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## PERIPHERAL NERVE ENTRAPMENT

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### INTRODUCTION

Peripheral nerve entrapment occurs when a nerve passes through a narrow anatomic tunnel or when a nerve becomes edematous. A compression injury to the nerve then occurs, which will lead to tingling and numbness in the distal distribution of the nerve. In severe cases, motor function can be lost. Permanent damage to the nerve will then occur if the entrapment is not decompressed in a timely fashion. There are numerous conservative approaches available, which include medications, such as gabapentin and pregabalin, vitamin supplements, topical creams, and electrical stimulation. Recently, more aggressive surgical decompressions have become mainstream and will be discussed below.

### ETIOLOGY

Peripheral neuropathy is a major burden for our patients and the health care system. It affects approximately 50% of the 25.8 million diabetic patients currently living in the US (1). Fifteen percent of these people will develop foot ulcers and 0.8% will require an amputation (1). The etiology of peripheral neuropathy is still often debated among the medical community but includes many possible causes including compact anatomic tunnels, trauma to specific areas, or systemic causes such as diabetes, hypothyroidism, arthritis, or many chemotherapy drugs (2-4). One known contributor is persistent hyperglycemia, which activates the polyol pathway. Sorbitol and fructose then increase in concentration within the nerve cell causing myoinositol uptake to decrease. In addition, the  $\text{Na}^+/\text{K}^+$  adenosine triphosphate pump is inhibited leading to an accumulation of sodium within the cell. Water then accumulates inside the cell causing edema within the nerve cell. Eventually, this fluid accumulation causes damage to the respective nerve that will lead to multiple symptoms including pain, paresthesias, burning, tingling, numbness, and diminished motor function (5).

A positive Tinel's sign over the suspected site of entrapment is essential to a diagnosis of peripheral nerve entrapment. Nerve conduction velocity and electromyographic studies are often inconclusive, but they can still

provide some information as to whether or not surgery is necessary (5). A pressure-specified sensory device will help detect early decrease in sensation. A Semmes-Weinstein monofilament is often not sensitive enough to detect early decreases in sensation (3). A magnetic resonance image (MRI) may show muscle atrophy in late stage nerve entrapment. The physician must look at the entire diagnostic picture, but must remember that the clinical presentation is the most important element needed for making a diagnosis of peripheral nerve entrapment.

In 1973, Upton and McComas presented the double crush phenomenon. They hypothesized that metabolic or mechanical trauma of a nerve proximally would make the nerve more susceptible to compression at more distal sites (2). Dellon and Mackinnon confirmed this in 1991 (2). In this study, the sciatic nerve of rats was banded with silicone tubes causing compression of the nerve. They found that if there were two separate sites of compression they would cause more neural damage than one site of compression. The double crush phenomenon holds true even if each point of compression is subclinical. This means that a person may have minimal compression proximally even if they are not experiencing pain in this area (2). Symptomatic nerve entrapment will occur more frequently due to this fact.

The extremities are natural sites for nerve entrapment. In both the upper and lower extremities, there are known anatomic sites where nerves must pass through compact tunnels or make acute turns. These anatomic sites may be sources of compression. In diabetics, even if the tunnel has not decreased in circumference, the edematous nerve may become compressed as it grows in size and passes through the inelastic tunnel. Similarly, trauma to the lower extremity, such as inversion ankle sprains, can cause tensing of the more proximal muscles causing increased traction on the more proximal nerves. Oppenheimer first described a classic example in 1911 using an inversion ankle sprain. This caused tensing of the peroneus longus muscle at the fibular neck, causing traction on the common peroneal nerve (4).

Early diagnosis of peripheral nerve entrapment is essential. After 2 months of compression, there is edema within the nerve. Within 6 months of entrapment, the myelin covering the nerve is damaged and by the end of 1 year, the

nerve will begin to die (3). As nerve tissue dies, scar tissue forms, leading to a cascade of further entrapment (3). The more quickly a nerve is decompressed, the more likely complete resolution of symptoms may be achieved (3).

Not all patients with traumatic injuries develop peripheral nerve entrapments, just as not all patients with diabetic peripheral neuropathy have peripheral nerve entrapments. The appropriate surgical candidate must have all of the following (6):

1. Clinical symptoms of paresthesias, pain, or numbness in the distribution of the suspected nerve;
2. Objective findings of decreased sensation;
3. Positive Tinel's sign over the site of the entrapped nerve;
4. Failure to resolve symptoms with nonsteroidal anti-inflammatories, opioids, neuropathic medications, and/or treatment of underlying systemic medical condition;
5. Adequate circulation;
6. Minimal lower extremity edema;
7. General medical condition acceptable for surgery;
8. Ability to comply with instructions;
9. Weight <140kg;
10. No history of failed back or previous nerve decompression surgeries.

## SURGICAL PROCEDURE

The common peroneal nerve (CPN) is palpated and marked at the fibula neck. The knee is then flexed and a 3-4 cm oblique incision is made over the area. The deep fascia is then carefully released, taking care not to cut any branches of the CPN. The nerve is then identified and carefully retracted so that any remaining restricting fascial bands may be released. Often, a small portion of the deep fascia is removed to prevent recurrence (6) (Figure 1).

The incision for the superficial peroneal nerve (SPN) is made approximately 10-12 cm proximal to the lateral malleolus and it is made in a vertical fashion. The nerve may be in the anterior or lateral compartment. Once the nerve is identified, the overlying fascia must be released for approximately 15 cm in a longitudinal fashion, continuing proximal and distal from the original skin incision site, in order to adequately release the nerve (6) (Figure 2).

A linear incision is then made at the junction of the first and second metatarsal bases and the medial cuneiform in order to identify the deep peroneal nerve (DPN). This nerve is often entrapped against the underlying bones by the



Figure 1. A small portion of the deep fascia is removed to free the CPN.



Figure 2. Once the SPN is identified, the overlying fascia must be released.

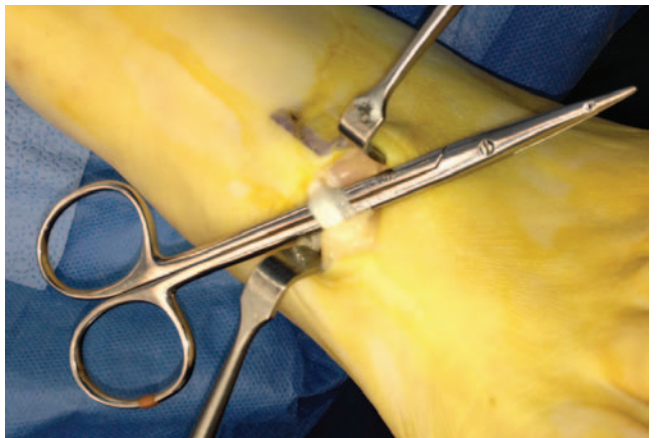


Figure 3. The EHB is sectioned to release the DPN.

extensor hallucis brevis tendon. This tendon is carefully identified (taking care to protect the deep peroneal artery) and a 1 cm portion is then removed. The DPN is then freed from any remaining entrapments in the area. In all 3 areas, the subcutaneous layer and skin are closed, but the underlying fascial areas are left open (6) (Figure 3).

Postoperatively the patient will be in a bulky compressive dressing made up of cast padding and ace bandages for approximately 3 weeks. The patient can bear weight during this time period and range of motion or nerve gliding exercises should be recommended.

## DISCUSSION

Over the past two decades, there has been much research on the efficacy of peripheral nerve decompressions for patients with peripheral nerve entrapments. In 1992, Dellon performed a landmark study on 60 diabetic patients with a positive Tinel's sign. After a follow-up of 2.5 years, 88% had improved sensation and positive subjective results, 10% were unimproved, and 2% were worse. Interestingly, in patients with bilateral nerve entrapments, only one leg was surgically decompressed. Of the limbs that were untouched, 50% became worse over the same follow-up period (7). This made a compelling argument that peripheral nerve decompressions can improve symptoms that may become worse if otherwise left untreated.

In 2000, Aszmann et al also had interesting results on 20 diabetic patients with a positive Tinel's sign over the posterior tibial nerve. Of the patients that had tibial nerve decompression, 79% demonstrated improvement, 21% were unchanged, and none became worse. The contralateral nerves that were not treated during the same time period demonstrated 9% improvement, 59% had no change, and 32% became worse (8).

Valdivia et al in 2005, did a prospective study of 100 patients, 40 of whom were non-diabetic. Valdivia and colleagues performed surgical decompression of the common peroneal, deep peroneal, and posterior tibial nerves in each patient. Improved sensation was reported by 87%, 92% had improved balance, and 78% stopped or decreased their pain medications (9). Valdivia discussed the importance of treatment protocols that will decrease the amount of pain medications that are needed by the patient (9).

In 2006, Siemionow et al performed a variety of surgical nerve decompressions on 32 patients, 20 of whom were nondiabetic. These decompressions included common

peroneal, deep peroneal, and posterior tibial nerve decompressions as necessary. Excellent or good results were reported by 100% of the diabetic patients, and 84% of nondiabetic patients had excellent or good results (10). While results were good in both diabetic and nondiabetic patients, this study led to a discussion about why the results may be better in diabetic patients. Further research is needed to determine the reason.

In 2007, Humphreys et al surgically decompressed the CPN on 48 patient. Humphreys and colleagues reported 49% improved sensation and 84% improvement in pain in this patient set (4). The majority of these patients had severe injury to the nerve that was evidenced by preoperative loss of motor function (4). The severity of these entrapments may explain the lower rates of improved sensation postoperatively.

A meta-analysis by Dellon et al in 2012, included 22 papers published over the past 2 decades, and showed an overall improvement of 80% in sensation and function after a tarsal tunnel release in patients with nerve compression in this area (6). This meta-analysis provides compelling support for surgical decompression for peripheral nerve entrapments.

Similarly, in 2012, Dellon et al also performed a multi-center prospective study showing that diabetic patients with ulcerations and peripheral neuropathy that underwent tibial nerve decompression had a 3.8% rate of recurrent ulcerations. This is a significant improvement versus diabetic patients with no treatment, which would likely result in a recurrent ulceration rate of 50-60%. Intensive primary foot care alone may reduce the recurrent ulceration rate to only 25-30% (11). Reducing the recurrent ulceration rate demonstrates how advantageous peripheral nerve decompressions may be in the appropriate patient to improve quality of life and reduce health care costs.

## CONCLUSION

As our knowledge of nerve anatomy and metabolism improves, the treatment options for neuropathic pain continue to improve exponentially. In addition to diabetic patients, there are many patients who suffer from peripheral nerve entrapments following chemotherapy or traumatic injuries. Larger randomized controlled prospective studies are needed to further our understanding of peripheral nerve decompression. For now, we confidently say that it is a viable treatment option to consider.

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## REFERENCES

1. 2011 National Diabetes Fact Sheet URL: [www.diabetes.org](http://www.diabetes.org).
2. Siemionow L, et al. Effect of early nerve release on the progression of neuropathy in diabetic rats. *Ann Plastic Surg* 2007;59:2.
3. Dellon AL. Pain solutions. URL: [www.dellon.org](http://www.dellon.org).
4. Humphreys, et al. Patient outcome after common peroneal nerve decompression. *J Neurosurg* 2007;107:7.
5. Rosson G, et al. Tibial nerve decompression in patients with tarsal tunnel syndrome: pressure in the tarsal, medial plantar, and lateral plantar tunnels. *Plastic Recon Surg* 2009;124:4.
6. Dellon AL. The Dellon approach to neurolysis in the neuropathy patient with chronic nerve compression. *Handchir Mikrochir Plast Chir* 2008;40:2.
7. Dellon AL. Treatment of symptoms of diabetic neuropathy by peripheral nerve decompression. *Plast Reconstr Surg* 1992;89:3.
8. Aszmann O, et al. Results of decompression of peripheral nerves in diabetics: a prospective, blinded study. *Plastic Recon Surg* 2000;106:4.
9. Valdivia J, et al. Surgical treatment of peripheral neuropathy: outcomes from 100 consecutive decompressions. *J Am Podiatry Med Assoc* 2005;95:5.
10. Siemiono W, et al. Clinical outcome of peripheral nerve decompression in diabetic and nondiabetic peripheral neuropathy. *Ann Plastic Surg* 2006;57:4.
11. Dellon AL, et al. Prevention of ulceration, amputation, and reduction of hospitalization: outcomes of a prospective multicenter trial of tibial neurolysis in patients with diabetic neuropathy. *J Recon Microsurg* 2012;28:4.