared with those of the contralateral extremity. Because the EDB may be innervated by an accessory peroneal nerve from the SPN in approximately one third of the population, stimulation of the peroneal musculature may be performed, as well.\(^2\)\(^4\)

Sensory nerve conduction studies should be used to evaluate both sensory branches of the SPN at the level of the ankle and the DPN. However, SPN conduction study findings may be normal in the setting of a neuropathy of the CPN secondary to the increased vulnerability of the deep nerve fibers to stretch and compression.\(^2\)\(^2\) These studies can also be specifically directed to further delineate findings from previous studies and the clinical examination. For example, conduction studies of the posterolateral cutaneous nerve of the calf may be performed in the setting of numbness in that area. Studies involving the tibial and sural nerve distributions may be performed to help rule out other causes of clinical findings, including plexopathy or injury to the sciatic nerve.\(^2\)\(^4\)

Needle EMG provides further detail to help the clinician identify the location and severity of peroneal nerve lesions. Muscles innervated by both the DPN and SPN should be tested, including the TA muscle, which is most commonly affected in patients with peroneal palsy.\(^2\)\(^2\) Studies may also be performed on the short head of the biceps and a tibial-innervated muscle distal to the knee to identify more proximal lesions and/or injury to the sciatic nerve. If the findings are abnormal, investigation should be extended to include more proximal sciatic-innervated muscles, gluteal muscles, and lumbosacral paraspinal muscles.

The results of NCV and EMG studies can help to dictate the course of treatment. When these findings suggest a more advanced disease state, surgical intervention may be indicated. At our institution, when EMG/NCV studies show severe changes, including severe conduction delay across the CPN (>50%) on NCV and evidence on EMG of substantial disruption of the CPN innervation to the musculature, surgical nerve decompression is indicated and may increase the likelihood of a favorable outcome.

**Management**

**Nonsurgical**

When left untreated, CPN palsy is associated with foot drop, equinovarus deformity, and limb disability ranging from 30% to 35%.\(^2\)\(^5\) Initial management of peroneal nerve palsy involves a nonsurgical approach because partial or full function often returns over time. In addition to knowledge of the causative positions or activities (eg, squatting, strenuous exercise) that contribute to peroneal nerve palsy, activity modification, such as cessation of leg crossing, is crucial. Padding of the prominent fibular head may be helpful, particularly after a direct traumatic injury, and may be worn at night to prevent compression while sleeping. Night
A more aggressive approach should be used for nerve injuries of any severity (including neurapraxia) that present with complete motor or sensory loss. Surgical exploration and decompression should be considered when a rapidly deteriorating lesion is present or there are no signs of improvement within 3 months. For open injuries with a suspected nerve laceration, the nerve should be explored within 72 hours, if possible, and if minimal gapping is found at the site of injury, primary repair should be attempted with an epineurial or fascicular technique. A clinical study of surgical nerve repair showed that there was no single superior epineurial or fascicular technique. In the setting of wound contamination, débridement of the wound and nerve edges, suturing of the local soft tissues, and a repair performed within 2 to 7 days is acceptable. If primary repair is not possible secondary to significant gapping or nerve damage, nerve grafting is indicated. This can be done as a primary procedure but is more commonly done as a delayed procedure. Autologous grafting is the standard of care, with sural nerve graft most commonly used. Alternatively, nerve conduits, including veins, bioabsorbable tubes, and pseudosheaths, may be used. These conduits have been shown to regenerate nerves across short gaps (<3 cm), with reported results comparable to those of autograft. Although the efficacy of nerve transfer remains unproven, this technique is an emerging option for irreparable nerve injuries, such as those with segmental nerve loss or a long regeneration distance. The principles of nerve transfer are similar to those of tendon transfer; an attempt is made to select the most synergistic nerve, and in the case of irreparable CPN nerve injury, this is most commonly a branch of the tibial nerve.

Splints may also be used to prevent contractures in cases of complete foot drop. Rehabilitation, including physical therapy and the use of orthotic devices, may be effective for managing the symptoms of foot drop and eversion weakness related to peroneal palsy. For significant dorsiflexion weakness, a custom ankle-foot orthosis may be helpful for foot clearance during ambulation. Physical therapy should initially focus on stretching the contralateral muscle groups, including the foot plantar flexors and inverters. In the setting of substantial muscle weakness, electrical stimulation may be used to initiate muscle contractions. Progressive strengthening of the dorsiflexors and evertors should begin once autonomous muscle contraction is present.

**Surgical**

**Acute Injuries**

Acute nerve injuries, including contusions, stretch injuries, lacerations, and crush injuries, should be assessed to determine the degree of functional loss. Injuries with neurapraxia should be monitored initially because excellent results have been obtained with nonsurgical management. A more aggressive approach should be used for nerve injuries of any severity (including neurapraxia) that present with complete motor or sensory loss. Surgical exploration and decompression should be considered when a rapidly deteriorating lesion is present or there are no signs of improvement within 3 months. For open injuries with a suspected nerve laceration, the nerve should be explored within 72 hours, if possible, and if minimal gapping is found at the site of injury, primary repair should be attempted with an epineurial or fascicular technique. A clinical study of surgical nerve repair showed that there was no single superior epineurial or fascicular technique. In the setting of wound contamination, débridement of the wound and nerve edges, suturing of the local soft tissues, and a repair performed within 2 to 7 days is acceptable. If primary repair is not possible secondary to significant gapping or nerve damage, nerve grafting is indicated. This can be done as a primary procedure but is more commonly done as a delayed procedure. Autologous grafting is the standard of care, with sural nerve graft most commonly used. Alternatively, nerve conduits, including veins, bioabsorbable tubes, and pseudosheaths, may be used. These conduits have been shown to regenerate nerves across short gaps (<3 cm), with reported results comparable to those of autograft.

**Compressive Masses**

When a mass is found to be the cause of compression, a thorough workup and evaluation should be performed before invasive treatment is initiated. As with any tumor, the clinician must assess the mass to determine whether it is malignant. Excision of a compressive mass is similar to excision of a mass elsewhere in the body, and lesions causing significant or progressive motor loss should be removed, as well. Extraneural lesions, such as fibular osteochondromas, vascular malformations, or extraneural cysts, should be resected in the standard fashion. Compression of the peroneal nerve by such benign extraneural masses is rare but should nonetheless be included in the differential diagnosis.

Intraneural lesions should be approached with caution because surgical dissection of these lesions is more meticulous and difficult (Figure 6). Referral to an orthopaedic oncologist or a specialist skilled in microsurgery should be considered. An experienced surgeon should perform resection of schwannomas and neurofibromas using an appropriate technique at the fascicular level and intraoperative nerve monitoring. Nerve monitoring can assist the clinician in localizing the lesion and confirming nerve function after dissection. Intraneural ganglion cysts originate from the superior
tibiofibular joint and should be dissected free, with the stalk traced and disconnected from the joint to prevent recurrence. Good outcomes can be obtained with surgical management of intraneural lesions by an experienced surgeon. Suspected malignancy should be confirmed with a frozen section unless a biopsy has been performed.

**Idiopathic/Postoperative Compression**

Surgical decompression should be considered for management of compressive lesions at the fibular head or fibular tunnel and nerve palsies that present after knee arthroplasty or high tibial osteotomy if no improvement is seen after a trial of nonsurgical management. Although the time frame for nonsurgical management is controversial, most authors agree that a minimum trial of 3 months should be attempted because improvement in nerve function may be seen for up to 6 months. However, if the patient fails to show clinical signs of improvement or if motor loss is rapidly progressive, decompression is warranted. Similarly, when EMG and/or NCV studies show evidence of severe conduction loss or disruption of motor innervation, surgical intervention should be more strongly considered over nonsurgical treatment. Favorable outcomes have been reported with the use of neurolysis, with return of function reported in up to 97% of cases in one study.

**Authors’ Preferred Technique**

Our preferred technique for CPN release at the proximal fibula provides comprehensive decompression and adequate exposure for concomitant procedures, such as excision of a mass. The patient is placed in the lateral recumbent position with the affected leg up, using a bean bag to secure the pelvis. The fibular head is marked, and the CPN can be palpated beneath the subcutaneous tissues just distal to this landmark (Figure 7). An oblique or curvilinear incision is made beginning posterior to the fibular head near the anterior popliteal fossa and courses anteriorly and distally for approximately 6 cm. Dissection is carried through the subcutaneous tissues, with care taken to avoid the lateral cutaneous nerve in the calf in the proximal aspect of the incision.

The CPN is palpated just distal to the fibular head, and the fascia overlying the nerve is incised and opened. The nerve is cautiously dissected and inspected in this region and then tagged with a vessel loop. The fascia overlying the peroneus longus muscle is then incised in line with the incision. After the nerve is exposed deep to the peroneus longus, three intermuscular septal planes are encountered and released. The posterior crural intermuscular septum is the first intermuscular plane encountered, and it is the most important to release because it is the most common site of nerve compression. When the nerve is free distal to the bifurcation into the SPN and DPN, neurolysis is adequate.
Tendon Transfer

Tendon transfer also may be used to restore function to the foot and ankle in refractory cases. This procedure typically involves transfer of the posterior tibial tendon (PTT) to the lateral cuneiform to restore ankle dorsiflexion. Goh et al. compared two methods of PTT transfer: (1) subcutaneous transfer around the medial aspect of the tibia and (2) transfer of the tendon through the interosseous membrane to the dorsal foot. Biomechanically, the transinterosseous membrane technique provided superior ankle dorsiflexion with minimum pronation. Although many variations exist, techniques for transferring the PTT through the interosseous membrane typically involve harvesting the tendon at its insertion, delivering it through a proximal incision, passing it through the interosseous membrane, and anchoring it into the dorsal midfoot (Figure 8).

An incision is made just distal to the medial malleolus, extending approximately 5 cm to expose the PTT. The tendon is then harvested subperiosteally from distal to proximal at the naviculocuneiform joint to ensure adequate tendon length. A second incision is made approximately 15 cm proximal to the tip of the medial malleolus. The PTT is delivered through the proximal incision and tagged with suture to facilitate transfer. The skin is marked for a 5-cm incision along the anterior border of the fibula. The PTT is transferred from the medial proximal incision to the lateral incision. (Reproduced with permission from Ho B, Khan Z, Switaj PJ, et al: Treatment of peroneal nerve injuries with simultaneous tendon transfer and nerve exploration. J Orthop Res Surg 2014;9:67-77.)

Intraoperative photographs of the ankle demonstrating the transfer of the posterior tibialis tendon (PTT) to the lateral cuneiform to restore ankle dorsiflexion. A, An incision is made just distal to the medial malleolus, extending approximately 5 cm to expose the PTT, and the tendon is harvested subperiosteally from distal to proximal at the naviculocuneiform joint to ensure adequate tendon length. B, A second incision is made approximately 15 cm proximal to the tip of the medial malleolus. C, The PTT is delivered through the proximal incision and tagged with suture to facilitate transfer. D, The skin is marked for a 5-cm incision along the anterior border of the fibula. E, The PTT is transferred from the medial proximal incision to the lateral incision. (Reproduced with permission from Ho B, Khan Z, Switaj PJ, et al: Treatment of peroneal nerve injuries with simultaneous tendon transfer and nerve exploration. J Orthop Res Surg 2014;9:67-77.)

Figure 8
Care must be taken to pass directly posterior to the tibia to prevent damage to the neurovascular bundle. The tendon is then passed subcutaneously to a dorsal incision made over the lateral cuneiform. The PTT is then anchored into the lateral cuneiform using interference screw fixation.

One potential downfall of this method is inadequate tendon length, which may require maximal dorsiflexion of the ankle or changing the location of the tendon insertion to achieve a stable transfer. Tendon-to-tendon suturing is an alternative to this technique. Direct tendon-to-tendon suturing, however, often decreases dorsiflexion strength because of dispersion of the force supplied by the PTT, and it may lead to an unbalanced foot. Vigasio et al described a technique in which the PTT is transferred to the anterior tibial tendon (rerouted through a new insertion on the lateral cuneiform) and the flexor digitorum longus is transferred to the EDL and extensor hallucis longus tendons (Figure 9). The technique was performed in 16 patients with complete CPN palsy, and at a minimum follow-up of 24 months, the authors noted good to excellent results in >80% of their patients. The authors concluded that this method is a reliable means for obtaining balanced dorsiflexion of the foot and toes, eliminating the need for orthoses during ambulation.

The timing of the procedure is somewhat controversial. Traditionally, this procedure has been performed in a delayed fashion to await possible return of nerve function. This often resulted in the patient waiting approximately 1 year after the initial injury or attempted nerve repair to monitor neurologic activity. Some studies, however, suggest that tendon transfers should be considered as early as 3 to 4 months after the initial injury to prevent development of significant equinus contracture and atrophy of the ankle dorsiflexors and to improve regeneration of nerve fibers when tendon transfer is combined with nerve repair and/or grafting procedures.

**Summary**

CPN palsy is the most common compressive neuropathy of the lower extremity. The most common presentation is acute foot drop, although symptoms may be progressive and may include sensory loss or pain. Most instances of CPN palsy improve or resolve over time with nonsurgical measures. Surgery is typically reserved for refractory cases, although some circumstances (eg, EMG and/or NCV findings, the presence of a compressive mass, an acute open injury) warrant earlier intervention. When nerve decompression or repair is unsuccessful or the prognosis for nerve recovery is poor, nerve or tendon transfers may restore some function.

**References**

Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, reference 10 is a level II study. References 4, 7, 12, 14, 16, 29, 34, 35, and 40 are level III studies. References 13, 17, 19, 31-33, and 36-38 are level IV studies. References 6, 15, 18, and 30 are level V expert opinion. References printed in bold type are those published within the past 5 years.

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