CHAPTER **35**

PERIPHERAL NERVE ENTRAPMENTS OF THE FOOT AND ANKLE

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Peripheral nerve abnormalities of the foot and ankle are relatively common, but can readily be misdiagnosed. Nerve entrapments of the foot and ankle are a frequently under recognized source of pain.[1] With the advent of improved knowledge, the clinician is becoming more and more familiar with this challenging condition. There are no nerves that are immune to injury by entrapment, traction, compression, or laceration. Their variable symptoms and often subtle clinical findings may make a diagnosis difficult. Familiarity with the nerve anatomy and known entrapment sites facilitates treatment. Local nerve pathology in the lower extremity must be distinguished from proximal nerve dysfunction and systemic diseases that may affect nerve function.

Kopell and Thompson[2] described peripheral nerve entrapment as "a region of localized injury and inflammation in a peripheral nerve that is caused by mechanical irritation from some impinging anatomic neighbor." Peripheral nerve injury following surgery or trauma to the lateral, dorsal or medial aspect of the foot and ankle can have painful neurologic consequences. This nerve pain is often diffuse and poorly defined.[1,3]

Treatment, both conservative and surgical, is tailored to the severity of the symptoms as well as to the objective evidence of a nerve entrapment, injury, or dysfunction. The topics in this section will address more common nerve pathology: interdigital neuromas and tarsal tunnel syndrome. And other less common nerve pathology: sural, saphenous, superficial peroneal and deep peroneal nerve entrapment, and entrapments of the motor branch to the abductor digiti quinti.

ETIOLOGY

Localized peripheral neuropathy may develop due to acute trauma or repeated microtrauma by either endogenous or exogenous stimuli. This trauma induces an inflammatory response that infiltrates the nerve trunk and surrounding tissues.

Endogenous sources include neighboring anatomic structures that repeatedly traumatize the nerve by direct pressure and inhibition of normal nerve mobility. These sources include: skeletal muscle, fibrous bands, osseous surfaces, and soft tissue masses (i.e. ganglion cyst, lipoma, neurolemmoma). Metabolic disorders like rheumatoid arthritis, diabetes, thyroid dysfunction, hyperlipidemia, and peripheral vascular disease may act as endogenous causes of peripheral nerve entrapment secondary to microvascular dysfunction and subcutaneous atrophy.

Exogenous sources include gross trauma, compartment syndrome,[4,5] fracture, dislocation, injection injury,[6-8] sprains, and type of footwear.

Iatrogenic causes include tourniquet compression,[9,10] bandage or cast pressure,[11] improper patient positioning in surgery, and surgical technique.[9,12,13] Finally, local infection, which causes inflammatory fibrosis, may affect peripheral nerves.

DIFFERENTIAL DIAGNOSIS

One must distinguish foot and ankle nerve entrapments from lumbosacral radiculopathy. In addition, symptoms of autonomic overtones with vasoconstriction, and decreased skin temperature may be suggestive of complex regional pain syndrome (CRPS). Infection (osteomyelitis or abscess) may mimic the pain of nerve entrapment. Patients who have tendonitis or ligamentous injury may also imitate a neurologic component. We must also differentiate in the foot between plantar fasciitis and nerve entrapment.

SIGNS, SYMPTOMS AND DIAGNOSIS

The diagnosis is usually made through a careful history and physical exam. The key diagnostic criterion is pain created by irritation of a specific nerve. You may ask the patient to draw out the specific area of pain. Sensory abnormalities usually

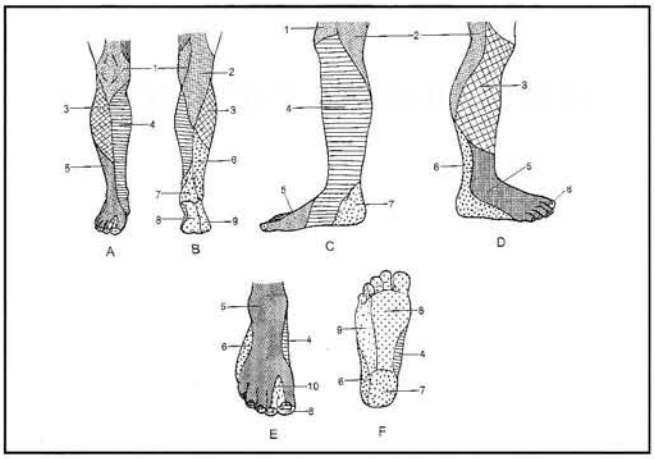

Figure 1. Peripheral nerve distribution.

predominate over motor dysfunction and pain is well localized over the sensory distribution of the involved nerve.[14] (Figure 1) Paresthesia, anesthesia, hyperesthesia, or dysesthesia are common complaints. A patient may complain of hypersensitivity or tenderness at a previous surgical site.

Pain associated with an entrapped motor component is less defined in terms of distribution and pain is usually a dull, aching sensation. In advanced cases disuse atrophy and weakness of a muscle may occur. The patient may not always recall a history of trauma, therefore, objective evaluation centers on the sensimotor evaluation.[15]

SENSIMOTOR EVALUATION

Two-point tactile dysfunction over sensory distribution of involved nerve (decreased), sharp-dull sensation (decreased)

- Percussion and moderate-deep palpation of nerve (elicits pain and paresthesia)
- Tinel's sign (distal radiation of pain) or Valleix's sign (proximal radiation of pain)
- Active or passive ROM of the extremity may exacerbate symptoms
- Nerve conduction velocity (NCV) and electromyography (EMG) measurements may be helpful

Note that manual muscle testing may not be helpful unless in seriously advanced conditions. In these cases muscle atrophy and weakness will be observed.

Diagnosis can be further achieved through a local anesthetic injection. Immediate resolution of symptoms indicates accurate localization of the correct nerve. This injection can also be both a diagnostic and therapeutic injection with the addition of corticosteroid.

Quantitative sensory testing (QST) has emerged as a useful adjunct in diagnosing suspected peripheral nerve entrapments. The use of QST can eliminate the need for invasive techniques. Not only may QST results be easier to interpret, you can use QST to evaluate nerves that are not conventionally accessible by electrodiagnostic techniques.[16] If larger nerves are involved, you may consider using electrodiagnostic studies or diagnostic imaging modalities, such as MRI, to help substantiate or support the diagnosis.

TREATMENT

The first course of treatment is to determine whether the cause is exogenous, endogenous, or iatrogenic. Conservative measures should include removal of extrinsic compression placed on the nerve and the use of anti-inflammatory medications (NSAIDs). If an offending object such as a stocking or a pressure point in a shoe can be identified, this should be removed or padded. Abnormal mechanical stress can be alleviated with the use of orthotics, careful casting, or splinting. Immobilization for 1-2 weeks can decrease local inflammation, and hence nerve irritation. Local infiltration of anesthetic and corticosteroid at the site of entrapment is the mainstay of conservative therapy.[13,17,18] Infiltration of steroid decreases both intraneural and extraneural inflammation and fibrosis, allowing axonal reorganization and remyelination within the nerve trunk. Furthermore, if hypertrophic scars or concern of significant scar tissue is considered, the addition of hyaluronidase can enhance breakdown of perineural fibrosis. Acupuncture, has also been known to help in the treatment.

Other conservative measures include topical medications (such as capsaicin), sclerosing injections, oral anti-inflammatories, oral medications or anti-depressants (such as Neurontin, Elavil), biomechanical support or control, and physical therapy modalities (including iontophoresis or phonophoresis, and desensitization techniques).

If the pain of nerve entrapment fails to respond to conservative treatment, if the clinical picture worsens with advanced sensory loss that threatens weight-bearing sensation, or if motor

Table 1

ETIOLOGY OF LOCALIZED ACQUIRED PERIPHERAL NEUROPATHY

ENDOGENOUS

I. Congenital
   - A. Anomalous development
   - B. Overuse

II. Neoplastic
   - A. Varix
   - B. Ganglion cyst
   - C. Lipoma
   - D. Neurilemoma (schwannoma)
   - E. Metastatic infiltration

III. Metabolic
   - A. Diabetes mellitus
   - B. Rheumatoid arthritis and other connective tissue diseases
   - C. Peripheral vascular disease
   - D. Thyroid dysfunction
   - E. Hyperlipidemia
   - F. Drug toxicity

EXOGENOUS

I. Traumatic
   - A. Laceration
   - B. Blunt trauma
   - C. Fracture/dislocation
   - D. Traction
   - E. Injection
     1. Puncture
     2. Chemical

II. Iatrogenic
   - A. Tourniquet compression
   - B. Surgical positioning
   - C. Cast or bandage constriction
   - D. Surgical technique
     1. Incision planning
     2. Dissection
     3. Hemostasis
     4. Nerve handling
     5. Suturing

III. Infectious
   - A. Local abcess
   - B. Postinflammatory fibrosis

weakness and atrophy develop, then surgical intervention is indicated. At this time, the risk of permanent nerve damage far exceeds the risk of surgical intervention.[19] It is important to inform the patient that symptoms may recur, worsen, or that residual anesthesia may develop after surgery.

Surgical neurolysis entails decompression of the nerve by releasing or removing any tightly bound structures on the nerve. A patient may be prepared to exchange hyperesthesia for anesthesia if it is necessary to resect the entrapped nerve. With surgical intervention it is imperative to perform proper incision planning, layer dissection, hemostasis, appropriate nerve manipulation, and wound closure.

When you see an entrapped nerve or neuroma that has a sufficient chance for recuperation, first consider an external and/or internal neurolysis.[20] However, if the peripheral nerve is damaged to the scope where conservative measures have been unsuccessful and neurolysis fails or it is unlikely to be beneficial, then you should consider performing a peripheral neurectomy. When conservative measures and neurolysis fail to make an impact, you may have to consider doing a peripheral neurectomy in order to treat a painful entrapped nerve or neuroma. With a neurectomy pain may be diminished but conversely, return of sensation is problematic.

Performing a neurectomy involves excising a portion of nerve. If the function of the entrapped nerve is not vital or if previous attempts at neurolysis or nerve reconstruction have failed, then you should resect the nerve with or without transposing the remaining nerve stump. To perform the neurectomy, first isolate the entrapped portion of nerve,

Table 2

SURGICAL TREATMENT OF TRANSECTED NERVE ENDING FOR INHIBITION OF AXONAL REGROWTH

Physical containment	Synthetic containment	Physiologic containment
Chemical treatment	Silicone caps	Epineuropathy
Alcohol	Rubber	Nerve grafting
Phenol	Plastic	
Formaldehyde	Lucite	
Nitrogen mustard	Polyethylene	
Pepsin	Collodium	Transection Away From Painful Stimuli
Resin	Cellophane	Excision and retraction
Hydrochloric acid	Metallic foil	Implantation into muscle
Iodine	Tantalum	Implantation into bone
Gentian violet	Glass	En bloc translocation
Steroids	Nerve glue	
Cautery		
Electrocoagulation		
Laser		
Radiofrequency current		
Cryosurgery		
Ligation		

(Adapted from Downey, MS: Management of neurologic trauma. In Scurran, BL: Foot and Ankle Trauma, New York, Churchill Livingstone, 1989, p.245.)

neuroma in-continuity, or stump neuroma. Then dissect proximally until you have identified the normal nerve. Once the normal nerve tissue is delineated, perform sharp release of the nerve as far distally as possible.

Once the entrapped portion of nerve is resected; there are numerous operative techniques to inhibit axonal regrowth and transpose the transected nerve ending away from painful stimuli. With these techniques, you can attempt to diminish stump neuroma formation and/or attempt to transpose the nerve to an area that is subjected to the least possible amount of mechanical stimulation.

To inhibit axonal regrowth or stump neuroma formation, one can utilize physical containment, synthetic containment, and physiologic containment. Physical containment is achieved with chemical treatment (alcohol, phenol, formaldehyde, nitrogen mustard, pepsin, hydrochloric acid, iodine, gentian violet or insoluble steroids), cautery (electrocoagulation, laser cautery, radiofrequency current and cryosurgery), and ligation after performing the neurectomy to try to achieve inhibition of further neuroma formation.

Synthetic containment is performed with the use of inert materials such as silicone caps, rubber, plastic, lucite, polyethylene, collodium, cellophane, silver and gold foil, tantalum, glass and nerve glues. Surgeons have also used physiologic containment with epineurorrhaphy and nerve grafting. (Table 2.)

Although long-term clinical studies with these varying methods are scarce, these approaches have reportedly had minimal success at diminishing recurrent stump neuroma formation and some have been associated with foreign body reactions.[21]

Transposing a resected nerve end away from potential irritation appears to be preferable to in situ containment. It may be beneficial to excise a neuroma and allow the nerve end to retract proximally, as it allows the nerve ending to rest in a proximal site away from the surgical incision and original site of entrapment. However, be aware that if the nerve end comes to rest in a poor soft tissue bed or continues to be irritated, this approach will be doomed to failure.[22]

Alternatively, transplantation of the resected end of the nerve into bone or muscle may be the best approach. The structure you use for transplantation of the nerve should be in close proximity to the nerve ending and subject the nerve to the least possible amount of mechanical irritation. When possible, implant the nerve ending into well-vascularized innervated muscle or bone that is away from denervated skin and scar tissue.

Mackinnon and Dellon coined the term "neurotrop(h)ism" to suggest influences that facilitate both nerve fiber maturation and appropriate direction of regeneration.[23] Recent research suggests that when you implant cut nerve endings into innervated muscle, they are less likely to demonstrate significant "neurotrop(h)ism." Therefore, the nerve is less likely to attempt regeneration in innervated muscle tissue.[24] To achieve this implantation, suture the epineurium into the belly of the muscle. If you prefer or if an appropriate muscle belly is not available, you can suture the epineurium into the bone.[25,26] Make a small trephine or drill hole into the bone and suture the epineurium into the opening you've created. Doing this allows you to bury the cut end of the nerve into the bone.

Finally, you may consider doing an en bloc transfer of an intact neuroma or resecting a neuroma with primary neurorhaphy or grafting. Herndon et al reported that 72% of patients had minimally painful results following an en bloc transfer of intact neuromas with their fibrous scar tissue encapsulation to an adjacent area that was more protective and free from scar tissue.[27]

Although these results are promising, doing an en bloc transfer does not appear to offer any advantage over implanting a freshly cut nerve ending into bone or muscle. Hattrup and Wood noted that 77% (10 of 13) of their patients had diminished symptoms after they performed a neurectomy with interfascicular grafting.[28] However, you would generally reserve nerve reconstruction for nerves with a major motor component, and when considering the foot and ankle, this would be limited to treating recalcitrant lesions of the posterior tibial nerve.[29]

After performing the neurectomy with transplantation, close the soft tissues in anatomic fashion. Prior to closure, the authors will commonly utilize an implanted slow-infusion anesthetic pump for further pain control and to enhance outcomes. After one week the pump is easily removed (similar to removing a TLS drain) during the first postoperative dressing change. Apply a standard compression dressing and use a closed suction drain if necessary. Consider protected weightbearing or nonweightbearing for the first two to four weeks. Institute range of motion exercises and rehabilitative modalities after one to two weeks. Once wound healing occurs, you may want to initiate physical therapy focusing on decreasing scar tissue formation and desensitization techniques.

One should typically consider peripheral neurectomy as a last resort for treating lower extremity nerve entrapments and neuromas. The exception to this rule is the classic Morton's neuroma. When you find it necessary to perform a peripheral neurectomy, the author's advocate implanting the transected nerve end into either innervated skeletal muscle or bone. This technique can help reduce or eliminate many of the complications associated with this procedure.

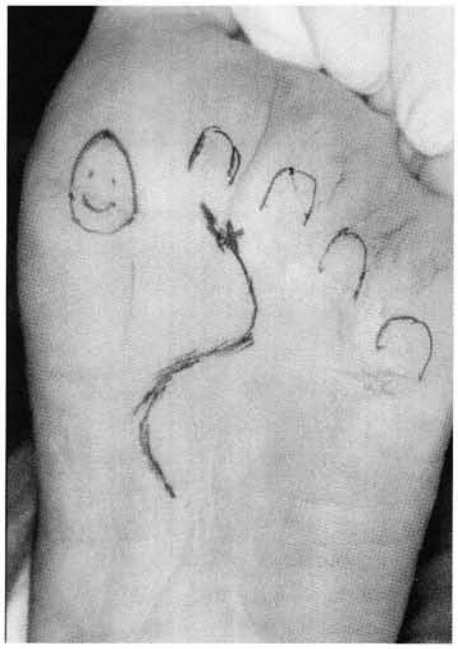

Figure 2. (Nerve 26) Incision made on the plantar foot focused at addressing a recurrent neuroma in the second interspace. A curvilinear incision is preferred to provide wider medial to lateral exposure.

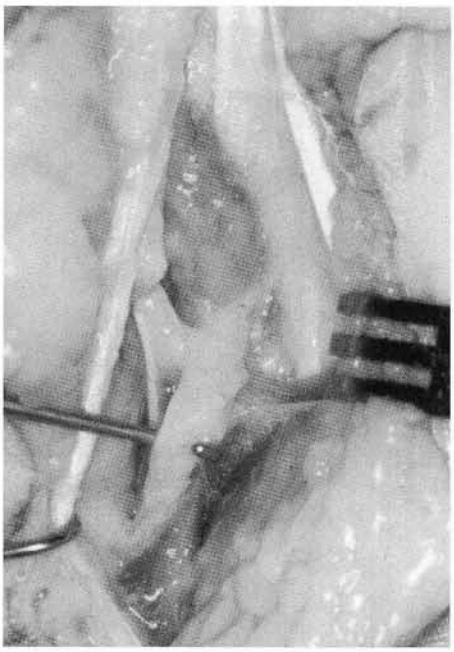

Figure 3. (Nerve 29) After a longitudinal incision through the deep fascia, the nerve should be visible without much dissection.

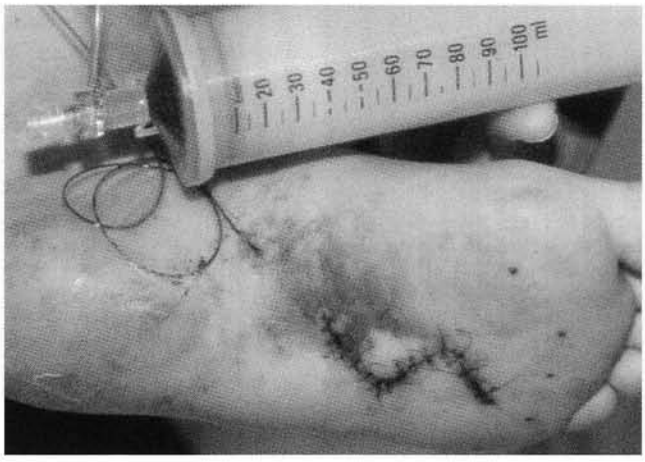

Figure 5. (Nerve 38) Three days postoperative with a pain pump still in place. The pump helps tremendously with analgesia within the first several days. The patients are instructed to remain non-weightbearing for the first 3 weeks.

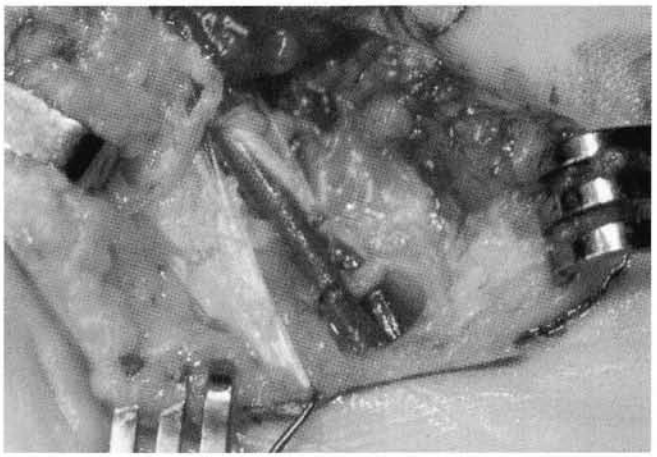

Figure 4. (Nerve 34) After resection, the nerve is translocated into a nearby skeletal plantar muscle and anchored there by 6-0 nonabsorbable suture. This will minimize the possibility of pulling out of the muscle.

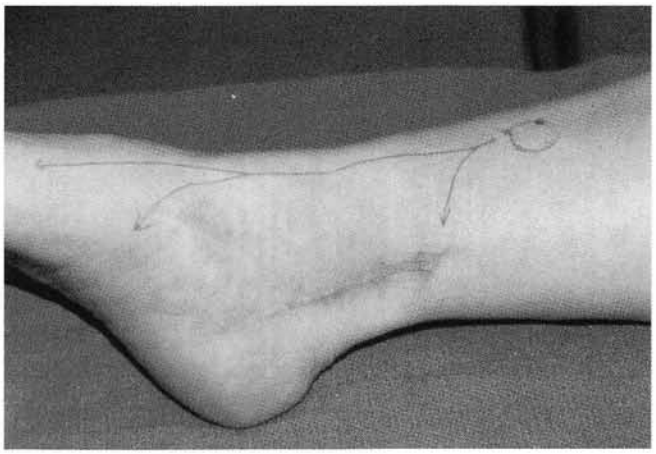

Figure 6. (Nerve 549). Saphenous nerve contribution to pain along the tarsal tunnel incision. This is a posterior branch of the saphenous that can become involved.

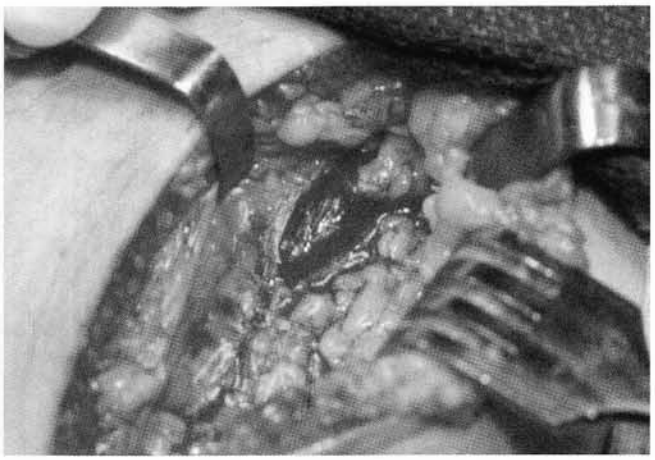

Figure 7. (Nerve 552). Implantation of the saphenous nerve into the soleus muscle.

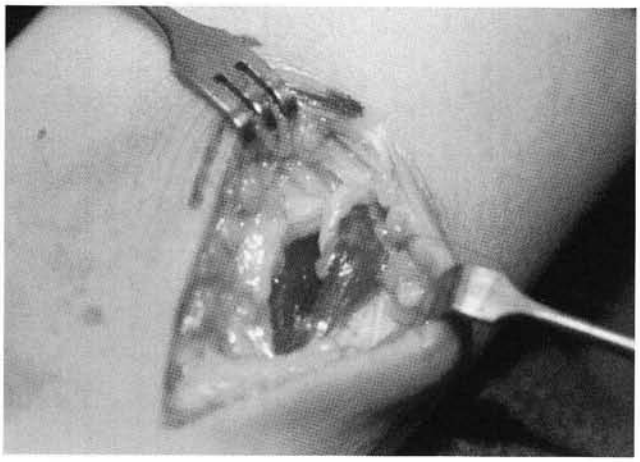

Figure 9. (Nerve 556) Placement of the sural nerve into the soleus muscle as well, this time from the lateral side. The soleus is ideal since it has the least amount of excursion compared to the other extrinsic muscles of the lower leg.

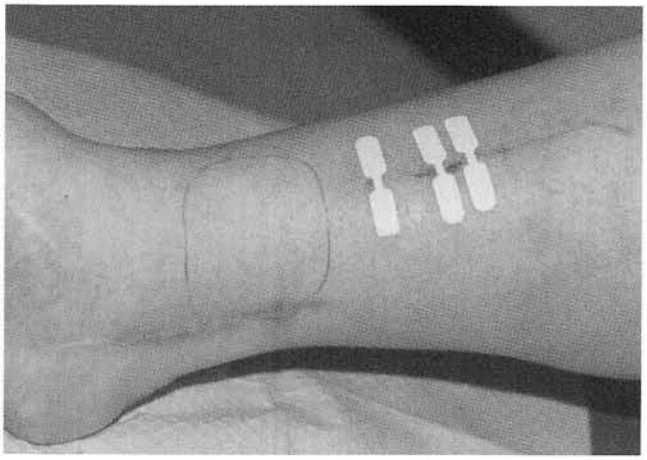

Figure 8. (Nerve 553) Three weeks postoperative with anesthesia noted in the incisional area of the posterior branch.

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