Peripheral Nerve Entrapments of the Lower Leg, Ankle, and Foot

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KEYWORDS

- Nerve entrapment • Peripheral nerve • Foot pain
- Anterior tarsal tunnel

Peripheral nerve entrapments of the lower extremity are a relatively rare and heterogeneous group of nerve disorders encompassing a wide variety of etiologies as well as clinical presentations. Such conditions often present a diagnostic challenge because of the diversity of patient presentations. As such, it is widely believed that these conditions are both underdiagnosed and underreported in the literature. Treatment for peripheral nerve entrapment is highly dependent upon proper identification of the involved nerve and determination of the anatomic location of compression. Fortunately, improvements to electrodiagnostic and imaging modalities have helped ease the previous near-total reliance on patient history and physical examination in diagnosis of these conditions. This determination allows one to choose the optimal treatment strategy tailored to the patient's individual pathology. In this article, we examine peripheral nerve entrapments in the lower extremity involving the sural, saphenous and common, superficial, and deep peroneal nerves. This article reviews the anatomy of the lower extremity as it relates to the course and common areas of entrapment for these nerves, illustrates characteristics of common presentations of each disorder, and examines operative and nonoperative options for treatment based on the best available evidence. A precise understanding of the anatomic course and motor/sensory distribution of each nerve, as well as recognition of the common areas of compression will aid in the correct diagnosis and optimal treatment for these disorders.

The authors have nothing to disclose.

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OVERVIEW OF NERVE ENTRAPMENT

Nerve entrapment is broadly defined as the compression or entrapment of a nerve as it passes through or by another anatomic structure, most commonly a fibro-osseous tunnel or fascial opening. Entrapment can occur through any of a number of mechanisms, which can be broadly grouped into internal or external factors. In addition, there are many medical conditions, particularly those leading to whole-body fluid accumulation, increased extracellular matrix production, or chronic inflammatory conditions that can cause nerve compressions. Thus, some nerve entrapments are directly related to the underlying condition and subsequently resolve as the offending condition is addressed. Optimal treatment for these patients, in many cases, is medical rather than surgical.

Although the entirety of the nerve appears compressed to the eye, microscopically the nerve fibers are not universally affected by compression. Superficially located fibers (the large diameter, heavily myelinated fibers such as light touch and motor fibers) tend to exhibit increased amounts of compression, while the deeper fibers (less myelinated fibers such as pain fibers) typically are less affected. Studies have shown that the area of greatest mechanical deformation occurs both in the superficial area of the nerve, as well as in the zone between compressed and uncompressed segments.[1] External pressures of 20 to 30 mm Hg have been shown to impair venular flow in the epineurium and to slow axonal transport. Pressures of 30 mm Hg result in changes to the permeability of these intraneural blood vessels, resulting in further increases of intraneural interstitial fluid pressure.[2] Experimentally induced higher pressures (130 to 150 mm Hg) resulted in an acute block to conduction[3] and resulted in changes in the morphology of nerve fibers, causing both epineural and perineural thickening and rapid loss of nerve function.[4] This provides a basic science basis for the common clinical observation that less "significant" nerve compression lasting shorter durations of time will more often result in quick recovery of function after decompression. In contrast, more "significant" nerve compressions of longer durations are more likely to require longer periods to restoration of function, or incomplete restoration of function, after decompression. Furthermore, these physiologic changes, particularly to the blood supply to the nerve, occur rapidly. Within hours edema can be seen in histologic samples, and within days and continuing for weeks there is inflammation, fibrin deposition, and axonal degeneration.[5]

Stages/Classification

Nerve compression often progresses through a series of stages, with characteristic symptomology related to both the duration and the degree of compression. Mild and brief compression, such as occurs when a limb "falls asleep," produces an interruption in axoplasmic flow. No major structural changes or damage occurs, and the nerve normalizes when pressure is relieved. In severe, acute compression there is often a sequential invagination of the myelin sheath seen on microscopy. As compression becomes chronic, segmental demyelination is seen, resulting in slowed conduction of action potentials and distal Wallerian degeneration of the nerve.

There are 3 described clinical stages in ongoing nerve compression in patients. Stage I is characterized by intermittent paresthesias and sensory deficits occurring primarily at night. Stage II occurs after continued progressive compression, which leads to more severe and consistent symptoms (such as paresthesias, numbness, impaired dexterity, and so forth) that fail to resolve during the day. Stage III is defined as altered nerve microcirculation and edema leading to pronounced morphologic changes including segmental demyelination. This results in more constant pain that