Peroneal neuropathy

Mamatha Pasnoor MD (Dr. Pasnoor of the University of Kansas Medical Center received medical advising fees from Alexion Pharmaceuticals, CSL Behring, and TerumoBCT and a consulting fee from Momenta Pharmaceuticals.)

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Introduction

This article includes discussion of peroneal neuropathy, lateral popliteal mononeuropathy, lateral popliteal nerve injury, lateral popliteal nerve palsy, peroneal mononeuropathy, peroneal nerve injury, and peroneal nerve palsy. The foregoing terms may include synonyms, similar disorders, variations in usage, and abbreviations.

Overview

This update provides additional information on usefulness of ultrasonography in the diagnosis of peroneal neuropathy.

Key points

- Peroneal mononeuropathy is the most common entrapment neuropathy in the lower extremity.
- Peroneal mononeuropathy presents with foot drop, sensory loss on dorsum of foot and lateral leg, and, rarely, pain.
- The most common etiology is external compression and trauma.
- Electrodiagnostic studies are useful for localization and assess the severity of peroneal neuropathy. High-resolution sonography also helps with assessment of peroneal neuropathy.

Historical note and terminology

The most common entrapment neuropathy in the lower extremity is common peroneal mononeuropathy at the fibular head. This syndrome can occur in both adults and children and also, rarely, in the neonate. Almost all patients presenting with common peroneal neuropathy will present with foot drop. Although the most common site of nerve entrapment is the fibular head, other sites within the calf, ankle, and foot may result in a portion of a common peroneal neuropathy syndrome.

Clinical manifestations

Presentation and course

Peroneal neuropathy usually presents with acute foot-drop. However, in some instances the foot-drop develops subacutely over days or even weeks. Foot-drop may be complete, with failure to dorsiflex the ankle and toes, or partial. The foot may get trapped or may cause the patient to fall. Numbness of the leg usually involves the dorsum of the foot and lower lateral leg; pain, however, is rare and, when present, is deep and ill-defined, usually located around the knee. On examination, the weakness is restricted to ankle eversion and ankle and toe dorsiflexion. Ankle inversion, toe flexion, and plantar flexion are normal. An apparent weakness of ankle inversion is common with complete foot-drop, since inversion is best obtained with the foot slightly dorsiflexed. In a large study of common peroneal neuropathy (Katirji and Wilbourn 1988), 43% of cases were clinically misdiagnosed by physicians, including neurologists. To avoid this misleading sign in a patient with foot-drop, the ankle should be dorsiflexed passively to 90 degrees while testing ankle inversion. Hypesthesia to touch and pain is limited to the lower two-thirds of the lateral leg and dorsum of foot. Tinel sign may be elicited by percussion of the peroneal nerve around the fibular neck. Knee and ankle reflexes are normal. The hamstrings, glutei, and quadriceps are normal. In selective deep peroneal neuropathies, which are much less frequent than common peroneal neuropathies, the sensory manifestations are lacking (except occasionally in the first web space), and ankle eversion is normal.

Overall, patients with common peroneal neuropathy complain of motor deficit in more than 99% of cases, sensory...
symptoms in 88% of cases, and pain in 20% of cases (Aprile et al 2005a).

**Prognosis and complications**

It is difficult to prognosticate based on clinical evaluation only. Since the vast majority of cases have prominent axonal loss, the prognosis in general is guarded and protracted. Also, in contrast to common belief, perioperative peroneal neuropathies, including the subgroup following anesthesia for coronary by-pass surgery, are frequently axonal and not due to demyelination (neurapraxia).

Electrodiagnostic evaluation is essential in the prognostic estimation of these lesions since they separate lesions with segmental demyelinating from those with axonal loss. Segmental demyelination has good prognosis with recovery occurring in 6 weeks to 8 weeks. However, the prognosis of axonal loss lesions is variable since it is dependent on the degree of structural nerve injury as defined by Sunderland (Sunderland 1991) and Seddon (Seddon 1943).

**Table 1. The Degree of Structural Injury as Defined by Sunderland and Seddon**

<table>
<thead>
<tr>
<th>Sunderland</th>
<th>Seddon</th>
<th>First degree</th>
<th>Second degree</th>
<th>Third degree</th>
<th>Fourth degree</th>
<th>Fifth degree</th>
</tr>
</thead>
<tbody>
<tr>
<td>Electrophysiology</td>
<td>Neurapraxia</td>
<td>Conduction block</td>
<td>Axonal loss</td>
<td>Neuromesis</td>
<td>Axonal loss</td>
<td>Neuromesis</td>
</tr>
<tr>
<td>Pathophysiology</td>
<td>Segmental demyelination</td>
<td>Loss of axons with intact supporting structures</td>
<td>Loss of axons with disrupted endoneurium</td>
<td>Loss of axons with disrupted endoneurium and perineurium</td>
<td>Unlikely without surgical repair</td>
<td>Impossible without surgical repair</td>
</tr>
<tr>
<td>Prognosis</td>
<td>Excellent recovery, usually in 2 to 3 months</td>
<td>Slow recovery, dependent on sprouting, reinnervation</td>
<td>Protracted and can fail due to misdirected axonal sprouts</td>
<td>Axonal loss</td>
<td>Axonal loss</td>
<td>Axonal loss</td>
</tr>
</tbody>
</table>

With common peroneal nerve injury secondary to knee dislocation, if the nerve is in continuity with less than 7 cm of nerve involved, complete recovery may occur over 6 to 18 months (Niall et al 2005). More partial recovery occurs in patients with more extensive common peroneal nerve lesions.

Also, as with other peripheral nerve injuries, partial axonal lesions fair better than complete lesions, since local sprouting reinnervates muscle fibers effectively. Also, children with peroneal nerve lesions do well (Jones et al 1993).

The nature of the traumatic injury in common peroneal neuropathy also impacts on outcome. Sharp injuries and severe dislocations of the knee tend to have an excellent recovery, whereas crush injuries and gunshot wounds lead to a good recovery much less often (Garozzo et al 2004). Common peroneal neuropathy due to prolonged squatting or posture tends to be a benign lesion and recovers with conservative therapy over 9 weeks in more than 90% of patients with avoidance of precipitating factors (Sangwan et al 2004). Complete recovery of peroneal neuropathy following hip arthroplasty ranges from 50% to 68% (Nercessian et al 2005). The mean time to recovery was approximately one year for partial peroneal palsy and one and one-half years for complete palsy. Weight of the patient has been observed as an important prognostic factor (Park et al 2013).

In nontraumatic acute-onset compressive peroneal mononeuropathy, patients with denervation on needle electromyography, older age, and severe initial weakness have a poorer prognosis (Bsteh et al 2013).

**Clinical vignette**

A 76-year-old woman underwent elective coronary bypass artery surgery for unstable angina. After waking from general anesthesia, she noted weakness of the left foot and numbness of dorsum of foot. She had no pain. When examined 2 months later, there was toe dorsiflexion, foot eversion, and a significant weakness of left ankle (Medical Research Council grade 3 of 5). Ankle inversion and plantar flexion were normal. Deep tendon reflexes, including the ankle jerk, were normal. There was a sensory impairment to touch and pin sensation over the dorsum of the left foot. Tinel sign was negative on percussion of the right peroneal nerve. The rest of the neurologic exam was normal.

An EMG examination revealed absent superficial sensory nerve action potential on the left and normal superficial sensory nerve action potential on the right. The distal peroneal compound muscle action potential recording from the extensor digitorum brevis and tibialis anterior was low compared to the left. Proximal stimulation at the knee (above the fibula head) revealed significant conduction block with minimal slowing of velocities. The needle EMG revealed that
all common peroneal innervated muscles, except the short head of biceps femoris, were abnormal as evidenced by the presence of fibrillation potentials and neurogenic recruitment. All tibial-innervated muscles and all muscles innervated by the L5 and L4 roots, including the paraspinal muscles, also were normal. This is consistent with a common peroneal neuropathy across the fibular head, mixed in type (segmental demyelination and axonal loss).

The segmental demyelination in this case is proven by the identification of conduction blocks across the fibular head 2 months following the onset of this acute lesion.

The presence of significant motor axonal loss is verified by finding low distal peroneal compound muscle action potential amplitudes stimulating distal to the lesion and the presence of fibrillation potentials in all the affected muscles.

The prognosis should be relatively good and is likely to be biphasic. The initial phase of recovery, dependent on remyelination, should be rapid, about 2 months to 3 months. The second phase is slower and more protracted since it depends on sprouting and reinnervation. Sprouting should be relatively productive in this patient since the lesion is partial, and reinnervation is likely to be effective since many of the clinically relevant affected muscles, such as the tibialis anterior, are situated relatively near the site of injury.

The patient was treated with an ankle brace and physical therapy. Two months later, she had significant improvement of foot-drop. The neurologic examination revealed residual mild left foot dorsiflexion and eversion weakness (Medical Research Council 4+ of 5) but more weakness of toes extensors (Medical Research Council 4- of 5). Sensation had improved with relative hypesthesia over the dorsum of the foot.

**Biological basis**

**Etiology and pathogenesis**

Peroneal neuropathy is usually caused by external compression around the fibular neck or by trauma (fibular neck fracture, tibio-fibular fractures or knee trauma). Less commonly, it may be due to intraoperative injury (hip and knee arthroplasty), benign nerve tumors, peroneal intraneural ganglia (Al Mufargi et al 2011), extraneural ganglion cysts (Ozden et al 2013), herpes zoster infection (Boylu et al 2010), and vasculitis. Peroneal nerve entrapments at the fibular tunnel are extremely rare.

A review of the anatomy of the peroneal nerve is essential to understanding the causes and the clinical manifestations. The sciatic nerve is composed of 2 separate nerves: the common peroneal nerve (also called lateral popliteal nerve) and the tibial nerve (also called medial popliteal nerve). Both share a common sheath within the sciatic nerve but do not exchange any fascicles. In the upper thigh, the common peroneal nerve innervates the short head of biceps femoris only, whereas all other hamstring muscles (long head of biceps femoris, semitendinosus, and semimembranosus) are innervated by the tibial nerve. The common peroneal and tibial nerves separate completely in the upper popliteal fossa. Soon after this split, the common peroneal nerve gives off the lateral cutaneous nerve of the calf, which innervates the skin over the upper third of the lateral aspect of the leg. It winds around the fibular neck and passes through a tendinous tunnel between the edge of the peroneus longus muscle and the fibula (the fibular tunnel). Near that point, the common peroneal nerve divides into its terminal branches, the superficial and deep peroneal nerves. The superficial peroneal nerve innervates the peroneus longus and brevis and the skin of the lower two-thirds of the lateral aspect of the leg and the dorsum of the foot. The deep peroneal is primarily motor; it innervates all ankle and toe extensors (tibialis anterior, extensor hallucis, extensor digitorum longus and brevis) and peroneus tertius, in addition to the skin of the web space between the first and second toes.

More common causes of peroneal neuropathy include the following:

**Knee dislocation and injury.** Injury to the common peroneal nerve occurs in 25% of patients with dislocation of the knee. All underwent ligament reconstruction. In patients presenting with anterior or anteromedial dislocation with associated disruption of both cruciate ligaments and the posterolateral structures of the knee, common peroneal nerve palsy occurs in 41% of patients (Niall et al 2005).

Posterolateral knee injuries with disruption requiring surgery can be associated with displaced common peroneal nerves in cases with biceps avulsions or avulsion-fracture of the fibular head, with nerve injury believed to occur due to the nerve being pulled anteriorly by the biceps tendon (Bottomley et al 2005).
**Knee surgery.** In an Italian study, 22% of common peroneal neuropathies were due to surgery (Aprile et al 2005). Knee surgery is the most common surgical procedure associated with development of common peroneal neuropathy, which appears to occur with 0.3% to 1.3% of all knee arthroplasties (Nercessian et al 2005). Possible risk factors for post-operative peroneal neuropathy include valgus deformity, post-operative epidural anesthesia, previous nerve disease, and rheumatoid arthritis (Nercessian et al 2005). The presence of a preexisting peripheral neuropathy appears to be a significant risk factor for peroneal nerve palsy after knee or hip arthroplasty (Dellon 2005).

The use of a tourniquet was previously thought to be a risk for peroneal neuropathy development, but this appears to be unlikely (Nercessian et al 2005).

**Other surgery.** Common peroneal nerve injury appears to be the most common traumatic nerve palsy after total hip arthroplasty, composing more than 50% of known motor nerve palsies (Farrell et al 2005). Risk factors for common nerve palsy post-hip arthroplasty include developmental dysplasia of the hip, post-traumatic arthritis, lengthening of the extremity, cement-less femoral fixation, and a posterior surgical approach (Farrell et al 2005).

Uncommonly, peroneal nerve lesions may occur with thoracoabdominal surgery, perhaps related to surgical positioning (Aprile et al 2005a). Improper positioning of patients during gynecologic surgeries can also lead to compression neuropathies (Bradshaw and Advincula 2010).

**Weight loss.** In a prospective series, 15% of common peroneal neuropathies were associated and possibly attributed to weight loss (Aprile et al 2005a). Some of these patients have a concurrent polyneuropathy (Cruz-Martinez et al 2000). Relatives of patients with weight-loss induced common peroneal neuropathy can have genetic abnormalities consistent with hereditary liability to pressure palsies (Cruz-Martinez et al 2000). Weight loss is often associated with anorexia nervosa. The exact cause of common peroneal neuropathy in weight loss, however, remains unknown.

**Prolonged maintenance of posture.** Patients who are bedridden represent 7% to 23% of all cases of common peroneal neuropathy (Aprile et al 2005a; Aprile et al 2005b); in 1 series (Aprile et al 2005b), this was the most common cause of common peroneal nerve palsy.

**External compression.** External compression composes 5.8% of cases of common peroneal neuropathy in 1 prospective series (Aprile et al 2005a). Intraneural ganglion cysts of the peroneal nerve can be associated with the ‘wishbone’ sign on MRI with the ascending limb of the peroneal intraneural ganglion intersecting with the longitudinal limb of the vascular adventitial cyst in the axial plane (Spinner et al 2006). Extraneural ganglion cyst causing peroneal neuropathy was reported (Ozden 2013). Arthrogenic cysts at the fibular head compresses the common peroneal nerve in 1.4% of cases (Aprile et al 2005a). Rare causes include extrinsic masses (osteomas, lipomas, ganglia, Baker cysts) or intrinsic nerve sheath tumors. Iatrogenic lesions include casts or tibial osteotomies. Bone tumors, such as osteochondromas, that cause peroneal neuropathies are more common than in adults.

Cast placement for broken bones may lead to as many as 6% of all common peroneal neuropathies (Aprile et al 2005b).

**Trauma.** This is responsible for 10% of cases of common peroneal neuropathy (Aprile et al 2005a) in 1 series and, likely, the second most common cause overall next to perioperative compression. Traumatic causes include fracture of the fibula, or tibio-fibular fractures (Kim and Jung 2011), knee surgery and arthroscopy, lacerations, and blunt injuries. Common peroneal nerve injury is the most common nerve injury resulting from varicose vein removal surgeries; the injury tends to occur just before or as it crosses the fibular neck (Giannas et al 2006). Stretch injuries of the peroneal nerve may occur following severe inversion sprains of the ankle.

Common peroneal mononeuropathies in children are similar to those of adults with compression and trauma as the leading causes (Jones et al 1993). Neonatal peroneal nerve injuries are uncommon, usually associated with breech presentations and malpositioning, and often result in intrauterine stretching of the infant’s peroneal nerves (Godley 1998). Superficial nerve palsy has been reported in 1 patient with nerve herniation through a fascial defect, which presented after exercise (Yang et al 2006). Compartment syndrome following pitviper envenomation has led to peroneal neuropathy (Hardy and Zamudio 2006).

Other rare causes include herpes zoster infection, tacrolimus neurotoxicity (Jain et al 2011), exertional compartment syndrome (Baker et al 2009), and hereditary neuropathy with pressure palsies.
Common causes of peroneal neuropathy are listed in Table 1.

**Table 2. Causes of Common Peroneal Neuropathy**

**Compression**
- During anesthesia
- Weight loss: 15% of cases (Aprile et al 2005)
- Habitual leg crossing
- Prolonged hospitalization
- Prolonged bed rest
- Tourniquet (Nercessian et al 2005)
- Improper bed habitus with intoxication, a variant of Saturday night palsy (Tacconi et al 2004)
- Prolonged posture (23% of cases) (Aprile et al 2005)
- Anorexia nervosa
- Coma
- Diabetes mellitus
- Peripheral polyneuropathy
- Prolonged squatting

- Yoga
- Crop harvesting (Sangwan et al 2004)
- Childbirth (Babayev et al 1998)
- Iatrogenic
- Knee cast (Aprile et al 2005b)
- Ankle-foot orthosis
- Pneumatic compression
- Anti-thrombotic stocking
- Bandage (Usmani et al 2004)
- Strap

- Lithotomy position
- Intrauterine (with breech presentation)
- Horse riding (Adam et al 2005).
- Acute exertional compartment syndrome (in a boxer taking anabolic steroids after heavy exertional activity) (Liem et al 2005)

**Trauma**
- Blunt
- Fibular fracture
- Ligamental knee joint rupture
- Knee dislocation
- Tibio-fibular joint dislocation

- Ankle sprain
• Open
• Laceration
• Gunshot wound
• Animal bite
• Compartment syndrome due to rattlesnake envenomation (Hardy and Zamudio 2006)
• Iatrogenic
• Conventional knee surgery
• Knee joint arthroplasty (Nercessian et al 2005)
• Arthroscopic knee surgery
• Varicose vein surgery
• Sural nerve biopsy when using a nerve stripper (Schubert et al 2005)
• Fascial defect (Yang et al 2006)
• Umbilical artery catheterization in a neonate (Giannakopoulou et al 2002)

Mass lesions
• Extrinsic with an arthrogenic cyst at the fibula (1.4%) (Aprile et al 2005a; Spinner et al 2006)
  - Synovial cyst of the proximal tibiofibular joint (Hersekli et al 2004)
  - Osteochondroma
  - Baker cyst
  - Ganglion cyst
  - Hematoma
  - Pseudoaneurysm
  - Infective sacular aneurysm (Ozcakar et al 2004)
  - Varicose Veins (Yamamoto and Koyano 2004)
• Intrinsic
  - Schwannoma
  - Neurofibroma
  - Neurogenic sarcoma

Other
• Rheumatoid vasculitis (Armstrong et al 2004)
• Leprosy (de Frietas et al 2004)
• Herpes zoster infection

In most cases, peroneal neuropathy results from compression of the peroneal nerve between an external object and
the fibular head. The second most common cause of acute peroneal neuropathy is trauma (second to perioperative
compression). This includes fracture of the fibula, knee dislocation, knee surgery and arthroscopy, lacerations, and
blunt injuries. Stretch injuries of the peroneal nerve may occur following severe inversion sprains of the ankle. Rare
causes include extrinsic masses (osteomas, lipomas, ganglia, Baker cysts) or intrinsic nerve sheath tumors. Peroneal
nerve entrapments at the fibular tunnel are extremely rare.

Common peroneal mononeuropathies in children are similar to those of adults with compression and trauma as the
leading causes (Jones et al 1993). Weight loss is often associated with anorexia nervosa. Iatrogenic lesions include
casts or tibial osteotomies. Bone tumors, such as osteochondromas, causing peroneal neuropathies are more common
than in adults. Neonatal peroneal nerve injuries are uncommon, usually associated with breech presentations and
malpositioning, and often result in intrauterine stretching of the infant’s peroneal nerves (Godley 1998).

Epidemiology
Peroneal neuropathy at the fibular head is the most common compressive neuropathy in the lower extremity;
however, its exact incidence and prevalence are unknown. Men are more frequent than women (male : female = 4:1:1)
(Aprile et al 2005b).

Prevention
Proper positioning of patients and protective padding of operating tables and hospital beds are essential in the
prevention of peroneal neuropathy induced by perioperative compression or compression during prolonged
hospitalizations (Anonymous 2000). It is not known whether avoiding leg crossing in persons with weight loss will
prevent peroneal neuropathy at the fibular neck.

**Differential diagnosis**

Foot-drop is a common presentation in neurologic practice. It may result from an upper or lower motor neuron lesion. Common causes of foot-drop are as follows:

- Deep peroneal mononeuropathy
- Common peroneal mononeuropathy
- Sciatic mononeuropathy
- Lumbosacral plexopathy
- Lumbar radiculopathy
- Motor Neuron Disease
- Parasagittal cortical or subcortical cerebral lesion

Lower motor neuron lesions causing foot-drop include common and deep peroneal neuropathy, sciatic neuropathy (especially when affecting the common peroneal nerve predominantly or exclusively), lumbosacral plexopathy (particularly with lumbosacral trunk lesion), or lumbar radiculopathy (particularly L5 radiculopathy, and less commonly, L4 radiculopathy).

**Table 3. Clinical Differential Diagnosis of Common Causes of Foot Drop**

<table>
<thead>
<tr>
<th>Common causes</th>
<th>Peroneal neuropathy at the fibular head</th>
<th>L5 radiculopathy</th>
<th>Lumbar plexopathy (lumbosacral trunk)</th>
<th>Sciatic neuropathy (mainly peroneal)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common causes</td>
<td>Compression (weight loss, perioperative), trauma</td>
<td>Disc herniation, spinal stenosis</td>
<td>Pelvic surgery, hematoma, prolonged labor</td>
<td>Hip surgery, injection injury, coma</td>
</tr>
<tr>
<td>Ankle inversion</td>
<td>Normal</td>
<td>Weak</td>
<td>Weak</td>
<td>Normal or mildly weak</td>
</tr>
<tr>
<td>Toe flexion</td>
<td>Normal</td>
<td>Weak</td>
<td>Weak</td>
<td>Normal or mildly weak</td>
</tr>
<tr>
<td>Plantar flexion</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal or mildly weak</td>
</tr>
<tr>
<td>Ankle jerk</td>
<td>Normal</td>
<td>Normal (unless with S1)</td>
<td>Normal (unless with S1)</td>
<td>Normal or depressed</td>
</tr>
<tr>
<td>Sensory loss distribution</td>
<td>Peroneal only</td>
<td>Poorly demarcated, predominantly big toe</td>
<td>Well demarcated to L5 dermatome</td>
<td>Peroneal and lateral cutaneous of calf</td>
</tr>
<tr>
<td>Pain</td>
<td>Rare, deep</td>
<td>Common, radicular</td>
<td>Common, can be radicular</td>
<td>Can be severe</td>
</tr>
</tbody>
</table>

**Table 4. Electrodiagnosis of Common Causes of Foot Drop**

<table>
<thead>
<tr>
<th>Common causes</th>
<th>Peroneal neuropathy at the fibular head</th>
<th>L5 radiculopathy</th>
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<th>Sciatic neuropathy (mainly peroneal)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peroneal motor study to extensor digitorum brevis and/or tibialis anterior</td>
<td>Low in amplitude or conduction block across fibular head or both</td>
<td>Usually normal, but can be low in amplitude</td>
<td>Low in amplitude</td>
<td>Low in amplitude</td>
</tr>
<tr>
<td>Superficial Peroneal sensory study</td>
<td>Low or absent</td>
<td>Normal</td>
<td>Low or absent</td>
<td>Low or absent</td>
</tr>
<tr>
<td>Sural sensory study</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal or low amp</td>
<td>Normal or low amp</td>
</tr>
<tr>
<td>Peroneal muscles *</td>
<td>Abnormal</td>
<td>Abnormal</td>
<td>Abnormal</td>
<td>Abnormal</td>
</tr>
<tr>
<td>Tibial L5 muscles + Other L5 muscles ^</td>
<td>Normal</td>
<td>Usually abnormal</td>
<td>Usually abnormal</td>
<td>Normal or abnormal</td>
</tr>
<tr>
<td>Biceps femoris (short head)</td>
<td>Normal</td>
<td>Normal or abnormal</td>
<td>Usually normal</td>
<td>Normal or abnormal</td>
</tr>
<tr>
<td>Paraspinal muscles fibrillations</td>
<td>Absent</td>
<td>May be absent</td>
<td>Absent</td>
<td>Absent</td>
</tr>
</tbody>
</table>

Radicular pain and positive straight leg test (Lasegue test) are common in L5 radiculopathy and may be present in plexopathy or sciatic neuropathy. Weakness of ankle inversion, toe or plantar flexion, or absent or depressed ankle jerk are not consistent with peroneal nerve lesion.
Partial sciatic nerve lesions usually affect the lateral division (peroneal nerve) more than the adjacent medial division (tibial nerve). These proximal (high) peroneal nerve lesions often present a diagnostic challenge since they imitate a distal selective peroneal nerve injury due to compression at the fibular head. The greater vulnerability of the peroneal division of the sciatic nerve to physical injury is related to 2 reasons (Sunderland 1991):

1. The difference between fascicular pattern and the cushioning effect of the perineurium of these 2 nerves in the upper thigh is that the peroneal nerve has fewer and larger fascicles with limited supportive tissue, whereas the tibial nerve is composed of many fascicles, well placed between the elastic epineurial tissue. This renders the peroneal division of the sciatic nerve more susceptible to external pressure.

2. The anatomical course of these 2 divisions: the peroneal nerve is taut and secured at the sciatic notch and fibular neck, whereas the tibial nerve is loosely fixed posteriorly. Hence, traction of the sciatic nerve in the upper thigh (such as during total hip replacement) will result in earlier and more extensive damage to the peroneal nerve than the tibial nerve.

Although the neurologic history is useful (such as following a gluteal injection or gunshot wound), the examiner should look for signs caused by tibial nerve involvement. Common manifestations of sciatic nerve involvement, which are inconsistent with a peroneal neuropathy at the fibular head, include severe foot pain, absent or depressed ankle jerk, weak ankle inversion, or sensory loss in the sole. When the tibial component is slightly involved, the electrophysiological findings often reveal asymmetrically low (or sometimes absent) sural sensory nerve action potential amplitude, borderline or low tibial compound muscle action potential amplitude, asymmetrically absent H-reflex, or minimal denervational changes in tibial innervated muscles (such as gastrocnemius, tibialis posterior, or flexor digitorum longus).

On rare occasions, the peroneal nerve is the only nerve injured, leaving the tibial nerve completely intact (Katirji and Wilbourn 1994). Distinguishing these cases (ie, sciatic nerve lesions affecting exclusively the peroneal component) from a distal peroneal neuropathy at the fibular head is almost impossible, particularly based on clinical grounds. The only useful clinical sign is detecting sensory loss in the upper later third of the leg, which corresponds to the territory of the lateral cutaneous nerve of the calf. This nerve originates from the peroneal nerve before the fibular head and should be spared in lesions at that site. Also, the nerve conduction studies are identical to an axon loss common peroneal mononeuropathy. However, needle EMG of the short head of biceps femoris will reveal signs of denervation only in proximal (high) peroneal neuropathies and should be spared in distal lesions such as at the fibular head. The short head of the biceps femoris is the only hamstring muscle innervated by the peroneal division of the sciatic nerve. It cannot be evaluated satisfactorily in isolation on manual muscle testing nor palpated during such testing because of its location deep to the long head. Its function is often concealed by the other more powerful hamstring muscles (semitendinosus, semimembranosus, and long head of biceps femoris), all innervated by the tibial nerve. However, it is easily accessible to the electromyographer during needle EMG. As a rule, the short head of biceps femoris should be sampled in all patients presenting with a foot drop but, in particular, when confronted with an axonal peroneal mononeuropathy where localization is dependent in its entirety on the needle EMG.

Acute peroneal neuropathy should also be distinguished from acute anterior compartmental syndrome of the leg. A compartmental syndrome is a condition in which increased pressure within a limited space (compartment) in a limb compromises the perfusion, circulation, and function of the contents of that space. Anterior compartmental syndrome of the leg often results in foot drop since the anterior compartment contains all muscles that function as ankle and toe extensors (tibialis anterior, extensor hallucis, and extensor digitorum longus), in addition to the deep peroneal nerve. Dysfunction of any of these structures (muscles with or without nerve) will result in weakness of foot and toe dorsiflexion. Acute compartmental syndrome occurs mainly following limb trauma (crush, contusion, fractures), but it may occur spontaneously after excessive exercise or spontaneous bleeding into a compartment. Acute compartmental syndrome is a state of emergency, requiring rapid diagnosis and surgical treatment. Prompt fasciotomy may prevent severe muscle necrosis and limb contractures (Matsen 1980).

Anterior compartmental syndrome manifests as follows:

1. Leg pain out of proportion to what is anticipated from the clinical situation (such as a tibial fracture), particularly if it occurs after a time interval from the primary etiological event (usually 1 day to 3 days).

2. Tenseness of the anterior compartmental fascia.

3. Hypesthesiain the distribution of the deep peroneal nerve (dorsal first web space), when nerve ischemia occurs.
Pain on passive flexion of toes and plantar flexion of ankle. Foot and toe dorsiflexion weakness. If there is concomitant nerve ischemia, muscles innervated by the crossing nerve beyond the compartment (deep peroneal nerve to extensor digitorum brevis) are also weakened.

Anterior compartmental syndrome of the leg may mimic a primary deep peroneal nerve lesion, particularly when associated with trauma (such as fibular fracture). Both will result in foot drop and sensory impairment. However, severe spontaneous pain, pain with passive stretching (passive toe flexion and foot plantar flexion), taut anterior compartment, and elevated tissue pressure (usually above 60 mm/Hg) are common findings in anterior compartmental syndrome and are not present in primary peroneal nerve lesions. Also, most primary peroneal nerve lesions involve the common peroneal nerve rather than the deep peroneal nerve selectively. This results in additional weakness of eversion (peroneus longus and brevis) and sensory loss over the dorsum of foot and lateral leg.

**Diagnostic workup**

The EMG examination is essential in the diagnosis and prognosis of peroneal neuropathy. The electrodiagnostic studies help confirm the site of the lesion (fibular head, thigh, deep branch), estimate the extent of injury (based on the conduction studies data) and its nature (demyelinating vs. axonal vs. mixed), and, hence, predict the expected course of recovery (weeks or months) (Katirji and Wilbourn 1988; Sourkes and Stewart 1991).

The peroneal motor and sensory nerve conduction studies should be obtained by recording extensor digitorum brevis and tibialis anterior since the extensor digitorum brevis muscle is commonly atrophic in normal subjects (Devi et al 1977; Pickett 1984).

The superficial peroneal sensory nerve action potential is commonly absent or low in amplitude except when the lesion is purely demyelinating or is restricted to the deep peroneal branch. In common peroneal neuropathy defined by electrophysiology, 88% of patients have clinical or electrophysiological evidence of superficial peroneal nerve involvement (Kang et al 2005). However, only 50% of these patients have a reduced superficial peroneal nerve sensory nerve action potential amplitude (Kang et al 2005). The superficial peroneal sensory nerve action potential amplitude is normal in radiculopathy but usually low or absent in lumbosacral plexopathy, sciatic neuropathy, or peripheral polyneuropathy. Therefore, the tibial motor and sural sensory studies and H-reflexes should be done to exclude these possibilities.

In all patients, fibrillation potentials are observed in peroneal innervated muscles when sampled at least 3 weeks after the onset of foot-drop. Testing nonperoneal muscles such as the tibialis posterior, flexor digitorum longus, or gluteus medius is essential. These are normal in peroneal lesions but abnormal in L5 radiculopathy and lumbosacral plexopathy. In axonal peroneal neuropathies, which are unable to be localized by nerve conduction studies, sampling the short head of biceps femoris is mandatory to rule out a high peroneal lesion (sciatic neuropathy affecting the peroneal predominantly or exclusively) (Katirji and Wilbourn 1994). In these lesions, the short head of the biceps femoris is abnormal, and when the tibial component of the sciatic nerve is involved, the other hamstrings, gastrocnemius, and abductor hallucis are also affected, but the glutei are spared. The findings on nerve conduction studies, are divided into several patterns characterized by conduction block (complete and partial), axonal loss (complete and partial), mixed pathology (conduction block and axonal), or selective deep peroneal lesions (Wilbourn 1986; Katirji and Wilbourn 1988; Raudino 1996; Katirji 1999).

<table>
<thead>
<tr>
<th>Pattern</th>
<th>Lesion site</th>
<th>Frequency</th>
<th>Superficial peroneal SNAP</th>
<th>Distal peroneal CMAPs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conduction block</td>
<td>Fibular head</td>
<td>20% to 30%</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Axonal loss</td>
<td>Mid-thigh and fibular head</td>
<td>45% to 50%</td>
<td>Usually absent</td>
<td>Low amplitude or absent</td>
</tr>
<tr>
<td>Axonal loss</td>
<td>Deep peroneal</td>
<td>5%</td>
<td>Normal</td>
<td>Low amplitude or absent</td>
</tr>
<tr>
<td>Axonal loss</td>
<td>Proximal</td>
<td>Less than 5%</td>
<td>Usually absent</td>
<td>Low amplitude or absent</td>
</tr>
<tr>
<td>Mixed</td>
<td>Fibular head</td>
<td>25% to 30%</td>
<td>Low amplitude or absent</td>
<td>Low amplitude</td>
</tr>
</tbody>
</table>

Table 5. Electrophysiological Patterns of Peroneal Mononeuropathies
Conduction block at fibular head | Focal slowing across the fibular head | Needle EMG of peroneus longus | Needle EMG of biceps femoris | Prognosis for recovery
---|---|---|---|---
Present | Rare | Abnormal | Normal | Excellent
Absent | Absent | Abnormal | Normal | Protracted
Absent | Absent | Normal | Abnormal | Fair
Present | Rare | Abnormal | Normal | Biphasic

Conduction block is a useful localizing electrodiagnostic sign that may improve with cooling (Rutkove 2001). Focal slowing is present in a minority of patients and is usually associated with conduction block. Conduction block can be found in 50% to 70% of patients with common peroneal nerve palsy related to postural positioning and weight loss (Aprile et al 2005b). Low amplitude or absent motor response (consistent with pure axonal loss) is observed in half of the patients, whereas pure conduction block and mixed lesions share the other half. At least three-fourths of patients have a significant degree of axonal loss based on a low distal compound muscle action potential.

MRI of the knee can be useful in the detection of tibiofibular joint cysts as a cause of previously idiopathic peroneal nerve palsies (Bakshi et al 2005; Iverson 2005).

Sonography can identify larger cross-sectional areas of the common peroneal nerve after injury post-knee subluxation, and may assist in the identification of peri-lesional fibrosis and hematoma formation (Gruber et al 2005). High-resolution sonography of the common peroneal nerve may identify structural lesions of the peroneal nerve such as intraneural ganglion (Visser 2006) and inflammatory changes in vasculitic neuropathy (Nodera et al 2006). In 1 study, sonography localized the lesion at the fibular head in 55% of patients and just above it in 71% of patients. Assessment of the most thickened part of the peroneal nerve resulted in a cut-off value of greater than 8 mm² with a sensitivity of 90% and a specificity of 69% (Visser 2013). This can also be used for diagnosis and intraoperative management of peripheral nerve lesions (Lee et al 2011). One study showed that extensor digitorum brevis thickness was closely associated with fibular nerve CMAP amplitude (Seok et al 2016). Another study looking at high-resolution sonography performed on 40 patients prospectively with suspected peroneal neuropathy allowed identification of single fascicular involvement in common peroneal neuropathy (Bignotti et al 2016). This study also showed that anterior fascicular involvement was present in up to 17% of patients with suspected common peroneal neuropathy.

Color duplex ultrasonography and angiography can reveal a popliteal artery pseudoaneurysm in the popliteal fossa (Ersozlu et al 2007).

**Management**

In managing acute compressive lesions, patients should be observed to allow for improvement by remyelination or reinnervation. Conduction block lesions (due to segmental demyelination) recover spontaneously in 2 months to 3 months as long as further compression is prevented. Ankle bracing is important when the foot-drop is profound to prevent ankle contractures and sprains. Surgical intervention is appropriate in certain situations (Kim and Kline 1996):

- When the nerve is lacerated and visibly discontinuous. This repair could be primary (at the time of laceration suturing) or secondary (if local infection is feared). In cases with open wounds and when a nerve transection was suspected, surgery can be performed on an emergent basis and may be advisable in some cases, although factors favoring surgery have not been determined (Garozzo et al 2004). Recovery in this group is uniformly poor.
- When clinical or EMG evidence for reinnervation has not been established in the tibialis anterior despite 4 months to 6 months passing since the time of injury. Here, the nerve lesion is likely severe, at least of the third degree. In such cases, neuroma formation occurs in 66%, and surgery consists of neuroma resection and grafted nerve repair (Garozzo et al 2004).
- In slowly progressive peroneal neuropathies, a nerve tumor, ganglion, cyst, or, rarely, true entrapment is suspected and the nerve explored after appropriate electrodiagnostic localization. Imaging studies, particularly MRI, are helpful in these situations.

One procedure for correction of such common peroneal nerve traumatic injuries is a 1-stage procedure of nerve repair and tibialis tendon transfer (Garozzo et al 2004). At 2-year follow-up, evidence of nerve regeneration is present in 90% of patients undergoing such a procedure.

Depending on the nature of the traumatic common peroneal nerve injury, different surgical procedures will be
Neurolysis in patients with knee-level common peroneal nerve lesions with recordable intraoperative nerve action potentials, 88% of patients recovered useful function (Kim et al 2004). In cases of nerve transaction, end-to-end suture repair can lead to good recovery in 84% of patients by 24 months (Kim et al 2004). The length of graft required in traumatic lesions where graft repair is necessary impacts on outcome. When grafts less than 6 cm long are required, 75% achieve good peroneal function, whereas only 38% of patients requiring 6 to 12 cm grafts, and only 16% of patients requiring 13 to 24 cm grafts attained good peroneal function (Kim et al 2004). Tibialis posterior tendon transfer is indicated primarily in the setting of a drop foot and a steppage gait with acceptable success in allowing patients to return to ambulation without an assistive device (Irgit 2012).

It has been suggested that the presence of a preexisting peripheral neuropathy is a risk factor for post-knee or hip arthroplasty development of peroneal neuropathy (Dellon 2005). A suggested algorithm states that if peroneal nerve palsy is still present 3 months after arthroplasty without nerve conduction study evidence of deep or superficial peroneal nerve territory reinnervation, then common peroneal neurolysis should be indicated (Dellon 2005).

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**References especially recommended by the author or editor for general reading.

**Former authors**

Bashar Katirji MD (original author) and Cory Toth MD BSc

**ICD and OMIM codes**

**ICD codes**

ICD-9:
Lesion of lateral popliteal nerve: 355.3

ICD-10:
Lesion of lateral popliteal nerve: G57.3

**Profile**

**Age range of presentation**

19-44 years
45-64 years
65+ years

**Sex preponderance**

male=female

**Family history**

none

**Population groups selectively affected**
none selectively affected

**Occupation groups selectively affected**

none selectively affected

**Differential diagnosis list**

- sciatic mononeuropathy
- lumbosacral plexopathy
- lumbar radiculopathy
- motor neuron disease
- parasagittal cortical lesion
- subcortical cerebral lesion
- lumbosacral trunk lesion
- L5 radiculopathy
- L4 radiculopathy

**Other topics to consider**

- Critical illness polyneuropathy
- Distal myopathies
- Femoral neuropathy
- Myopathies associated with parathyroid disorders
- Sarcoïd neuropathy
- Tibial nerve injuries
- Uremic neuropathy