

# RECURRENT TARSAI TUNNEL SYNDROME: What Now?

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## INTRODUCTION

Tarsal tunnel syndrome, first described independently in 1962 by Keck<sup>1</sup> and Lam,<sup>2</sup> is an entrapment neuropathy of the posterior tibial nerve or one of its branches created by compression of the nerves within the fibro-osseous tunnels posterior and inferior to the medial malleolus. Once diagnosed, surgical decompression of tarsal tunnel syndrome has often been necessary and is a frequently performed surgery. Recurrent tarsal tunnel syndrome then is defined as continuing tarsal tunnel syndrome following previous surgical decompression or tarsal tunnel release. Recurrent tarsal tunnel syndrome is a frustrating condition and can be difficult to treat. The treatment results for recurrent tarsal tunnel syndrome are less predictable than with the initial conservative or surgical treatment.<sup>3</sup> We will discuss the etiologies, clinical presentation, diagnosis, and treatment options for recurrent tarsal tunnel syndrome.

## ETIOLOGIES

There are many potential etiologies of recurrent tarsal tunnel syndrome. These can be loosely subdivided into 6 categories: incorrect initial diagnosis; incomplete release; adhesive neuritis; intraneural damage; double crush syndrome; and, idiopathic or other causes.<sup>4</sup> Differentiating between these different causes of failed tarsal tunnel syndrome can aid the clinician in guiding proper additional treatment and can increase the likelihood of treatment success for this recalcitrant condition.

## CLINICAL PRESENTATION

Recurrent tarsal tunnel syndrome generally presents in 3 distinct ways: the patient shows no improvement or worsens after the initial procedure, the patient shows only partial improvement after the initial procedure, and the patient has temporary relief over a period of weeks to months with subsequent recurrence of all or part of their symptoms.<sup>4</sup> Common clinical features seen in most patients with recurrent tarsal tunnel syndrome include burning, tingling, and numbness over the distribution of

the involved nerve or nerves. With plantar nerve involvement, symptoms are localized to the toes, distal sole of the foot, and the abductor canal in the proximal medial arch. When the medial calcaneal branch is involved, symptoms are localized to the heel.

## DIAGNOSIS

A thorough history and physical examination is critical in the patient with recurrent tarsal tunnel syndrome. This should include an investigation of the initial complaints before the primary release contrasted with the current complaints. Attention should focus on the nature, location, duration, onset, and course of the presenting complaints. Evidence of metabolic disorders should be investigated and ruled out.

If the clinician examining the patient did not perform the initial release, the operative report from the primary decompression should be obtained and reviewed, if possible. The operative report may provide evidence that an incomplete release was previously performed.

Physical examination should include an assessment of the prior surgical incision(s). The location and length of the incision can provide clues as to whether a complete release was previously performed. A thickened scar, hypertrophic scar, or keloid may provide evidence of underlying excessive scar formation and the possibility of adhesive neuritis. The clinician should palpate and percuss the posterior tibial nerve. Point tenderness over the nerve or distal tingling upon percussion (i.e., Tinel's sign) can be strong clinical indicators of continuing tarsal tunnel syndrome. Sensorimotor testing should be done to assess areas of decreased two-point discrimination, decreased light touch, and paresthesias. Stance and gait evaluation should be performed to assess the extent any concomitant foot deformity or antalgic gait pattern has on the clinical findings. The physical examination should also include an assessment of the proximal peripheral nerves of the ipsilateral lower extremity and spine to rule-out double crush syndrome as an etiology for the recurrent symptomatology.

Magnetic resonance imaging (MRI) studies should be considered in most cases of recurrent tarsal tunnel

syndrome. This is especially true if an MRI study was not performed prior to the initial tarsal tunnel decompression. MRI can also help identify any space-occupying soft tissue lesions that might be contributing to recurrent tarsal tunnel syndrome.<sup>5-9</sup> Erickson and others,<sup>5</sup> using MRI, were able to identify neurilemmomas, tenosynovitis, ganglion cysts, post-traumatic fibrosis and post-traumatic neuromas causing tarsal tunnel syndrome. Kerr and Frey<sup>7</sup> found lesions in 82% of their cases with the most common causes being a focal mass lesion and varicose veins. Another study by the same authors studied 40 symptomatic feet using MRI.<sup>9</sup> Electrodiagnostic studies confirmed the diagnosis in 20 feet. Seventeen of these 20 (85%) had positive MRI findings with the most common being flexor hallucis tenosynovitis. Surgery was required in 21 feet and confirmed MRI findings in 19 of the 21. Downey<sup>8</sup> also described the combined use of the Perthes' test and MRI to confirm venous insufficiency of the venae comitantes as a cause of tarsal tunnel syndrome. Finally, MRI has been shown to be effective in evaluating incomplete release of the flexor retinaculum.<sup>10</sup>

Electrodiagnostic studies are often insufficiently sensitive to assess tarsal tunnel syndrome and offer little predictive value to the prognosis of recurrent tarsal tunnel syndrome. Despite this reality, they should still be obtained in most cases to evaluate and rule out the presence of systemic disease, intraneural damage, or more proximal sites of compression.

## TREATMENT

The treatment of recurrent tarsal tunnel syndrome is typically oriented towards the etiology of the continuing symptomatology. Hopefully, the etiology has been elucidated by the diagnostic work up. Many times, the etiology of the recurrence will be multi-factorial and the treatment approach may not be entirely straightforward.

### Incorrect Initial Diagnosis

If the initial diagnosis of tarsal tunnel syndrome was incorrect, then the symptoms will invariably continue or recur postoperatively. The most common reasons for misdiagnosis include conditions that mimic or secondarily cause tarsal tunnel symptomatology, such as a space-occupying lesion (e.g., a ganglion within the tarsal tunnel), tenosynovitis (e.g., tendon inflammation and fluid accumulation associated with tibialis posterior tendon dysfunction), or mechanical abnormality (e.g., severe valgus hindfoot or ankle deformity causing eversion stress on the posterior tibial nerve). An MRI study can be critical to establishing the correct diagnosis in such cases. If a

recurrent tarsal tunnel syndrome is due to an initial misdiagnosis, the continued treatment should address the underlying correct pathology.

### Incomplete Release

Initial surgery has traditionally focused on releasing the flexor retinaculum without addressing the plantar branches distally. In the literature, inadequate distal decompression has been reported as the most common indication for revisional surgery.<sup>11-12</sup> Skalley and associates<sup>11</sup> analyzed clinical results following revisional tarsal tunnel release. Revisional surgery was performed a mean of 3.5 years after initial release. They identified three groups of patients based on intraoperative findings and clinical outcome. The first group (4 feet), which did poorly, revealed posterior tibial nerve scarring and inadequate distal release at the initial surgery. The second group (5 feet), which overall were improved, had scarring of the nerve, and an adequate distal release. The third and final group (4 feet), which did well, had no posterior tibial nerve scarring but inadequate distal release. The authors concluded that the results of epineurolysis from scarring of the posterior tibial nerve are less predictable and they would not recommend surgical exploration following a previous tarsal tunnel surgery with an adequate distal release. Their technique did note the use of intraoperative nerve stimulation to identify the nerve encased in scar tissue.

An incomplete distal release should be suspected if the initial release was done through an endoscopic approach or if the healed surgical incision from the initial release is of inadequate length and/or location for a full release to have been performed. Typically, the patient with an incomplete release describes no improvement or only partial improvement after the initial procedure, depending on the extent of the initial release and what remains compressed. If the distal portion of the tarsal tunnel was not released initially, the patient will often have minimal or no symptomatology over the proximal portion of the posterior tibial nerve, but will have point tenderness and/or a positive Tinel's sign over the abductor hallucis muscle distally.

As noted, if an incomplete distal release was performed at the time of the initial surgery, the prognosis for improvement is fair to good with revisional surgery that includes adequate distal decompression (Figure 1). When performing the revisional surgery, the initial surgical incision should be expanded both proximally and distally. The posterior tibial nerve should be identified proximal to any scar tissue and carefully re-released throughout the tarsal tunnel region. The posterior tibial

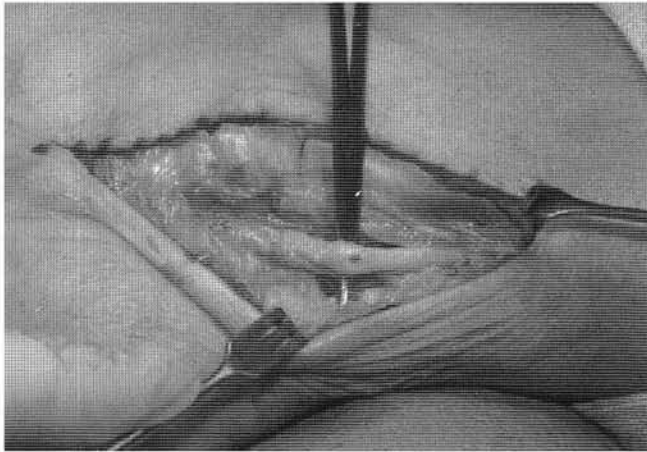


Figure 1A. Revisional tarsal tunnel decompression with distal release. Incision made from approximately 2 cm above the flexor retinaculum to the inferior margin of the abductor hallucis muscle. The posterior tibial nerve has been identified in the third compartment of the tarsal tunnel.

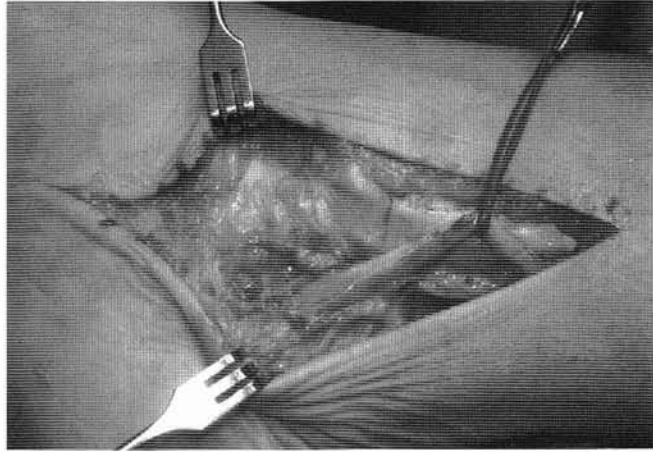


Figure 1B. The posterior tibial nerve is gently manipulated with a vessel loop and the dissection is carried distally.

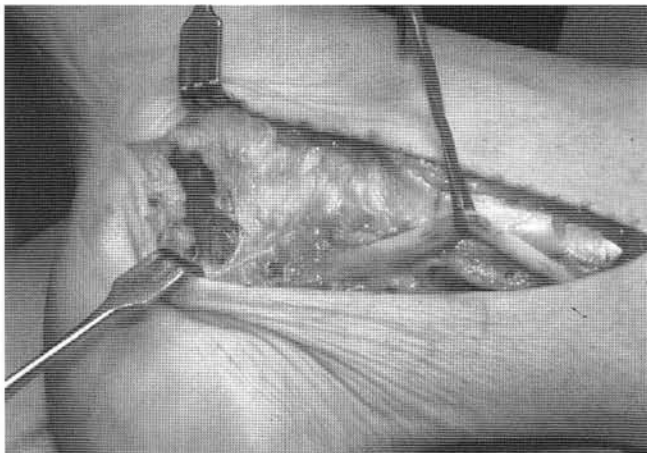


Figure 1C. Dissection to the level of the abductor hallucis muscle. The porta pedis may be dilated or incised.



Figure 1D. The deep fascia overlying the abductor hallucis muscle is incised and the abductor muscle retracted inferiorly. The fascia deep to the abductor hallucis muscle is then visualized. This forms the roof of the distal tarsal tunnel or porta pedis.



Figure 1E. Incision of the deeper fascia exposes the medial and lateral plantar nerves.

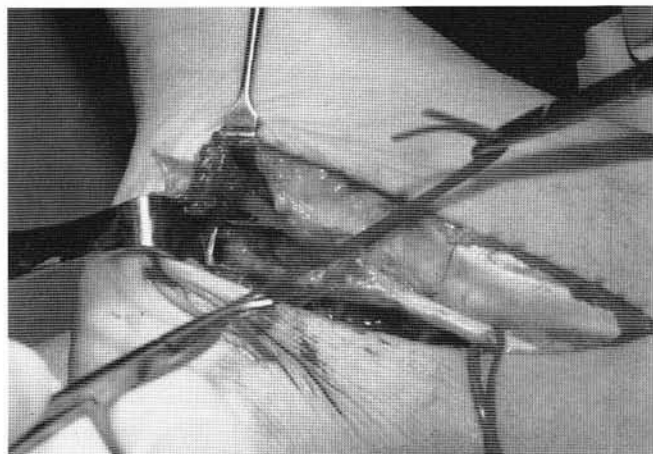


Figure 1F. The septum between the nerves is incised creating a large common tunnel for the two nerves. This completes the tarsal tunnel release.

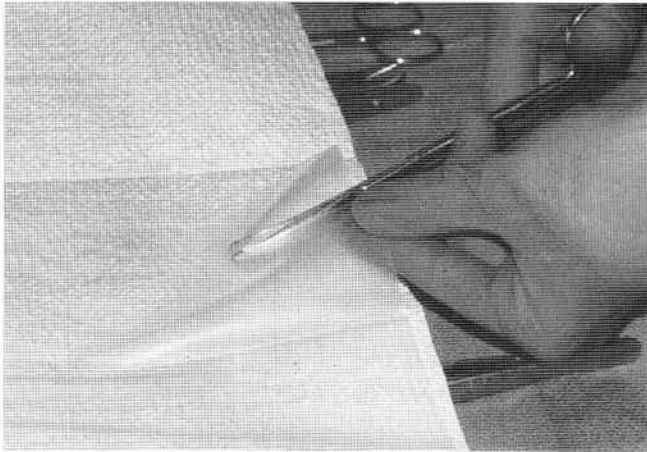


Figure 2A. Silicone entubulation of the posterior tibial nerve done in a patient with recurrent tarsal tunnel syndrome. 0.007 surgical grade silicone sheeting is used and cut to the appropriate size.

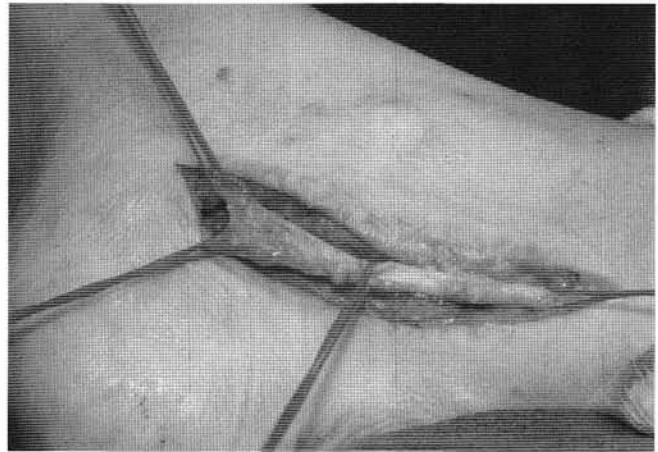


Figure 2B. Revisional tarsal tunnel decompression is performed.

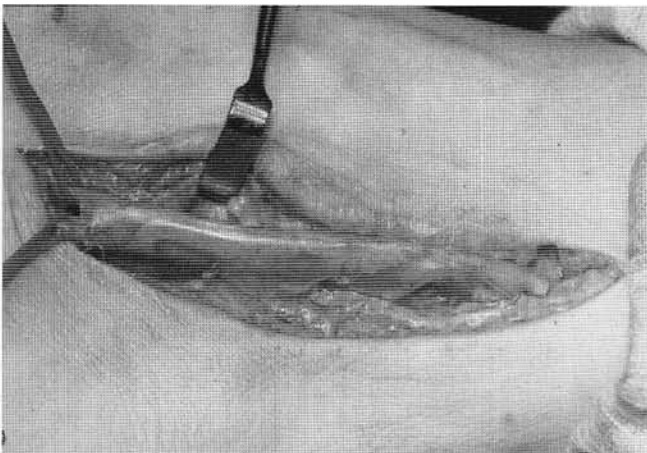


Figure 2C. The posterior tibial nerve is ensheathed with the silicone sheeting.

nerve should then be tracked distally to expose and decompress the terminal branches of the nerve, including the medial and lateral plantar nerves, the medial calcaneal nerve branches, and the first branch of the lateral plantar nerve (i.e., Baxter's nerve).

### Adhesive Neuritis

Adhesive neuritis occurs when scar tissue forms externally around the nerve or internally within the nerve. The goal of the initial tarsal tunnel release is to decompress the nerves associated with tarsal tunnel syndrome and free them from any impingement or scar tissue. However, any surgery necessarily results in some scar tissue formation postoperatively. The amount of scar tissue formation that occurs varies depending on a multitude of factors, including factors inherent to the patient, patient compliance, and postoperative complications (e.g., postoperative hematoma formation or infection). When the

scar tissue that forms limits the gliding of the nerve or damages its vascularity, recurrent pain can ensue. This adhesive neuritis may be external or internal to the nerve.

Adhesive neuritis should be strongly suspected if the patient had temporary relief of their symptoms following the initial release, only to experience a return or worsening of their symptom complex over the ensuing weeks or months. Adhesive neuritis is also more likely if the patient has a postoperative complication resulting in prolonged swelling, wound healing problems, or has hypertrophic or keloid scar formation.

Revisional surgery for recurrent tarsal tunnel syndrome due to adhesive neuritis involves a complete release of the nerves with the inclusion of techniques to prevent or minimize recurrent scar tissue formation. Many surgeons have advocated barrier techniques designed to insulate the posterior tibial nerve from recurrent scar tissue incarceration. One such method is silicone entubulation or ensheathment, which has been used for treating peripheral nerve entrapments (Figure 2).<sup>3,13-14</sup> This type of barrier technique however, has been found to cause a fibrous capsule around the nerve, and ultimately lead to re-entrapment. Novotny and associates<sup>12</sup> reported excellent results in two patients with a recurrent tarsal tunnel syndrome utilizing a radial forearm free-flap. They found this to be effective in limiting scar tissue formation. More recently the use of autogenous saphenous vein graft wrapping has been advocated as a barrier technique for recurrent nerve entrapments (Figure 3). Sotereanos et al<sup>15</sup> described the procedure for recurrent entrapment of the median nerve in the upper extremity secondary to scar formation. Under loupe magnification, the greater saphenous vein is wrapped around the nerve with its endothelial surface

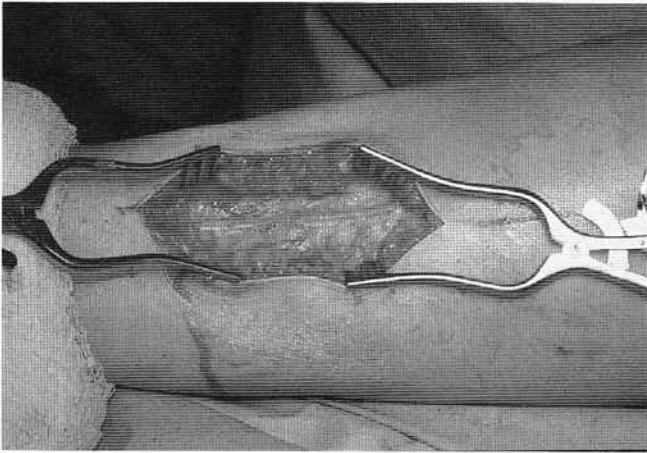


Figure 3A. Autogenous vein wrapping of the posterior tibial nerve with the greater saphenous vein in a patient undergoing revisional tarsal tunnel decompression. The greater saphenous vein is harvested. The vein length needs to be roughly 3x the length of the nerve area to be covered.

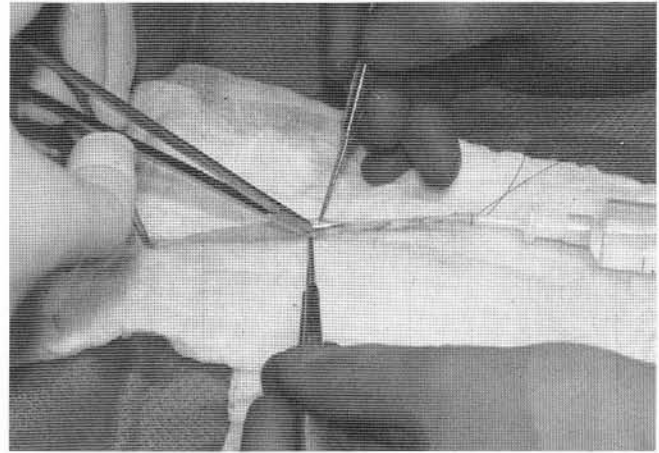


Figure 3B. The harvested vein is dilated with a syringe filled with plain lidocaine and the vein is split longitudinally.

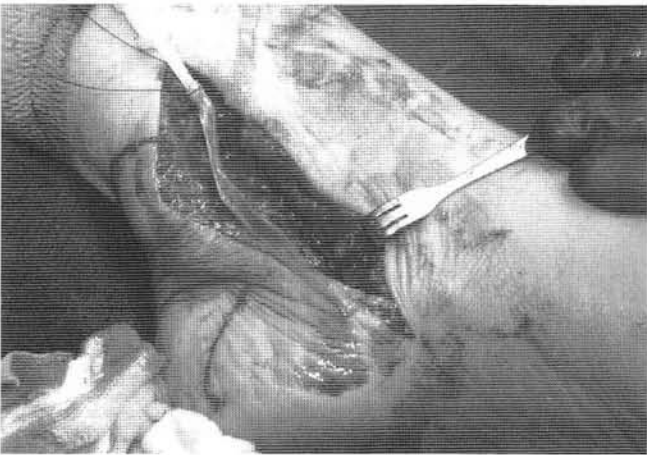


Figure 3C. The vein is then wrapped around the entire exposed portion of the posterior tibial nerve by either simply enclosing the nerve inside the vein (if the vein is large enough), or by coiling it around the nerve in "barber pole" fashion (if the vein is smaller). The intima of the vein is wrapped against the nerve.



Figure 3D. Under loupe magnification, the vein is then sutured to itself with 7-0 non-absorbable suture. The subcutaneous layer and skin are then closed.

against the nerve. Theoretically, the autogenous vein is believed to provide an external barrier against scarring of the nerve and the surrounding tissues allowing improvement in the vascular supply to the nerve. They also espoused that the vein acts as a gliding conduit for the nerve and that the vascular endothelium prevents internal scar formation. The use of this technique for recurrent tarsal tunnel syndrome has been examined in several clinical series.<sup>16-18</sup> Gould<sup>16</sup> reported wrapping a total of 65 nerves in the lower extremity, including the posterior tibial, superficial peroneal, common and deep peroneal, sural, and intermetatarsal. He reported 63% good or excellent results (i.e., no pain or occasional pain with exertion) and 37% fair to poor results. Seventy-five percent of Gould's patients were gratified with the results. The patients with the best outcomes were noted to have external adhesions and those with internal scarring did

the poorest.<sup>19</sup> Interestingly, Campbell et al<sup>17</sup> incorporated histological findings 17 months after surgery to validate theoretical mechanisms. Specifically, histologic evaluation demonstrated viable vein graft with adequate vascularity evidenced by patent adventitial lumens. No degeneration of the vein graft was noted. While internal scar within the nerve was not detected, no obvious gliding surface between the nerve and vein graft was identified, suggesting this to be a less likely mechanism. Easley and Schon<sup>18</sup> reviewed their series of vein wrapping procedures used for adhesive neuralgia in 25 patients. They used the saphenous vein in 19 cases and a fetal umbilical vein in 6 cases. Twenty-one of their 25 cases were entrapment neuropathies of the posterior tibial nerve. Seventeen of the 25 (68%) patients were satisfied with the procedure (with or without reservations), while 8 (32%) patients gained minimal or no relief of their symptoms. Their

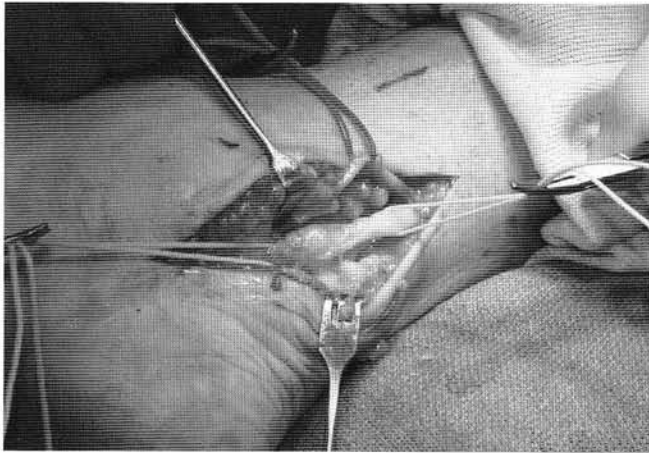


Figure 4A. Neurectomy of the posterior tibial nerve with implantation into distal tibia. Exposure of posterior tibial nerve with a non-repairable neuroma-in-continuity.

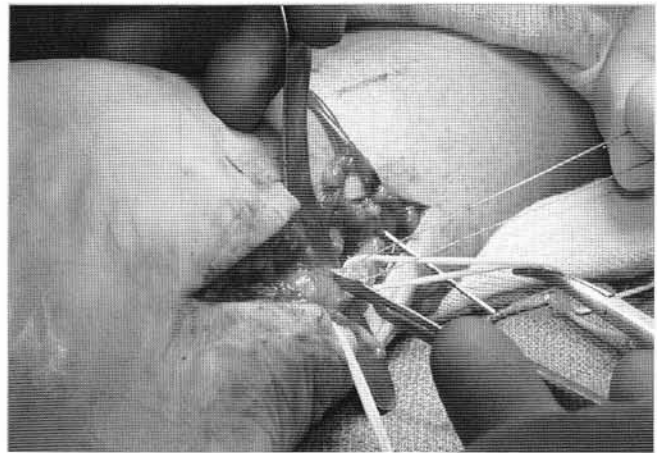


Figure 4B. Sharp sectioning of the posterior tibial nerve through a normal segment proximal to the neuroma-in-continuity.

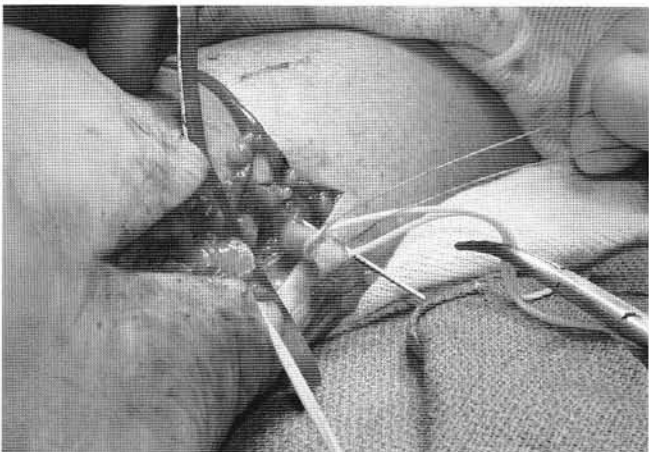


Figure 4C. Freshly sectioned end of the posterior tibial nerve. The diseased segment (i.e., neuroma-in-continuity) is also excised.

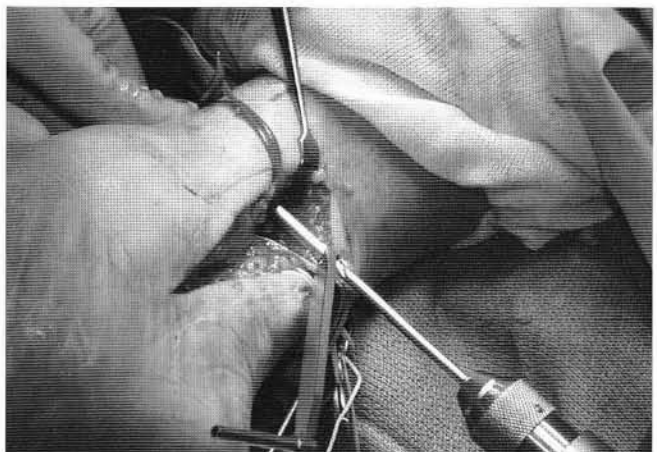


Figure 4D. Preparation of drill hole in tibia. Diameter of the hole will be larger than the diameter of the sectioned nerve.

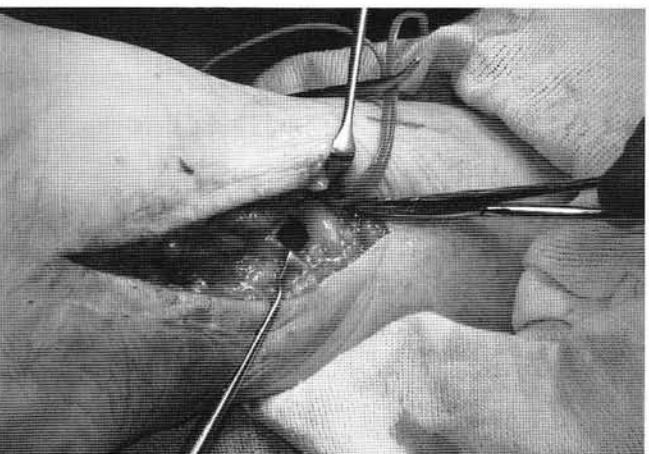


Figure 4E. Nerve is placed into the drill hole under minimal tension and a suture through the epineurium is used to maintain its position.

indications for vein wrapping were intractable neurogenic pain; failure of the nonoperative management protocol; temporary relief of symptoms after previous neurolysis with subsequent recurrence; and clinical findings consistent with adhesive neuralgia. More long-term studies are needed to accurately assess the benefits of this technique for recurrent tarsal tunnel syndrome.

#### Intraneural Damage

Recurrent tarsal tunnel syndrome associated with intraneural damage carries the worst prognosis. Intraneural damage results from axonotmesis (i.e., axonal or Wallerian degeneration) caused by a stretching or crushing injury, chronic external compression causing intraneural ischemia, or systemic disease. Intraneural

damage often results in a “neuroma-in continuity,” which is a mixture of intrafascicular fibrosis and poorly myelinated regenerating axons. Despite the continued gross continuity of the nerve itself, spontaneous recovery may or may not occur.

Patients with recurrent tarsal tunnel syndrome due to intraneural damage usually describe their pain as occurring spontaneously and often at rest or at night. Their pain can be termed spontaneous or ectopic neuralgia as it is not typically triggered by movement, position, or activity.

Since the recurrent tarsal tunnel syndrome in cases of intraneural damage is caused by internal nerve damage at the axonal level, external neurolysis is going to be of minimal benefit. If possible, the treatment should be oriented towards the cause of the intraneural damage. If a neuroma-in-continuity is identified, an attempt at internal neurolysis might be considered. However, the results from this approach have been less than promising. More likely the patient might benefit from insertion of a direct peripheral nerve stimulator, resection of the damaged section of the nerve with nerve grafting, or from resection of the damaged section of nerve with implantation of the remaining proximal nerve end into innervated skeletal muscle or bone (Figures 4). Insertion of a direct peripheral nerve stimulator involves the insertion of an electrode directly onto the involved peripheral nerve proximal to the area of the neuroma-in-continuity or nerve injury. Each of these approaches should be considered a “last resort” approach, and generally are associated with a poor to guarded prognosis.

### Double Crush Syndrome

Double crush syndrome involves the concept that a proximal site of nerve compression will render a peripheral nerve more susceptible to chronic nerve compression. Systemic diseases, such as diabetes mellitus, may also be considered a lesion or “crush” leading to a double crush syndrome. Double crush syndrome embodies the idea that each site of compression, in and by itself, might not be sufficient to produce symptoms, but that the two sites of compression or the “double crush” could summate to produce symptomatology. In this way, for example, a L5-S1 radiculopathy could cause a tarsal tunnel syndrome to become symptomatic much more easily.

A patient with recurrent tarsal tunnel syndrome secondary to a double crush syndrome will typically have no improvement or only partial improvement following his or her initial tarsal tunnel release.

If a double crush syndrome is present, then any other nerve lesions (proximal or distal) should also be addressed. Any spine pathology should be assessed and treated. If a systemic disease is thought to be contributing to the double crush syndrome, it should be medically managed as closely as possible.<sup>20</sup>

### Idiopathic or Other Causes

Other factors that have been associated with failure of tarsal tunnel release include idiopathic cases,<sup>21</sup> older patients,<sup>22</sup> chronic disease,<sup>23</sup> and postoperative trauma. Zahari and Ly<sup>24</sup> reported two cases of recurrence related to postoperative ankle injuries. They re-released the retinaculum and injected steroid to retard scar formation. In most cases of recurrent tarsal tunnel syndrome with these etiologies, further surgery is not likely to be of significant benefit.

## CONCLUSION

Recurrent tarsal tunnel syndrome is a challenging condition that can frustrate both the patient and the clinician. Without a doubt, failed tarsal tunnel surgeries are better prevented than treated. A thorough history and physical examination, appropriate diagnostic studies including MRI, and a complete initial decompression can decrease the likelihood of recurrent tarsal tunnel syndrome. When recurrent tarsal tunnel syndrome does occur, an appropriate re-evaluation and work up should be done. The prognosis for success of revisional tarsal tunnel surgery depends directly upon the etiology of the recurrent problem. If the cause of the recurrent tarsal tunnel syndrome is inadequate or incomplete release, and no intraneural damage is present, the prognosis is good. On the other hand, if extensive intraneural damage is present, the prognosis is poor. If external adhesive neuritis is the primary cause of the recurrent symptomatology, the prognosis may be satisfactory with a barrier technique approach. Future outcome studies should compare and contrast the treatment modalities currently available for the management of this perplexing condition.

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