Nerve decompression in diabetics with chronic nerve compression: update 2022

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Abstract
The number of people with diabetes worldwide has reached epidemic proportions. Diabetics are well-known to have chronic nerve compressions, and the prevalence of compressions exceeds 50% in those with neuropathy. The loss of sensation in the feet of people with diabetic neuropathy is the primary cause of their ulceration and amputation, as well as pain. The aim of this article is to update the reader on the current status of lower extremity nerve decompression in patients with diabetic neuropathy. A review of the history and literature related to the current approach to the patient with chronic nerve compression plus diabetic neuropathy was undertaken. The current evidence is overwhelmingly clear, in diabetics with neuropathy and a positive Tinel sign over the tibial nerve at the tarsal tunnel, that decompression, by neurolysis of lower extremity nerves, can relieve pain, restore sensation, and prevent ulceration and amputation. Furthermore, economic cost-benefit analysis by the Markov technique demonstrates that lower extremity nerve compression is not only cost-effective compared to standard medical care, but also increases the quality of life and life expectancy. The remaining barriers to acceptance and implementation of this proven surgical approach must lie in the education of physicians in training and re-education of diabetes educators, primary care providers and endocrinologists.

Keywords: Diabetic neuropathy, neurolysis, decompression, Markov analysis, amputation

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INTRODUCTION

Since about 50% of people with diabetes develop neuropathy\(^1,2\), the worldwide explosion of diabetes\(^3\) means that there will be an explosion of patients with diabetic neuropathy. About 50% of patients with diabetic neuropathy have multiple chronic lower extremity nerve compressions [Figures 1 and 2]\(^4,5\). In 1982, I began doing decompression of lower extremity nerves in diabetics and the results of this work were reported in 1992\(^6\), demonstrating that 80% of patients with a positive Tinel sign of the tibial nerve in the tarsal tunnel had improved sensation, and decreased pain. Subsequent studies demonstrated that there was significantly less (\(P < 0.001\)) ulceration and amputation for a patient that had nerve decompression than the “control” that did not have that surgery\(^7\). In 2012, a multicenter prospective study of 38 surgeons, including 800 patients with a 3-year follow-up, demonstrated that 80% of patients had a significant (\(P < 0.001\)) relief of pain by six months after surgery, sensation recovered in the feet, 0.3% vs. the expected 15% of patients developed ulceration, 0.2% vs. the expected 15% had an amputation, and 0.6% vs. the expected 3.7% had a hospitalization for foot infection\(^8\).

There have been four independent basic science studies that demonstrate that rats with streptozotocin-induced diabetes will not develop a neuropathic walking track pattern\(^9,10\), and will have improved lower extremity motor function\(^11\) and improved perception of pain\(^12\). These studies confirm experimentally the hypothesis I proposed in 1988\(^13,14\), that the double crush concept did apply to diabetes clinically: the underlying pathophysiology of diabetes acts as the first crush, or site of compression, making the nerve susceptible to more distal sites of compression.

Subsequently, 19 Level IV studies\(^6,8,15-29\), two systematic reviews\(^30,31\), and three Level I studies\(^32-34\) have all concluded that decompression of lower extremity nerves in diabetics relieves pain, improves sensation, and prevents ulcers and amputations. There have been two economic cost-benefit analyses using the decision-tree approach that concluded that the cost of surgical decompression is less than the cost of standard medical care for the treatment of diabetic neuropathy\(^25,26\). The two most recent approaches, using the highest form of analysis, Markov analysis, not only confirmed that the cost of surgical decompression is less than the cost of standard medical care for the treatment of diabetic neuropathy, but also proved that surgical decompression of lower extremity nerves improves the quality of life and even prolongs life\(^37,38\).

The results of the Markov cost-effectiveness studies are worth further comment. For example, from the study from the Johns Hopkins School of Hygiene and Public Health\(^37\): when compared to standard medical “prevention” (treatment), for a patient population of 10,000, surgical decompression of lower extremity nerves with the Dellon Approach prevented a total of 1447 ulcers and 409 amputations over a period of 5 years. The quality-adjusted incremental effectiveness (QALY) was 0.41. A QALY of 1.0 means a perfectly healthy life without disability for 1 year. To put this increase of 0.41 into perspective, the QALY for a person with diabetic neuropathy is 0.40, so that this surgery doubled the quality of life for the person who has undergone surgery. In relation to survival, given the difference in death rates between the two prevention strategies, medical vs. surgical, survival was 73% for those receiving medical prevention compared to 95% for those undergoing surgery. The surgery-treated group lived longer because they had fewer ulcers, fewer hospital admissions for infection, and fewer amputations.

THE DELLON APPROACH TO NERVE DECOMPRESSION IN THE DIABETIC

Making the diagnosis of neuropathy

What is the approach to the person who has any symptom of numbness, tingling, pain, or any combination of these in the feet? If the symptoms are bilateral and include the dorsum and the plantar aspect of the foot, we have little doubt that the diagnosis is neuropathy. As a Peripheral Nerve Surgeon, the person you are
Figure 1. Relationship between severity of neuropathy, determined by the Michigan neuropathy screening instrument and the number of Tinel signs in a population of diabetics. The worse the neuropathy clinically, the more sites of compression in the given patient, with a correlation coefficient of 0.94 and a value of significance of $P < 0.001$\cite{4}.

Figure 2. Relationship in the upper and the lower extremity between the number of nerve compressions in diabetics with and without neuropathy for a given site of nerve entrapment\cite{4}.
seeing may not have been evaluated appropriately for the presence of a medically treatable etiology of neuropathy. Before assuming the patient has diabetic neuropathy, it is critical to do a two-hour oral glucose tolerance test (OGTT). All too often, a patient will see me who has been told they have “idiopathic neuropathy”, meaning the usual tests for serum folate/B12 levels, autoimmune disease, heavy metal toxicity, hypothyroid, HbA1c and serum glucose are normal, yet they have not had this OGTT, a stress test. Up to 56% of idiopathic neuropathy patients will have an abnormal OGTT, meaning that they have impaired glucose tolerance\(^\text{[39]}\). Whether their medical doctor will or will not tell them they have diabetes, their peripheral nerves are subjected to a hyperosmolar condition related to excess serum glucose, causing the nerve to swell. This swelling, in the presence of a known site of anatomic narrowing, like the tarsal tunnel region or the fibular tunnel, is the usual cause of chronic compression.

Of course, the history is important too in making the diagnosis. For example, chemotherapy-induced neuropathy, today usually due to treatment with a “platin” or “taxol” drug that binds to tubulin in the axoplasm of the nerve, can cause neuropathy. The results of lower extremity nerve decompression for the treatment of chemotherapy-induced neuropathy, neuropathy that persists for more than six months after the cessation of chemotherapy, can be as successful as for the treatment of diabetic neuropathy. This has been proven experimentally\(^\text{[40]}\) and clinically\(^\text{[41]}\). Indeed, the Dellon Approach is as successful in the treatment of idiopathic neuropathy as it is in treating diabetic neuropathy\(^\text{[27,42]}\). Furthermore, once the patient with Leprosy has received triple antibiotic therapy and is no longer infectious, but has a disability related to the immune-mediated swelling along the course of superficial peripheral nerves, like branches of the peroneal nerve at the knee (fibular tunnel), leg (superficial peroneal nerve) or foot dorsum (deep peroneal nerve), then neurolysis of these lower extremity nerves can also be successful\(^\text{[42,43]}\).

Electrodiagnostic testing will most likely have been done prior to the patient ever seeing the surgeon, and is quite successful at identifying a symmetrical neuropathy, especially if there is decreased number of axons present. However, electrodiagnostic testing, even in the upper extremity, where it is most sensitive, is often unable to identify a chronic nerve compression in a person who does not have neuropathy and is very unlikely to identify a superimposed chronic nerve compression in the presence of an underlying neuropathy due to the inherent nature of the neuropathy upon the axons\(^\text{[44-46]}\). Similar reasoning applies to the lower extremity, where 50% of asymptomatic people over the age of 55 have no medial plantar response and have motor fasciculations\(^\text{[47-49]}\).

**MAKING THE DIAGNOSIS OF CHRONIC NERVE COMPRESSION**

If the electrodiagnostic testing has demonstrated a superimposed chronic nerve compression at a known site of anatomic narrowing, then the diagnosis has been confirmed objectively. Jules Tinel, MD, a Neurologist in France, and Paul Hoffman, PhD, a Physiologist in Germany, both described, independently, in 1915, the tingling sign that results from a peripheral nerve regenerating distally\(^\text{[50]}\). Beginning with Phalen’s description of carpal tunnel syndrome in 1966, the Hoffman-Tinel sign (hereinafter referred to as the Tinel sign) has been accepted as identifying the site of a nerve compression along the course of a peripheral nerve\(^\text{[51]}\). In my experience, this sign is sufficient if the site of compression is close to the target skin territory, like the dorsum of big toe/1st web space when the deep peroneal nerve is lightly tapped at the junction of the first and second metatarsal and the cuneiform bone, or the tibial nerve in the tarsal tunnel. However, for the common peroneal nerve at the fibular neck, there is often NOT a distally radiating perception, but just tenderness of the nerve or proximal radiation of the tingling. This sign, described by Francoise Louise Isidore Valleix, MD, in 1841\(^\text{[52]}\), has been proven to be valid in my clinical experience at localizing a site of nerve compression for the common peroneal nerve at the fibular neck.
The reason there has been reported so much lack of sensitivity and specificity of the Tinel sign is that the presence of a positive Tinel sign varies according to the degree of compression, that is, the underlying pathophysiology present at the time of testing. Early in nerve compression, there is just endoneurial edema, and there may be no symptoms or just paresthesias, but once demyelination begins, the Tinel sign appears, and once an advanced axonal loss occurs, the Tinel sign can disappear\cite{53-55}. The presence of a positive Tinel sign can also vary depending upon how hard the nerve is palpated and the experience of the examiner\cite{56}.

Therefore, over the past more than 40 years, if I can elicit a Tinel sign at a known site of anatomic narrowing, I am confident that there is a chronic nerve compression present. In the lower extremity, the presence of a positive Tinel sign over the tibial nerve in the tarsal tunnel has proven to have at least an 80% positive predictive value for good to excellent results from lower extremity peripheral nerve compression from the now “classic” Dellon Triple nerve decompression (neurolysis of the common peroneal nerve at the knee, the deep peroneal nerve at the foot dorsum, the release of the tarsal tunnel with its included neurolysis of the medial, lateral and calcaneal nerves)\cite{41,57,58}.

**THE IDEAL SURGICAL CANDIDATE**

An ideal patient is a person with diabetes who has symptoms of numbness and tingling with or without pain. The person who has just pain, without numbness, may not be an ideal candidate as that patient might have small fiber neuropathy. Small fiber neuropathy is rare but can be identified by a skin biopsy that quantitates unmyelinated nerve fibers in the dermis of the leg and thigh in the presence of normal tests for large fiber function. The skin biopsy will likely be positive in patients with the commonest form of diabetic neuropathy (a mixed large and small fiber neuropathy), and also in patients with an advanced form of chronic nerve compression without diabetes. In chronic nerve compression, both large, myelinated fibers and small, unmyelinated fibers can undergo degeneration\cite{59}. What must be present in the ideal candidate for surgery are large fiber symptoms, such as tingling and numbness, and some measurement of large fiber function that is abnormal, such as vibratory perception threshold (quantitative vibrometry), or touch perception threshold (Pressure-Specified Sensory Device™ or Semmes-Weinstein monofilaments), or abnormal static or moving two-point discrimination\cite{60-63}. An abnormal electrodiagnostic test that shows nerve compression is also a form of large fiber evaluation.

The ideal candidate for decompression should have had an extensive medical trial of neuropathic pain medications, such as Neurontin, Gabapentin, Duloxetine, Elavil, or a combination of these drugs, and found that they had too many side effects or else were ineffective.

The ideal candidate for decompression, in addition to having the correct diagnosis, must have good circulation in the lower extremities. This can be determined by palpating the pulse, and if this examination is not clearly in favor of good arterial inflow, then Doppler flow studies, ankle/brachial index, and, if needed, cutaneous oxygen levels can be ascertained. The surgical incisions clearly may not heal in the absence of sufficient circulation. The same can be said for pedal edema. In the presence of cardiac or renal problems, edema is present, which is a contra-indication to surgery.

In addition to the positive Tinel sign of the tibial nerve, as discussed above, a good prognosis can be expected if the patient has had previously successful carpal tunnel surgery\cite{64}. From a database of 300 patients who had a Dellon Approach to their lower extremity nerve compressions, 35 patients were found who had previous carpal tunnel release. The presence of a good response to median nerve decompression at the wrist gave an 88% positive predictive value to response to the decompression of the tibial and peroneal nerves.
SURGICAL DECOMPRESSION

The main concept is that the stocking distribution of symptoms present in diabetic neuropathy can be understood as compression of the peroneal and tibial nerves. The original approach I took to surgical decompression required understanding that the tarsal tunnel is NOT the carpal tunnel anatomically [Figure 3]^[65], but is the forearm, with the carpal tunnel being the medial plantar tunnel, the Guyon’s canal being the lateral plantar tunnel and the compression of the palmar cutaneous branch of the median nerve being homologous with compression of the medial calcaneal nerve^[66]. Therefore instead of simply decreasing the tarsal tunnel, the operative treatment must be conceived of as releasing the tarsal tunnel and a neurolysis of the medial and lateral plantar and calcaneal nerves; four medial ankle tunnels, or *tarsal tunnel syndrome^[67]. To perform this surgery successfully, the septum between the medial and lateral plantar tunnels must be removed [Figure 4], as has been demonstrated by measuring compartment pressures in both cadavers^[68] and patients having this surgical decompression^[69].

The anatomical basis of these techniques has been described for the deep peroneal over the dorsum of the foot [Figure 5]^[70], the common peroneal nerve at the fibular neck^[71], and variations of the superficial peroneal nerve in the leg [Figure 6]^[72]. The actual surgical techniques for the four medial ankle tunnels are illustrated in Figure 4 and have been described in detail in the past^[73-75]. Videos of the surgery can be viewed on YouTube.com for the tarsal tunnel decompress (https://www.youtube.com/watch?v=coBfi9NDjUM&t=36s) and the peroneal nerve decompressions (https://www.youtube.com/watch?v=Qlte57IHBzE&t=37s).

When should you consider neurolysis of the superficial peroneal nerve? The superficial peroneal nerve most always exits from the fascia of the lateral compartment about 6 to 8 cm proximal to the lateral malleolus. Sometimes there is a small bulge marking the spot. If there is a positive Tinel sign at that site, then neurolysis of this nerve should be added to the classic Dellon Triple. This can easily be added to the operation without additional post-op rehabilitation or morbidity but remember to use the bipolar cautery along the edge of the fascia before dividing the fascia to minimize post-operative bruising.

For the peroneal nerve, the multiple crush concept extends the double crush concept as follows: The first source of reduced axoplasmic flow is diabetes itself, with compression of the common peroneal nerve being the second crush, the superficial peroneal nerve being the third crush site, and the deep peroneal nerve being the fourth site. The first site cannot be changed. Theoretically, the vertebral foramen and the sciatic notch could be added, but as a matter of practicality, these sites are not easily decompressed, so they are not included. What can be approached safely are the common, superficial, and deep peroneal nerves. Although it is not clear experimentally that all need to be decompressed, if there has been a Tinel sign present, then that site is included in the surgical approach. Also, remember to open the anterior compartment, as 25% of people will have a branch of the superficial peroneal nerve located in this compartment [Figure 6]^[76].

For the tibial nerve, a site proximal to the tarsal tunnel had not been identified until 2009^[76,77]. This clinical problem was raised not in the setting of neuropathy but in the setting of “failed tarsal tunnel decompression”. It is well documented in upper extremity surgery that a “failed” carpal tunnel release can be due to proximal compression beneath the pronator teres deep head. A similar site in the lower extremity would be the fibrous arcade of the soleal sling, which gives origin to the soleus muscle. This site is at the soleal sling [Figure 6] and is identified clinically by two physical exam findings: weakness of the flexor hallucis longus and tenderness in the calf about 8 cm below the knee joint, a site similar to that of the Homan’s sign for deep vein thrombosis. Sometimes a 3T MRI will show changes in the tibial nerve at this location, but it is unusual for the electrodiagnostic testing to be abnormal. The surgical technique has been
Figure 3. Illustration of a cross-section through the tarsal tunnels to compare to the carpal tunnel. Note that the flexor/abductor muscles in the foot correspond to the thenar muscles in hand, and cover the roof of the medial plantar nerve, homologous to the median nerve, and the lateral plantar nerve, homologous to the lateral plantar nerve. The septum between medial and lateral plantar tunnels is homologous to the hook process of the hamate carpal bone.

described as well as the clinical results [78-80]. While the surgical exposure for this neurolysis is extensive and risks injury to the popliteal vessels, the post-operative course is easier than for tarsal tunnel decompression (see below). This raises the question of if a patient has both tarsal tunnel and soleal sling sites of compression, should both sites be operated upon at the same time. My current approach is to consider that tarsal tunnel release will not recover toe flexion, but will require the use of a walker for three weeks, and that it is conceivable that the proximal neurolysis will restore sensation to the plantar aspect obviating the need to do the tarsal tunnel decompressions (TTD). If the plantar sensation is not recovered, TTD can be done three months later.

REHABILITATION AFTER DELLON DECOMPRESSION

The first reported tarsal tunnel decompressions were case reports by Lam and Keck in 1962 [80,81]. They recommended the use of crutches and non-weight bearing for three weeks post-operatively. We know today that if a peripheral nerve is immobilized after neurolysis, it is likely to become adherent during wound healing. Therefore, my post-op regimen requires the use of a bulky “Robert Jones” type dressing [82] and immediate walking using a walker [Figure 7]. The patient is instructed to “march”, lifting the operated foot from the knee, thereby avoiding much ankle flexion, and preventing sutures from pulling out of what is often skin with poor sensibility. Steps can be climbed sideways. The dressing is removed on post-op day #7 and betadine is applied twice a day thereafter. Sutures at the knee and leg are removed on post-op day #14, and at the ankle on post-op day #21. Water walking is begun on post-op day #22, three to four times per week, 15 to 20 minutes per time, and increased thereafter as tolerated.

BARRIERS TO ACCEPTANCE & IMPLEMENTATION OF THIS SURGICAL APPROACH

Lack of awareness of the Dellon Approach to decompress peripheral nerves in diabetics with chronic nerve compression is probably the biggest obstacle to implementation today. The study by Melenhorst et al., from Zwolle in the Netherlands, best illustrates this problem [31]. In that study, they asked physicians and diabetes nurse educators the following questions:
Figure 4. Illustration of decompression of the tarsal tunnels. The tarsal tunnel itself is opened to identify the anatomy leading to the more distal tunnels, and anomalous anatomy. The muscles are retracted to reveal the roof of the medial and roof of the lateral plantar tunnel. These roof structures are released distally and the septum between the tunnels removed. Finally, the calcaneal tunnel is released.

1. “Are you aware of the theory that nerve compression injury plays a part in the pathophysiology of diabetic neuropathy?”

2. “Do you think that nerve compression injury may play a role in the development of diabetic neuropathy?”

3. “Are you aware of the potential value of nerve decompression surgery in the treatment of peripheral diabetic neuropathy?”

4. “Do you refer to other medical professionals when considering diabetic neuropathy?”

5. “Do you explain to the patient that peripheral diabetic neuropathy is an irreversible condition?”
Figure 5. The deep peroneal nerve has been released over the dorsum of the foot by resecting the extensor digitorum brevis to the hallux. This tendon compresses the underlying nerve against the cuneiform bone.

Table 1 contains the results of their study. On average, just 35% of health care practitioners were aware of this approach and just 9% appreciated its value. Only 3% of these health care providers referred patients to a surgeon for the surgical approach. This study was done in the Netherlands, where the first randomized control study was reported using the Dellon Approach.

The questions to be addressed today are “How can we best educate those health care professionals throughout the world to learn that (1) diabetics have an increased prevalence of chronic nerve compressions, (2) how to diagnose a chronic nerve compression, and (3) how to train surgeons interested in doing these procedures? I am hopeful that through a program aimed at providing this information to medical students early in their educational curriculum, an approach described elsewhere in an article in this issue of this journal, the future may hold optimism for those suffering from diabetic neuropathy[83].

CONCLUSION
The current evidence is overwhelmingly clear, in diabetics with neuropathy and a positive Tinel sign over the tibial nerve at the tarsal tunnel, that decompression, by neurolysis of lower extremity nerves, can relieve pain, restore sensation, and prevent ulceration and amputation. Furthermore, lower extremity nerve
Table 1. Includes data from the study performed by Melenhorst et al. evaluating the awareness of the general medical community of the surgical approach to peripheral neuropathy.[31]

<table>
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<th></th>
<th>Aware of theory</th>
<th>Aware of surgery value</th>
<th>Explains neuropathy is reversible</th>
<th>Refers to surgeon</th>
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<td>General practitioner</td>
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<td>23%</td>
<td>9%</td>
<td>35%</td>
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Modified from Melenhorst et al.[31]. NA: Not applicable.

Figure 6. The superficial peroneal nerve usually exits the fascia of the lateral compartment, where it becomes entrapped. In about 25% of patients, there is a high division of this nerve such that it has a branch in both the lateral and anterior compartment. Therefore, in every patient, we teach to open both compartments. An example of such a patient is shown here after decompression.

Compression in diabetics with neuropathy and superimposed chronic nerve compression is not only cost-effective compared to standard medical care, but also increases the quality of life and life expectancy.
Figure 7. In order to facilitate the gliding of the decompressed nerves, immediate mobilization is permitted by using a bulky Robert Jones type dressing, and a walker. In this figure, the patient is seen walking to the toilet prior to going home via wheelchair. The dressing limits ankle range of motion to less than 30 degrees, reducing the chance of the sutures tearing through the skin that has very little sensation. This dressing is used for the first week. The walker is used for three weeks until the sutures are removed at the ankle.

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The author contributed solely to the article.

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Ethical approval and consent to participate
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