18 Nerve Injury After Open and Arthroscopic Surgery of the Ankle and Foot, Including Morton Neuroma

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18.1 Risks/Incidence/Mechanism of Nerve Injury

Arthroscopic surgery of the ankle has continued to increase in popularity among foot and ankle surgeons in the past several decades as it allows for preservation of the soft tissue envelope, direct viewing of the internal joint structures, and earlier return to athletic activity compared to open approaches [1]. Prior to modern intraoperative distraction techniques, incidence of surgical complications with ankle arthroscopy in the literature was recorded as 24.6% [2]. However, a variety of invasive and noninvasive distraction techniques exist today which, in conjunction with standardization of portal placement and continued evolution of safe arthroscopic practices, has decreased the overall complication rate to 3–10% [3–5] Nevertheless, nerve injury following ankle arthroscopy remains the most common complication [6] and can have devastating consequences on postoperative outcome.

Anterior ankle arthroscopy is commonly performed for both diagnostic and therapeutic indications. Evaluation and treatment of ankle impingement from bony and soft tissue etiologies, removal of osteochondral lesions, assessment of ankle instability, and management of arthritis and chronic synovitis are all common conditions where anterior ankle arthroscopy use has been supported by literature [7]. The most common portal sites are the anteromedial (between the tibialis anterior and great saphenous vein) and anterolateral (lateral to the peroneal tertius tendon if present, or the extensor digitorum longus between the medial and lateral dorsal cutaneous branches of the superficial peroneal nerve) (Fig. 18.1). Injury to these branches of the superficial peroneal nerve are the most reported neurologic complication of anterior ankle arthroscopy [6], and great care during placement of the anterolateral portal is taken to prevent permanent neurologic sequelae. Injury to the saphenous nerve, which runs longitudinally with the greater saphenous vein, is at risk of injury during insertion of the anteromedial portal. A previously described anterocentral portal, which lies between the extensor hallucis longus and extensor digitorum longus, has fallen out of favor due to unacceptably high risk to the deep peroneal nerve and dorsalis pedis artery [8].

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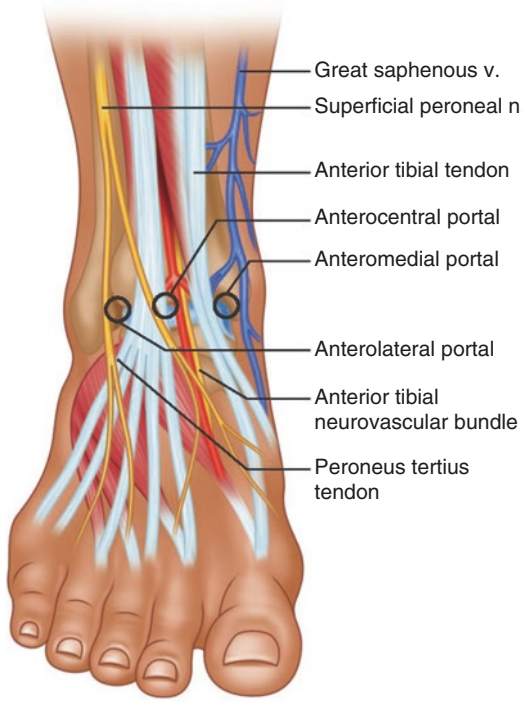

Fig. 18.1 Anterior ankle arthroscopy portals and critical nearby anatomic structures

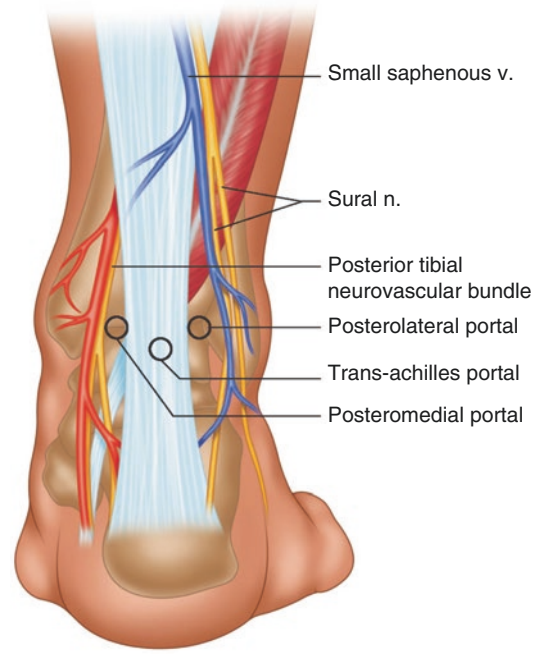

Fig. 18.2 Posterior ankle arthroscopy portals and nearby critical anatomic structures

Posterior ankle arthroscopy, first described by van Dijk et al. in 2000 [9], allows for greater viewing of the tibiotalar and subtalar joints and has gained increased support for diagnosis and treatment of causes of posterior ankle impingement and cartilage and soft tissue disorders of the posterior hindfoot. The two portals used in posterior arthroscopy are the posteromedial (medial to the Achilles tendon) and posterolateral (lateral aspect of Achilles tendon) (Fig. 18.2). The medial portal risks injury to the main posterior tibial neurovascular bundle to the foot, while the lateral portal can injure the sural nerve and lesser saphenous vein.

18.2 Pertinent Anatomy

18.2.1 Superficial Peroneal Nerve

The superficial peroneal nerve arises from the common peroneal nerve at the fibular head and runs longitudinally in the lateral compartment of the lower leg. As it descends, it becomes more superficial within the compartment until approximately 4–5 cm above the ankle joint where it pierces the crural fascia to enter the subcutaneous plane. At the ankle, the nerve has been described by Takao et al. to have five branching patterns [10]. The most common branching pattern is type 2, with division into a medial dorsal cutaneous nerve and intermediate or lateral dorsal cutaneous nerve at the ankle, with further divisions into the terminal branches occurring more distally. The intermediate dorsal cutaneous nerve runs over the lateral two rays in the direction of the third metatarsal space before dividing into the lateral dorsal digital branches (Fig. 18.3), while the medial dorsal cutaneous nerve passes over the common extensor digitorum longus tendon to run with the extensor hallucis longus tendon and divides into the medial dorsal digital branches (Fig. 18.4). The variability of the branching patterns at the level of the ankle joint places the nerve at risk during creation of the anterolateral portal.

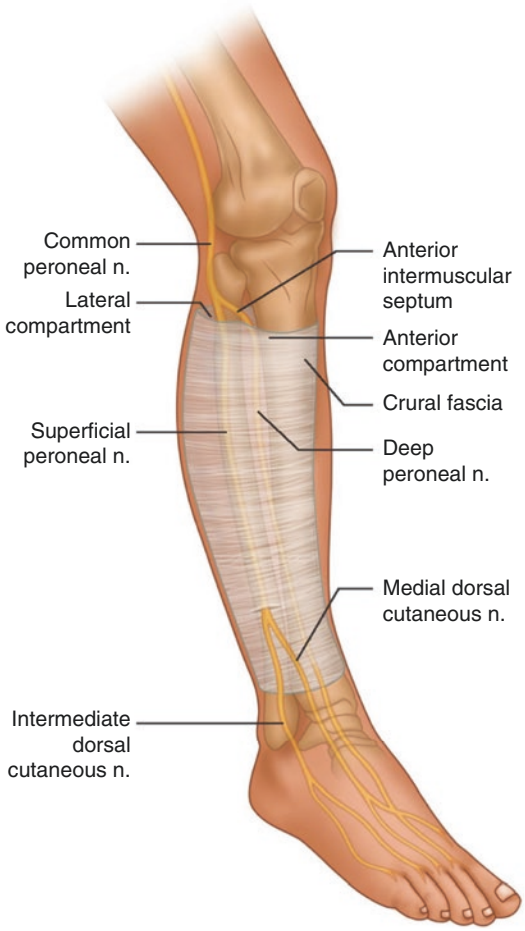

Fig. 18.3 Course of the peroneal nerve in the lower extremity

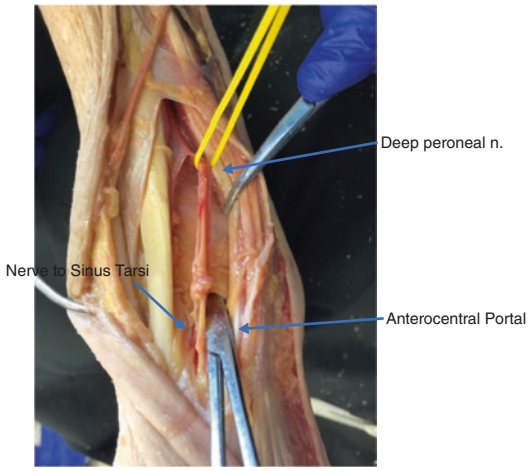

Fig. 18.4 Deep anterior ankle dissection demonstrating the deep peroneal nerve and its relationship to the anterocentral portal

18.2.2 Deep Peroneal Nerve

The deep peroneal nerve begins as a branch of the common peroneal nerve in the proximal leg and then courses through the anterior compartment between the fibula and peroneus longus, where it gives off several muscular branches as well as a branch to the articular surface of the ankle. At the ankle joint, it lies beneath the crural fascia, lateral to the dorsalis pedis artery between the extensor hallucis longus tendon and first tendon of the extensor digitorum longus. The previously described anterocentral portal places the deep peroneal nerve at great risk (Fig. 18.5). For this reason, the anteromedial portal, first described by Buckingham et al. [11], has replaced the anterocentral portal for general use.

18.2.3 Posterior Tibial Nerve

The tibial nerve arises as a branch of the sciatic nerve in the distal thigh, continues through the popliteal fossa, and then traverses the lower extremity on the deep surface of the soleus until it traverses posterior to the medial malleolus with the posterior tibial artery. At this point, the continuation of the tibial nerve is described as the posterior tibial nerve. The posterior tibial nerve then branches in the hindfoot to give rise to the medial and lateral plantar nerves, the main motor nerves of the foot musculature. Injury to the posterior tibial nerve is an uncommon complication of posteromedial portal insertion during posterior ankle arthroscopy.

18.2.4 Sural Nerve

The sural nerve is formed by fusion of the medial sural cutaneous nerve, a branch of the tibial nerve at the head of the lateral gastrocnemius, and the lateral sural cutaneous nerve, a cutaneous branch of the common peroneal nerve at the fibular head prior to division into its superficial and deep branches. The medial and lateral sural cutaneous nerves travel distally and join at the distal third of the gastrocnemius via the sural communicating branch where it pierces the muscular fascia and enters the subcutaneous

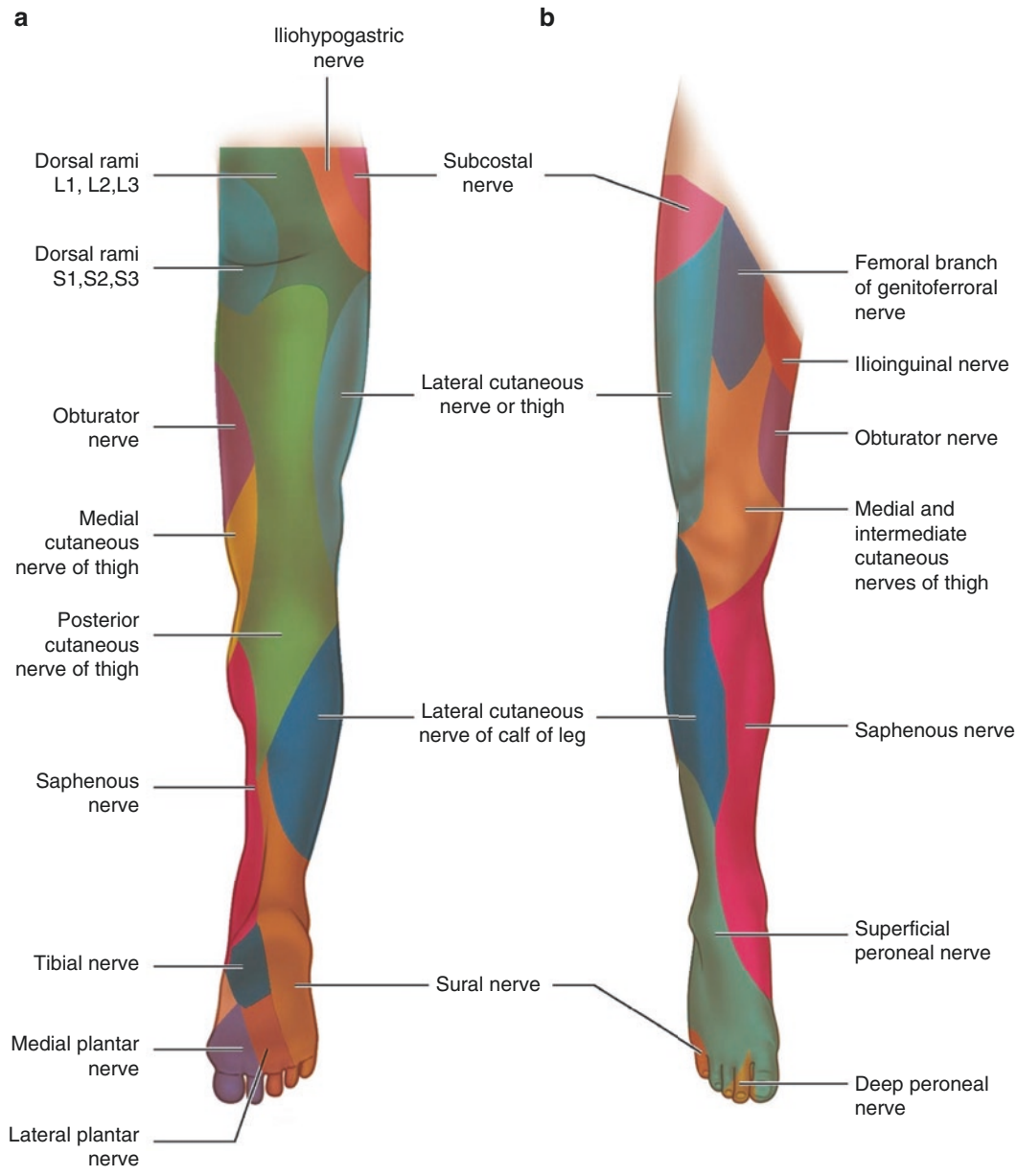

Fig. 18.5 Dermatome distribution of the anterior and posterior lower extremity

plane. It travels along this plane with the lesser saphenous vein and traverses the ankle 1.5 cm posterior to the lateral malleolus, posterior to the peroneal tendons. The nerve runs anterior to the short saphenous vein and then divides into the medial and lateral terminal branches at the base of the fifth metatarsal (Fig. 18.6). Insertion of the posterolateral portal places the sural nerve at risk during posterior ankle arthroscopy.

18.2.5 Saphenous Nerve

The saphenous nerve arises from the femoral nerve in the anterior thigh, where it passes deep to the sartorius muscle, travels through the adductor canal, and pierces the deep fascia 10 cm above the level of the knee between the tendons of the sartorius and gracilis where it enters the subcutaneous plane. The nerve then

travels along the tibial side of the leg in close proximity to the great saphenous vein and then descends posterior to the medial border of the tibia where it gives off anterior and posterior branches approximately 3 cm proximal to the tip of the medial malleolus. These branches provide sensation to the medial leg and ankle (Fig. 18.6). The saphenous nerve may be injured from insertion of the anteromedial portal during anterior ankle arthroscopy, or loss of sensation could also occur following harvest as a donor nerve graft (Fig. 18.7).

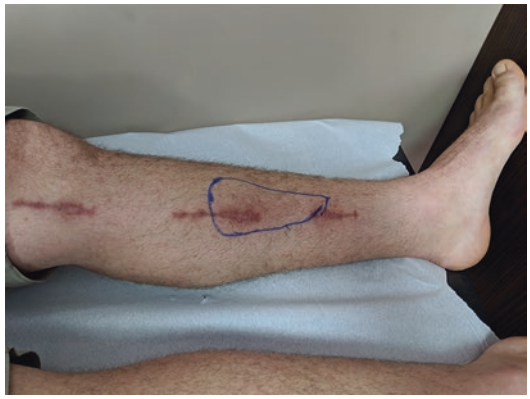

Fig. 18.6 Sensory deficit following saphenous nerve harvest for brachial plexus reconstruction. The marked area shows the resultant sensory deficit 2 years after donor harvest

18.3 Prevention Strategies

- A well-padded pneumatic tourniquet on the thigh, proper positioning of the extremity with the hip abducted, the knee flexed, and a knee holder for anterior ankle arthroscopy (removed for posterior ankle arthroscopy).
- Noninvasive ankle distractors using the dorsiflexion method or an ankle strap for longitudinal traction to widen the ankle joint space during surgery.
- Plantarflexion and inversion of the ankle can assist with surface identification of the intermediate dorsal cutaneous nerve, which can be marked preoperatively to prevent inadvertent injury.
- Placement of the anteromedial portal first and then placement of the anterolateral portal under inside-out visualization during anterior ankle arthroscopy.
- Placement of the posterolateral portal first and then placement of the posteromedial portal under inside-out visualization during posterior ankle arthroscopy.

18.4 Typical Course/Natural History

Injury to the superficial peroneal nerve or its branches during anterior ankle arthroscopy will

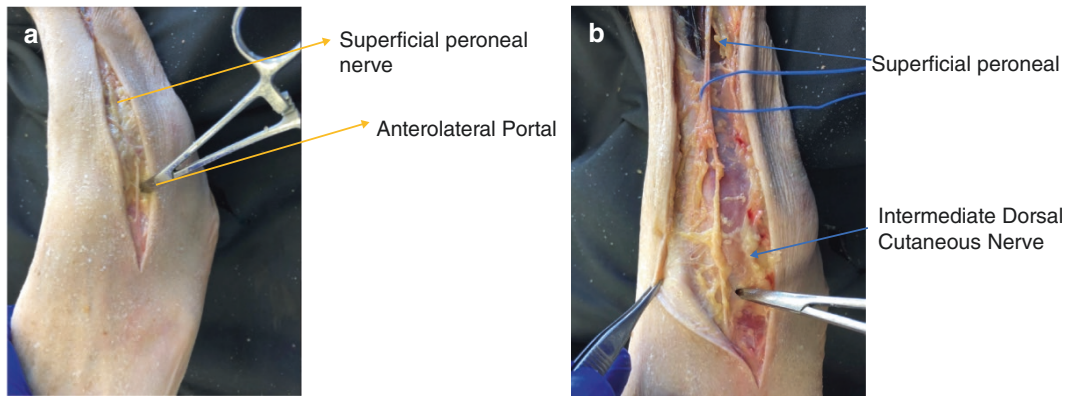

Fig. 18.7 Surface anatomy (**a**) and superficial dissection (**b**) showing the course of the superficial peroneal nerve and the relationship with the anterolateral portal

present immediately with loss of sensation within the affected dermatome on the lateral dorsal aspect of the foot and "wicked" neuroma pain. Severe axonotmesis or neurotmesis injury from portal placement will not allow for regeneration of normal sensation in this distribution, and permanent sensory loss will develop. Alternatively, aberrant nerve fibers may develop into a cutaneous neuroma over the subsequent months, which will present with paresthesia and palpable tenderness manifesting as a shock-like sensation at the neuroma site. Similar effects will be seen in the dermatome distribution for the other sensory nerves of the foot and ankle at risk during arthroscopy.

Damage to the posterior tibial nerve during posterior ankle arthroscopy is a much more devastating complication, as immediate loss of plantarflexion and the intrinsic motor function of the foot will occur. Patients will complain about inability to plant their foot during athletic activities and while driving an automobile. In addition, patients will exhibit sensory loss on the plantar surface of the foot, important for proprioception and protective movements.

18.5 Initial Evaluation/Exam

If a patient presents with concern for neurologic injury following anterior or posterior ankle arthroscopy, clinical examination with meticulous documentation is the key to determining the etiology of their symptoms and likelihood for improvement:

- Examination of surgical scars created from portal sites
- Tinel's sign at patient-directed affected areas to identify neuroma
- Sensation testing via light touch, two-point discrimination, proprioception, and monofilament threshold testing
- Motor testing of plantarflexion, inversion, and abduction/adduction of phalanges

18.6 Diagnostic Tests/Imaging

- Ultrasonography can be helpful to assist with identification of discontinuity at surgical site and surrounding scar tissue. If unable to visualize using ultrasound, magnetic resonance imaging can better identify areas of compression or neuroma locations.
- Diagnostic nerve blocks using lidocaine or bupivacaine can be beneficial to determine cause-effect alleviation of patient symptomology in nerve distribution and can rule out nerve injury as a potential cause of pain if the diagnosis of postoperative neurologic complication is unclear. Patients are encouraged to keep a diary documenting pain intensity at regular intervals to monitor pain relief. The diary is brought to the next consult and scanned into the medical record.
- Electromyography and nerve conduction studies can complement the physical examination, helping to localize the level of injury to the sensory nerves of the foot and ankle, as well as the posterior tibial nerve, and predict the likelihood of spontaneous recovery.

18.7 Surgical Techniques

Principles of peripheral nerve injury management have been largely elucidated as pertains to upper extremity injuries and similarly apply to foot and ankle surgery as well. If a peripheral nerve injury is recognized at the time of the index procedure, it is important to return to the operating room for re-exploration within 72 hours. Within this timeframe, the distal nerve ends contain neurotransmitters, and motor end plates can be stimulated intraoperatively, [12] critical for proper alignment of the posterior tibial nerve topography during repair. More often, iatrogenic nerve injuries go unnoticed during the immediate postoperative period and only become apparent when a painful neuroma has formed. Prior to surgical management of painful sensory neu-

roma, conservative measures such as desensitization protocols and neuropathic medications do no harm and may be occasionally beneficial. These should be employed for at least 6 weeks, depending on patient response [13].

When the decision to move forward with surgical intervention is made, careful preoperative planning of location to explore, modalities of repair, and likelihood of successful outcomes must be considered prior to entering the operating room. The first critical step in any nerve injury exploration is identification of the proximal and distal ends. Knowledge of nerve course is essential for this, as surgical planes will often be obfuscated by scar formation. Exploration should be performed under loupe magnification, and a well-padded tourniquet is preferred to provide a bloodless field during exploration. The primary goal is to reconnect the proximal and distal stumps whenever possible and allow the proximal axons to reach their targets. Once the proximal and distal stumps are identified and trimmed properly to healthy fascicles, tension-free coaptation may be attempted, to restore continuity. However often the resultant gap will demand a graft. Neurolysis from the surrounding scar tissue and anatomic positioning of the ankle (i.e., dorsiflexion for dorsal branches) can be helpful to decrease nerve gap; however, nerve gaps should not be directly repaired under tension as traction ischemia and contraction during healing will decrease likelihood of proper regeneration [14].

18.8 Salvage Techniques

In a delayed re-exploration, direct repair is usually not possible, as retraction of the proximal end and perineural scarring will make tension-free repair exceedingly difficult. In these cases, gap management using nerve substitutes must be considered. Nerve autograft is considered the gold standard for gap management, as it contains the necessary components for nerve regeneration including viable Schwann cells, endoneurial tubes, and extracellular matrix [15]. Commonly used autografts within the surgical field include the sural nerve and saphenous nerve, with the medial or lateral antebrachial cutaneous nerves easily harvested at a second surgical site. However, increased operative time along with sensory nerve loss and neuroma formation at the donor site preclude the use of autografts in small gaps <3 cm when other options are available [12].

Nerve substitutes include synthetic conduits and processed nerve allografts (PNAs). Both conduits and PNAs are appealing due to their "off-the-shelf" availability in a variety of lengths and diameters, ability to relieve tension off a direct repair, prevention of surrounding scar tissue formation, and support regeneration across a nerve gap without need for a donor site. Success of nerve regeneration with conduits and PNAs has been demonstrated in small sensory nerves such as the digital nerves of the hand; however, failed regeneration has been reported with larger diameter nerves and greater gap lengths. While nerve substitutes retain normal nerve architecture such as endoneurial tubes and extracellular matrix, [16] they lack the Schwann cells and nerve vasculature that aid in nerve regeneration [17].

When approaching a patient with peripheral nerve injury, repair is commonly performed utilizing the following procedural steps:

- Identification of proximal and distal ends of the injured peripheral nerve, and neurolysis within the surgical field to allow for free movement of the severed ends.
- Resection of scar tissue until visualization of healthy fascicular bundles in the cut ends.
- Measurement of nerve diameter and gap length while under minimum tension.
- If direct repair can be performed (<1 cm gap), place two to three 9/0 or 10/0 nonabsorbable monofilament sutures in the epineurium to coapt the ends with minimal trauma. Then, use an appropriately sized nerve conduit to wrap the coaptation site and protect from sur-

rounding scar tissue infiltration and take the tension off the repair ("remote detensioning").
- If direct repair cannot be performed without tension and nerve gap measures <3 cm, then utilization of a conduit (for gap <5 mm) or PNA is warranted. Coaptation of the proximal and distal ends to the substitute should be performed similarly to direct repair, with the minimal number of epineural sutures needed to secure the substitute across the nerve gap.
- If a conduit is used, the proximal and distal ends can be telescoped into the conduit via 9/0 or 10/0 nonabsorbable monofilament sutures in order to decrease the nerve gap and take tension off the severed ends.

Neuroma formation occurs following any failure of nerve regeneration; therefore, neuromas may be seen following the index procedure or after failed nerve repair from the aforementioned methods. Upon exploration of the surgical field, the proximal end may be found to end in a "scar ball," or the proximal and distal ends may be joined by a thickened segment of scar tissue, known as a neuroma-in-continuity. Following identification of the neuroma, it should be resected back to healthy nerve fascicles. When resection length is in question, frozen sections of nerve margins have been utilized for histologic evaluation of the margin of resection, with 75% of axonal elements in the stump being the threshold for adequate repair [18].

After resection is performed, multiple management options exist, including nerve repair using autografts or nerve substitutes; placement of an acellular nerve allograft "cap"; transposition of the proximal end into surrounding tissue such as muscle, bone, or veins; use of regenerative peripheral nerve interfaces; or targeted muscle reinnervation [18, 19]. While the data for each of these methods are largely based on studies on neuromas of the upper extremity and following lower extremity amputations, each technique has shown good to excellent results, and at this time no head-to-head studies for iatrogenic foot and ankle neurologic injuries have confirmed benefit of one technique over others [20].

18.9 Outcomes

In the foot and ankle literature, Souza et al. used PNAs to treat iatrogenic painful neuromas in 22 patients, most commonly of the sural and superficial peroneal nerve branches. After excision of end-neuromas and neuromas-in-continuity, the average gap spanned by PNAs was 3.3 cm, and after a minimum of 6 months' follow-up, their average pain scores decreased by a statistically significant and clinically important proportion with decreased ordinal pain and less interference with activities of daily living. While this retrospective review was limited by its small sample size, inherent bias due to its retrospective nature, and lack of comparison treatment outcome, it validated the use of PNAs for treatment of neuromas following neurologic injury in foot and ankle surgery [21].

Alternatively, Bibbo et al. described a methodology for treatment of severe recalcitrant superficial peroneal neuromas following anterior ankle arthroscopy via nerve transfer to the deep peroneal nerve using an allograft conduit in the mid-leg. Upon dissection and neurolysis of the superficial and deep peroneal nerves, stimulation was used to ensure the motor branches of the deep peroneal nerve had emanated proximally to their planned recipient site. Once the deep peroneal nerve sensory branch was confirmed, both nerves were divided proximally to the neuroma site, and a PNA with nerve wrap was used to span the gap between them for neurorrhaphy. After performing this transfer in 11 patients with a mean follow-up of 31 months, a statistically significant decrease in neuropathic pain was recorded by each patient, with all patients responding that they would choose to undergo the procedure again [22].

18.10 Technical Pearls and Pitfalls

18.10.1 Tarsal Tunnel Syndrome

18.10.1.1 Risks/Incidence/Mechanism of Nerve Injury/Structures at Risk

The tarsal tunnel is a fibro-osseous space located posterior to the medial malleolus. It has a bony floor formed by the medial talar surface, the sustentaculum tali, and the medial calcaneal wall. The roof of the tarsal tunnel is formed by the flexor retinaculum which is the thin fibrous tissue that has its origin from the medial and inferior aspect of the medial malleolus and inserts into the periosteum of the medial tuberosity of the calcaneus. The base of the flexor retinaculum corresponds to the superior border of the abductor hallucis muscle [23] (Fig. 18.8).

The posterior tibial, flexor digitorum longus, and flexor hallucis longus tendons are located within the tarsal tunnel, each with its own syno-

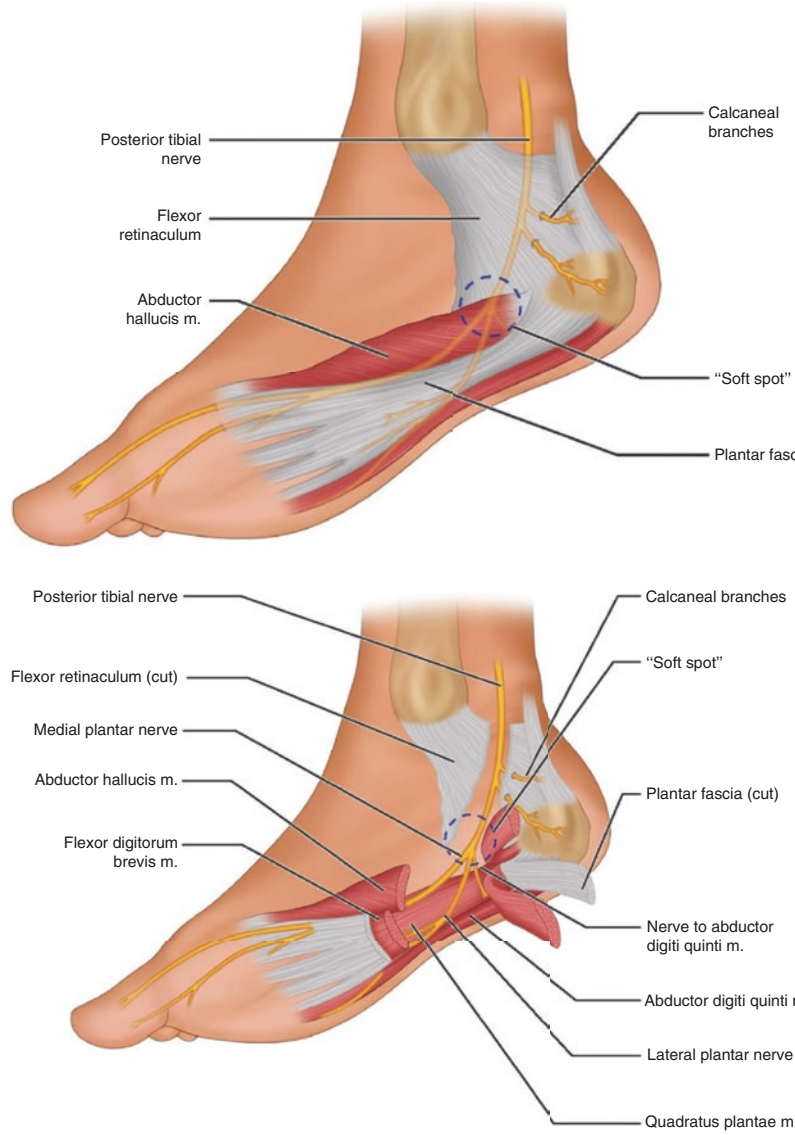

Fig. 18.8 Tarsal tunnel anatomy

vial sheath. The tendons are contained within a separate compartment formed by the fibrous projections from the undersurface of the flexor retinaculum. The tibial nerve enters the tarsal tunnel between the overlying flexor retinaculum and the underlying tendon sheath of the posterior tibial flexor digitorum longus and flexor hallucis longus muscles. The tibial nerve and artery are often attached to these sheets through surrounding areolar tissue. The tarsal tunnel is narrowest at its distal portion where it is conjoined with the fascia of the abductor hallucis longus muscle. The nerve at this level can become trapped causing tarsal tunnel syndrome, the most common entrapment neuropathy of the tibial nerve.

Tarsal tunnel syndrome was described in 1962 and was thought to be analogous to carpal tunnel syndrome with the flexor retinaculum being comparable to the transverse carpal ligament [24]. However, the medial plantar nerve, lateral plantar nerve, and the calcaneal nerve frequently reside in their own tunnels, making this analogy inaccurate [25].

18.11 Pertinent Anatomy

The tibial nerve arises from the medial half of the sciatic nerve, usually at the middle to distal one third of the thigh. The nerve is deep to the hamstring muscles, which are on either side of the posterior compartment of the thigh, and in the popliteal fossa the nerve lies posterior to the popliteal artery and vein. A medial hamstring branch occasionally leaves the tibial nerve at this level. More commonly, sensory branches to the proximal calf may arise before the nerve reaches its first major target as it courses through the popliteal fossa. The tibial nerve runs beneath the gastrocnemius soleus muscle group giving an abundance of branches to it and the plantaris, popliteus, and tibial muscles. Such branches begin to define themselves as separate tibial branches proximal to the superior edge of the gastrocnemius soleus complex [26].

A deeper posterior tibial branch accompanies the tibial artery and vein and runs through the leg medial and posterior to the tibia and posterior to the intermuscular septum, separating the anterior from the posterior compartments. The posterior tibial nerve carries fibers destined for the foot but gives off branches in the more proximal leg to supply the flexor digitorum longus and flexor hallucis longus muscle. As the posterior tibial nerve approaches the ankle, it courses inferior to the medial malleolus. At this level, it passes beneath the flexor retinaculum and branches into medial and lateral plantar nerves, although these nerves can also arise and be well defined proximal to the malleolus [27]. The lateral plantar nerve, which is comparable with the ulnar nerve at the hand, runs deep in the instep and supplies the second to fourth lumbricals, the adductor hallucis, and all interossei except that of the fourth metatarsal. It also supplies the skin of the fifth toe and the lateral half of the fourth toe.

The medial plantar branch provides sensation to the medial plantar surface of the foot and innervation to the abductor hallucis and flexor digitorum brevis muscles. A third branch – the calcaneal nerve – can usually be found either arising proximal to these nerves or branching from the medial plantar nerve. The calcaneal nerve can have numerous anatomic variations [28]. Injury to the medial and lateral plantar nerves may spare sensation on the heel of the foot as the calcaneal nerve provides cutaneous innervation to this region.

The tarsal tunnel may be divided into a proximal zone which extends from the retinaculum to the origin of the abductor muscle and a distal zone which begins at the fibrous origin of the abductor hallucis muscle and extends through this muscle. The distal zone may contain three additional separate tunnels: the medial plantar tunnel, the lateral plantar tunnel, and the calcaneal tunnel. The goal of surgery is to decompress all four tunnels: the tarsal tunnel and the three separate distal tunnels [29] (Fig. 18.9).

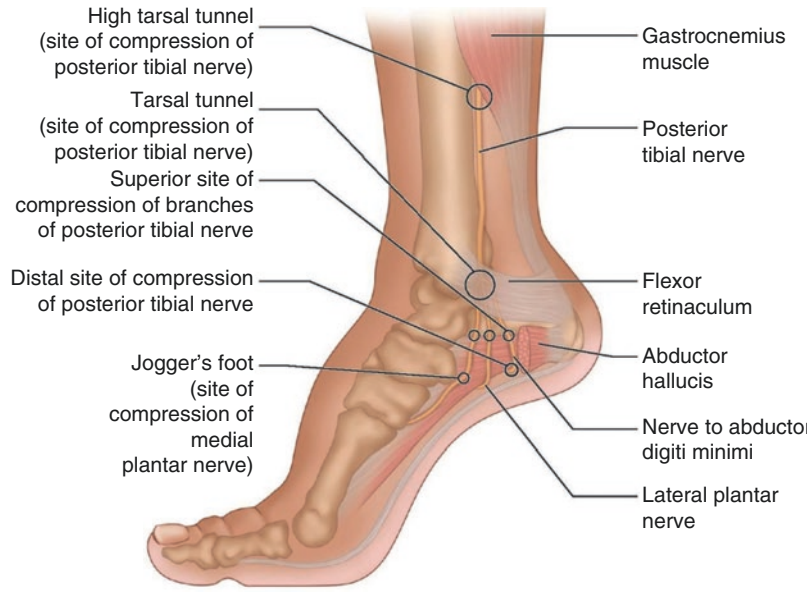

Fig. 18.9 Artist depiction of the four compression sites at tibial nerve branches

18.12 Typical Course/Natural History

Tarsal tunnel syndrome is a spontaneous and slowly progressive condition. Common associations include obesity, decreased elasticity of collagen, or a progressive flatfoot in an adult [23]. Other causes of the syndrome are a deep medial ganglion from the subtalar joint, an adjacent chronic tenosynovitis, or partial rupture of the posterior tibial tendon with secondary compression of the nerve [30].

18.13 Initial Evaluation

An accurate diagnosis depends on a detailed history and meticulous clinical examination, with adjunctive electrical studies and occasionally advanced imaging. Patients complain of burning plantar heel pain, often in the metatarsal area and occasionally radiating to the medial calf. This may be alleviated by rest and aggravated activity, although some patients report night pain [31]. The main physical examination finding is a Tinel sign producing paresthesias on the plantar surface of the foot and usually elicited inferior to the medial malleolus and sometimes proximally or distally in the region of the instep.

A positive Tinel sign test proximal to the point of compression usually means that the nerve is compressed about 2 cm distal to the enlarged tibial nerve. Sometimes there is either a mild hypoesthesia or mixed hypo- and hyperesthesia on the sole or heel of the foot. Toe flexion and foot intrinsic function are usually spared in the majority of cases unless there has been a prior operation, ankle or foot injury as a precipitating factor, or if symptoms have been long-standing. The presence of Tinel's sign is predictive of a positive response to nerve decompression [32].

18.14 Diagnostic Tests

Perineural infiltration of 1% lidocaine with or without cortisone via local injection may diminish paresthesias and pain with weight-bearing, and although anesthesia is only temporary, the relief of pain and discomfort provides diagnostic

information [33]. Patients are advised to keep a "pain diary" and document pain symptoms following the injection. Pain relief after lidocaine injection may be therapeutic for a varying period of time and signals that surgical intervention in the future may resolve the symptoms. However partial response may signify different etiology or a second nerve compression at a different anatomical site.

18.14.1 Electrodiagnostic Studies

Electrodiagnostic studies may be used to supplement the clinical examination findings [23] but are not essential to make the diagnosis in every case. A recent review could not determine the sensitivity and specificity of electrodiagnostic studies in the diagnosis of tarsal tunnel syndrome [34]. As with other compression neuropathies, it is important to compare values from the involved foot with those in the contralateral unaffected limb. The normal reference limits are far less precisely defined for tibial nerve compression than they are for carpal tunnel studies.

Since nerve compression is dynamic in its early stages, it is not unusual to have a negative electrical study unless the patient undergoes 10–15 minutes of walking, standing, or tiptoeing before the tests are performed. Typically, if exercise causes paresthesias, compression is usually present [23, 35].

18.15 Surgical Techniques

Nonoperative treatment for tarsal tunnel syndrome includes ankle immobilization, anti-inflammatory medications, and frequent use of a wide comfortable shoe. An orthosis with a relief within the medial arch may be effective if distal tarsal tunnel is suspected or if symptoms are worsened by longitudinal arch support orthosis.

It is important to exclude diabetes and alcoholic neuropathies and to be certain that the foot has sufficient blood supply to heal the surgical wound before performing tarsal tunnel release. Metabolic neuropathy with secondary nerve compression is not a contraindication to surgery, as evidence supports the role of nerve decompression in this patient population as an adjunct to medical optimization [36, 37]. Contraindications for surgery include morbid obesity, severe venous stasis, and insufficient blood supply to heal surgical wounds. Caution should be exercised in patients older than 60 and those with no identifiable cause of the symptoms.

18.15.1 Tarsal Tunnel Release

The procedure begins with a curvilinear skin incision about 5 cm proximal and posterior to the medial malleolus, curving anteriorly to the heel. Alternatively, two incisions can be made – the one proximal to the malleolus and the second distal to the malleolus to expose the three tarsal tunnels. Magnification and the use of the tourniquet, medium-sized tenotomy scissors, microbipolar electrocautery, and Penrose drains or vessel loops for retraction are helpful. Stepwise and patient dissection is essential, especially as the dissection progresses distally. Initially, the posterior tibial nerve is found medial to the Achilles tendon and proximal to the medial malleolus. The nerve is then traced beneath the medial malleolus by dividing the overlying flexor retinaculum. Exposure of the posterior tibial nerve at this level is often compared with that of the median nerve, though the tarsal tunnel is much more complex, and dissection is more tedious. The tibial artery has a serpiginous course and arterial and venous branches are intertwined with the nerve as it forms the medial and lateral plantar and calcaneal nerves. The medial and lateral plantar nerves are traced distally as they reach the medial border of the abductor hallucis and continue plantarward deep to the muscle. As dissection continues distally, three fascial layers require release: superficial abductor fascia, deep abductor fascia, and septum anchoring the deep fascia of the muscle to the calcaneus (Fig. 18.10).

The lateral plantar nerve is identified first and followed into its separate tunnel by dividing the

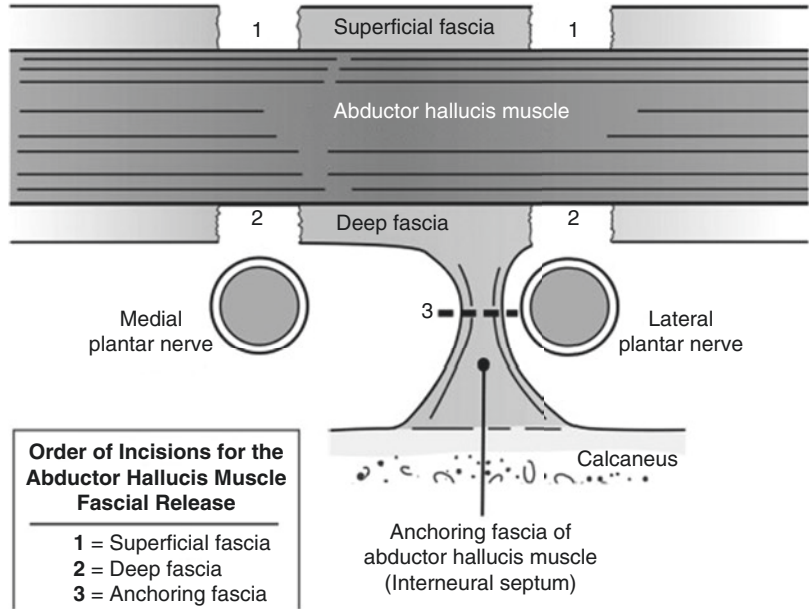

Fig. 18.10 Cross-sectional illustration of the medial and lateral plantar tunnels with interneural septum

fascial origin of the abductor hallucis brevis, which is the roof of the tunnel. The medial plantar nerve is more anterior and is unroofed in its separate tunnel as well. Care is taken to avoid injury to the little unnamed branch from the medial plantar nerve into the skin of the medial arch [38]. Injury to this nerve will lead to chronic pain at the distal aspect of the tarsal tunnel skin incision.

The branches are separated and must be entirely unroofed and exposed circumferentially to provide a bed for the nerves and branches free of scar or compressive tissue. This must include sectioning of the overlying muscle and its fascial edge in the instep portion of the foot. The septum between the medial and lateral plantar tunnels is longitudinally released. The calcaneal branch should be decompressed in its tunnel, especially in patients reporting heel pain (Fig. 18.11). Complete external neurolysis is performed at this stage. Successful release of the distal tunnels allows the surgeon's small finger to pass into the plantar aspect of the foot (Fig. 18.12). The wound is then closed in a standard fashion. The patient is encouraged to ambulate after surgery in order to prevent scarring of the nerve.

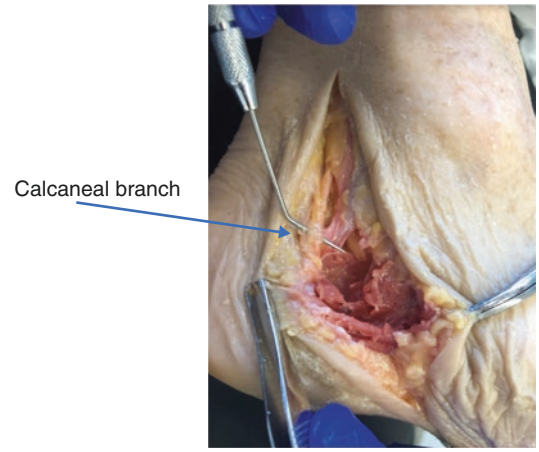

Fig. 18.11 Anatomic dissection of the calcaneal branch of the posterior tibial nerve. Isolated decompression of the calcaneal nerve should be performed especially in patients with heel pain. Unnamed proximal branches of this nerve (not shown) can lead to painful neuroma if cut unintentionally

18.16 Outcomes

Mullick and Dellon summarize their long-term outcomes after 87 release procedures with an average follow-up of 3.6 years. They reported

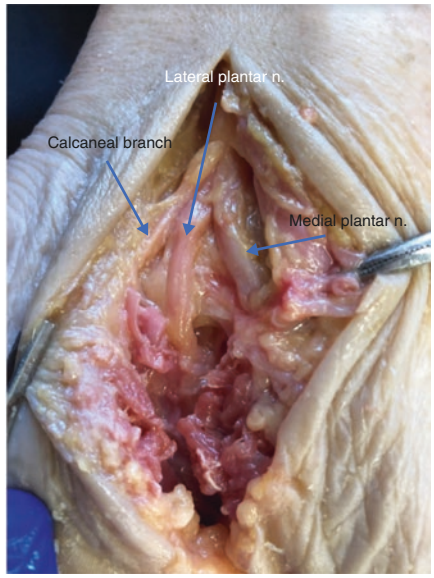

Fig. 18.12 Anatomic dissection demonstrating decompression and neurolysis of all branches of the posterior tibial nerve

resolution of symptoms in 82% of patients, with slight residual numbness and tingling in 11% of the patients who still were able to return to work [39]. Kim and Murovic reported their results in 46 feet from 43 patients with the diagnosis of tarsal tunnel syndrome who underwent decompression with complete external neurolysis, sectioning of the flexor retinaculum and origin of the abductor hallucis muscle, and splitting of the muscles of the instep. In 28 patients without prior surgery, the outcome following external neurolysis was excellent in 22 (79%) and fair to poor in 6 (21%) [40]. Lack of response following nerve decompression is well-described and may signify a second compression site or severe neuropathy. We tell our patients that decompressive surgery does not cure neuropathy, and regeneration may take up to 1 year. We encourage them to walk frequently postoperatively in order to prevent adhesions and mobilize the released nerves.

18.17 Technical Pearls

- Successful relief of symptoms may be offered to the patient with tibial nerve entrapment in the tarsal tunnel, using the same principles that were developed to treat the upper extremity nerves.
- Careful surgical technique can lead to successful outcomes even in patients with superimposed metabolic neuropathy predisposed to compression neuropathy.
- Clinical history, the presence of Tinel's sign, and response to local nerve blocks are the cornerstones of diagnosis.

Decompression of the proximal tarsal tunnel as well as the three distal tunnels is essential along with external neurolysis to separate the nerve from surrounding structures.

18.17.1 Hallux Valgus

18.17.1.1 Risks/Incidence/Mechanism of Nerve Injury

Hallux valgus or "bunion deformity" is one of the most common disorders of the forefoot. The hallmark of the condition is lateral deviation of the great toe phalanges (hallux) and medial deviation of the first metatarsal. The disease is progressive with multiple stages, eventually leading to progressive subluxation of the first metatarsophalangeal (MTP) joint [41]. As the disease progresses, symptoms such as poor fitting shoes, plantar foot, medial first MTP joint pain, decreased athletic performance from loss of propulsion and abnormal weight-bearing distribution, [42] and first MTP joint destruction are all seen. While the disease is commonly seen in adults, juvenile hallux valgus can occur. Furthermore, women are diagnosed more frequently than men, with some studies quoting a F:M ratio of 15:1; women are also more likely to have surgery. This disparity is theorized to be the consequence of more frequent use of tight fitting and high-heeled shoes [43]. While restrictive footwear is thought to play a role in development of the disorder, intrinsic factors such as genetics, pronation of the hindfoot, pes planus (flat foot), hypermobility, Achilles tendon contracture, cerebral palsy, and previous strokes have all been associated with hallux valgus onset [44].

Diagnosis of hallux valgus includes a thorough history, including duration of symptoms,

footwear, activity modification, and family history. Physical examination should test observance of gait, alignment, range of motion of the first MTP joint, specific areas of tenderness, presence of calluses or bunions, and presence or absence of Achilles tightness. Weight-bearing radiographs are also necessary for diagnosis to view the angle between the longitudinal axis of the first metatarsal and first proximal phalanx, known as the hallux valgus angle, and the intermetatarsal angle between the longitudinal axis of the first and second metatarsal. Typically, a hallux valgus angle >15 degrees or an intermetatarsal angle >9 degrees has been defined as abnormal with varying degrees of severity as these angles increase [45]. The radiographic classification of hallux valgus divides the deformity into mild, moderate, and severe based on these angles and the degree of subluxation of the lateral sesamoid on anteroposterior view.

Management of hallux valgus always starts conservatively, with modalities such as avoidance of tight-fitting high-heeled shoes and use of wide-toed soft footwear, as well as various inserts/pads such as bunion shields and toe spacers for support and comfort. Physical therapy is prescribed for stretching and balance correction [46]. When patients fail nonoperative management and continue to have symptoms of the progressive deformity, surgical intervention is indicated to improve athletic performance and alleviate disruption of lifestyle and activities of daily living.

Over 100 options for surgical intervention of hallux valgus exist, with severity of disease dictating choice of treatment. Mild to moderate deformity is typically treated by distal procedures such as simple bunionectomy, the modified McBride procedure, or distal Chevron osteotomy. Severe deformity usually involves surgical treatment of the MTP joint, and procedures such as the proximal Chevron osteotomy, proximal oblique ("Ludloff") osteotomy, proximal crescentic osteotomy, and opening wedge proximal first metatarsal osteotomy all have been described and advocated by different surgeons. Additionally, minimally invasive percutaneous surgery has become increasingly popular, with proponents touting quicker surgical and recovery times with an overall decrease in morbidity [47]. The descriptions of the many individual operations are beyond the scope of this chapter, but at this time no consensus has been made as to which open surgical technique [48], or minimally invasive procedure [49], provides the best outcomes.

Neurologic injury following surgical correction of hallux valgus is a rare complication, as a recent systematic review by Bard et al. that evaluated 229 studies for outcomes analysis found only 3% of patients suffered intraoperative nerve injury [50]. The nerve most commonly injured in these cases was the dorsomedial cutaneous nerve (DMCN), which innervates the medial surface of the hallux (Fig. 18.13a, b). Despite the many operative techniques for correction of the deformity, exposure of the underlying anatomic structures typically requires a dorsomedial incision which places the DMCN at risk. Damage to this nerve can result in a very painful postoperative course following a relatively benign procedure and must be avoided at all costs.

18.17.1.2 Pertinent Anatomy

As described previously in this chapter, the superficial peroneal nerve divides into multiple branches that give sensation to the dorsum of the foot (Fig. 18.3). The medial most branch, termed the medial dorsal cutaneous nerve (MDCN), typically branches from the superficial peroneal nerve near the ankle. The MDCN then further branches and gives off the dorsomedial cutaneous nerve (DMCN), which travels superficial to the extensor hallucis longus (EHL) tendon, before terminating near the distal dorsomedial aspect of the first metatarsal [51]. Solomon et al. reported that the DMCN independently supplies the cutaneous innervation to the first metatarsal and medial aspect of the great toe in 100% of cadaver specimens [52]; thus, the DMCN is also referred to as the proper dorsal digital nerve to the great toe. Additionally, Solomon et al. determined the DMCN supplies sensation to the lateral aspect of the great toe and the medial aspect of the second digit in 41% and 47% of specimens, respectively, in conjunction with branches from the deep peroneal nerve. This emphasizes the

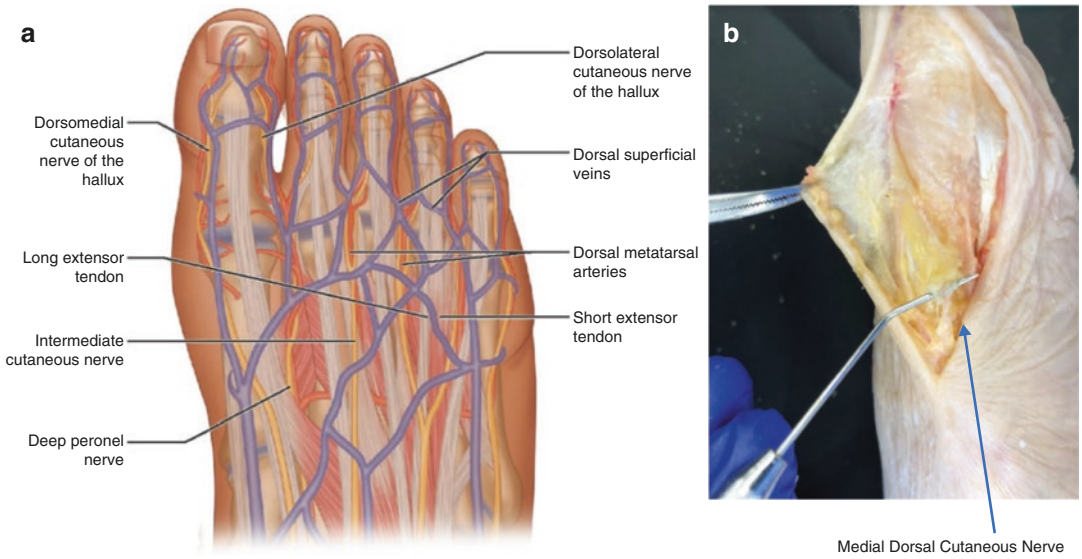

Fig. 18.13 (**a**) Artist depiction of superficial anatomy of the dorsal forefoot; (**b**) anatomic dissection of the dorsomedial cutaneous nerve

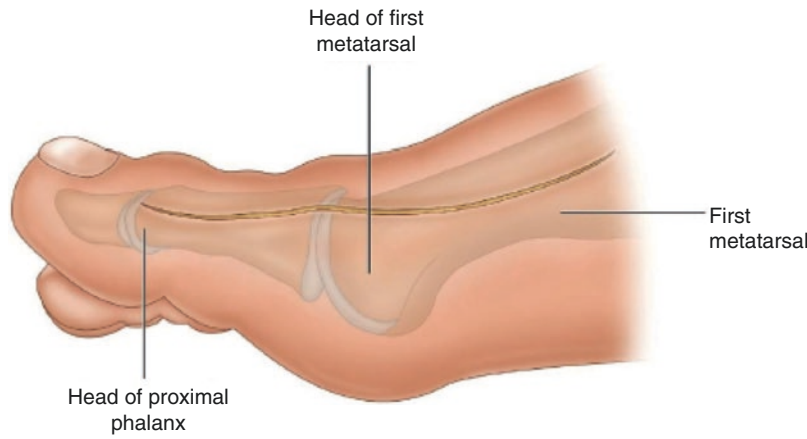

Fig. 18.14 Illustration demonstrating the dorsomedial incision with nearby bony landmarks

clinical importance of the DMCN during operations on the forefoot.

18.17.1.3 Prevention Strategies

- Use of a well-padded pneumatic tourniquet and loupe magnification to aid in visualization of neurovascular structures.
- A mid-dorsal incision should be made at the junction of the dorsal and plantar skin (Fig. 18.14).
- Identification of subcutaneous superficial veins and careful division under direct vision will avoid injury to the DMCN in the direct vicinity.
- When performing MTP arthroscopy, placement of dorsomedial and dorsolateral portals to the MTP joint should be approximately 0.5 cm from the margins of the EHL to avoid injury to the DMCN and terminal branches to the peroneal nerve [53] (Fig. 18.15).

18.17.1.4 Typical Course/Natural History

Intraoperative injury to the DMCN will lead to immediate numbness over the medial aspect of the hallux and in some patients may cause numbness of the lateral aspect of the hallux as well. Within subsequent weeks, sensation can

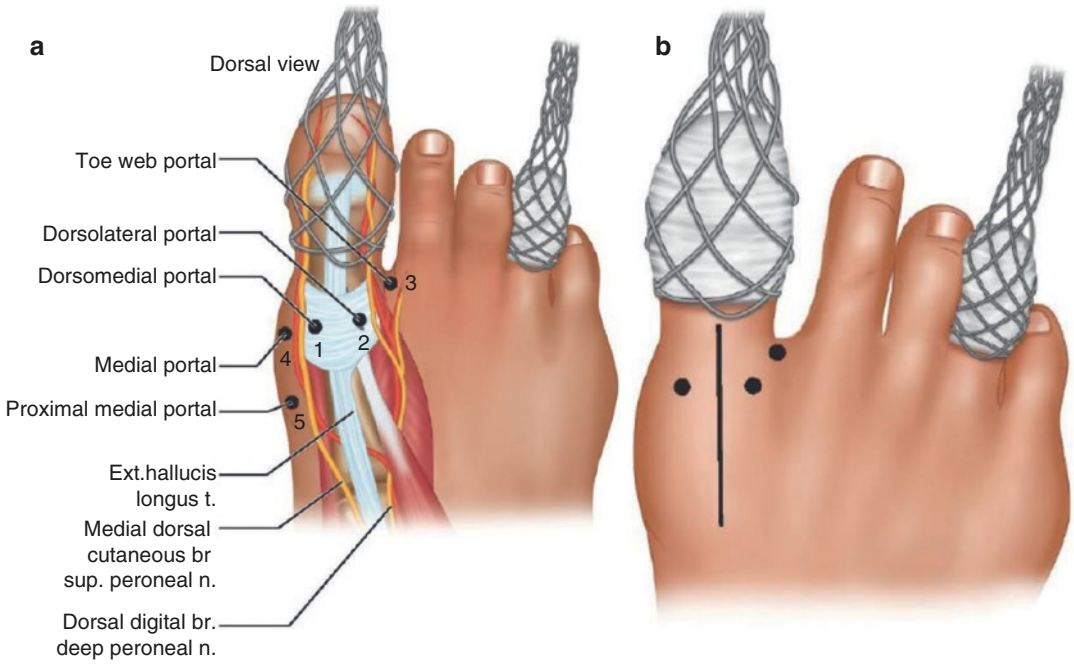

Fig. 18.15 (**a**) Underlying anatomic structures and (**b**) superficial skin landmarks for first MTP arthroscopy

remain absent or patients can begin to develop what's known as DMCN syndrome: neuropathic pain at the site of injury with numbness or paresthesia distally along the nerve innervation course. A Tinel sign may be present at the surgical scar line where the nerve injury occurred, with hyperesthesia and a shock-like sensation occurring from even minimal palpation. This can often cause difficulty with activities of daily living, as patients may be unable to wear shoes or place weight on the affected limb, leading to an overall useless limb secondary to pain intolerance.

18.17.1.5 Initial Evaluation

If a patient presents with concern for neurologic injury following surgical correction of hallux valgus deformity, clinical examination is the key to determining the etiology of their symptoms and likelihood for improvement.

- Examination of surgical scars
- Tinel's sign at patient-directed affected areas to identify neuroma
- Sensation testing via two-point discrimination, proprioception, and monofilament esthesiometer sensation (standard monofilament values 3.5–4.5 for feet) [54–56]

18.17.2 Diagnostic Tests/Imaging

- Ultrasonography can be helpful to assist with identification of discontinuity at surgical site and surrounding scar tissue. If unable to visualize using ultrasound, magnetic resonance imaging can better identify areas of compression or neuroma locations.
- Diagnostic nerve blockade using lidocaine can be beneficial to determine cause-effect alleviation of patient symptomology in nerve

distribution and can rule out nerve injury as a potential cause of pain if the diagnosis of postoperative neurologic complication is unclear.
- Electromyography and nerve conduction studies can assist with identification of injury to the sensory nerves of the foot and ankle.

18.18 Surgical Techniques

Identification of injury to the DMCN intraoperatively necessitates repair. If the ends are sharply transected during exposure, simple nerve repair with or without conduit assistance should be performed using 2–3 9/0 nylon sutures in the epineurium. If the nerve is injured via crush or thermal damage, then the affected segments should be excised, and the gap should be spanned with peripheral nerve allografts (PNAs) or nerve conduits. These products can be coapted to proximal and distal nerve ends in similar fashion with minimal amount of 9/0 or 10/0 nylon in the epineurium of the nerve ends.

More often, damage to the DMCN goes unnoticed during the index surgery, and discovery of nerve injury is recognized once patients present with medial hallux numbness or DMCN symptoms such as paresthesia and shock-like sensation from neuroma formation. Initial numbness should not immediately lead to re-exploration, as neurapraxia may resolve over the subsequent months. However, patients with persistent symptoms should undergo re-exploration if pain and sensitivity cause excessive morbidity and therefore unfavorable outcomes.

When approaching a patient with peripheral nerve injury, repair is commonly performed utilizing the procedural steps detailed previously in this chapter (see "Ankle Arthroscopy" section, "nerve repair").

18.19 Salvage Techniques

After neuroma resection is performed, multiple options of management exist, including nerve repair using autografts or nerve substitutes; placement of an acellular nerve allograft "cap"; transposition of the proximal end into surrounding tissue such as muscle, bone, or veins; use of regenerative peripheral nerve interfaces; or targeted muscle reinnervation [18, 19]. While the data for each of these methods are largely based on studies on neuromas of the upper extremity and following lower extremity amputations, each technique has shown good to excellent results, and at this time no head-to-head studies for iatrogenic foot and ankle neurologic injuries have confirmed benefit of one technique over others [20].

18.20 Outcomes

Miller [57] published his data of a small cohort of nine patients with DMCN syndrome following hallux valgus surgery which were treated with reoperation following a minimum of 4 months' symptom duration. After identification of the DMCN neuroma, the neuroma was resected, and the proximal end of the nerve was buried into nearby bone, preferably the base of the first metatarsal, but also the cuneiform or navicular bones if the neuroma was found more proximally. Nerve burial was performed by exposing a small area of denuded bone and drilling a 3.5 mm hole into the bone 1.5 cm in depth. After placement of the proximal end in this burial site without substantial tension, a 5/0 absorbable suture was used to secure the epineurium to nearby periosteum. Following closure of superficial tissue, the patient began weight-bearing in a hard-soled shoe at 2 days postoperatively, which they continued for 4 weeks.

At a mean follow-up of 20 months, all patients had a substantial decrease in pain symptoms, with all stating they could walk much better and would likely undergo the surgery again if given the choice. However, these results may be somewhat confounded as all patients underwent concurrent surgery at the time of nerve burial, most commonly bunionectomy or arthrodesis. At follow-up, patients felt confident that they could distinguish neuropathic pain from other pain sources. Furthermore, all postoperative pain scoring was performed by the operative surgeon,

a potential source of bias. Nevertheless, this study proved the feasibility of nerve transection and osseous implantation in patients suffering from DMCN syndrome. Invariably, the end of the nerve will attempt to regrow (albeit in its new position inside the bone) and may then form a recurrent symptomatic neuroma. Although this is the surgical technique with the longest track record, it does not address the nerve end. Although there are no randomized trials comparing this technique to others, there is rationale to believe that it may be inferior to more active methods of neuroma treatment such as coaptation-based techniques.

18.21 Technical Pearls and Pitfalls

18.21.1 Interdigital Neuritis (Morton's Neuroma)

18.21.1.1 Risks/Incidence/Mechanism of Nerve Injury

The painful forefoot condition known as Morton's neuroma presents with sharp or burning pain in the second or third webspace, often radiating to one or two toes. As previously reported, this is a compression neuropathy of the common digital nerve involving the distal transverse metatarsal ligament (DTML), making terms like Morton's metatarsalgia or interdigital neuritis (IDN) more appropriate [58].

The terminology used to describe IDN and its treatment is replete with confusion.

Morton's neuroma is the most commonly used term despite the fact that the condition is not the neuroma per se, lacking the haphazard proliferation of axons seen in a neuroma. This leads to additional misleading terminology regarding treatment.

Neuroma excision is a misnomer and should be termed neuroma production; *recurrent Morton's neuroma* is the result of excision becoming a symptomatic neuroma and not a recurrence. The true meaning of these terms should be kept in mind while reading the literature and counseling patients.

18.21.2 Pertinent Anatomy

The medial plantar nerve enters the foot between the abductor hallucis and quadratus plantae and then continues distally to give off four digital branches (Fig. 18.16). The most medial branch is the proper digital nerve to the medial aspect of the big toe. The other three branches including the first, second, and third common digital nerves supply sensation to the first, second, and third interspaces. The lateral plantar nerve divides into the proper digital nerve to the lateral side of the fifth toe and the common digital nerve to the fourth interspace, which has a communicating branch that passes to the third digital branch of the medial plantar nerve in the third interspace. This makes the third interspace dually innervated from both medial and lateral plantar nerves.

18.21.3 Prevention Strategies

Nonoperative treatment should always be the first line in treatment of IDN, with the goal of avoiding surgery or delaying it as much as possible. Nonsurgical interventions to treat a compression neuropathy of a sensory nerve in the foot come in different varieties. These interventions may address the weight-bearing environment and the surrounding local irritation or modulate the production of pain. Orthosis to offload the forefoot, injections of different materials, and shockwave therapy are some common methods. Special attention should be given to calf muscle stretching to offload the forefoot and ameliorate the gait abnormalities associated with calf muscle tightness.

A recent literature review demonstrated corticosteroid injections to be effective for 12 months, with the response rate declining to 50% after 12 months, leading to surgical excision in 33% [63]. Alcohol injection resulted in pain relief in 29% of patients at 5 years but was also associated with burning pain. Shockwave therapy, botulinum toxin injection, capsaicin injection, and laser therapy had little or no evidence.

Since a satisfying surgical solution does not exist, surgery should be thought of as the final

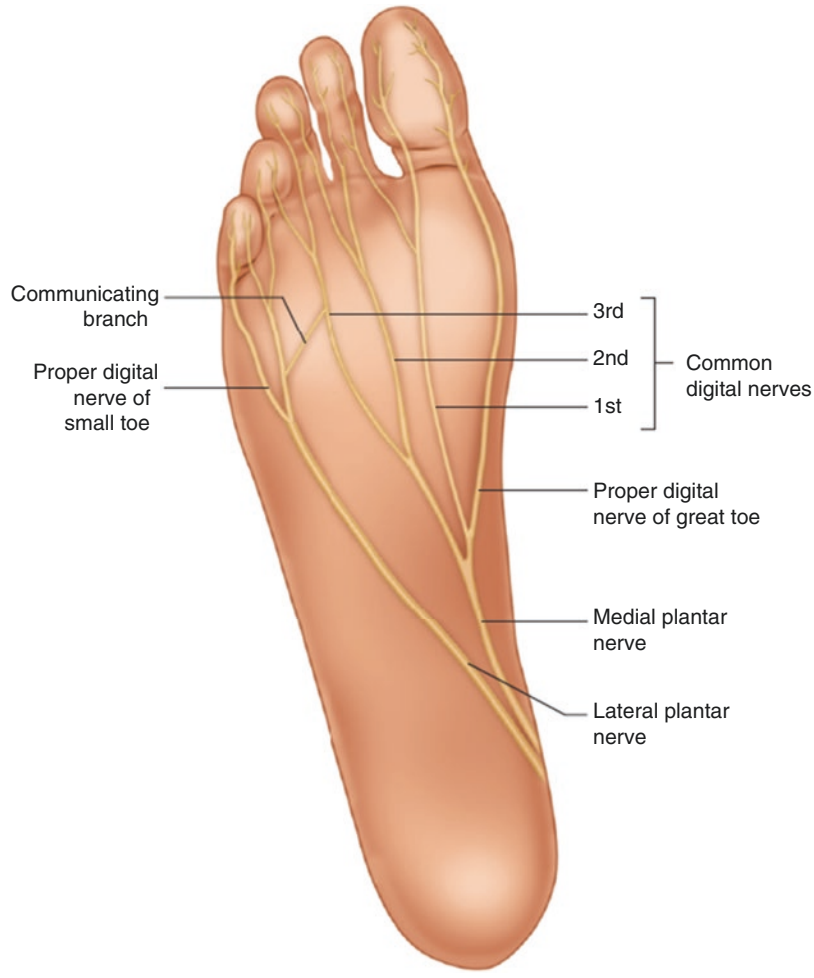

Fig. 18.16 Plantar nerve anatomy of the foot

resort in patients with persistent pain, and every effort should be made to maximize nonoperative treatment.

18.21.4 Typical Course/Natural History

The typical patient with Morton's neuroma will report footwear-related pain and frequent need to remove shoes and massage his or her foot. Nerve quality is sharp, with tingling in the toes. Women are more commonly affected than men.

18.21.4.1 Initial Evaluation/Exam Findings

The physical examination of Morton's neuroma begins with localizing tenderness and dorsal bulging of the affected webspace (typically the third) between the metatarsal heads but not at the heads themselves. Pain is induced with compression of the intermetatarsal space or with tightening the metatarsals to one another which may be associated with a painful click (Mulder's sign) [59]. The thumb and index finger squeeze test, which is simply squeezing the webspace between the thumb and index, has a

96% sensitivity for the diagnosis of Morton's neuroma [60].

Calf muscle tightness should be examined and every patient with forefoot pathology. This reduces the pressure in the heel and instead transfers pressure distally to the metatarsal heads. Gastrocnemius contracture is assessed using the Silfverskiold test: assessing ankle dorsiflexion with the knee in full extension and 90° of flexion, with the foot locked in subtalar neutral position. Equinus contracture is noted by lack of dorsiflexion past neutral.

18.21.5 Diagnostic Tests/Imaging

Although imaging is not indicated for diagnosis of Morton's neuroma, plain weight-bearing foot radiographs are helpful to exclude other causes of pain such as stress fractures and degenerative MTP changes or subluxation and in order to assess the relative length of the metatarsals (which may contribute to metatarsalgia). These diagnoses are not mutually exclusive and may coexist with IDN.

In cases of clinical uncertainty and multiple webspace involvement, ultrasound has been suggested as the imaging modality of choice [61].

A diagnostic block using 1–2 mL of local anesthetic with or without cortisone may be useful in equivocal cases; however, caution must be exercised in interpreting the results since the local anesthetic may diffuse toward neighboring structures such as joint capsule and can therefore limit its diagnostic utility [62].

18.22 Surgical Techniques

18.22.1 General Considerations

The mainstay of surgical treatment is sharp division of the nerve proximal to the enlarged nerve segment, such that the proximal stump retracts to the level of the muscle bellies. This converts a peripheral compression neuropathy into a true stump neuroma. For comparison, compression neuropathies in other locations are treated with decompression procedures alone and release of the offending structures. There are no other reports of compression neuropathy treated with nerve resection. This led to the development of alternative approaches to nerve resection and prevention of painful stump neuromas.

18.22.2 Decompression Alone

Decompression involves incision of the DTML and decompression of the common digital nerve [64], thereby interrupting the pathophysiology of the Morton entrapment process (Fig. 18.17).

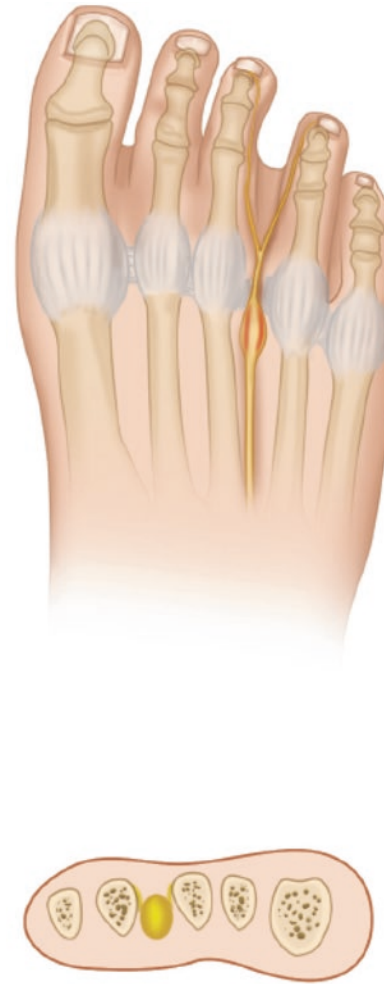

Fig. 18.17 Schematic of dorsal deep transverse metatarsal ligament division and nerve decompression. Note that the nerve crosses plantarward under the ligament

Dellon in 1992 performed decompression on five patients, with pain relief in 80% (four) of the patients. Earlier series suggested partial split of the DTML with good results in 83% of cases, improved results in 14.5%, and 2.5% failure rate [65].

Others have recommended against simple resection of the ligament due to recurrent compression from the regenerated soft tissue [66]; however, their study involved patients who underwent excision making that conclusion questionable. Although this consideration may be anatomically correct, this has not been demonstrated in clinical studies.

Okafor reported the results of neurolysis 1 cm distal to the DTML and 3 cm proximal to it, with patient satisfaction noted to be "extremely high," and complete pain relief in 17 out of 35 patients [67]. Zelent reported on the results of nerve decompression using a device designed for carpal tunnel release [68], resulting in absent symptoms in 11 of the 14 patients at 25 months' follow-up.

Villas [69] reported similar pain relief with neurolysis and neurectomy in a group of 69 feet. They concluded that neurolysis is a valid option if the nerve is not macroscopically thick, which was their selection criterion for neurectomy. Recent report on nerve decompression using Dellon's DTML sectioning technique in 12 patients showed improved foot functional scores and pain scores over the median follow-up period of 37 months [70].

Song described decompression and dorsal suspension of the nerve using the dorsal transverse ligament and compared it to standard neurectomy, with a mean follow-up of 34 months [72]. They showed comparable results with fewer complications of paresthesia and numbness in the dorsal suspension group, essentially relocating the nerve to a more dorsal position away from the weight-bearing surface.

A recent systematic review included neurectomy in 14 studies and decompression in four studies [72]. The authors reported 88% success rate for neurectomy versus 94% success rate for decompression, with no difference between dorsal and plantar approaches, over 46 months' follow-up.

Taken together, these studies demonstrate favorable outcomes for nerve decompression, supporting the notion that IDN is an entrapment neuropathy. The results are comparable with nerve resection, without the risk of creating a stump neuroma and sensory loss. Simple decompression is a straightforward and easy to master procedure with minimal risk to surrounding tissues and low morbidity. Cadaver study has demonstrated negligible and clinically undetectable widening of the intermetatarsal angle and metatarsal alignment [73].

18.22.3 Neurectomy

Resection of the common digital nerve is the most common procedure for interdigital neuritis although the procedure converts an irritated nerve into an inevitable stump neuroma (Fig. 18.18).

Nerve excision can be performed from plantar or dorsal approach. The dorsal approach (Fig. 18.19a) allows release of the DTML, as well as neurolysis. It is considered technically easy and does not involve a scar at the sensitive plantar surface. This approach is usually recommended for primary cases. Plantar incision is more direct as the common digital nerve is more superficial at this location. Its drawbacks include a sensitive plantar scar and delayed weight-bearing. This approach is usually reserved for revision or "recurrent" cases. The plantar longitudinal incision is designed proximally and between the metatarsal heads so that any scarring will not be directly under the weight-bearing area but instead over the intermetatarsal spaces (Fig. 18.19b, c).

18.22.4 Nerve Excision and Interpositional Nerve Grafting

Ratanshi reported their experience with nerve excision and interpositional nerve grafting in eight patients with nine neuromas, after failure of nonoperative treatment with a minimum of 1-year follow-up [77]. The neuroma was excised and a

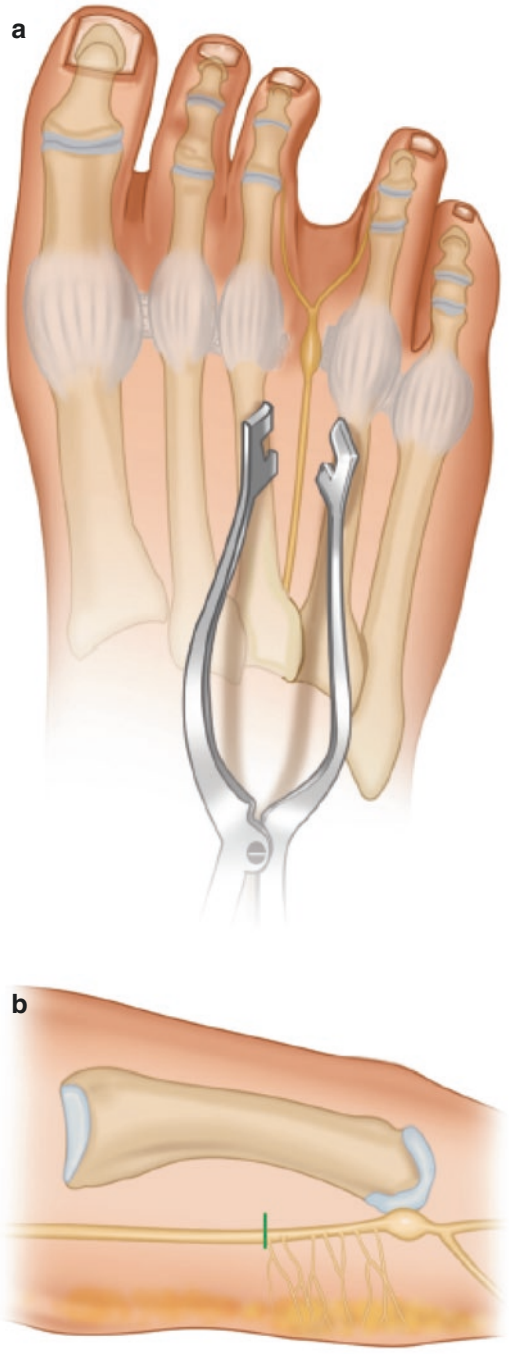

Fig. 18.18 (**a**) Exposure of the common digital nerve through the soft tissues between the metatarsal heads. A Weitlaner is placed between the metatarsal heads to gain optimal exposure. (**b**) Lateral view of the common digital nerve with the level of resection marked in green

segment of the proper digital nerve to one of the toes just distal to the excised neuroma was harvested, reversed, and interposed as a nerve graft between the common digital nerve stump and the adjacent distal proper digital nerve.

The authors reported pain relief in all patients with no recurrence as well as return of sensation. The authors recommended excision and nerve grafting as the primary treatment for cases that failed nonoperative treatment, given the above advantages.

18.22.4.1 Salvage Techniques

Nerve resection is associated with a 14%–21% failure rate [78]. In general, patients with symptoms following nerve resection can be classified into three groups: (1) Patients describe the same symptoms postoperatively with no period of relief; (2) the period of relief followed by recurrence of the same or worse symptoms; (3) patients describe new symptoms following surgery.

In the first group of patients where symptoms never subsided, the problem may have been initially thought to be IDN but is in fact a different condition that mimics the symptom complex (wrong diagnosis). In these patients, the correct diagnosis should be sought, such as tarsal tunnel syndrome, other causes of metatarsalgia, or IDN at an adjacent space (wrong interspace).

In the second group of patients, symptoms are related to the inevitable neuroma formed at the proximal stump by the excision of the common plantar digital nerve. This is erroneously termed "recurrent neuroma." The stump may have not retracted proximal enough, became adherent to the plantar plate or skin and irritated with cyclic weight-bearing.

The third group of patients is challenging to manage since they describe a new symptom complex: either hypersensitivity from disruption of the small plantar branches of the digital nerve or chronic regional pain syndrome (CRPS).

In patients with a symptomatic proximal stump neuroma, associated conditions such as tight calf muscles should be optimized. A local anesthetic injection with or without cortisone and

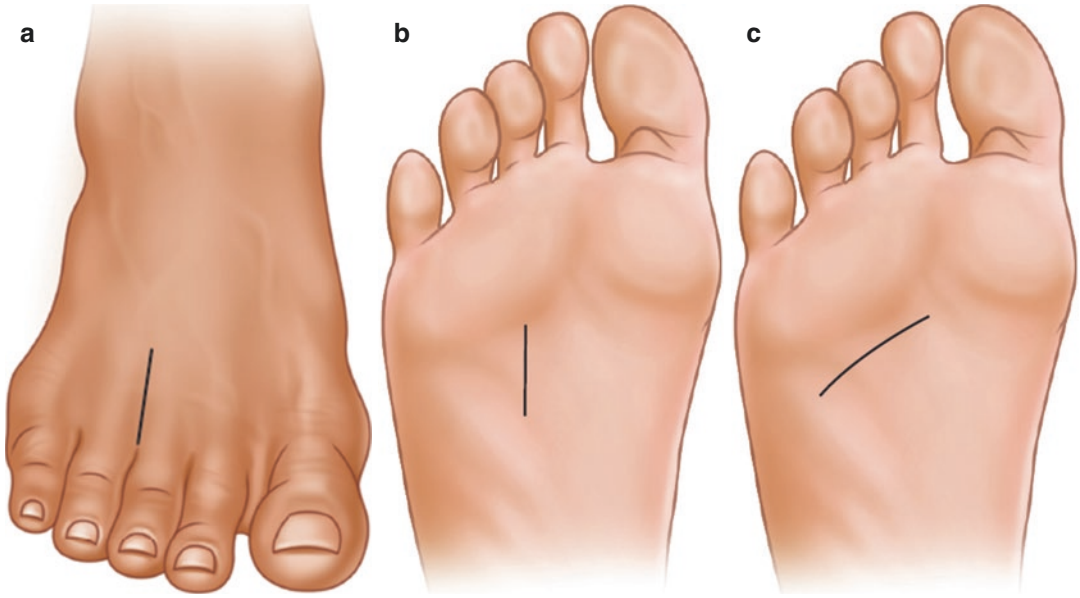

Fig. 18.19 Dorsal and plantar longitudinal incisions. (**a**) The dorsal incision is begun in the webspace and carried proximally and midline for about 3 cm to the level of the metatarsal heads. (**b**) The plantar longitudinal incision centered over the intermetatarsal space approximately 1 cm proximal to the metatarsal heads. (**c**) The plantar horizontal incision allows access to adjacent intermetatarsal space, also located approximately 1 cm proximal to the metatarsal heads

documentation of pain intensity using a pain diary reviewed at a follow-up consultation are essential in confirming the proximal stump as the offending agent. A trial of nonoperative treatment is appropriate before embarking on revision surgery. This will typically include medications such as pregabalin or nortriptyline that may be helpful in decreasing neuropathic pain. This group of patients is historically reported to do poorly with re-resection, with unsatisfactory results in 20–40% of patients [79].

Given the significant morbidity and unsatisfactory results of re-resection, alternative surgical techniques have been developed to alleviate pain with minimal morbidity and complications.

The approach to the symptomatic stump neuroma has been revolutionized in the past few years, incorporating new surgical approaches and techniques, which may be employed following IDN resection. The two major categories of stump neuroma management are passive-ablative and active-reconstructive interventions [19]. These are based on the presence or absence of the distal stump. If on exploration the distal proper digital nerve is available, autograft allograft or conduit reconstruction may be considered, to complete the nerve circuit. This potentially restores sensation to one or two toes. If however the distal end is unavailable, reconstruction is deemed unfeasible, or the distal nerve is extremely small or scarred, the proximal stump may be re-resected and implanted dorsally into muscle, adjacent soft tissues or bone, away from the weight-bearing area. There are no randomized controlled trials of these methods in IDN. Additionally, these methods relocate the neuroma without addressing the nerve's potential for regrowth and development of a symptomatic neuroma in a different location.

With this in mind, active methods of treatment have been developed, to provide a neural pathway for the regenerating axons, thereby decreasing the potential for regrowth and symptomatic neuroma formation. These include relocation nerve grafting using the long nerve graft, capping the nerve with a vein or conduit to ameliorate regrowth, or coapting the nerve stump to a nearby muscular branch. This last technique (targeted

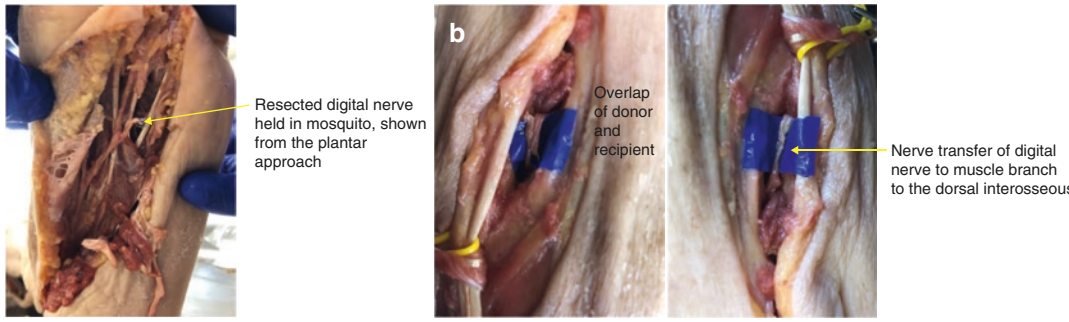

Fig. 18.20 (**a**) Nerve transfer for persistently painful neuroma stump, following primary nerve resection – plantar view. (**b**) Nerve transfer for persistently painful neuroma stump, following primary nerve resection – dorsal view

muscle reinnervation) provides the nerve with a denervated, vascularized target to grow into. Taking into account the unique local anatomy of the foot, nerve transfers to dorsal interossei motor branches can be designed to relocate the nerve away from the susceptible weight-bearing area and provide it with a target for regeneration. If a gap exists between the donor and recipient, due to the very proximal excision, nerve graft is necessary to overcome the segmental nerve loss. In this regard, commercially available cadaveric nerve allograft is ideal as it is available off the shelf, has good handling properties, and avoids creating a donor site neuroma as with autograft. However, its use is associated with increased cost.

Figure 18.20 shows a cadaver dissection with a simulated nerve transfer of the cut proximal stump of the third common digital nerve to a nearby dorsal interosseous motor branch. The motor nerves to the interossei foot muscles are of adequate size to facilitate nerve rotation to the common digital nerve. Further anatomic studies are underway to map the location of the motor entry points in the foot.

Alternatively, a muscle graft may provide a pathway for neural regeneration from the distal nerve stump into empty motor endplates of the denervated muscle graft. This technique is known as regenerative peripheral nerve interface (RPNI), found to be useful in reduction of neuroma pain and phantom limb pain in major limb amputations [80].

18.22.4.2 Outcomes

After a thorough and accurate diagnosis, nerve excision results in considerable improvement of IDN symptoms in the majority of patients, around 80% [58]. In Mann's series, 65% of patients still noted local plantar tenderness after surgery and 20% noted the improvement to be less than 50% [66]. Womack reported 51% good to excellent results, 10% failure results, and 40% poor results on long-term follow-up of 120 patients [74]. Other authors reported 15-year follow-up of nerve excision, with 76% good or excellent result, fair in 15%, and poor in 8% [75]. Finally, a prospective study reporting the pre- and postoperative patient-reported outcomes and satisfaction scores following nerve excision reported 9% poor and very poor results and pain relief in only 63% of patients. The authors concluded that patient-reported outcomes after nerve excision are acceptable but may not be as good as earlier studies suggested [76].

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