

Common Fibular Nerve Compression

Anatomy, Symptoms, Clinical Evaluation, and Surgical Decompression

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KEYWORDS

- Common fibular nerve • Common peroneal nerve • Peripheral neuropathy
- Nerve decompression surgery • Dropfoot • Proprioception • Decompression
- Wallerian regeneration

KEY POINTS

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- Common fibular nerve decompression may provide significant relief to patients suffering from drop foot. Motor improvement has been shown to be rapid in many cases.
- Common fibular nerve decompression may be the primary procedure of choice in the different treatment options for those suffering from chronic ankle instability.
- Common fibular nerve decompression has been shown to successfully address drop foot that has resulted from intraoperative traction of the sciatic nerve during hip or knee replacement surgeries.
- Common fibular nerve decompression can increase ankle stability by improving proprioceptive ability on the anterior lateral aspect of ankle and dorsal aspect of foot.

BACKGROUND

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Nerve decompression for diabetic and nondiabetic neuropathy was introduced to podiatric surgeons in the early 2000s by plastic surgeons who were performing these procedures. These decompressions were typically performed on other areas of the body, in particular the carpal tunnel area. Notably, plastic surgeon Dr Lee Dellon, a prominent clinical researcher who performed nerve decompressions on the upper limb, expanded his research to include tunnels in the lower extremity to improve peripheral neuropathy symptoms among diabetic patients. Dellon introduced and trained podiatric surgeons to perform these decompression procedures. This

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introduction, however, was received with skepticism because many podiatrists were unaware or slow to embrace the compression component that could be contributing to patients' neuropathy symptoms. In addition, many podiatric surgeons had fairly limited exposure to nerve surgery beyond decompression of the tibial nerve in the tarsal tunnel and excision of interdigital forefoot neuromas. As more podiatrists began to perform the surgery and research began to appear in the podiatric literature proving the efficacy of the procedures,¹ many more podiatric surgeons began to use the technique. Contemporary podiatrists have evolved from exclusive decompression of the tarsal tunnel to include the common fibular nerve tunnel, the superficial fibular nerve tunnel, the deep fibular nerve tunnel, and the soleal sling to treat peripheral nerve entrapment. A careful physical examination and a thorough medical history determine which nerves are being affected and, therefore, which nerve tunnels are involved. This article discusses the anatomy of the nerve and nerve tunnel, the symptoms associated with common fibular nerve compression, and the proper surgical technique to decompress the nerve.

INTRODUCTION

To properly introduce the common fibular nerve, a brief background on the history of nomenclature may be necessary. The term peroneal is often used interchangeably with fibular; however, the adjective peroneal was officially replaced with fibular by the International Federation of Associations of Anatomists and, therefore, fibular is used throughout this article when referencing the aforementioned nerve.

The common fibular nerve is an important nerve to consider when performing a complete neurologic evaluation in the lower extremity.² This is because the common fibular nerve can elicit a host of problems if damaged, including tingling, numbness, or prickling sensations. More severe common fibular nerve impairments can also affect motor function causing gait disturbances, including drop foot.^{2,3} These problems, although significant, may be commonly overlooked or misdiagnosed by the podiatric physician. The podiatrist may assume the problem is originating from the lower back, resulting in radiculopathy. The physician may also mistakenly assume that after a knee or hip replacement surgery the common fibular nerve is not a relevant component of the differential diagnosis that could be the cause of a postoperative drop foot. A physician may also misattribute ankle instability to frequent ankle sprains or ligament laxity rather than nerve entrapment of the common fibular nerve. The unaware clinician may investigate the possibility of other nerve disorders, such as multiple sclerosis or amyotrophic lateral sclerosis. However, a knowledgeable clinician who understands both the anatomic nerve tunnel and symptoms associated with a damaged common fibular nerve will be able to implement an appropriate diagnostic evaluation.

DESCRIPTION

The common fibular nerve is 1 of 2 primary branches that arise from the sciatic nerve. The common fibular nerve is composed of the spinal nerves from the fourth lumbar nerve through the second sacral nerve. The sciatic nerve divides into the tibial nerve and common fibular nerve immediately proximal to the popliteal fossa. The common fibular nerve then courses distally and laterally entering deep into the lateral leg compartment over the neck of the fibula. It lies beneath a fascial layer before it enters the lateral leg compartment. There are 2 sensory branches found in this area: the lateral sural cutaneous nerve and the recurrent articular nerve. The lateral sural cutaneous nerve forms the sural nerve more distally, whereas the recurrent articular nerve innervates the anterior aspect of the knee. As the common fibular nerve continues

100 more distally, it enters into the lateral leg compartment. At this anatomic location
101 another fascial layer is present. The fascial layer that lies superficial to the nerve but
102 deep to the peroneus longus muscle is the posterior crural intermuscular septum.
103 This fascial tissue separates the muscles of the anterior compartment from the poster-
104 rior compartment. This anatomic location is believed to cause significant compression
105 of the common fibular nerve.⁴ After the nerve exits the fibrous tunnel made of the deep
106 fascial layer of the peroneus longus muscle, it divides into the deep and superficial
107 fibular nerves. The deep fibular nerve then sends efferent signals via motor branches
108 to innervate the tibialis anterior, extensor digitorum longus, extensor digitorum brevis,
109 extensor hallucis longus, and peroneus tertius. The superficial fibular nerve courses
110 down the lateral compartment carrying efferent signals to innervate the peroneus lon-
111 gus and peroneus brevis muscles. Most of these motor nerve branches are in the
112 proximal portion of the leg.

113 During surgery, it is possible to use intraoperative electromyography (EMG) to
114 monitor nerve function. In the case of the common fibular nerve, electrodes are placed
115 in both the tibialis anterior and the peroneus longus muscles. A stimulating electrode is
116 then used to artificially innervate the nerve and recordings are gathered as part of the
117 nerve monitoring protocol. (See Anderson and Yamasaki: Intraoperative nerve moni-
118 toring, in this issue.) It has been observed that motor fascicles located in the anterior
119 superior region of the nerve innervate the tibialis anterior, whereas motor fascicles
120 innervating the peroneus longus lie more posterior and inferior. These observations
121 agree with results published in 1948 by Sunderland and Ray⁵ that investigated the
122 intraneural topography of the common fibular nerve. Conflicting information does,
123 however, appear in the literature. For example, a paper was published in 2007 by
124 Kudoh and Sakai,⁶ and then another in 2012 by Gustafson and colleagues,⁷ suggest-
125 ing a location 90° from what Sunderland and Ray⁵ observed. Intraoperative nerve
126 testing also provides evidence that the peroneus longus demonstrates more improve-
127 ment after decompression than the tibialis anterior.⁸ A proposed theory that may
128 explain this phenomenon is that the change in traction occurs on the posterior or infer-
129 rior nerve fascicles rather than the anterior or superior fascicles as the leg changes
130 from flexion to full extension. During cadaver dissection, it was observed by the clinicians
131 Dr James Anderson and Dr James Wilton that the motor branch arising from the
132 common fibular nerve frequently courses along the anterior fibular ridge to innervate
133 the extensor hallucis longus. These physicians also clinically observed that in early
134 stages of drop foot the extensor hallucis longus is affected earlier than other muscles
135 being innervated by the common fibular nerve. It should be noted that after the com-
136 mon fibular nerve passes through its nerve tunnel, a motor branch that innervates the
137 extensor hallucis longus courses over the anterior crest of the fibula. It is suggested by
138 these clinicians that this bony edge may have an additive compressive effect on the
139 motor branch, resulting in this muscle being one of the first affected.

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141 SYMPTOMS

143 Patients affected by common fibular nerve entrapment may exhibit an array of symp-
144 toms that manifest through sensory and motor system abnormalities as well as func-
145 tional impairments.⁹ Patients suffering from sensory impairments may have burning,
146 tingling, numbness, and pain in the region innervated by the common fibular nerve.¹⁰
147 This innervation zone most often extends from the anterolateral aspect of the leg
148 from just below the nerve tunnel to the dorsal aspect of the foot (Fig. 1). A common
149 complaint may also be pain at night when blankets touch the anterior part of the leg
150 or dorsum of the foot. Patients may also complain of having to reposition themselves

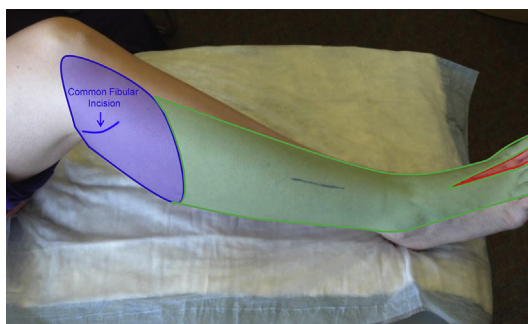


Fig. 1. Common fibular nerve distribution. Markings for the 3 incision sites for the common fibular nerve decompressions (*blue*). The shaded regions represent the cutaneous sensory innervation for the lateral sural cutaneous nerve (*blue*), superficial fibular nerve (*green*), and deep fibular nerve (*red*).

to be more comfortable. During examination, patients suffering from motor impairments will demonstrate abnormalities of the dorsiflexors (ie, tibialis anterior) and evertors (ie, peroneus longus) of the foot and ankle. Severe damage to the common fibular nerve may limit the ability to dorsiflex and evert the foot (ie, drop foot)³ and this could lead to a clinical presentation of an abnormal gait (ie, steppage gait pattern). Due to the lack of muscle strength in the tibialis anterior, which normally provides an eccentric lengthening function, there may be reduced ability to control plantar flexion of the foot due to loss of antagonistic muscle innervation.¹¹ This may lead to a very antalgic gait and instability. Patients who do not demonstrate weakness may still present some gait disturbances due to lack of afferent proprioceptive feedback that arises from muscle spindles of the tibialis anterior and peroneus longus. Lack of proprioception is especially apparent if the nerves being affected innervate the plantar aspect of the foot. This is a more subtle complication than motor weakness and can be assessed using gait analysis and proprioceptive evaluation techniques (ie, Romberg). Proprioception and gait impairment is currently being investigated by the author in collaboration with the Neuromuscular Function Lab at Colorado State University.

CAUSE OF NEURAL ENTRAPMENT

Damage to or entrapment of the common fibular nerve can have multiple causes. They may include trauma to the nerve, including blunt trauma, proximal fibular fracture, surgical complications; or compression from an improperly positioned cast. Drop foot may be a potential complication of total hip or knee replacement arthroplasty^{12,13} and the mechanisms of this could be due to the traction that is placed on the sciatic nerve during surgery. Because the common fibular nerve is a distal extension of the sciatic nerve, it is thought that, if damage occurs to the sciatic nerve, the common fibular nerve may be damaged, resulting in a drop foot.¹⁴

In the case of diabetic neuropathy it has been shown that metabolic nerve tissue is likely to swell as a result of sorbitol in the nerve tissue.¹⁵ This swelling can result in a greater potential for nerve compression in the aforementioned anatomic tunnel caused by increase nerve diameter. In the case of idiopathic neuropathy, the patient may be predisposed with slightly smaller nerve tunnels or there may be mechanical stress to the nerve via bone or muscle.

CLINICAL EXAMINATION

Before nerve testing (eg, nerve conduction, pressure specified sensory device, nerve density testing) is ordered, a thorough clinical examination will indicate if there is an underlying cause for their symptoms other than nerve entrapment. This examination will provide a holistic perspective on how to treat the patient and will also determine if the clinician should proceed with nerve testing. The neurologic examination should assess different sensory modalities including sharp, dull, and vibratory sensation in both limbs. (See Wilton JP: Lower extremity focused neurological examination, in this issue.) This will determine if there is compromised sensory function throughout the distribution of the suspected nerve. Muscle testing should also be performed bilaterally among all muscle groups to detect weakness or motor impairment in the lower extremities. A gait evaluation, in addition to a Romberg test, will help determine if the patient is exhibiting signs of drop foot or impaired proprioceptive ability, which could indicate common fibular nerve entrapment. A pressure-specific sensory device test could also be used to quantify the patient's sensitivity to pressure and assess their 2-point discrimination. This test may also be performed along with EMG and nerve conduction testing. Lumbar radiculopathy and history of spinal surgery or lumbosacral pathologic condition must also be considered because these complications may present similar symptoms throughout the common fibular nerve innervation area. If the differential diagnosis implies a peripheral neural entrapment, a diagnostic injection may be used to confirm the diagnosis. In most cases, the injection will consist of lidocaine and dexamethasone, and should be injected near the common fibular nerve tunnel. Following the diagnostic injection, a cam walker or ankle brace may be needed to protect the ankle from an inversion injury until the effects of the anesthesia has worn off.

NERVE DECOMPRESSION SURGERY

If the clinical evaluation determined that nerve entrapment is the cause of the patient's symptoms, then nerve decompression surgery may be an appropriate avenue for treatment. Intraoperative nerve monitoring may be used during the surgery and, if so, use of a thigh tourniquet should be avoided due to its propensity to alter nerve monitoring recordings. The patient is placed in a supine position with the knee flexed at approximately 45°. The bend in the knee enhances the surgeon's ability to localize the common fibular nerve and increases the laxity of the nerve to prevent damage to it and promote nerve gliding. It is important to use the head of the fibula as a reference point for incision placement (Fig. 2). Palpation of the fibular head may be difficult among obese patients. Therefore, a C-arm may be needed to mark the location on the skin. This extra step will help to prevent a misplaced incision. The incision begins approximately 1 cm distal and anterior to the area where the nerve passes over the fibula and continues proximally from anterior distal to posterior proximal approximately 4 cm (see Fig. 2). After the incision is made, dissection is carried down through the subcutaneous adipose tissue to identify the fascial layers over the nerve and the lateral leg compartment. It is necessary to use the head of the fibula as a landmark to guide the surgeon throughout the dissection (Fig. 3). It should be noted that there may be more adipose tissue over the fascial layers, which will have a more yellow appearance. The lateral leg compartment will appear either white with a thick fascial layer or as muscle if the fascia is thin. At this point in the surgery there will be 2 defined sections: a more proximal fascial layer composed of 2 layers superficial to the nerve and a defined lateral leg compartment more distally (Fig. 4). The fascial layer comprises a thinner superficial layer and a thicker deeper layer that is adjacent to the

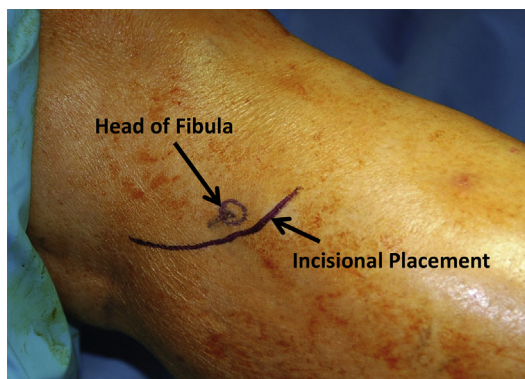


Fig. 2. Common fibular nerve tunnel. Markings for the head of the fibula and the incisional placement for the common fibular nerve.

nerve. The surgeon should be able to locate the nerve by direct palpation or with the nerve stimulator when nerve monitoring is used. The first step in the decompression is the release of the 2 fascial layers over the common fibular nerve. This is accomplished up to where the nerve passes beneath the lateral leg compartment. The surgeon may also elect to divide the 2 fascial layers using digital palpation and separate the fibers proximally (see Fig. 4). Once the fascial layer has been released, the second portion of the procedure is performed by decompressing the tissues that form the proximal portion of the lateral leg compartment. As the dissection proceeds more distally, the surgeon must be meticulous to avoid unintentional damage to motor nerve branches in this area. Before decompressing the leg compartment, care should be taken to identify the direction the nerve courses as it dives beneath the muscle compartment over the fibular neck. The dissection should be made directly over the midline of the nerve. With dissection scissors, a release of the anterior compartment is then done (Fig. 5). The fibers of the peroneus longus muscle are then retracted distally and the deep fascial layer over the nerve will then be observed. This fascial layer can vary in

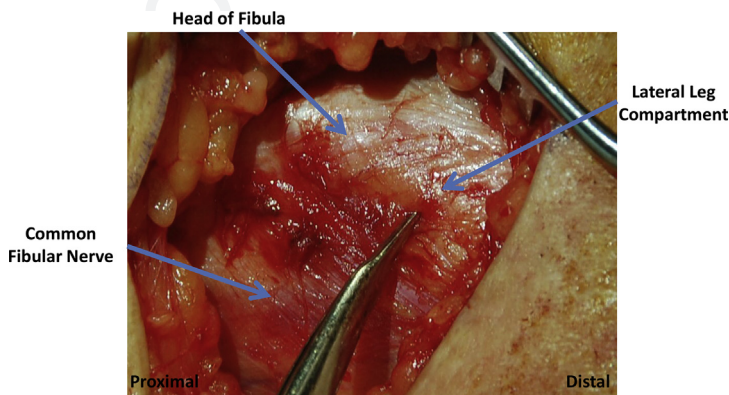
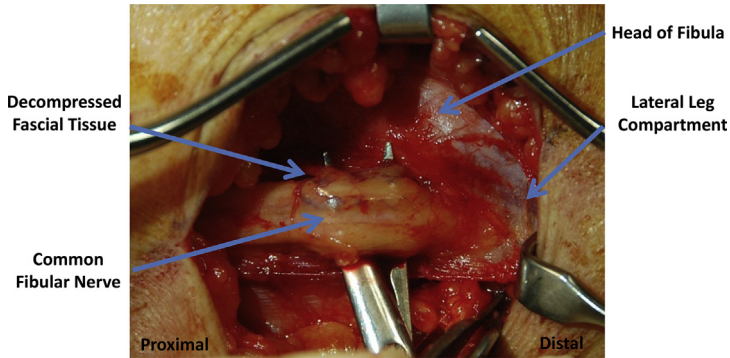
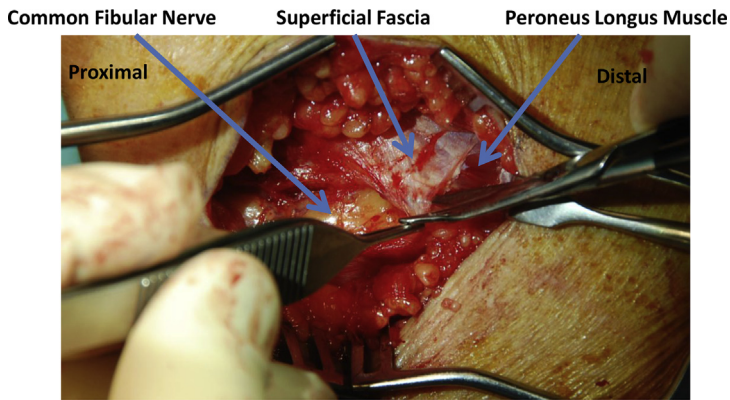


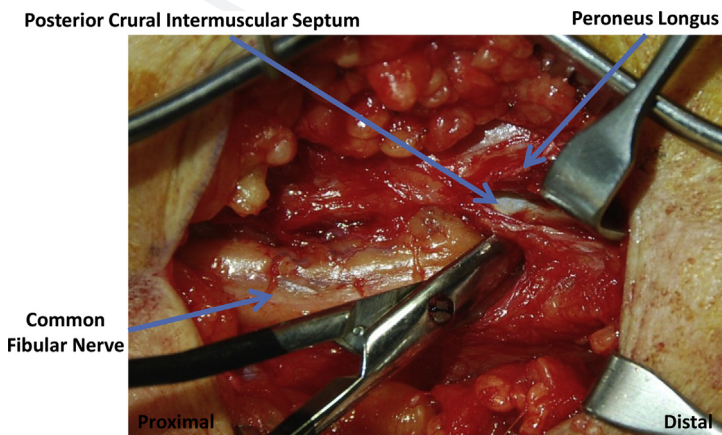
Fig. 3. Head of fibula (landmark). The anatomy of the surgical site before decompression. The fascial layer and lateral leg compartment will be decompressed during surgery.



317 **Fig. 4.** Postfascial decompression and prelateral leg compartment decompression. The surgical site postdecompression of the proximal fascial layer and predecompression of the lateral leg compartment.



335 **Fig. 5.** Decompression of lateral leg compartment. Dissection through the superficial fascial layer over the peroneus longus muscle.



353 **Fig. 6.** Decompression of lateral leg compartment. Retraction of the peroneus longus muscle distally and the entrapment site of the posterior crural intermuscular septum.

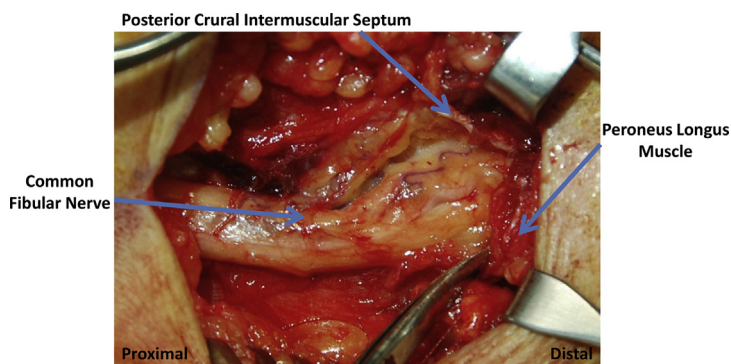


Fig. 7. Postdecompression. Completion of the decompression of the fascial layers proximally and the lateral leg compartment distally.

length. It should be noted that the tightest region of the nerve tunnel will be here, where the superficial fascial layer and the deep fascial layer merge to form a tight band. This band is the posterior crural intermuscular septum and is the fascial layer between the anterior and posterior leg compartments (Fig. 6). Beneath the nerve, there may also be another fibrous band in this same area called the posterior deep fascial arch. Release of this tissue may also be necessary if it is compressing the nerve. At this point, the nerve decompression surgery has been completed (Fig. 7). The surgeon should then use subcuticular sutures and a skin closing medium of his or her choice. If a local anesthetic is used, a possibility for postoperative foot drop exists. Therefore, a patient should be weight-bearing in a cam walker to protect them from an inversion sprain until the anesthesia entirely dissipates. Early ambulation is important to reduce potential for scar adhesions that could have a detrimental effect on the outcome of the surgery. These scar adhesions could compromise nerve gliding as it courses throughout the limb, affecting the success of the surgery.

SUMMARY

The common fibular nerve is an important part of the lower extremity nerve anatomy and needs to be considered by clinicians. It is frequently under-recognized. Conducting a thorough medical history and lower extremity neurologic examination is vital. Excellent anatomic knowledge and surgical technique is essential in preventing an adverse event such as a drop foot. Surgical treatment of common fibular nerve impairment can provide for a much more stable and pain-free lower extremity, leading to improved quality of life for the patient.

CASE STUDY

A case study relevant to this article appears in this issue. (See Barrett SL: Case study for clinics in podiatric medicine, in this issue.)

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