Surgical approach to lower extremity nerve decompression in the patient with diabetic neuropathy

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Surgical approach to lower extremity nerve decompression in the patient with diabetic neuropathy

Chirurgische benadering van decompressie van perifere zenuwen in de onderste extremiteit bij de patiënt met diabetische neuropathie

(met een samenvatting in het Nederlands)

Proefschrift

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door

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Chapter 1

Introduction

DEFINITION

Diabetic polyneuropathy, in its commonest form is a bilateral, symmetrical, diffuse, sensorimotor, mixed (both large and small fiber) neuropathy that affects the lower extremity more than it does the upper extremity.[1-5] For the purposes of this manuscript, it will be called “diabetic neuropathy”, recognizing however, that many different types of neuropathy exist within the disease entity of diabetes mellitus. (see **Table 1** for abbreviated classification.)

Table 1
Types of neuropathy within diabetes mellitus

Focal	
Mononeuritis	
Compressive	
	Upper Extremity: Carpal and Cubital Tunnel Syndrome
	Lower Extremity: Fibular and Tarsal Tunnel Syndrome
Autonomic	
	Gastroporesis
	Cardiac
Vascular	
	Cranial nerve: VI palsy, III palsy
	Amotropy
Mononeuritis Multiplex	
Diffuse	
Large or Mixed Fiber	
Small Fiber	

MAGNITUDE OF THE PROBLEM

Diabetes mellitus is on the rise in epidemic proportions.[6,7] It is estimated that by the year 2030 there will be 300 million people in the world with diabetes. In the United States of America in the year 2005, it was estimated that there was a population of 300 million people. Of these 6% of Caucasians, 10% of African Americans, 15% of Hispanics, and 50% of Native Americans have diabetes. It is estimated that 6% of Europeans, 12% of Asians, 22% of Caribbeans, and 50% of those in the United Arab Emirates. Of those with diabetes, 8% will have diabetic neuropathy at the time of diagnosis, and 50% will have diabetic neuropathy by the time they have had diabetes for more than 25 years.[8] The prevalence of diabetic neuropathy may in fact be higher, since the studies on which these estimates were made used screening instruments of low sensitivity.

In practical terms, today, in the United States, there are likely to be 9 million people with diabetic neuropathy. Among this group there are 80,000 amputations per year. These numbers can be extrapolated world wide.[9,10]

The present state of medical knowledge offers neither prevention of diabetic neuropathy nor cure of diabetic neuropathy.[4,5,11,12] The present medical approach must deal with the treatment of foot infections, ulcerations, amputations, and pain.[13-17]

The natural history of diabetic neuropathy is that 15% of patients will develop an ulceration. Of these, 15% will have an amputation. Of those with an amputation, 50% will get an ulceration in the contralateral extremity within three years. Of those with an amputation, 63% are dead within five years.[18-20]

ECONOMIC BURDEN OF THE PROBLEM

In the United States, one of every seven health care dollars is spent on diabetes. It has been calculated that the average cost to heal an ulceration is $27,500, for an amputation is $40,000.[7,17] In his presidential address to the American Diabetes Association in 2004, Eugene Barnett, MD, said,[21]

It is estimated that the cost of caring for people
with diabetes and obesity will, as the population ages,
be a dominant factor in bankrupting
the Medicare Trust Fund by the year 2019.

THE CONTROVERSY

The correct and respected, traditional, medical approach to the treatment of diabetic neuropathy is an attempt to achieve a euglycemic state, and to obtain regular care of the feet.[22-24] Regular care includes foot inspection daily for the presence of erythema, yearly sensory testing to detect neuropathy, and provision of special protective footwear. If there is a painful neuropathy component, burning, dysesthetic feet, then the traditional medical approach includes both non-narcotic and narcotic medication, which all too often is not successful in relieving the pain.[25,26] Diabetic neuropathy occurs in a stocking and glove distribution, consistent with a systemic metabolic disease. Since there is no known medical treatment for diabetic neuropathy, the disease progresses over time. Sensory loss in neuropathy provides the basis for infection, ulceration and amputation.

The controversy begins in medical school with the classic teaching that diabetic neuropathy is progressive and irreversible. It follows immediately then, that there should be no role for surgery in the prevention or treatment of this diabling coniditon.

MY CLINICAL EXPERIENCE CHALLENGED TRADITION

The genesis of my concept, that *the symptoms of diabetic neuropathy could be treated if they were related to superimposed nerve compressions upon an underlying metabolic neuropathy*, relates to my training in Plastic Surgery. Plastic Surgery, to me, is really an approach to problem solving.

When I finished my Plastic Surgery training in 1978, which included a Hand Surgery Fellowship, it was natural for patients with diabetes to be referred to me for the treatment of their carpal tunnel syndrome. Some of these patients had an isolated carpal tunnel syndrome, but some had the symptoms of carpal tunnel syndrome in the presence of their diabetic neuropathy. Throughout my eight years of surgical training after medical school, I had not encountered patients with symptoms of neuropathy. Certainly, I had been referred patients with diabetic foot infections, ulcers, and gangrene, requiring the classic procedures for drainage, closure and amputation. My attention had been directed to technical procedures and not to the patient's neuropathy complaints, per se. Now, the similarity of the symptoms of chronic nerve compression and those of diabetic neuropathy became apparent to me, and these symptoms seemed almost the same. In deed, in many patients they were identical. When a diabetic patient would tell me that their thumb, index, and middle finger felt "better" or even "normal" after carpal tunnel decompression, but that their little and ring finger still felt numb, tingling, burning or cold, it seemed reasonable to me to consider that these symptoms in the diabetic might represent a superimposed cubital tunnel syndrome, rather than neuropathy alone. If I could identify a positive Tinel sign over the ulnar nerve in the post-condylar groove, then it was possible that there was another nerve compression at this location of a known site of anatomic narrowing. Although traditional ulnar nerve decompression techniques have varied success depending upon the degree of nerve compression,[27] I developed an approach to submuscular transposition of the ulnar nerve that included a lengthening of the flexor/pronator muscle mass.[28-31] I found this approach to give excellent relief of symptoms despite advanced degrees of nerve compression, even in diabetics.

The diabetic patients who had their median nerve decompressed at the wrist and their ulnar nerve transposed at the elbow might still complain of numbness and burning over the dorsal radial aspect of their hand. This was presumed still to be due to their diabetic neuropathy. Could there be a site of nerve compression responsible for these symptoms too? While working anatomically on the treatment of painful neuromas of the radial sensory nerve,[32] in became clear that the radial sensory nerve could become entrapped in the fascia connecting the brachioradialis to the extensor carpi radialis longus. Although Wartenberg, a neurologist, had reported in 1934 that these symptoms were due to Cheiralgeia paresthetica, an inflammation,[33] I reported in 1986, in the hand surgery literature, that this was the site of radial sensory nerve compression in the forearm.[34] Thereafter, decompression of this nerve was added to my approach to the diabetic with painful upper extremity complaints, and with the result that these symptoms also could be relieved. **Anatomically, the sensory territories of the median plus the ulnar plus the radial sensory nerves, when combined, gives the pattern of a glove. I realized that multiple chronic peripheral nerve compressions in the upper extremity give the appearance of neuropathy.**

THOUGHT TRANSITION FROM UPPER TO LOWER EXTREMITY

Patients with diabetes who had been referred to me for the treatment of upper extremity neuropathy symptoms, and who found relief after upper extremity peripheral nerve decompression, would ask, "Doctor Dellon, can you do the same thing for my feet?" Traditionally I would answer, "no, your symptoms in the foot are due to neuropathy. I am sorry I cannot help you. No one decompresses nerves in the leg." One day, when a resident was with me, and my mind was in its teaching

or more contemplative mode, instead of the mode that just regurgitates what we have been taught classically, I responded to this same question by saying, “Maybe I can help. Maybe the nerves behave the same way in the lower extremity as they do in the upper extremity”.

I then had to begin to examine what had been written about the presence of nerve compression in neuropathy. In fact, there have been many reports suggesting that nerve compression is not uncommon in patients with diabetes (**see Table 2**).

Clearly, the metabolic disease, diabetes mellitus, must have some underlying susceptibility that predisposed the peripheral nerve to chronic nerve compression, for, as **Table 2** demonstrates, between 25 to 30% of patients in studies of nerve compression had a nerve compression. In deed, one study, as early as 1961, had identified a lower extremity peripheral nerve compression of the common peroneal nerve at the fibular head. “What if the tibial nerve were compressed at the ankle, in the tarsal tunnel,” I asked myself. If it could, then it would follow that **if you combine the skin territories of the peroneal and tibial nerves, you get a stocking sensory distribution of skin**. It followed, theoretically, that **if sensation could be restored to the foot, then ulceration and amputation could be prevented. Without realizing that I was beginning a journey that would continue more than a quarter of a century, most of most my professional life, I went to the anatomy lab to identify sites of compression for the nerves of the lower extremity, and to evaluate the basis for nerve decompression in neuropathy.**

Table 2
Prevalence of diabetics in nerve compression cohorts

Author	Year	Nerve Compression Site	Diabetics in Study	
Mulder, et al[35]	1961	carpal tunnel	(9/103)	9%
		cubital tunnel	(5/103)	5%
		peroneal n., knee	(13/103)	13%
			total	**27%**
Brown & Asbury[36]	1984	carpal tunnel	(15/38)	40%
Comi, et al[37]	1985	carpal tunnel	n.a.	23%
Wada, et al[38]	1997	carpal tunnel	(21/65)	32%
Greenwald, et al[39]	1999	cubital tunnel	(6/24)	25%

HYPOTHESES TO BE TESTED IN THIS THESIS

1) Nerves in diabetics are susceptible to chronic nerve compression.
2) There are anatomic sites of narrowing in the lower extremity that can locations of chronic nerve compression.
3) Decompression of a peripheral nerve in the lower extremity of a person with symptoms of neuropathy can relieve the symptoms of numbness and pain.
4) Restoration of sensibility to the foot of a person with diabetic neuropathy will prevent ulceration.
5) Restoration of sensibility to the foot of a person with diabetic neuropathy will prevent amputation.
6) Restoration of sensibility to the foot of a person with diabetic neuropathy will restore balance.

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Chapter 2

Approach to the peroneal nerve

Based on

Dellon AL, Entrapment of the deep peroneal nerve on the dorsum of the foot.
Foot and Ankle 11:73-80, 1990

Dellon AL, Ebmer J, Swier P, Anatomical variations related to decompression of the common peroneal nerve at the fibular head.
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The first nerve entrapment site for the peroneal nerve to be described was for the common peroneal nerve at the fibular neck. In this location, the common peroneal nerve transitions from the popliteal fossa, posteriorly, to travel laterally across the neck of the fibula and into the anterior and lateral compartments of the leg. In 1897, a woman having a gynecologic procedure awoke from surgery with foot drop. During surgery she had been positioned with stirrups in the classic lithotomy position.[1]

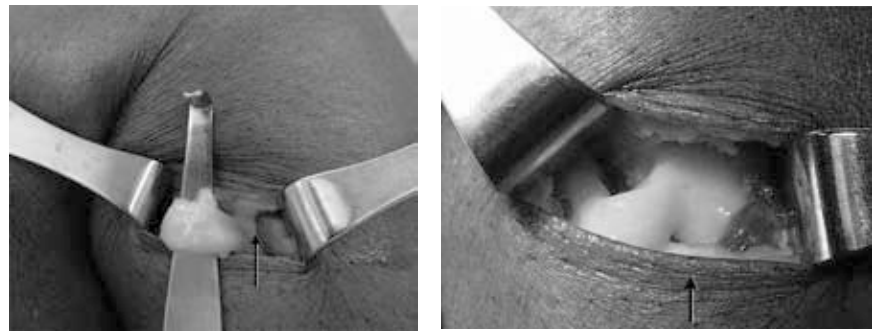
COMMON PERONEAL NERVE ENTRAPMENT

Surgical considerations related to the common peroneal nerve are classic within Orthopedic Surgery, as it is recognized that this nerve can be injured concomitantly with knee joint and ankle joint injuries.[2-8] Stretch and traction injuries can be sufficient to give foot drop, with complete peroneal motor and sensory loss, to less severe gradations of chronic nerve compressions. Surgical approaches have been described variously from 6 months of observation, awaiting spontaneous recovery, to surgical exploration. At surgery, the approach varies from neurolysis, to nerve repair, to nerve grafting of the common peroneal nerve depending upon the pathology observed.[9-13]

My first surgical approaches to the common peroneal nerve involved primarily division of the superficial fascial of the peroneus longus muscle, and our reported series was the largest at that time.[14] That series of 31 patients was a retrospective review of patients from 1980 through 1990. Following neurolysis of the common peroneal nerve at the fibular neck, 90% of the patients had improvement in peroneal nerve motor function, and early intervention in patients with post-traumatic peroneal palsy was recommended.

During my early procedures upon the common peroneal nerve, I observed that there was most often a fibrous band of varying width *deep* to the peroneus longus muscle. This was a band not normally seen during an anatomy dissection. This band clearly had to be divided, and deep to this band was often a definitive indentation in the common peroneal nerve, with the nerve deep to this band being flattened, soft and consistent with axonal loss. There was also a loss of the vascular marking on the nerve. In the trauma cases, without neuropathy, the nerve was white in appearance, except at this site of compression (**Figure 1**).

Figure 1



Exposed right common peroneal nerve held beneath the small retractor. Arrow identifies the deep white fibrous band beneath the retracted peroneus longus muscle (muscle retracted beneath large retractor to the right). Note white color of nerve. Right: after division of the deep fibrous band, the indentation of the nerve by the band is noted by arrow. Note absence of vascular markings on the nerve.

A comparison study of 29 bilateral cadaver dissections and 65 unilateral clinical decompressions was undertaken then to identify the anatomic variations about the common peroneal nerve at the fibular neck.[17] This study demonstrated that while the fibrous band deep to the peroneus longus muscle was present in only 30% of cadavers, it was present in 78.5% of patients requiring neurolysis of the common peroneal nerve for clinical symptoms of nerve compression. Additional findings were that the lateral gastrocnemius muscle may have a thick fascial origin deep to the common peroneal nerve (43% of cadavers and 20% of patients) that would require division (**Figure 2**), and that the entrance of the common peroneal nerve into the anterior and lateral compartments of the leg can be tight due to a proximal origin of the soleus muscle (9% of cadavers and 6% of patients). These observations require a surgical approach for neurolysis of this nerve to be adjusted accordingly to search for each of these variations.

SUPERFICIAL PERONEAL NERVE ENTRAPMENT

Entrapment of the superficial peroneal nerve as it transits from below the fascia and muscles of the lateral compartment of the leg and into the subcutaneous tissue was first described by Henry in 1945.[18] Little however has been written about

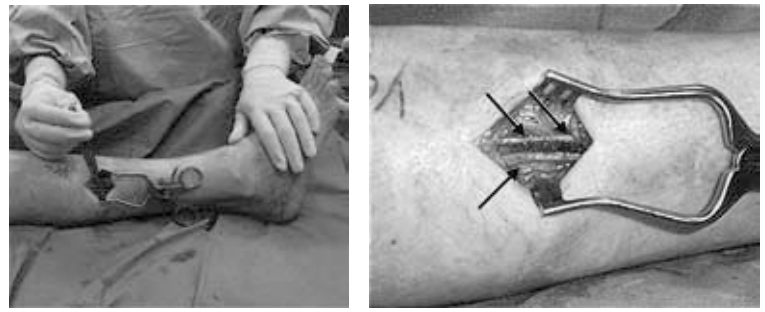
Figure 2

Right knee with exposure of common peroneal nerve. Patient had previous blunt trauma to the knee. The nerve is held beneath small retractor. Fibrous band seen in Figure 2.1 has been released. The white thickened fascia of the lateral gastrocnemius (arrow), seen deep to the common peroneal nerve, remains to be decompressed.

chronic compression of this nerve. In 1981, Banerjee and Koons described two patients with entrapment of the superficial peroneal nerve.[19] As recently as 1997, Styf and Moberg reported an incidence of superficial peroneal nerve entrapment as a source of pain in just 3.5 % or 480 patients with lower extremity pain.[20] The most commonly understood cause of this nerve entrapment is an induced compartment syndrome due to exercise, described first in 1977 by Gafins, Murbarak, and Owen,[21] and then popularized by Rorabeck, Bourne and Fowler in 1983.[22] This condition continues to be reported extensively today, for example with a series of 50 patients in whom the specificity and sensitivity of different diagnostic techniques were evaluated.[23]

My own involvement with this nerve began in attempting to treat patients with dorsal foot pain due to neuromas of the peroneal nerve distal branches. Ultimately an approach was described that required first resecting the distal neuromas of the deep and/or superficial peroneal nerves, and then translocating the proximal ends of these nerves into a muscular environment in the anterior compartment of the leg, away from the movements of the ankle joint.[24] During the care of these patients, there were some who remained with some degree of pain in the distribution of the superficial peroneal nerve despite my having personally identified and resected the superficial peroneal nerve in its traditional location, the lateral compartment.[25-29] I found that a local anesthetic block of the superficial peroneal nerve at the ankle would relieve the persistent pain, suggesting that there was another anatomic route to innervate this region. My own subsequent dissections identified the presence of a branch of the superficial peroneal nerve in these patients, located in the anterior compartment of the leg. An example of a subsequent patient having a branch of the superficial peroneal nerve in both the anterior and the lateral compartment of the leg is give in **Figure 3**.

Figure 3



Left: Overview of surgical site in the lower extremity. Right: The fascia of the anterior and lateral compartment has been removed, as has the septum between the anterior and lateral compartments. The larger portion of the superficial peroneal nerve is noted by a single arrow in the lateral compartment, while the portion in the anterior compartment is noted by a double arrow.

The presence of a nerve in the anterior compartment was not described in an early anatomic report of this region by Kosinski in 1926.[30] A report of the clinical success of decompression of the superficial peroneal nerve by Styf in 1989 noted that 6 of 22 patients had a branch of the superficial peroneal nerve located outside of the lateral compartment and within the anterior compartment.[31] Subsequently, variations in the locations of branches of this nerve was reported by Adkinson, et al in 85 legs: the superficial peroneal nerve was within the lateral compartment only in 75% of their dissections.[32] I have subsequently studied this in both cadaver dissections and clinical explorations of the superficial peroneal nerve.[33-35] These results are summarized in **Table 1**.

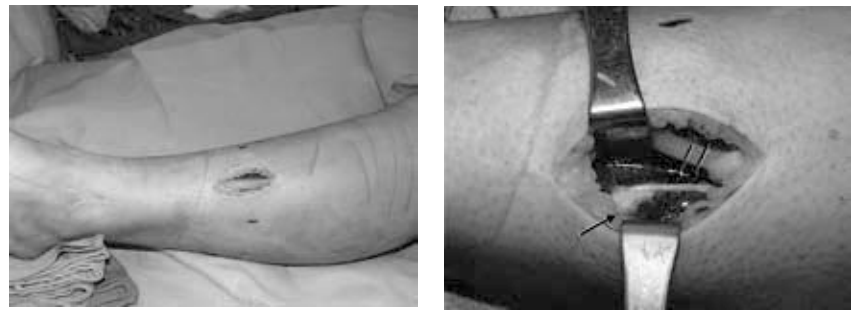
Table 1
Anatomic variability of superficial peroneal nerve

Study	Number legs	Lateral	Anterior	Lat + anter	Subcut
Adkinson, et al, 1991[32]	cad 85	73%	12%	14%	0%
Styf, 1989[31]	clin 22	73% (16/22)	22% (5/22)	5% (5/22)	0%
Rosson, et al, 2005[33]	clin 35	57% (20/35)	21% (6/35)	26% (9/35)	0%
Barrett, et al, 2006[34]	cad 75	72% (54/75)	17% (23/75)	5% (4/75)	6%
Ducic, et al, 2006[35]	cad				

the results of the clinical explorations[33] and cadaver dissections[34,35] carried out in my own series of studies confirms the earlier observations of Adkinson et al,[32] and of Styf[31] in terms of the variability of the superficial peroneal nerve. The clinical implication of these anatomic studies is that if the superficial peroneal nerve requires decompression, neurolysis or resection, then the surgeon must evaluate both the anterior and the lateral compartments of the leg. An example of the superficial peroneal nerve being located completely in the anterior compartment is given in **Figure 4**.

From **Table 1**, it may be calculated that percentage of patients with the superficial peroneal nerve located only within the lateral compartment is 43% in the two clinical series, whereas it is 72% in the cadaver series. This difference is significantly different at the $P < .05$ level by chi-square analysis. This reinforces the need to explore both compartments at the initial operation. This suggests, furthermore, that patients with failure to recover from a traditional neurolysis of the superficial peroneal in the lateral compartment should be re-explored looking for a remaining branch of the nerve that is still entrapped. Indeed, a recent study of 18 patients who failed to improve from their first surgical attempt to treat their exertional compartment syndrome found that 75% did improve after additional fasciectomy and neurolyis.[36]

Figure 4



Overview of surgical site in the lower extremity. Right: After fasciectomy, a portion of septum between the anterior and lateral compartment (arrow) is left to identify the location of the entire superficial peroneal nerve within the anterior compartment (double arrow).

THE DEEP PERONEAL NERVE ENTRAPMENT

During my training in Hand Surgery, surgically transferring a toe to the hand by microsurgical transfer focused my attention of the relationship between the tendon of the extensor hallucis brevis and the deep peroneal nerve. During the 1980's, as I was increasingly being referred patients to treat foot pain, it became clear to me that localized dorsal foot pain, or radiation of pain between the first and second toes might be due to compression of the deep peroneal nerve at the location where the tendon of the extensor hallucis brevis crosses the deep peroneal nerve in close association to the first and second metatarsal junctures with the cunnieform bones.

In 1990, my report of compression of the deep peroneal nerve was published along with my technique for the neurolysis.[37] The neurolysis included resection of a segment of the tendon of the extensor hallucis brevis where it caused the compression (**Figure 5**). There is also frequently a small distal fascial band at the site at which the nerve becomes superficial to enter the skin.

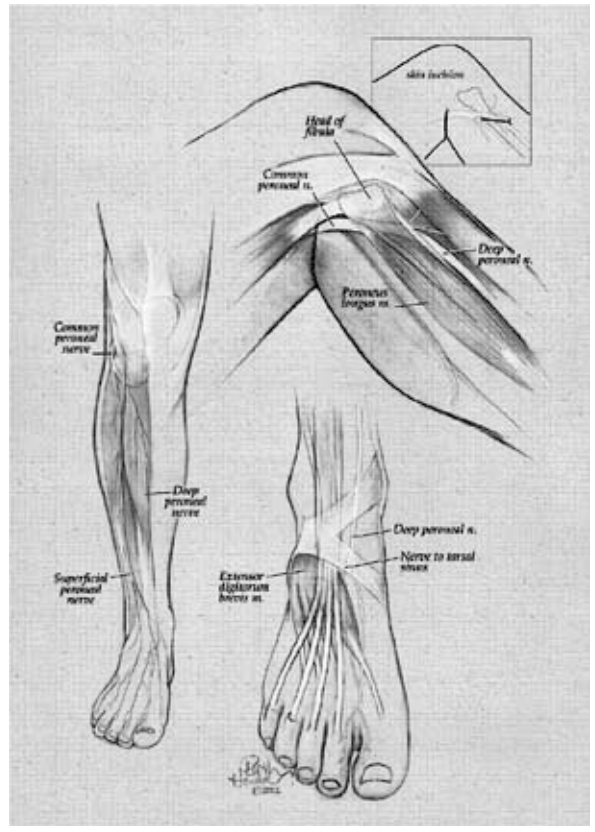
Figure 5

Dorsum of foot after excision of portion of tendon of the extensor digitorum brevis that compressed the deep peroneal nerve (overlying the marker). Note the indented area and widening of the nerve proximal to the site of compression (arrow).

SUMMARY

Three sites of anatomic narrowing must be evaluated by the physician who suspects the patient of having sensory or motor symptoms related to the peroneal nerve. Because there are no useful surgical illustrations that demonstrate the anatomic observations made in the above studies, **Figure 6** was commissioned.[38]

Figure 5



Anatomic sites of compression along the peroneal nerve include the common peroneal nerve at the knee (incision site shown in inset), the superficial peroneal nerve in either the anterior or lateral compartments (or both), and the deep peroneal nerve over the dorsum of the foot, caused by compression of this nerve branch by the tendon of the extensor digitorum brevis (http://www.dellonipns.com/peroneal_nerve_compression.php)

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Chapter 3

Approach to the tibial nerve

Based on

Dellon AL, Dackinnon SD, Tibial nerve branching in the tarsal tunnel.
Arch Neurol, 41:645-646, 1984

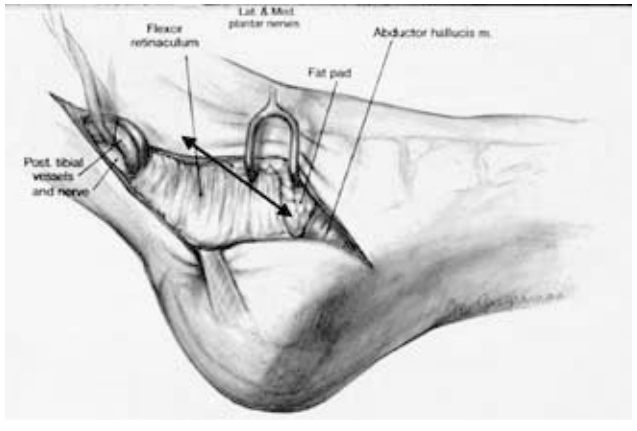
Dellon AL, Computer-assisted sensibility evaluation and surgical treatment of tarsal tunnel syndrome.
Adv Podiatry, 2:17-40, 1996

MY FIRST TARSAL TUNNEL EXPERIENCES

After the popularization of sensory symptoms in the hand being related to compression of the median nerve at the wrist, called the carpal tunnel syndrome by George Phalen, MD, of Cleveland, Ohio in the 1950's, it was only a matter of time until similar symptoms in the foot were to be related to a peripheral nerve compression. In 1962, independent observations led to the publication of case reports in England, by Lam,[1] and in the United States by Keck,[2] of compression of the tibial nerve at the ankle in the region known as the tarsal tunnel. Both authors drew the analogy of tibial nerve compression in the tarsal tunnel to the median nerve in the carpal tunnel. Each author described the flexor retinaculum (previously termed the lancinate ligament) crossing from the medial malleolus to the calcaneus as being the unyielding structure which was responsible for compressing the tibial nerve against the underlying bones. (**Figure 1**)

In 1980, I was referred a patient with sensory symptoms in the legs who had been evaluated by a Vascular Surgeon and found to have excellent circulation. The Vascular Surgeon suggested to me, knowing of my interest in upper extremity peripheral nerves, that this patient might have the newly described lower extremity peripheral nerve compression called tarsal tunnel syndrome. When I saw this patient, he did have a positive Tinel sign over the tibial nerve in the tarsal tunnel as well as symptoms related to the plantar aspect of his foot associated with nighttime symptoms. As I prepared to operate on this patient, a retired United States Senator, I did the mental comparison of the tarsal tunnel and the carpal tunnel and realized that this was not an appropriate analogy. I did a cadaver dissection which documented the appropriate analogies, given in **Table 1**.

Figure 1



The flexor retinaculum covers the posterior tibial artery and veins and the tibial nerve within the tarsal tunnel (double headed arrow). This region is analogous with the forearm, and not the carpal tunnel.

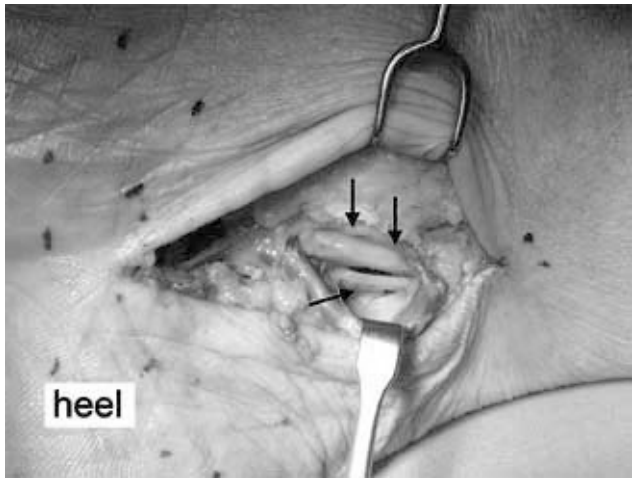
Table 1
Homologies related to nerve compression:
Median nerve at wrist and tibial nerve at ankle

Upper Extremity	Lower Extremity
Forearm	Tarsal Tunnel
Antebrachial Fascia	Flexor Retinaculum
Carpal Tunnel	Medial Plantar Tunnel
Flexor Retinaculum	Roof of Medial Plantar Tunnel (deep fascia of Abductor Hallucis)
Guyon's Canal	Lateral Plantar Tunnel
Hook Process of Hamate	Septum between Medial and Lateral Plantar Tunnels
Tunnel for Palmar Cutaneous branch of Median Nerve	Medial Calcaneal Nerve Tunnel

During the cadaver dissection and during the patient's surgery, it was clear that the flexor retinaculum at the ankle is loose, and, in the absence of trauma or a space occupying lesion within the tarsal tunnel, this thin ligament could not in and of itself be the primary cause of chronic tibial nerve compression. Rather, I believed that it was the thick unyielding layer of deep fascia which originates on the calcaneus and gives origin to the abductor hallucis muscle, that was the source of pressure. In the hand, for patients with pain after nonunion of fractures of the

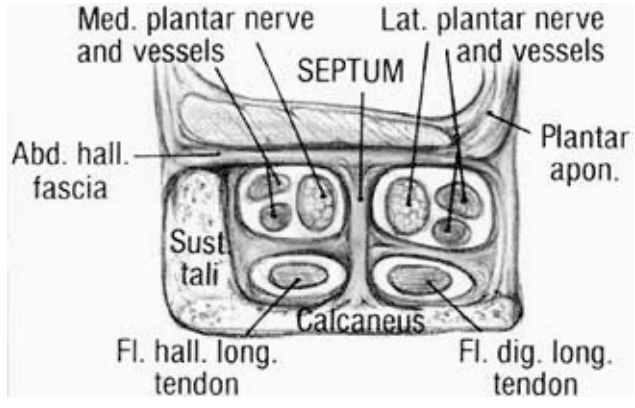
hook process of the hamate, it was appropriate to excise the bone fragment, allowing the median and ulnar nerves to merge their respective carpal and Guyon's canals into one larger tunnel. I decided that this would be the appropriate approach to this patient's symptoms, namely, to excise the septum between the medial and lateral plantar tunnels, creating one large physiologic space for the medial and lateral plantar nerves. That was the surgery I did. The patient recovered from this procedure, with great symptomatic relief. The tibial nerve normally divides into the medial and lateral plantar nerves within the tarsal tunnel. During the first operation I did for tarsal tunnel decompression, I found an anatomic anomaly. The tibial nerve was in fact present as two separate nerves at the time it entered the tarsal tunnel. An example of this anomaly from a recent patient is seen in **Figure 2**. I had seen this occur with the median nerve at the wrist, where it had a high division in the forearm, so that at the time it entered the carpal tunnel it occupied more volume than normal, perhaps predisposing the person to get carpal tunnel syndrome. I hypothesized that this might be the situation for this patient as well.

Figure 2



Approach to the medial ankle tunnels. Within the tarsal tunnel, the posterior tibial artery and veins are being retracted posteriorly to reveal the tibial nerve. In this example, rather than dividing into the medial and lateral plantar nerves within the tunnel, the medial plantar nerve (double arrows) and the lateral plantar nerve (single arrow) are both present, having divided anomalously more proximally in the leg.

Figure 3



Cross-section, transversely through the region of the foot just distal to the tarsal and directly through the abductor hallucis muscle. The flexor retinaculum splits to creat a layer of fascial superficial and deep to the abductor hallucis. The superficial fascia must be released during the surgery and the abductor muscle retracted to demonstrate the deeper, thick fascial layer that forms the roof of the medial and lateral plantar tunnels. There is a septum between the two tunnels that arises either from the calcaneus or from the flexor sheaths, or both. The operation that I designed removes this septum, which is analogous to the hook process of the hamate in the hand, the process which separates the carpal tunnel (like the medial plantar tunnel) from ulnar nerve in Guyon's canal (like the lateral plantar tunnel).[5]

Inspired by the success of my first tibial nerve decompression in this first patient, and intrigued by the anatomy variability, I began my first series of cadaver dissections into this anatomy. This was published in 1984,[3] and documented that the tibial nerve divided within 1cm on either side of a line drawn from the medial malleolus to the calcaneus, a line I termed the medial-calcaneal axis. In the 20 cadavers dissected, one specimen had the high origin observed in my first patient, or a 5% incidence. Furthermore, description of the medial calcaneal nerve arising with three possible variations was described as well; an origin from the proximal tibial nerve, an origin from the lateral plantar nerve, and an origin from both. This was in the first time that this variability was described. The medial calcaneal nerve(s) had its (their) own separate tunnel with a roof again formed by the origins of the fascial layer that gives rises to the abductor hallucis muscle and forms the roof of the medial and lateral plantar tunnel.

In 1987, the analogies in **Table 1** were published[4] in the hopes of drawing recognition to the fact that simply releasing the tarsal tunnel would not have a high prob-

ability of success in relieving the patient's symptoms (**Figure 3**).

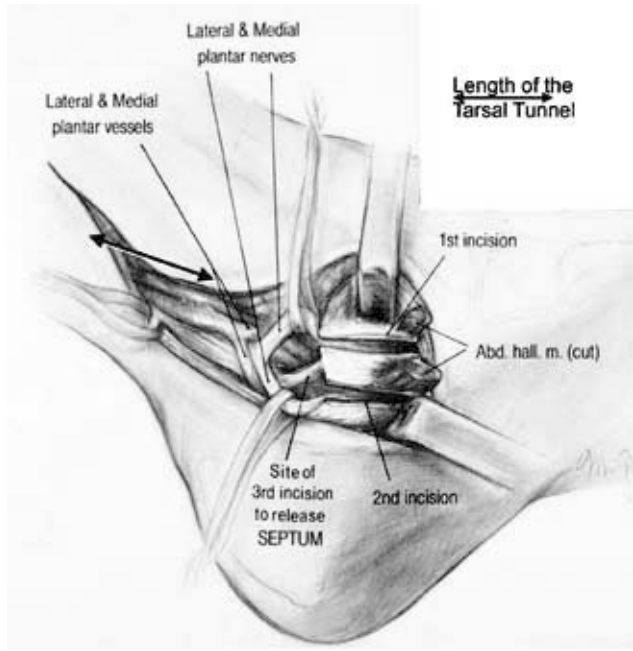
In 1988, my book, co-authored with Susan E. Mackinnon, MD, was published. In *Surgery of the Peripheral Nerve*, Chapter 12 is entitled Tarsal Tunnel Syndrome. This was the first textbook to contain anatomic drawings of the technical approach that I had developed for decompression of the tibial nerve in the four medial ankle tunnels.[5] That chapter reviewed previous experiences with tarsal tunnel decompression. There were actually not many reports of tarsal tunnel decompression, and those reports in general did not give expectation for much success when the operation that was performed was to just divide the flexor retinaculum. **Figure 4** further illustrates the difference in approaches.

INNERVATION OF THE HEEL

Knowledge of the innervation of the medial calcaneal region is important for the diagnosis and treatment of heel pain, tarsal tunnel syndrome, soft tissue and bony ankle injury and the treatment of secondary heel pain due to traumatic or iatrogenic neuroma. This subject is inadequately considered in standard anatomy textbooks, which generally illustrate a single medial calcaneal nerve originating from the lateral plantar nerve within the tarsal tunnel.[6-8] As discussed briefly above, in 1984, we reported three patterns of origin of the medial plantar nerve after dissecting 20 embalmed cadavers.[3] One of these patterns had two different calcaneal nerves; one originating from the lateral plantar and one originating from the posterior tibial nerve, both within the tarsal tunnel. In 1988, Havel, et al, described nine patterns of origin of the calcaneal nerve after dissecting 38 pairs of embalmed cadavers.[9] His increased number of patterns resulted from noting variations in which a calcaneal nerve originated proximal to the tarsal tunnel and also by describing an origin of a medial calcaneal nerve from the medial plantar nerve.

The publications of Baxter since 1984 have popularized the concept that heel pain is due to the "first branch of the lateral plantar nerve".[10-13] This is actually a small sensory branch arising from the motor branch of the lateral plantar nerve that innervates intrinsic muscles. Clearly these branches exist within the lateral plantar nerve within the tarsal tunnel, and do not go to the skin of the heel itself. Most recently, a microdissection of fresh cadavers has described the innervation of the skin of the medial ankle region,[14] but did not consider the medial calcaneal innervation. It was therefore necessary to describe the variations in the origin of the medial calcaneal nerve from dissections done in living tissue during tarsal decompression in humans. Because the work to be described later in this thesis on patients with diabetic neuropathy provided the opportunity for many dissections of the medial ankle, it was possible to record detailed notes from 85 dissections done in a bloodless field using the tourniquet in the year 2000. These were finally reported in 2002.[15] From computer records of tarsal tunnel decompressions done

Figure 4



Overview of anatomy of the medial ankle. The tarsal tunnel contains the posterior tibial vessels and the tibial nerve. The length of the tarsal tunnel is noted by the double headed arrow. Note that the tibial nerve divides into its branches within the tarsal tunnel and that a medial calcaneal branch is shown, as is traditional, to arise proximally and not have a tunnel. Alternatively, the calcaneal branch has been shown to arise from the lateral plantar nerve. For the traditional tarsal tunnel decompression, only this length is released. The true pressure upon the tibial nerve however is to its branches that are more distal. Distal to the tarsal tunnel are the medial and lateral plantar tunnels deep to the fascial of the abductor hallucis muscle. After the tarsal tunnel has been opened, then the roof of both the medial and lateral plantar tunnels is divided. The septum is excised to create one large tunnel for both of these nerves. In this early illustration, the muscle has been shown, mistakenly, divided. The muscle is retracted but not divided. Also in this early illustration, the tunnel for the medial calcaneal nerve is not shown.[5] **The main distinction from tradition that I introduced was to appreciate that there were four medial plantar tunnels that required release, and not just the tarsal tunnel.**

between 1998 and April of 2000, operative notes were reviewed concentrating on the description of the origins of the calcaneal nerve(s). The surgical procedure was done in a bloodless field using 3½ power loupe magnification. In particular, the site of origin with respect to the tarsal tunnel, the posterior tibial nerve, and the medial and lateral plantar nerves was noted in the reports of 85 tarsal tunnel decompressions. The nerve described by Baxter, that arises from the first motor branch of the lateral plantar nerve, was not dissected in these patients and is not included in any of the numbers or statistics describing the innervation of the calcaneal skin.

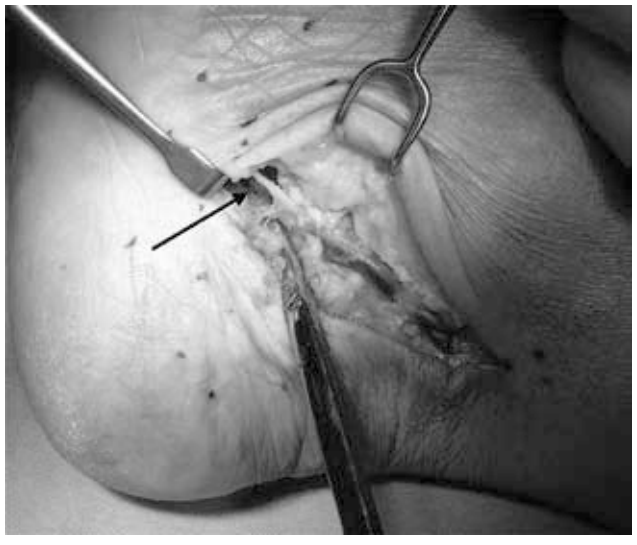
A nerve not previously described in detail, a branch from the medial plantar nerve that crosses anterior to the tibial vessels to innervate the skin of the posterior-medial arch and calcaneal skin, was noted in 46% of the feet (**Figure 5**)

In 36 of the 39 (92%) feet in which this branch, shown in **Figure 5**, was found, the nerve crossed the vessels in a distal direction, and pierced the fascia of the abductor hallucis to innervate the skin of the posteromedial arch, just anterior or distal to the true region of the skin of the heel. In three feet, this nerve did not innervate the skin of the posteromedial arch, but rather entered the tunnel usually occupied by the medial calcaneal nerve, and was the primary innervation of the heel skin.

Table 2 and 3 lists the variations found in this study.

Twenty-two percent of the feet had at least one calcaneal nerve originating proximal to the tarsal tunnel. In 7% of the feet the division of the posterior tibial nerve into the medial and lateral plantar nerves occurred more than 3 cm proximal to the malleolar-calcaneal axis. **Figures 6 through 9** illustrate the different patterns of origin of the calcaneal nerve(s).

Figure 5



"New" nerve to the medial ankle/arch skin arising from the medial calcaneal nerve anterior to vessels (arrow). It is at risk for injury during plantar fasciotomy and tarsal tunnel surgery.[15]

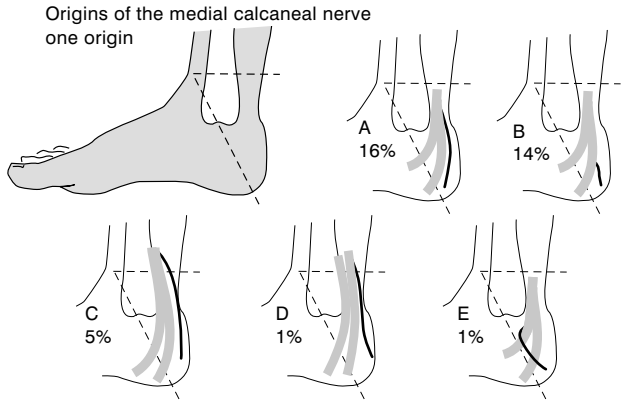
Table 2
Site of origin of medial calcaneal nerves[15]

Nerve of Origin*	Percentage of Feet
Lateral Plantar	(56/85) 66%
Posterior Tibial	(48/85) 56%
Medial Plantar	(39/85) 46%

Table 3
Variation in number of calcaneal nerves per foot[15]

Number of Calcaneal Nerves	Percentage of Feet
1	(31/85) 37%
2	(35/85) 41%
3	(16/85) 19%
4	(3/85) 3%

Figure 6



Variations origin of medial calcaneal nerve when there is just one medial calcaneal nerve. Variation D is similar to that found in the first patient I operated on for tarsal tunnel syndrome, described above, with the high division of the tibial nerve.[15]

Figure 7

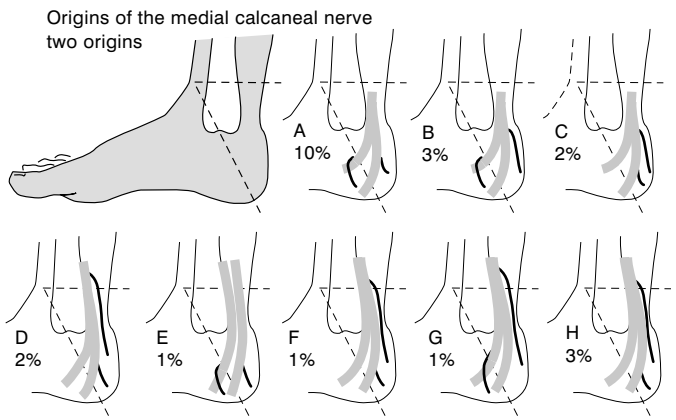


Figure 8

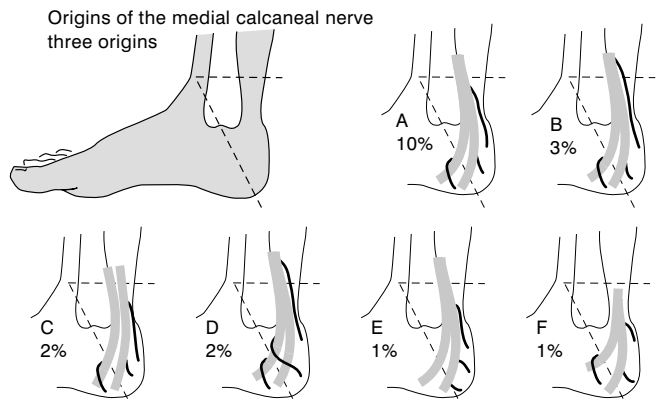
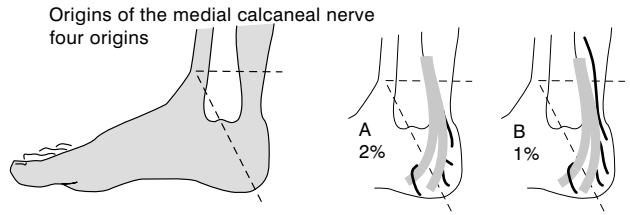


Figure 9



The results of this study demonstrate a great variability in the site of origin of the medial calcaneal nerve(s). This knowledge will provide the surgeon with a previously unavailable guide during dissections in this area. For example, Cunningham's anatomy text does not describe or illustrate the origin of the medial calcaneal nerve, simply indicating that it pierces the fascia and its branches are distributed to the skin of the heel.[7] In figures in the Cunningham text neither the medial nor the lateral plantar nerves are demonstrated as giving origin to a calca-

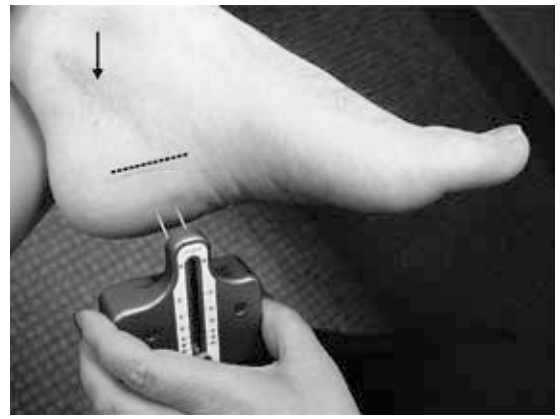
neal branch. In contrast, Pernkopf's textbook suggests that there is a branch from the medial plantar nerve to the skin of the heel area, but this branch is not named.[8] This same illustration demonstrates the medial calcaneal branch as arising from the posterior tibial nerve, again at a proximal level. An origin for the medial calcaneal nerve from the lateral plantar nerve is not illustrated. In further contrast, Grant's Atlas of Anatomy demonstrates the medial calcaneal nerve to consist of two branches arising from the posterior tibial nerve, probably proximal to the tarsal tunnel.[6] A recent textbook demonstrates the medial calcaneal nerve to arise as a single branch from the posterior tibial nerve within the tarsal tunnel.[12] The results of the present study demonstrate that 63% of feet having surgery to decompress the tarsal tunnel will have more than one origin for the medial calcaneal nerve (**Table 2**) and that these nerves may originate from either the posterior tibial nerve (56%), the lateral plantar nerve (66%) or the medial plantar nerve (46%) (**Table 2**).

The anatomic patterns described in this study hopefully will provide a knowledge base that may be used as more surgery in this medial ankle region is done in the future. This increased surgery in this region may be predicted from the recent publications on surgical decompression of the calcaneal nerve to treat recalcitrant heel pain[16,17], the recent publications, to be discussed later in this Thesis that suggest that decompression of the tarsal tunnel can restore sensation to diabetic feet, and the publications that have documented neuromas of nerves that innervate the medial calcaneal region.[18,19]

The results of this study emphasize that the medial plantar nerve gives origin to a nerve that innervates the skin in the region in which incisions are made commonly for calcaneal spur removal or plantar fasciotomy. This nerve, which has branches on the order of 0.9 mm is at risk for injury during these procedures. Awareness of its existence, and use of magnification during surgery in this area is the only hope for prevention of painful neuromas as a complication to heel pain surgery. As just discussed, this nerve can cause heel pain that is misinterpreted as recurrent or persistent plantar fasciitis when it is a true neuroma of a medial calcaneal nerve.[18] In 3% of the feet in this study, the main innervation of the heel skin was from a branch of the medial plantar nerve that crossed the vessels superficially to then enter into the tunnel usually occupied by a branch of the posterior tibial or lateral plantar nerve. The location of this nerve, superficial to the vessels, places it at risk for injury during division of the flexor retinaculum.

With regard to teaching an approach to diagnosis and treatment of heel pain, it is

Figure 10



The Pressure-Specified Sensory Device™ shown being used to measure sensibility of the heel in a patient with persistent heel symptoms following a tarsal tunnel decompression (arrow) and previous open plantar fasciotomy (scar ….. line).

probably most appropriate not to attribute heel pain to any one nerve, as Baxter has,[10-13] because that misdirects the therapy and the surgeon. Since it cannot be known without a surgical dissection what the innervation pattern of the heel is, it is most reasonable to suggest that heel pain that does not respond to non-operative measures be approached surgically through an incision that permits the surgeon access to all the described variations in heel innervation as shown in **Figures 6 through 9**.

The pattern of origin of medial calcaneal nerves arising proximal to the tarsal tunnel suggests that the involvement of heel symptomatology in tarsal tunnel syndrome can vary based upon the origin of this nerve. For example, from **Figure 6C and D**, it may be estimated that 6% of tarsal tunnel syndrome patients may have no heel symptomatology and/or normal neurosensory testing with the Pressure-Specified Sensory Device™ of the medial heel due to the origin of the medial calcaneal nerve proximal to the site of compression.[20-22] It may be predicted that there will be a group of patients whose forefoot symptoms in tarsal tunnel syndrome will dominate over their heel symptoms because there is a dual innervation of the medial calcaneal region, in which a portion of the innervation does arise within the tarsal tunnel (**Figures 7F, G, H, Figure 8B, E, and Figure 9B**.) This group, based

upon the observations reported here comprises 15%, and with neurosensory testing Pressure-Specified Sensory Device™ (**Figure 10**) may have abnormalities that are not as advanced as those found in skin innervated by the medial plantar nerve, which, in all patients will pass through both the tarsal tunnel and the medial plantar tunnel.

RESULTS OF DELLON APPROACH TO THE FOUR MEDIAL ANKLE TUNNELS

The commonest nerve entrapment in the lower extremity, tarsal tunnel syndrome, is controversial in terms of diagnosis, surgical approach and post-operative rehabilitation. Despite being described more than 40 years ago,[1,2,23] and despite many reviews of the subject,[24-28] tarsal tunnel syndrome remains debated as to the appropriate method for electrodiagnosis, as to whether the site of compression is within the tarsal tunnel or distal to the tarsal tunnel, and as to rehabilitation of the patient post-operatively. For example, two recent textbooks differ as follows: Haddad, in Myerson's *Foot and Ankle Disorders*,[29] states "perhaps no greater source of controversy exists with respect to tarsal tunnel syndrome than the value of electrodiagnostic studies", while Richardson, in Canale's edition of Campbells's *Operative Orthopedics*,[30] states "any patient suspected of having compression of the tibial nerve beneath the flexor retinaculum should undergo electromyography and nerve conduction studies." With regard to the operative technique, Haddad recommends "the dissection be carried distally to the level of the abductor fascia, [and] routinely release this fascia by tracing the medial plantar nerve distally to the point where it plunges beneath the muscle into the plantar surface of the foot",[29] without describing a release of the lateral plantar or the calcaneal nerves. In contrast, Richardson recommends "the release must include…following both the medial and lateral plantar nerves beneath the abductor hallucis, since one or both of these branches may pass through fascial slings as they enter the plantar surface of the foot,"[30] without describing a release of the calcaneal nerve. Post-operatively, while Haddad recommends "a posterior plaster splint and stirrup for ten days followed by a controlled-ankle walking boot locked at neutral dorsiflexion"[29] for an unspecified additional length of time, Richardson recommends "a bulky compression dressing for 7 to 10 days in equinovarus…[then] the foot is brought to a neutral position, and a fiberglass prefabricated short leg cast-brace is worn for an additional 10 to 14 days while the wound matures."[30]

There are few surgical series reported for tarsal tunnel syndrome, and none with more than 68 procedures (**Table 1**).[31-44] A frequently quoted study, in which long-term followup was obtained, reported just 44% of the patients with excellent outcomes, and a 13% complication rate,[38] whereas a more recent report indicated 72% of the patients with satisfactory results but a 30% complication rate.[41] The most recent report, using outcome assessment, found 51% of the patients having a marked improvement in the quality or their life despite 85% of the patients stating they had excellent relief of their pain, and that study had a 7% rate of complications.[44] These make the 35 year old observation of Linscheid et al prophetic: "An excellent category for results was intentionally deleted because so many of our patients had some residual symptoms from this entrapment syndrome".[33] Clearly, there was room for improvement in the surgical approach to this clinical syndrome.

In my approach, as described above, it was first appreciated in 1980 that the tarsal tunnel anatomically would represent the distal forearm and not the carpal tunnel in the human upper extremity (**Table 1**). Therefore, it was hypothesized that simple release of the flexor retinaculum (the former lancinate ligament) across the tarsal tunnel was likely to be ineffective, and that the surgical approach should include decompression of all four of the medial ankle tunnels. My approach also differed in postoperative rehabilitation. From upper extremity peripheral nerve surgery, it was clear that post-operative immobilization permitted the nerve to become adherent to the surgical bed during the 7th to 21st day when collagen was forming and cross-linking,[45] and that immediate mobilization of the peripheral nerve would permit a gliding bed to form in the surgical plane of the neurolysis.[46] Therefore, it was hypothesized that post-operative rehabilitation of the patient having decompression of four medial ankle tunnels must be permitted immediate ambulation without completely immobilizing the ankle.

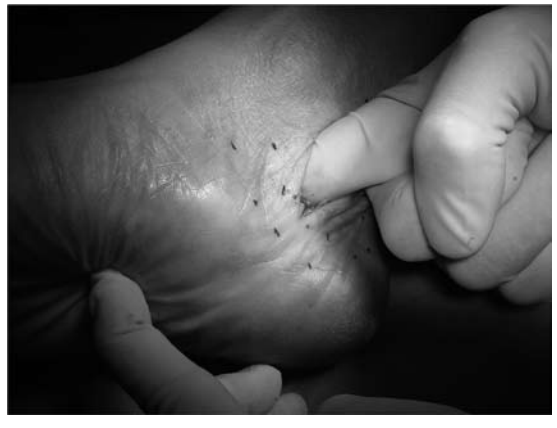
A consecutive series of patients with tarsal tunnel was accrued beginning in January of 1987 through December of 1994. The diagnosis was based upon a history of sensory disturbances in the toes, the ball of the foot, and/or the heel, with the symptoms usually worsening throughout the day, made worse with standing, and often becoming worse during the night. The physical examination included sensibility testing with the 256 Hz tuning fork, measurement of two-point discrimination with a Disk-Criminator™, and evaluation of intrinsic muscles for strength and the presence of muscle wasting of the abductor hallucis brevis and of clawing of the 2nd through 5th toes. Presence of a Tinel sign over the posterior tibial nerve

in the tarsal tunnel was required. Patients were not sent for electrodiagnostic testing, but 46 of them already had them done at the time of their initial consultation, and 24 of these (49%) were positive. There were 77 patients, of whom ten patients had bilateral tarsal tunnel syndrome, for a total of 87 tarsal tunnel decompressions. The mean age of the patient population was 42 years. Among the 77 patients were 34 who had a history of trauma and 32 who were diabetic and had symptoms of neuropathy. To be included in the final analysis, a minimum of two years postoperative follow-up was required.

The surgical technique, in brief, is that an incision is begun 4 cm proximal to the medial malleolus, and, staying well posterior to the medial malleolus, extends towards the plantar aspect of the foot, curving anteriorly at the heel. The deep fascia is entered proximally, identifying the posterior tibial artery and vein. This fascia is divided and continuing with the division distally, as it thickens to become the flexor retinaculum. When the fascia covering the abductor hallucis brevis is encountered, the end of the tarsal tunnel has been reached. The distal dissection requires that the fascia of the abductor hallucis is divided superficially. The muscle is then retracted toward the plantar surface of the foot. In this location, about 50% of patients will have a small, perhaps 1mm wide, branch of the medial plantar nerve crossing the vessels anteriorly to enter through the fascia into the skin of the medial arch of the foot,[15] as shown in **Figures 5, 7-9**. Then the medial and lateral plantar tunnels are identified by inserting a straight clamp into each. The roof of each tunnel, which is the fascial origin of the abductor hallucis brevis muscle, is divided all the way to the plantar surface of the foot. The septum that separates the two tunnels is divided longitudinally from its connection to the calcaneus or to the sheath of the flexor hallucis longus. Additional fibrous bands are released until the surgeon can slide his finger into the plantar aspect of the foot (**Figure 11**)

The one or more branches of the calcaneal nerve are identified, and their tunnel(s) released by dividing the fascia of the abductor hallucis. Finally, the posterior tibial vessels are elevated from the posterior tibial nerve, and the division into the medial and lateral plantar nerves identified. If there is intraneural fibrosis, an intraneural neurolysis is indicated. Post-operatively, the foot is placed into a bulky, supportive dressing (Robert-Jones type) for one week (**Figure 12**). Partial weight bearing is allowed immediately using a walker. This permits mobilization of the nerves within the tunnels, but protects the suture line. Full weight bearing without

Figure 11



Surgeon's finger passes through released medial ankle tunnels and into the plantar aspect of the foot.

Figure 12

The post-op dressing allows immediate post-operative ambulation, permitting the tibial nerve to glide, and preventing post-operative adherence of the tibial nerve to the surgical environment.

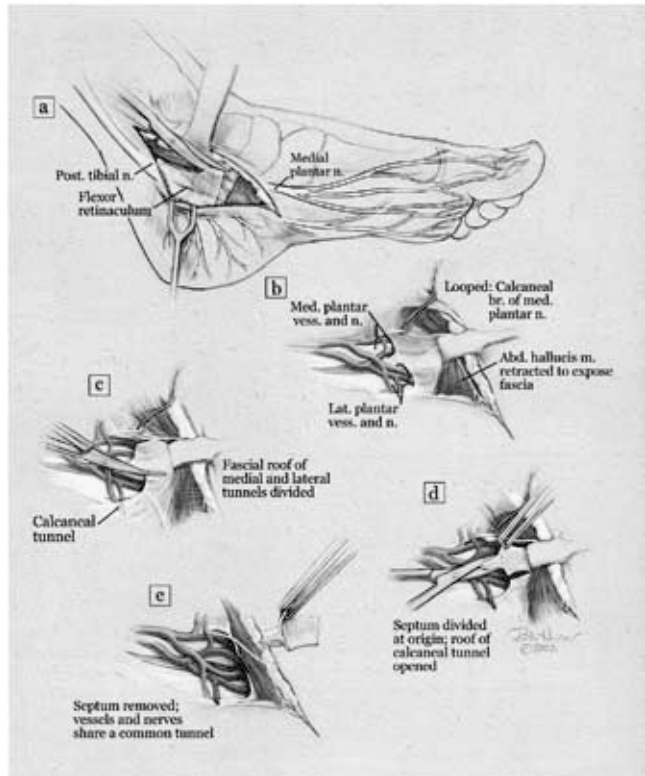
a walker is begun after the ankle sutures are removed on the 21st day. Entire operative approach is given in **Figure 13**.

The results of the decompression of the peripheral nerve were grading traditionally into excellent (no remaining symptoms, able to do all desired activities, no pain medication), good (slight residual numbness and tingling, intermittently, returned to usual occupation, no pain medication), fair (residual muscle wasting and or residual pain requiring medication, not able to work at previous job), and poor (no improvement). The results were also determined using a numerical grading scale[48] given in **Table 4**. Analysis using non-parametric statistics (Wilcoxon's rank sum) was applied to the numerical grading scale to permit evaluation of the improvement possible between different stages of nerve compression and to analyze separately motor from sensory involvement.

Table 4
Numerical grading system[48]
Applied to the distal posterior tibial nerve

Grade	Description
0	Normal
1	Intermittent symptoms of numbness, tingling, paresthesias in toes, "ball" of foot, and/or heel
2	Abnormal vibratory or pressure threshold, mild
3	Increased motor threshold (weakness) abductor hallucis brevis
4	Abnormal vibratory or pressure threshold, moderate
5	Persistent symptoms of numbness, tingling, paresthesias
6	Abnormal static two-point discrimination, mild age: <45 (7-10 mm), >45 (9-12 mm)
7	Muscle atrophy, mild (abductor wasting)
8	Abnormal static two-point discrimination, moderate age: <45(11-15 mm), >45(13-17 mm)
9	Anesthesia (no two-point discrimination)
10	Muscle atrophy, severe (any clawing)

Figure 13



Dellon Approach to Decompression of Four Medial Ankle Tunnels.[49]
Note excision of the septum between the medial and lateral plantar tunnels in "e".

At a mean of 3.2 ± 1.2 years, the results of decompression of the four medial ankle tunnels combined with immediate post-operative ambulation gave an excellent result in 82%, a good result in 11%, a fair result in 5%, and a poor result in 2% of the limbs. The analysis by numerical grading scale demonstrated that with a mean pre-op sensory grade of 8, the mean post-op sensory grade was 0, giving a $P < .001$. With a mean pre-op motor grade of 5, the mean post-op sensory grade was 1, giving a $P < .001$. There was no difference in the results between those patients who had diabetes and those who did not have diabetes.[47]

The present study represents the largest reported series of patients with tarsal tunnel syndrome, and the results are among the best reported outcomes (**Table 5**). When the techniques of previous studies are compared to that reported in the present study, it is clear that previous studies primarily focused upon decompression of just the tarsal tunnel, and do little, if anything, to the structures distal to the tarsal tunnel other than indicating that the medial and lateral plantar nerves are "followed" distally or the fascia of the abductor hallucis brevis is "divided". The results reported here are believed to be improved over those reported in the past due to the conscious decompression of four medial ankle tunnels, including excision of the septum between these two tunnels such that the surgeon's finger can pass into the plantar aspect of the foot. And, as again can be seen in **Table 5**, the inclusion of ambulation early after surgery.

The optimum evaluation technique to determine the success of treatment of tarsal tunnels syndrome is not clear. Some studies determined improvement by a better (lower, decreased distance) in two-point static-touch, and this has a basis in improvement in innervation density, and reversal of the nerve compression. Some studies have talked about improvement in pain, and yet typically chronic nerve compressions, such as carpal tunnel syndrome, have numbness and paresthesias, but not pain, which is usually a sign of acute nerve compression or neuropathy. Baille and Kelikian attempted to add a symptom score and a functional foot outcome score to their evaluation system.[42] They demonstrated that each of these numerical scoring systems permitted statistical analysis, that there was a statistically significant improvement in each of these in their patients, and that these scores correlated with a patients satisfaction questionnaire. This is important information, however, neither of these scoring systems has been validated for use with the foot. In addition to the traditional evaluation system used by surgeons in the past, the present study used a numerical scoring system based upon the known pathophysiologic effects of chronic nerve compression upon peripheral nerve sensory and motor function.[48,50-52] This numerical grading system has been reported previously for evaluating the results of the surgical treatment of recurrent carpal tunnel syndrome[53] and the non-operative treatment of cubital tunnel syndrome.[54] The advantage of such a numerical scoring system is that it permits non-parametric statistical evaluation of the results of peripheral nerve surgery. A similar approach was taken by Takakura et al in 1991 in the evaluation of their results.[55] The

Table 5
Summary of "therapeutic level 4" tarsal tunnel studies

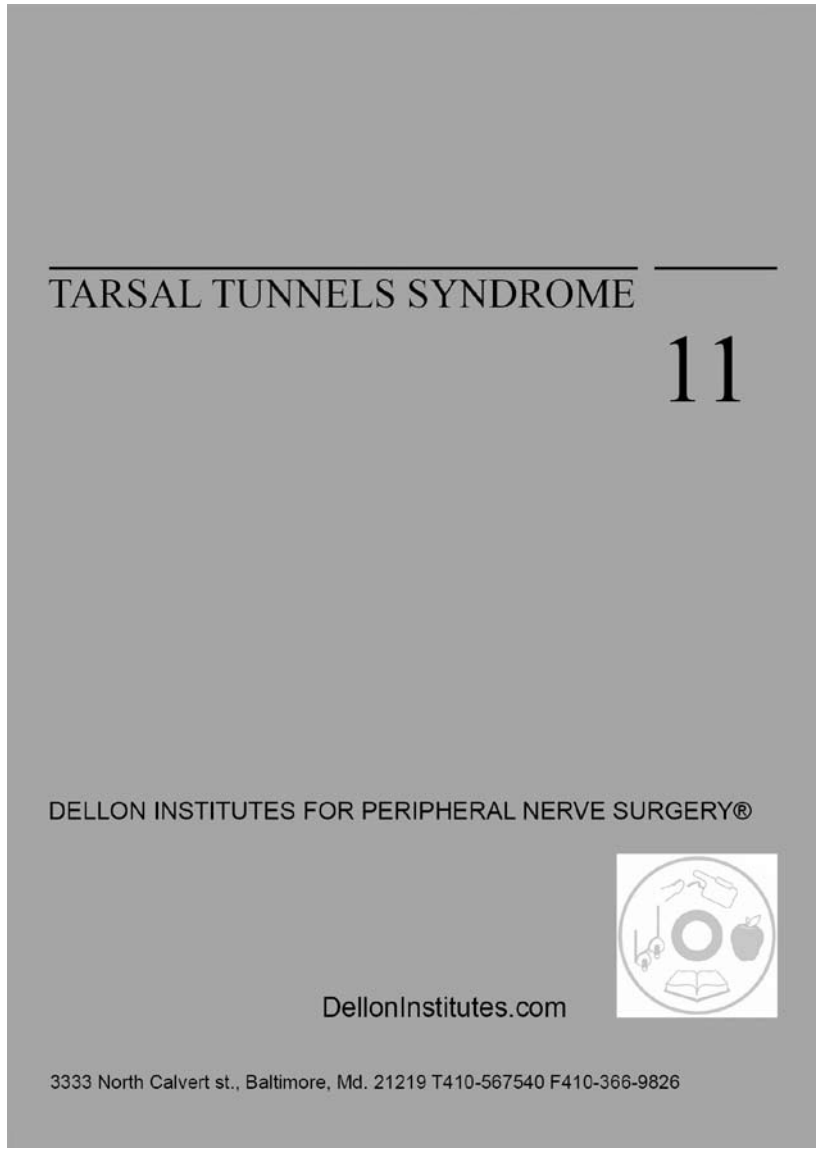
Study	Number of patients	NCV EMG*	Tinel Positive	Tunnels Released	Immobilized Post-Op	Results (%) Exc	Good	Fair	Poor
1967, Lam[31]	13	no	yes	3?	NA	86	7	7	0
1969, Edwards et al[32]	16	33%	yes	1	cast, ? time	88	6	0	6***
1970, Linscheid et al[33]	24	100%	86%	1	NA	0	50	30	20
1974, Mann[34]	9	90%	yes	3	3 weeks	78	0	11	11
1989, Stern,Joyce[35]	15	40%	yes	4	10 dys	54	20	20	6
1992, Byank,Curtis[36]	49	100%	yes	1	NA	26	53	12	9
1993, Sammarco et al[37]	5	100%	yes	4	NA	20	80	0	0
1994, Pfeiffer,Cracchiolo[38]	32	100%	yes	3	10 dys	15	29	23	33
1996, Mahan et al[39]	45	NA	yes	3	NA	24	36	11	29
1997, Turan et al[40]	18	0%	yes	4	2 weeks	61	22	0	17
1997, Baba, et al[41]	34	100%	yes	3	3 weeks	70	16	8	6
1998 Bailie,Kelitian[42]	36	80%	yes	3§§	2 weeks	57	16	11	16
2000, Kohno et al**[43]	12	40%	yes	1	NA	50	34	8	8
2003, Gondring et al**[43]	68	100%	yes	2?	3 weeks	51§	0	0	49
2005, Mullick/Dellon[47]	87	50%	yes	4+	none	92	11	5	2

* percentage of patients in series who did have electrodiagnostic testing.
For Pfeiffer/Cracchiolo series, 81% were positive. For Linscheid, et al, 68% were positive.
For Baile and Kelikian, 81% were positive.
In none of these studies did the NCV/EMG result correlate with surgical outcome.
** a pneumatic tourniquet was not used . *** follow-up was just 3 to 4 months.
§ outcome: patient reported improvement. For surgeon reported pain relief, it was 85% relief of pain and 15% not relieved of pain. §§ the inter-tunnel septum was excised

basis of their system however was not pathophysiology, but rather upon “spontaneous pain, pain on movement, burning pain, Tinel’s sign, sensory disturbance, and muscular atrophy or weakness.” Since they did not give a percentage of their patients improved, their data could not be included in **Table 5**. A combination of the traditional method and a numerical scoring system based upon pathophysiology seems to be the best approach, permitting a reader to know that the improvement is statistically significant as well as knowing what percentage of the patients are improved. Adding a validated outcome questionnaire would add a final dimension to the results reporting.

Finally, decompression of just the tarsal tunnel assumes that this is the anatomic location of the site of pressure that is causing the symptoms. At present there is just one study of the pressure within the tarsal tunnel, done in cadavers.[56] That study demonstrated the pressure to be 2 ± 1 mmHg in neutral ankle position, to increase in eversion to 32 ± 5 mmHg and in inversion to 17 ± 5 mmHg ($P < .005$ and $< .05$ respectively, but no significant difference between eversion and inversion). Tarsal tunnel syndrome is considered a chronic nerve compression, and yet in the first two reported cases, each patient had an acute increase in pressure, one related to forced marches during the first weeks of military training[1] and the second related to working as a “docker”.[2] At surgical exploration, dilated veins were identified in the first, and nothing identified in the second patient, though the posterior tibial nerve was noted to be “fusiform” in each case, and the authors introduced the analogy to the median nerve in the carpal tunnel. It remains for a study to demonstrate in cadavers, and then in humans intra-operatively, what the pressure measurements are in the medial and lateral plantar, and calcaneal tunnels. Perhaps this clinical condition now should be called “Tarsal Tunnels Syndrome”. This is exactly what I have called our new patient information brochure (**Figure 14**).[57]

Figure 14



New Tarsal Tunnels Syndrome brochure[57] of the Dellon Institutes for Peripheral Nerve Surgery® uses a new name that reflects the four tunnels to decompress.

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Chapter 4

First lower extremity decompressions for neuropathy

Based on

Dellon AL, Treatment of symptoms of diabetic neuropathy by peripheral nerve decompression.
Plast Reconstr Surg 89:689-697, 1992

Dellon AL, A numerical grading scale for peripheral nerve function.
J Hand Ther 6:152-160, 1993

FIRST LOWER EXTREMITY DECOMPRESSIONS FOR NEUROPATHY

Getting started

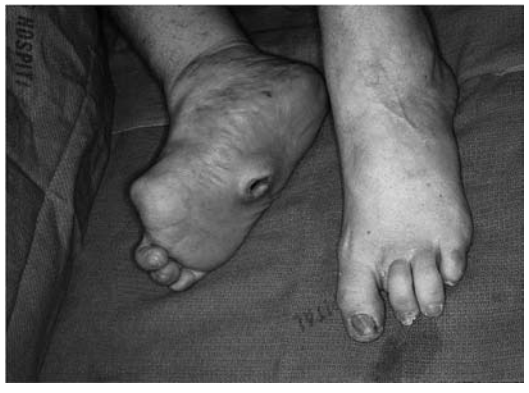
I began a prospective study in 1982 that would not appear in print until 1992.[1] Included were upper extremity nerve decompressions as well as the lower extremity nerve decompressions. The approach that I used for the lower extremity nerve decompressions included the technical aspects covered in Chapter 2 and 3. This first group of patients had the surgical concept come from the insight discussed in Chapter 1, that if a patient with neuropathy could obtain relief of symptoms for nerve decompressions in the hand then there was the possibility that they could obtain relief of symptoms by decompressing nerves in their legs. But how to find the first patients!

It was difficult for me, as a Plastic Surgeon, to have a group of patients for lower extremity peripheral nerve decompressions. Patients are typically referred to a Plastic Surgeon if they were a diabetic only if they needed to have an infection drained, a wound débrided or closed, or to have an amputation. A typical patient is noted in **Figure 1**.

I did get started therefore by doing the lower extremity peripheral nerve decompressions in some patients who were coming to surgery to get their wounds treated. For example, the woman with the Charcot foot and plantar ulcer, noted in **Figure 1**, required resection of the cuboid bone, muscle flap rotation, and skin graft to obtain wound closure. At the same time, I did a neurolysis of the tibial nerve in the four medial ankle tunnels. My expectation was just to recover protective sensibility. In **Figure 2** her recovery of sensibility has charted on the plantar surface of her foot, 8 months post-op.

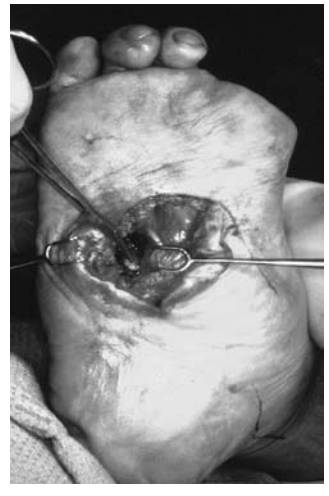
A second patient that provided early encouragement to me was the patient in **Figure 3**. He was referred for debridement and grafting of the dorsal foot wound. At the same surgery, I did a neurolysis of the tibial nerve in the four medial ankle tunnels, and removed the septum between the medial and lateral plantar tunnels. The skin graft took, and in time protective sensibility returned. At three years after the skin grafting, he had no new ulcerations on that foot as demonstrated in **Figure 3** at the bottom.

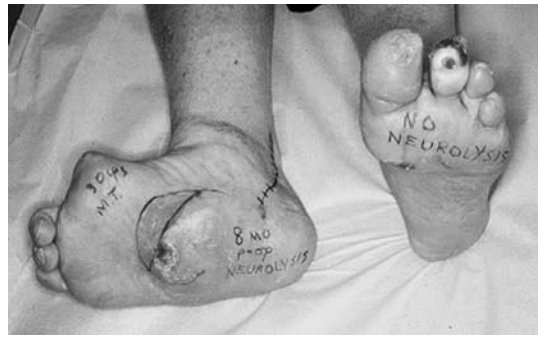
Figure 1



Typical patient referred to a Plastic Surgeon requires drainage for infection, debridement and wound closure, or amputation. They are not referred to restore sensation or prevent ulceration.

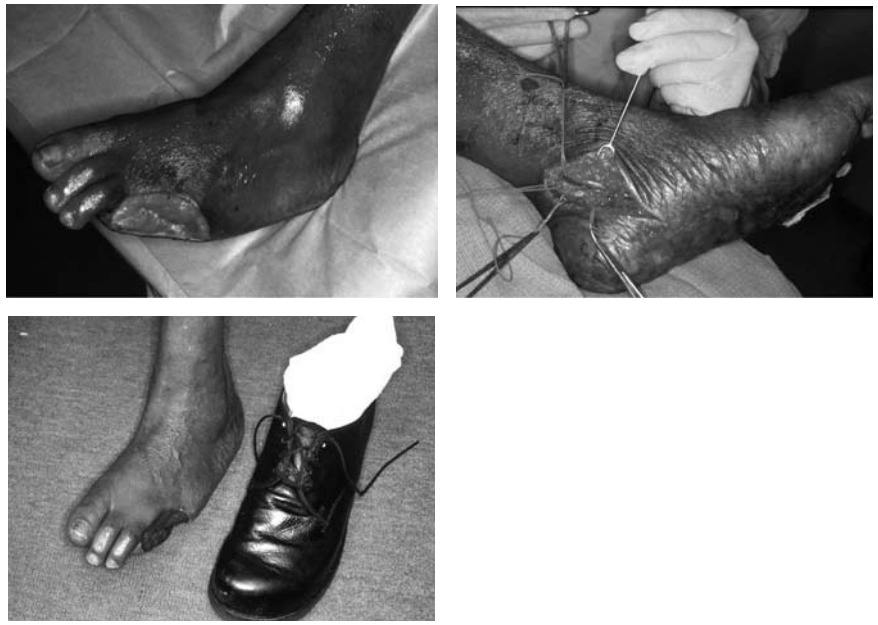
Figure 2





Patient from Figure 1. Left, at surgery, after debridement, after flap rotation. Right: Eight months after wound closure and neurolysis of the tibial nerve in the four medial ankle tunnels. Possibly one of my first patients to have this procedure. She is anesthetic in both feet. Note that perception of moving touch stimuli and perception of the 30 Hz tuning fork has regenerated to the stump of the hallux, consistent with a rate of neural regeneration of 1 mm per day. This woman was 45 at the time of this photo, and a non-compliant type I diabetic for 30 years. Note ulcerations continue in the left , non-operated foot. There are no new ulcers on the right.

Figure 3



Patient with anesthetic foot referred for wound closure of the wound on the Left. On the right, at the time of debridement and skin grafting of the dorsal wound, a neurolysis of the four medial ankle tunnels and excision of the inter-tunnel septum was done to restore protective sensation. Below, at three years after surgery, the skin remains, no other ulcers have occurred, protective sensibility has been restored and he walks with normal fitted shoes.

THE CLASSIC 1992 ABSTRACT

"Symptomatic diabetic sensorimotor polyneuropathy is considered progressive and irreversible. The hypothesis that symptoms of diabetic neuropathy may be due to entrapment of peripheral nerves was investigated in a prospective study from 1982 to 1988 in which diabetics (38 type I, 22 type II) had surgical decompression of 154 peripheral nerves in 51 upper extremities and 31 lower extremities. Mean postoperative follow-up was 30 months (range 6 to 83 months). Considering the entire series, an excellent final result was noted for motor function in 44 percent and for sensory function in 67 percent of the decompressed nerves. Ten percent of the patients were not improved, and 2 percent were worse in sensorimotor

function. Upper extremity nerve decompressions achieved better results than lower extremity nerve decompressions. Improvement in postoperative electrodiagnostic studies varied in relationship to the preoperative electrodiagnosis. Improvement was noted in 100 percent of those nerves with the preoperative diagnosis of "localized entrapment," 80 percent for "peripheral neuropathy with superimposed entrapment," and 50 percent for "peripheral neuropathy." Progressive neuropathy occurred in a non-treated limb of 50 percent of those patients whose surgically treated limb maintained improvement. The results of this study suggest that symptoms of sensorimotor diabetic neuropathy may be due partly to compression of multiple peripheral nerves. The results further suggest that surgical decompression of such nerves may result in symptomatic improvement."[1]

SELECTION OF THE FIRST GROUP OF PATIENTS

During the beginning of my work with diabetics with symptoms of neuropathy, I would evaluate each patient for upper and lower extremity superimposed peripheral nerve compressions. It was clear to me that clinically traditional electrodiagnostic nerve conduction and electromyographic studies could not identify superimposed nerve compressions easily in this patient population. In deed, these tests have trouble identifying these nerve compressions in patients without neuropathy, and this subject is treated more extensively in the Discussion, Chapter 10. It was also clear to me, however, that a) many patients already had these tests done prior to seeing me for a consultation, and b) doctors reviewing my initial work would ask for "objective" proof, i.e., electrodiagnostic studies, to prove the patient's original condition as well as to document improvement. For these reasons, I decided to include as much information as possible in this first study on electrodiagnostic testing as was available. It was also clear to me that I needed to have a population of patients with a criteria clinically that could be used to identify the presence of a localized site of nerve compression. As a Hand Surgeon, the presence of a positive Tinel sign was extremely reliable for me in predicting success for a proposed surgical decompression. If the nerve in a patient with neuropathy had a localizing sign, such as a Tinel sign, over a known site of anatomic narrowing, that for me would constitute an inclusion criteria for operating on the nerve at that site. An exclusion criteria for the lower extremity patients was a history of previous ulceration or amputation, as I wanted to see what could be achieved in patients who were not as far advanced as the few I started with, as exemplified in **Figures 1 and 2**.

Table 1
Electrodiagnostic studies: pre-op diagnoses

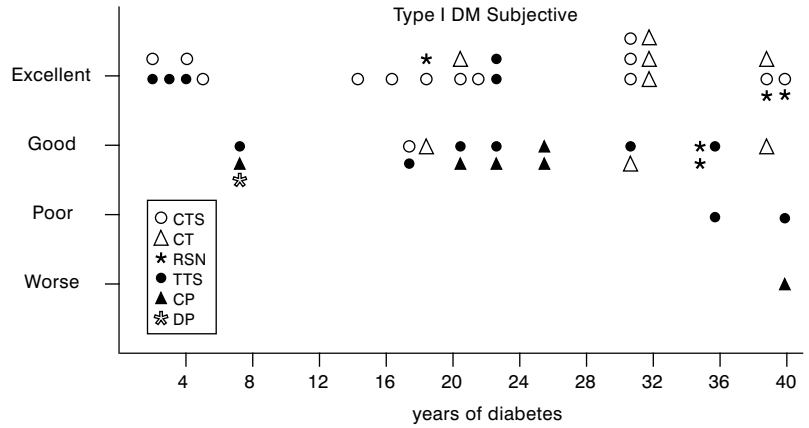
Pre-operative diagnosis	percent
Normal	8%
Localized Nerve Compression	11%
Neuropathy with Superimposed Nerve Compression	43%
Diffuse Neuropathy	38%

The first cohort of patients included both Type I (38) and Type II patients (22) diabetics, whose history of diabetes varied from 5 to 41 years in length. It was clear that type II diabetics did not always have a clear starting point for their disease. These 60 patients included a mean age for the Type I diabetics of 44.9 and for the Type II diabetics of 48.6 years. The age range in the series was 38 to 74 years. The breakdown into upper versus lower extremity patients for surgery was that 51 patients had an upper extremity and 31 patients had a lower extremity nerve decompression. Therefore, some patients had both an upper and a lower extremity peripheral nerve decompression. The total number of different nerves decompressed was 154, each with a positive Tinel sign, so that there were patients who had multiple peripheral nerves decompressed simultaneously in upper as well as lower extremities. With respect to electrodiagnostic studies, 94% of patients had a pre-operative test. Of these 80% had a post-operative test. **Table 1** has the stratification of pre-operative electrodiagnostic results.

RESULTS IN DETAIL

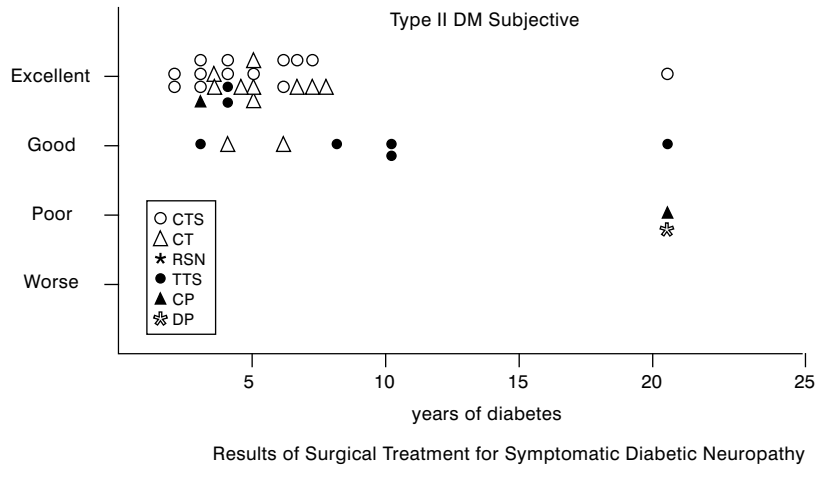
The results for each patient in this study were evaluated in terms of sensory recovery, motor recovery, and also by asking the patient if they thought they were improved, which today would be considered a legitimate outcome measure. The results of the outcome survey for each patient is given in **Figure 4** for the Type I diabetics, and **Figure 5** for the Type II diabetics. Follow-up after surgery was a mean of 30 months, range 6 to 83 months.

Figure 4



Outcome study of nerve decompression for each Type I diabetic. The *y*-axis is the number of years of diabetes, and not the length of follow-up. Note that some patients had neuropathy at the time of diagnosis. Each color represents a different nerve decompression site. CTS: carpal tunnel, CT: cubital tunnel, RSN: radial sensory nerve, TTS: tarsal tunnel, CP; common peroneal nerve, DP; deep peroneal nerve

Figure 5



Outcome study of nerve decompression for each Type II diabetic. The y-axis is the number of years of diabetes, and not the length of follow-up. Note that some patients had neuropathy at the time of diagnosis. Each color represents a different nerve decompression site. CTS: carpal tunnel, CT: cubital tunnel, RSN: radial sensory nerve, TTS: tarsal tunnel, CP; common peroneal nerve, DP; deep peroneal nerve

Table 2
Electrodiagnostic studies: post-operatively

Pre-operative ncv/emg diagnosis	improved post-operatively by electrodiagnostic testing
Localized Nerve Compression	100 %
Neuropathy with Superimposed Nerve Compression	80 %
Diffuse Neuropathy	50 %

Overall, 88% of patients were improved, 10% were not improved and 2% were made worse. The 2 patients who were worse included one man whose touch perception improved in terms of two-point discrimination but who had such painful nerve regeneration that he insisted he had been made worse by the surgery. The second patient, who was among the first patients to have a common peroneal nerve procedure by me, lost motor function related to the foot everters. This was because I slid my finger alongside the superficial peroneal nerve into the lateral compartment, injuring a small nerve branch. She had diabetes for 41 years (note the red triangle in **Figure 4** under "worse"). (There have since been no motor nerve downgrading in any of my patients since that patient.)

No patient had progressive neuropathy in any of the limbs operated upon. In contrast, 50% of patients had progressive neuropathy in a contralateral, non-operated, extremity. (This was the first suggestion that peripheral nerve decompression could change the natural history of peripheral neuropathy.)

No patient developed an ulceration or amputation or wound infection during the post-operative follow-up period in the leg that had a nerve decompression.

CONCLUSIONS FROM THIS EARLY EXPERIENCE

This was a difficult research project to get published. It was sequentially rejected from three leading medical journals, a process which delayed publication for several years. The medical journals are not surgical journals. A Plastic Surgeon, which is what I am, has little credibility as a basic scientist or even as a surgical scientist.

Table 3
Diabetic neuropathy results
Peripheral nerve decompression

Median nerve: carpal tunnel syndrome

number of:		subjective results:		two-point discrim.:		recurrence
Nerves	Patients	excellent	good	excellent	good	
44	34	96%	2%	96%	4%	0%

Ulnar nerve: cubital tunnel syndrome

number of:		subjective results:		two-point discrim.:		strength	
Nerves	Patients	excellent	good	excellent	good	excellent	good
11	8	82%	18%	82%	9%	54%	36%

Posterior tibial nerve: tarsal tunnel syndrome

number of:		pre-operative:		results:	recurrent
Nerves	Patients	Ulcers	Amput	Improved	Ulceration
31	22	0	0	Pain 85% 2PD 72 %	0%

Reviewers of this manuscript repeatedly asked me questions like: "What is the evidence the patient is a diabetic?", "What was the mean fasting glucose, and HbA1?", "What is the evidence the patient's have neuropathy?" Clearly the results of this study that demonstrated improvements in symptoms in patients with diabetic neuropathy was a challenge to traditional teaching in medicine.

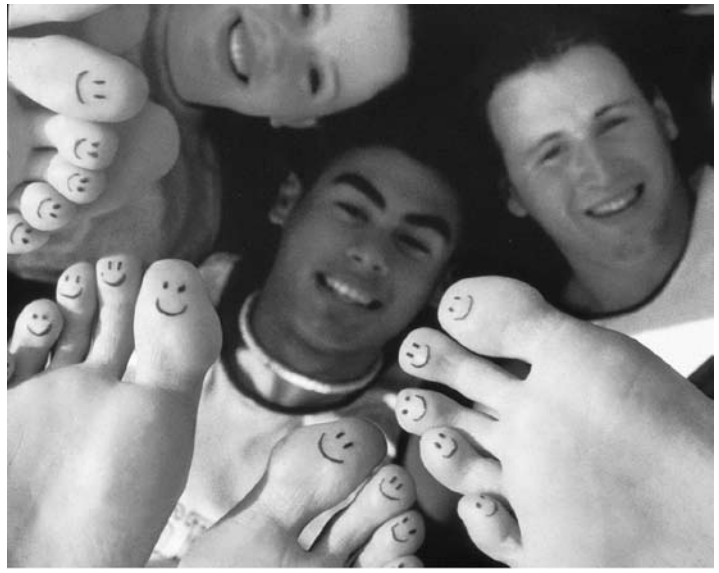
This study demonstrated that symptoms of neuropathy in a patient with diabetes mellitus, who can be shown to have a superimposed nerve compression, can be improved by decompression of the entrapped peripheral nerves. This study demonstrated that in each extremity there could be more than one entrapped nerve. This study demonstrated that in the limbs with a nerve decompression, progres-

sive neuropathy did not occur, whereas, during the period of post-operative observation, a mean of 30 months, 50% of the contralateral, non-operated extremities, did have progression of neuropathy.

The main criticism of this study was that it was single authored by the surgeon who did the surgery, and who, therefore, was clearly biased. Given even this criticism, 78% of those patients with post-operative, objective, electrodiagnostic testing, were improved.

Based upon this first clinical study, which was based upon anatomic studies discussed in Chapters 2 and 3 related to peripheral nerve anatomy and entrapment sites, it was necessary to go into the basic science laboratory to develop models for chronic nerve compression, models for diabetic neuropathy, models for the double crush syndrome, and thereby form the metabolic and neurophysiologic basis for these clinical observations. It is that part of the story that is described in Chapter 5. The clinically successful outcomes that followed this “detour” into to laboratory will be described in Chapters 6, 7, 8 and 9. International confirmation of this work followed, and is described in Chapter 10.

Figure 6



Symptoms of neuropathy can be relieved by decompression of peripheral nerves in the lower extremity. This can prevent ulceration and amputation.

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Chapter 5

Modeling chronic compression in diabetic nerves

Based on

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Dellon AL, Mackinnon SE, Seiler WA IV, Susceptibility of the diabetic nerve to chronic compression.
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THE NERVE COMPRESSION MODEL

The first attempt to treat an ulnar nerve compression at the elbow was by B.F. Curtis, a subcutaneous transposition, in 1874,[1] with the first carpal tunnel being compressed for the median nerve possibly as early as 1924.[2] Yet most aspects of chronic nerve compression have been taught traditionally based upon the experience of the previous surgeon. An experimental model of chronic nerve compression did not exist. The problem with the treatment ulnar nerve entrapment is a good example, in which today we are still without a prospective randomized clinical study to support the use of one of the seven currently used operative approaches **(Table 1)**

The first meta-analysis of the results of ulnar nerve decompression at the elbow documented that if you stage the degree of compression, results varied significantly according to the degree of compression, and that the failure or recurrence for any technique with a severe degree of ulnar nerve compression approached 33%.[3] A recent meta-analysis of this same problem came to the same conclusion.[4] The only experimental model, in which intraneural pressures were measured, in fresh cadavers, demonstrated that only the anterior submuscular transposition by Dellon's musculofascial lengthening technique reduced the pressure on the ulnar nerve in all degrees of elbow flexion along three points of the ulnar nerve.[5] Dellon's technique has been documented recently.[6,7] This highlights the need to have an experimental model for chronic nerve compression.

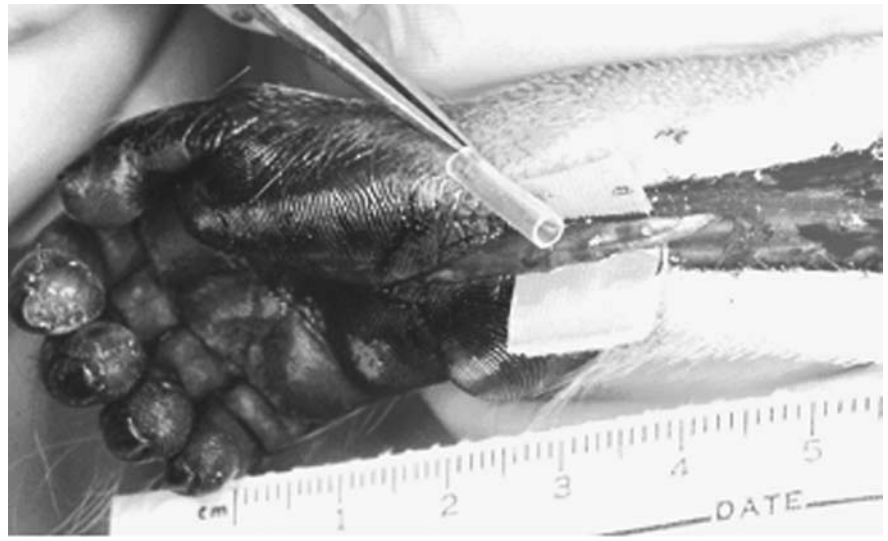
Beginning in 1982, during the time that Susan E. Mackinnon, MD, was a Fellow at

Table 1

Decompression In Situ
Open
Endoscopic
Anterior Transposition
Subcutaneous
Intramuscular
Submuscular (traditional Learmonth)
Submuscular (Dellon fascial lengthening)
Medial Epicondylectomy

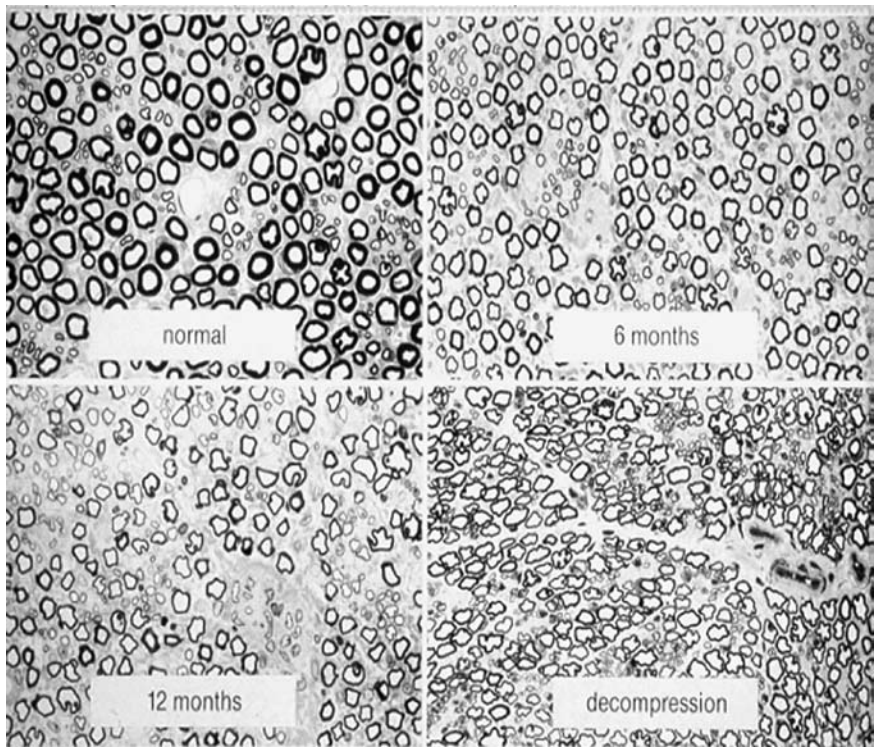
the Raymond M. Curtis, MD Hand Center at Union Memorial Hospital in Baltimore, she and I worked in the basic science area to develop a model of chronic nerve compression. The only previous work had utilized a ligature about a nerve, which created a high pressure applied to a short interval of nerve. This was a model of acute nerve compression. In humans, the peripheral nerve is compressed along a length that is relative long with respect to its width, and the condition of chronic nerve compression can exist for many months through years before the patient seeks help. Therefore we constructed a model that would a use a silicone tube of about two to three times the in length of the width of the nerve in diameter, and the tube would be placed just to fit, but not to directly compress the nerve. This model was first begun in the rat,[8,9] and then progressed to the subhuman primate, the monkey.[10] A summary slide from the work in primates is given in **Figure 1a and 1b** that demonstrates the model and the critical results.

Figure 1a



Primate model for chronic nerve compression. Above, a silicone tube, twice the diameter of the median nerve, is going to be slipped around the median nerve to creat the site for chronic compression. The histology of the median nerve is below for the time frames 6 months and 12 months, during which progressive thinning of myelin can be seen compared to normal. The results of treatment can be observed after 3 months after removing the silicone from the median nerve. The loss of fibers seen at 12 months is reversed.[10]

Figure 1b



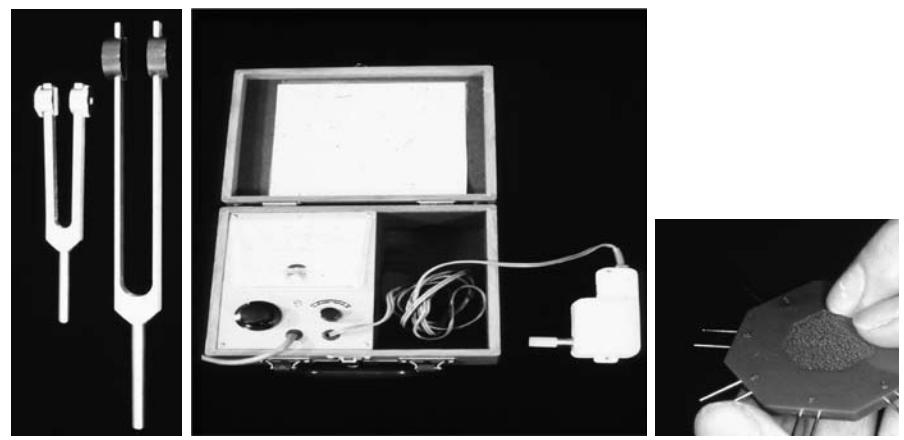
With the development of a reproducible model, it was possible to describe the pathophysiology of chronic nerve compression. After two months of compression in the rat model, there was a breakdown of the blood nerve barrier, as demonstrated by the leakage of Evan's blue albumin dye from the intravascular space into the endoneurial space.[8] This would increase intraneural pressure. By six months of compression in this model, myelin thinning began.[9,10] By 12 months of compression in this model, there was a drop out, or loss of large myelinated fibers.[9,10] This experimental histopathology was confirmed in three examples of chronic human nerve compression: the tibial nerve in the tarsal tunnel,[11] radial sensory nerve in the forearm,[12] and the ulnar nerve in the cubital tunnel.[13]

CONCLUSIONS FROM THE EXPERIMENTAL MODEL OF COMPRESSION

With an experimental model of chronic nerve compression [8-10] and confirmatory clinical histopathology,[11-13] it was possible to investigate therapeutic options, such as the effectiveness and safety of microsurgical internal neurolysis.[14-16]

It was concluded that microsurgical internal neurolysis was safe in a region of already scarred nerve,[15,16] that it did not create new intraneural scar tissue postoperatively, that it improved histopathology in the rat model [15] but not in the primate model.[16] From the combined experimental and human studies, conclusions could be drawn with regard to the physical examination of the patient with chronic nerve compression:[17,18] Chronic nerve compression progresses overtime. The type of instruments needed to measure this change would require ability to measure threshold changes for the large myelinated fibers in the early stages, and threshold plus innervation density changes in the latter stages. At the time of these studies of chronic nerve compression, the only available instruments to measure the large myelinated fibers' threshold changes for stimulation were the non-quantitative tuning fork [19] and the quantitative vibrometer [20] to measure vibratory threshold, and the nylon monofilament to estimate the pressure threshold. At the time of these studies of chronic nerve compression, the only available instrument to measure the large myelinated fibers' innervation density was the Disk-Criminator™.[21] **(Figure 2)**

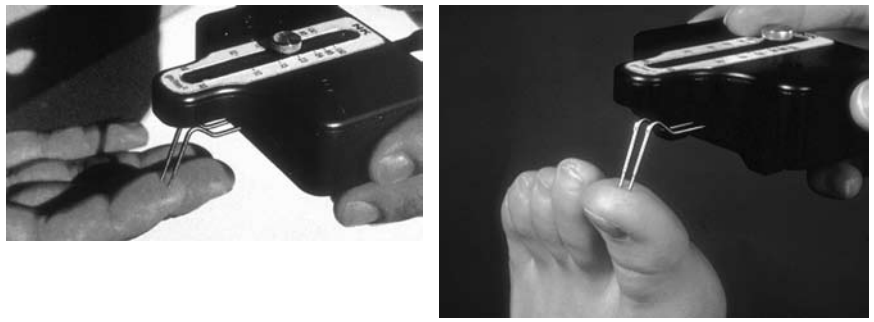
Figure 2



A B C

Prior to 1990, the only instruments available to measure the progressive changes of chronic nerve compression upon the large myelinated fibers for touch were the A) tuning forks and B) vibrometer, and C) the Disk-Criminator™.

Figure 3



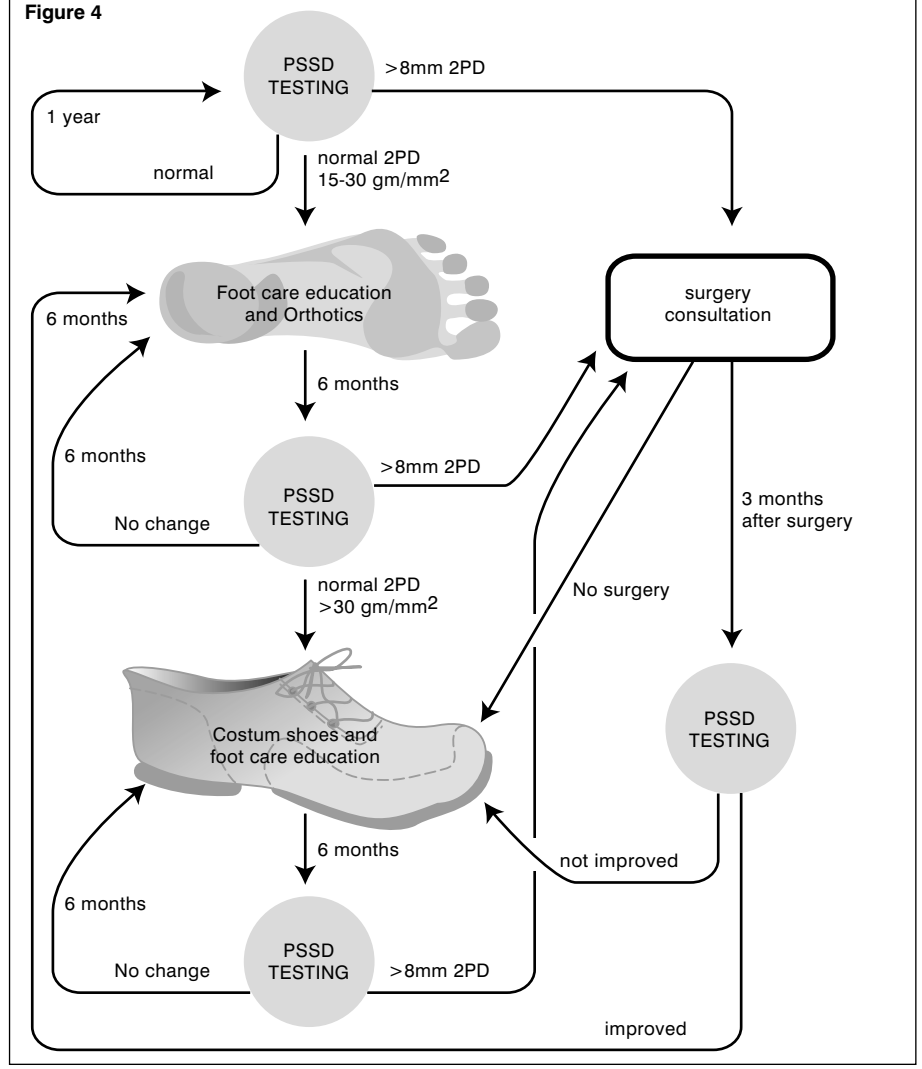
The Pressure-Specified Sensory Device™ was invented to provide the ability to measure the changes in sensibility throughout the range of histopathology created by chronic nerve compression. It can be used to measure the sensibility of any piece of skin.

The implication of the histopathology of chronic nerve compression was that there needed to be an instrument that could measure the thresholds for both the pressure of application of the stimulus and the distance between the prongs that applied the pressure stimulus. Such an instrument did not exist. While the vibrometer could give a measure of stimulus intensity, its sinusoidal waveform stimulated skin at a distance, so that if the thumb were being tested the sensation would be transmitted through both the radial and the median nerve for the thumb and through both the peroneal and the tibial nerve for the big toe. A new instrument was therefore invented to provide this need, The Pressure-Specified Sensory Device™. It was invented by myself and an aerospace engineer, Nebosja Kovocivic, PhD. He created a device to meet my specifications **(Figure 3)**

When the first diabetics were evaluated in the first study described in Chapter 4, the only instruments for their evaluation were those in **Figure 2**. Success could be measured only with regard to percentage of patients improved, but without numerical evaluations, statistical analysis was difficult.

The Pressure-Specified Sensory Device™ has now been demonstrated to be of value in the measurement and documentation of both upper and lower extremity chronic nerve compression for both post-traumatic and neuropathic causes of patient symptoms.[22-26] This enables the development of a numerical grading scale to document and stage the degree of nerve compression and neuropathy.[27] An example of this type of staging system for the tibial nerve at the ankle level is given in **Table 2**. Based upon these measurements, it is possible to create an algorithm for management of the patient with diabetic neuropathy as in **Figure 5**.

Figure 4



Algorithm for neurosensory testing with the Pressure-Specified Sensory Device™ in patients with neuropathy. Yearly testing is advised for all diabetics. Once neuropathy is diagnosed, the testing is every 6 months. When neuropathy progresses but before axonal loss, then special shoes are advocated. Once there is evidence of neural degeneration, then a surgical consultation is advised to consider nerve decompression.

Table 2
Numerical grading scale for the tibial nerve at ankle

Numerical score		Description of impairment
Sensory	**motor**	
0		no symptoms
1		paresthesia, intermittent
2		abnormal pressure threshold, (Pressure-Specified Sensory Device™) < 45 years old; ≤ 6.3mm, at 6.8-30 gm/mm^2 ≥ 45 years old; ≤ 8.3mm, at 25-40 gm/mm^2
	3	weakness, abductor hallucis
4		abnormal pressure threshold, (Pressure-Specified Sensory Device™) < 45 years old; ≤ 6.3mm, at > 30.1 gm/mm^2 ≥ 45 years old; ≤ 8.3mm, at > 40.1 gm/mm^2
5		paresthesias, persistent
6		abnormal innervation density (Pressure-Specified Sensory Device™) < 45 years old; ≥ 6.3mm < 10mm, at any gm/mm^2 > 45 years old; ≥ 8.3mm < 11mm, at any gm/mm^2
	7	muscle wasting or clawing (1+ or 2+ of 4+)
8		abnormal innervation density (Pressure-Specified Sensory Device™) < 45 years old; ≥ 10.1mm, at any gm/mm^2 ≥ 45 years old; ≥ 11.1mm, at any gm/mm^2
9		anesthesia
	10	muscle wasting, clawing (3+ or 4+ of 4+)

SUSCEPTIBILLITY OF THE DIABETIC NERVE TO COMPRESSION

It is clear and accepted now that the patient with diabetes will have an increase in the amount of chronic nerve compressions compared to the non-diabetic. In Chapter 1, in Table 2, a series of patients with nerve compression were reviewed

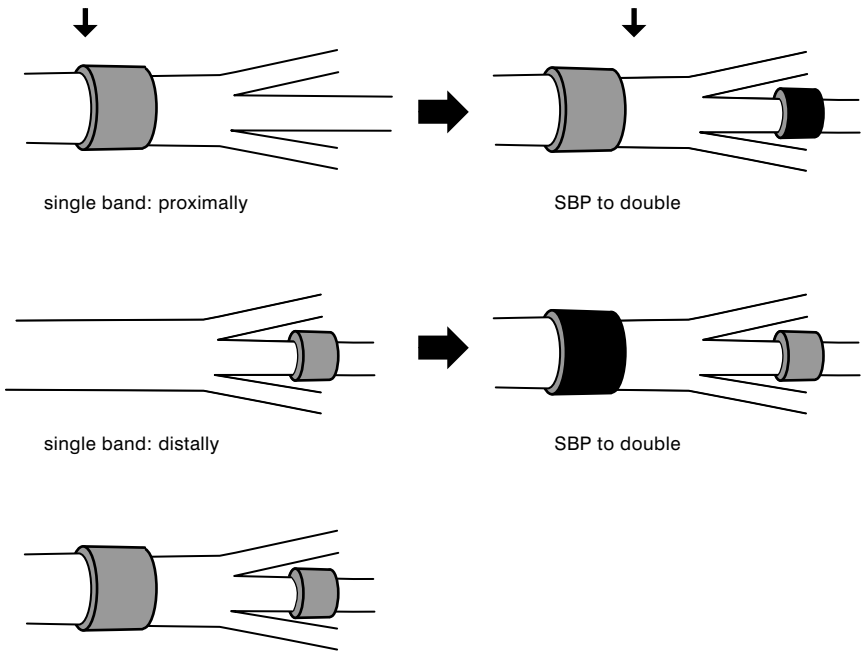
with the percentages of those patients having diabetes ranging from 23 to 40% of the total population of patients with nerve compression.[28-33] A paper that makes this perfectly clear is one based upon a Canadian population of patients:[34] The non-diabetic population had an prevalence of carpal tunnel syndrome of 2%, the population of diabetics without neuropathy had an prevalence of 14%, and the population of diabetics with neuropathy had a prevalence of 30% carpal tunnel syndrome. The question then becomes, why do diabetics have a susceptibility to chronic nerve compression?

THE DOUBLE CRUSH HYPOTHESIS

In 1973, Upton and McComas, hypothesized, based upon their clinical observations, that a proximal site of nerve compression makes the person more likely to have another chronic nerve compression more distally along that same nerve.[35] The observed the common occurrence of carpal tunnel syndrome and a C6 nerve root compression. They observed the common occurrence of cubital tunnel syndrome and patients with compression of the lower trunk of the brachial plexus. From these observations they finally hypothesized that perhaps even the presence of a metabolic neuropathy would act as the “proximal crush”, and make that patient more susceptible to nerve compressions in the extremities. Their postulated mechanism for the double crush phenomenon was a proximal decrease in axoplasmic flow. Two brief reports on the association of brachial plexus compression and carpal tunnel syndrome had been recorded earlier in the British literature.[36,37] This implied that while neither crush in and of itself might be sufficient to create clinical symptoms, the two would have an additive effect that would summate to give symptoms. To me this implied further that perhaps not all sites had to be decompressed to relieve symptoms and that this hypothesis might be the basis of my clinical observations (see Chapter 1 and 4) of patients with diabetic neuropathy and their superimposed nerve compressions.

As a good hypothesis is likely to do, the Upton and McComas double crush concept was investigated in several animal models by Gilliat’s group in England, and they did confirm in their models, which were models of acute compression related to creating a constriction about the nerve, that a proximal constriction did predispose to distal peripheral nerve problems.[38-40] Refinements in peripheral nerve compression were then evaluated by Lundborg’s group in Sweden, showed directly that small amounts of pressure applied to a peripheral nerve would create in-

Figure 5



Experimental model of the double crush hypothesis using bands to create chronic compression in the rat sciatic nerve model. This was the first experimental study to confirm the double crush hypothesis as it relates to chronic nerve compression.[51]

creased intraneural pressure, decreased blood flow, and decreased axoplasmic flow.[41-44] With time, other experimental models were created,[45] and many clinical observations were repeated on the association of cervical disc and brachial plexus compression with carpal tunnel syndrome.[46-50]

In 1983, during the same time period that Lundborg's group was using the application of direct pressure to a peripheral nerve and looking at blood flow, our group applied the model of chronic nerve compression described above [8,9] to the rat sciatic nerve model to the double crush concept. Our original silicon tube placed about the sciatic nerve acted as the first crush, and now a second silicon tube could be placed about the tibial nerve distally, with function being measured electrophysiologically.[51] We compared a single crush proximally or distally to two crush sites placed simultaneously and two a two sites placed at different times **(Figure 5)**.

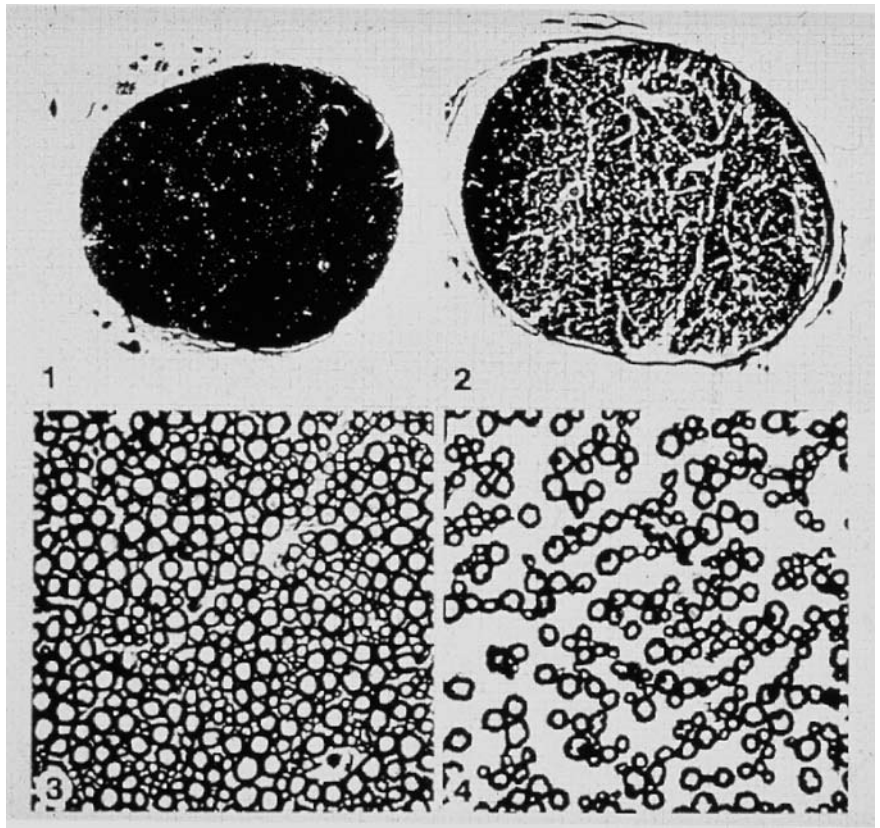
We demonstrated that the proximal site of compression does make the distal nerve more susceptible to a second site of compression, thereby proving the double crush hypothesis for the first time in a model of chronic compression.

It remained now just to apply this double crush model from the normal rat to the diabetic rat. Rats were made diabetic with an intraperitoneal injection of 60 mg of streptozotocin. Their blood sugar reaches 400 mg/dl by the third week. At that time an experiment similar to the one just described was done in diabetic and non-diabetic rats. That study demonstrated that the diabetic banded rat was statistically significantly more likely to develop a worse degree of electrical function of the sciatic and tibial nerves than the non-banded diabetic rat, and then the banded and non-banded non-diabetic rat.[52]

OPTIMISM IN DIABETIC NEUROPATHY

In 1988, I wrote an article published in the Annals of Plastic Surgery entitled "Optimism in Diabetic Neuropathy."[53] This article was the first to articulate my hypothesis that the underlying metabolic problems in diabetes mellitus created the environment in which the peripheral nerves in the upper and lower extremities would be likely to become compressed at the multiple sites of known anatomic narrowing, like both the wrist and the forearm/elbow level in the upper and the ankle and knee level in the lower extremities. Furthermore, that while the metabolic abnormalities alone might not be sufficient so to cause symptoms, the distal nerve compressions, even when early in the course of nerve compression, would summate to give symptoms that appeared to be neuropathy. Finally, decompression of the peripheral nerves might be sufficient, even thought the metabolic abnormalities were still present, to relieve the patient of their symptoms. This was described in greater detail in Chapter 14 of our book, Surgery of the Peripheral Nerve, published in the same year, 1988.[54] The publication of our first series of patients with decompression of multiple peripheral nerves for the treatment of their symptoms of neuropathy in 1992[55] (Chapter 4) confirmed this new optimism.

Figure 6



Rat sciatic nerve is shown in non-dehydrated osmium fixed (myelin is dark) preparation. Normal is on the left. On the right is nerve from a streptozotocin-induced diabetic rat. Same magnification left and right. Note subperineurial edema (arrow) right and increased endoneurial edema in the bottom right. Adapted from Jakobsen, 1978.[56]

METABOLIC FACTORS INTRINSIC TO DIABETIC NERVE SUSCEPTIBILITY TO CHRONIC COMPRESSION

There are now identified three metabolic problems related to diabetes mellitus that render the peripheral nerve susceptible to chronic peripheral nerve compression.

1. Diabetic nerve has increased water content

In 1978, Jakobsen demonstrated that metabolism of glucose into sorbitol by the nerve through the enzyme aldose reductase causes water to be pulled into the nerve resulting in subperineurial and endoneurial edema.[56] This is illustrated in **Figure 6**. In the simplest terms, a nerve within a normally tight anatomical space increases in volume due to increased water content, causing increased endoneurial pressure, decrease blood flow, and symptoms of neural ischemia.

2. Diabetic nerve has decreased axoplasmic flow

The Upton and McComas hypothesis suggested that the underlying cause for the susceptibility of the distal nerve to chronic compression was a decrease in axoplasmic flow proximally.[35] The fast component of anterograde axoplasmic flow transports neurotransmitters. The slow component of anterograde axoplasmic flow transports structural proteins. In a region of chronic compression there is structural damage requiring proteins for repair. Jakobsen and Sidenius demonstrated in 1980 that it is the slow anterograde component of axoplasmic flow that is reduced in diabetic rats, thereby documenting that there is decreased axoplasmic flow, as required by the Upton and McComas hypothesis, and further documenting that the peripheral nerve in the diabetic rat will not be able to repair itself optimally.[57]

3. Advanced glycosylation end-products are in the nerve

The elevated glucose content in diabetes mellitus causes a non-enzymatic reaction between the glucose and collagen. Glucose binds to collagen. This is probably the reason for increased thickness in the flexor tendon sheaths and increased incidence of trigger fingers in diabetics.[58] Today these products are referred to as AGE products, for advanced glycosylated end-products. This has been extensively described.[59] Although not proven yet for the diabetic nerve, I believe that these products occur in the endoneurium, altering the stress-strain relationships of the nerve, making it more stiff. Therefore it is harder for the nerve to glide across joint surfaces, increasing its susceptibility to chronic compression. Demonstrating this remains an excellent future research project.

Figure 7

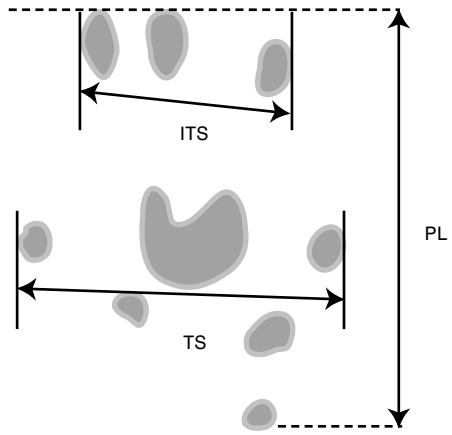
Walking tracks can demonstrate disease states. Tracks can be obtained by dipping the rat's hind feet in to water-color paints and having the rats walk on white paper. Reported in 1991.[60]

FINAL EXPERIMENTAL PROOF OF VALUE OF NERVE DECOMPRESSION

It remained to prove that decompression of a nerve in a diabetic rat would alter the natural history of progressive neuropathy. To do this, the streptozotocin-induced diabetic rat model was used, and walking track analysis was employed as the assay for neuropathy. The demonstration that progressive neuropathy can be documented and related to serum glucose levels is given in **Figure 7, 8, and 9**.[60]

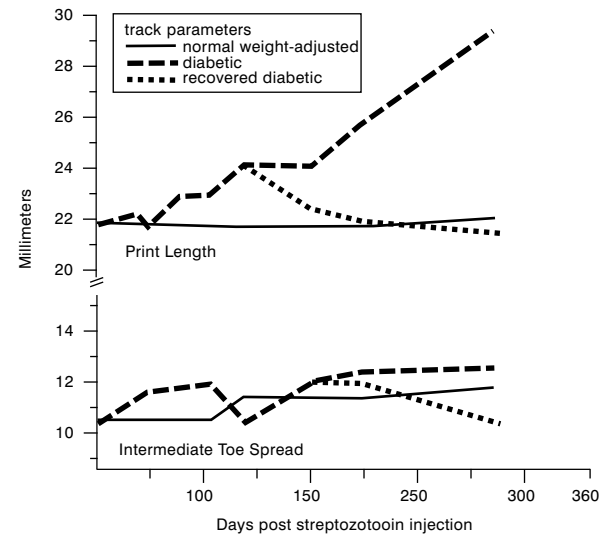
The rat has a tarsal tunnel, and for this study, rather than create an abnormal site of compression on the sciatic nerve with a silicon tube, it was assumed that the tarsal tunnel in the rat might serve as a site of anatomical narrowing just as it does in the human. Therefore, the next stage in the study was to decompress the tarsal tunnel in the rat prior to giving streptozotocin to create a group of rats without a tarsal tunnel. **Figure 10**. This was done, and it was now possible to compare the natural history of rat walking tracks in diabetic rats with **(Figure 11)** and without a tarsal tunnel **(Figure 12)**.[61]

Figure 8



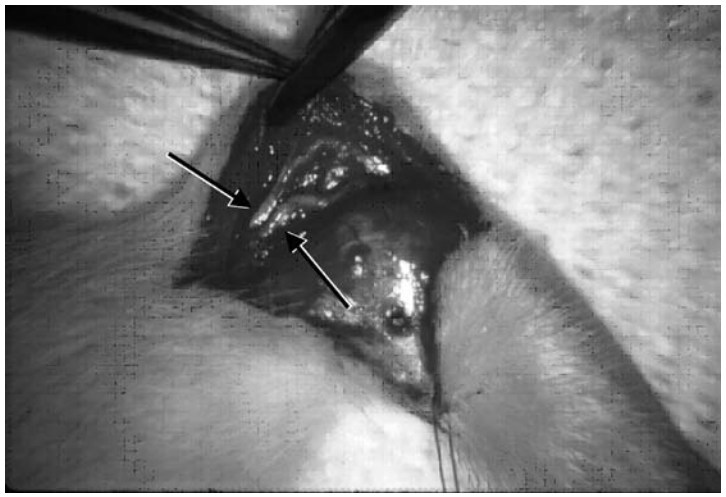
Walking tracks can be digitized and the print length, intermediate and full toe spread distances measured and graphed.

Figure 9



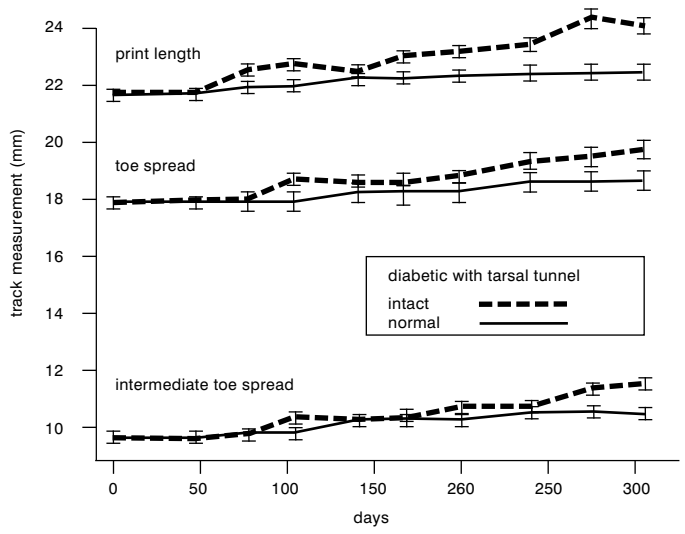
Graph of diabetic rat walking tracks over a period of one year after a single injection of streptozotocin. The solid line is the normal rat walking track. The dashed line is for rats with a glucose of 400 mg/dl. The dotted line is for rats whose pancreas has begun to produce insulin again (recovered from the streptozotocin injection) with a glucose of 90 gm/dl. This demonstrates that measuring changes in the walking track correlates with glucose level and that the abnormal measurements are related to neuropathy.[60]

Figure 10



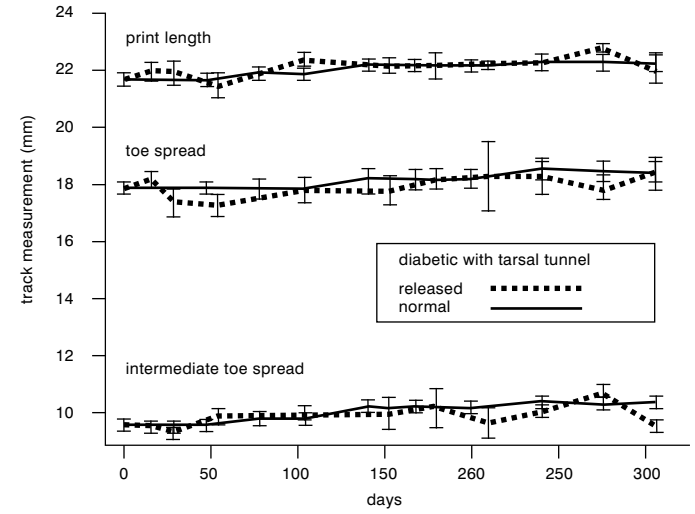
Decompression of tarsal tunnel in the rat. Note the medial and lateral plantar nerves can be seen (white arrows) in the open tunnel.

Figure 11



Walking track analysis of diabetic rats (red line) with blood sugars of 440 mg/dl and an intact tunnel, compared to normal rats (white line). The differences between these two groups document development of diabetic neuropathy over the course of one year (half the rat's life time in the lab). This is confirmation of the pattern observed first in Figure 5.9 and reported in 1991 was reported for this portion of the study in 1994.[61]

Figure 12



Walking track results comparing a group of diabetic rats (yellow line) with blood sugars of 400 mg/dl for one year who have had their tarsal tunnel decompressed prior to becoming diabetic. This group of diabetic rats does not have a site of anatomic compression. When compared to the normal rats (white line), the diabetic rats without a tarsal tunnel are not significantly different from normal. The diabetic rats with blood sugars of 400 mg/dl for one year did not develop neuropathy. Reported in 1994.[61]

CONCLUSION

The peripheral nerve in the diabetic is susceptible to chronic compression due to metabolic problems intrinsic to the disease. Surgery cannot change those factors. Metabolic problems serve as the "first crush" to make the nerve susceptible distally to compression at known areas of anatomic tightness. Decompression of these tight areas can prevent the chronic nerve compression, thereby preventing the development of the symptoms of neuropathy. This is a source of clinical optimism.

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Chapter 6

Predictive value of a positive Tinel sign

Based on

Lee CL, Dellon AL, Prognostic Ability of Tinel Sign in Determining outcome for Decompression Surgery in Diabetic and Non-Diabetic Neuropathy.
Ann Plast Surg 53:523-527, 2004

ABSTRACT

Over the past 12 years, six studies reported that restoration of sensation and relief of pain in the foot by decompression of tibial nerve and its distal branches in diabetic neuropathy. While a positive Tinel sign related to favorable outcomes in some of the reports, this relationship was not evaluated specifically. In this study, the presence of the Tinel sign, positive or negative, over the tibial nerve was recorded in 46 patients with diabetic neuropathy and in 40 patients with idiopathic neuropathy. Outcome were dichotomized into either a good/excellent or failure/poor category. Post-operative data was analyzed at one year. In diabetic neuropathy, presence of a positive Tinel sign had a sensitivity of 88%, a specificity of 50% and a positive predictive value of 88% in identifying patients who would have a good/excellent outcome. In idiopathic neuropathy, the presence of a positive Tinel's sign had a sensitivity of 95%, a specificity of 56%, and a positive predictive value of 93% in identifying patients who would have a good/excellent outcome. It is concluded that a positive Tinel sign is a reliable indicator of successful outcome from decompression of the tibial nerve in diabetics with symptomatic neuropathy, and also in patients with symptomatic idiopathic neuropathy.

INTRODUCTION

Over the past twelve years, there have been six reports published that, taken together, demonstrate that in the patient with diabetic neuropathy decompression of the tibial nerve and its distal branches (in the four medial ankle tunnels) can restore sensation and relieve pain the in the foot.[1-6] The basis for this surgery is that in diabetes, the peripheral nerve is susceptible to chronic compression at known sites of anatomic narrowing.[7-11] The surgical procedure is a neurolysis done at a known site of nerve entrapment. In **Table 1**, the cumulative experience of the six clinical reports is summarized. In three of the six reports,[1,4,6] none of the patients had a history of ulceration or amputation, and the presence of a positive Tinel sign over the site of nerve compression was required as an indication for surgery. In the other three reports,[2,3,5] the patients had a more advanced degree of neuropathy, as indicated by the presence ulceration or amputation in some of the patients, and the presence or absence of a pre-operative Tinel sign was either not mentioned or not taken into consideration regarding the indication for surgery. The present investigation is a prospective study of the prognostic ability of the presence of a pre-operative Tinel sign, at the site of nerve compression, upon the outcome of the peripheral nerve decompression surgery. This experience is reported for patients with neuropathy due to diabetes, and, for the first time, in patients with neuropathy of unknown etiology.

METHODS AND MATERIALS

A prospective study was begun in January of 1997 and continued through December of 2000. Patients with neuropathy due to diabetes mellitus or with neuropathy of unknown etiology were included. Excluded were patients with neuropathy due to alcoholism, chemotherapy, collagen vascular disease, thyroid disorders, gamma globulinopathy, autoimmune or inflammatory diseases. Neuropathy was documented by electrodiagnostic studies and neurosensory testing with the Pressure-Specified Sensory Device' (Sensory Management Services, LLC, Baltimore, Maryland).[12-16] The patients with neuropathy of unknown etiology did not have a small fiber neuropathy, as proven by the presence of large fiber abnormality on electrodiagnostic or neurosensory testing.

Table 1
Results of tibial nerve decompression in diabetic neuropathy

	number of:		pre-operative:			
Study	**Nerves**	**Patients**	**Ulcers**	**Amputation**	**Results, Improved**	**Recurrent ulceration**
Dellon[1] 1992	31	22	0	0	pain, 85% 2PD, 72%	0%
Wieman[2] 1995	33	26	13	0	pain, 92% 2PD, 72%	7%
Chaffe[3] 2000	58	36	11	6	pain, 86% touch, 50%	0%
Aszmann[4] 2004	16	12	0	0	2PD, 69%	0%
Tambekar[5] 2001	10	10	10	n.a.	pain, n.a. touch, 100%	0%
Wood[6] 2003	33	33	0	0	pain, 90% 2PD, 67%	0%
Biddinger and Amend[33] 2004	15	22	0	0	pain, 86% 2PD, 80%	0%

n.a. = information not available in manuscript

The Tinel sign was done by tapping over the tibial nerve from just proximal to the medial malleolus to the juncture of the medial ankle with the sole of the foot. A reflex hammer not used. Gentle tapping of the examiner's finger was used, and not sufficient pressure to cause pain. A "positive" Tinel sign was taken to be a response that indicated either a tingling or radiating electric-like perception either into the heel, the arch, or the toes (the most common responses), or proximally up the inside of the ankle (least common response). A reply that consisted of the patient stating "I feel that", was considered a negative response. There had to be some radiation of the perception after the percussion of the nerve.

During the course of the enrollment period, 46 diabetics and 40 non-diabetics with neuropathy were included, and went on to have a surgical decompression of the tibial nerve and its branches in the four medial ankle tunnels (tarsal tunnel, medial plantar tunnel, lateral plantar tunnel, calcaneal tunnel). This technique has been described previously.[17-21] The period of follow-up ended in December of 2001, so that the minimum follow-up was one year.

Outcome for the surgery was predicated upon improvement in pain and/or recovery of sensibility, as determined by the patient's office visit interview, amount of pain medication being taken, and neurosensory testing. Outcome was dichotomized into good/excellent and failure/poor categories to permit statistical evaluation. Statistical analysis was done for sensitivity, specificity, positive and negative predictive value according to **Table 2**.

Table 2
Statistical analysis definitions

Tinel Sign	Good/Excellent	Failure/Poor
Positive	A	C
Negative	B	D

Sensitivity = A/(A + B)
Specificity = D/(C + D)
Positive Predictive Value = A/(A + C)
Negative Predictive Value = B/(B + D)

Table 3
Results in diabetic neuropathy

Tinel Sign	Good/Excellent	Failure/Poor
Positive, n	37	3
Negative, n	2	4

Sensitivity = 95%
Specificity = 56%
Positive Predictive Value = 92%
Negative Predictive Value = 67%

RESULTS

In the patient with diabetic neuropathy, the prognostic ability of the pre-operative positive Tinel sign is given in **Table 3**. The most important implication of this result is that in the patient with symptoms of diabetic neuropathy in the foot, the presence of a positive Tinel sign demonstrates that 92% of the patients can expect a good to excellent outcome, and that, conversely, if the pre-operative Tinel sign is negative, just 33% of the patients can expect a good to excellent outcome. The difference in the percentage of patients who achieve an excellent result with a positive Tinel sign is significantly better ($P < .003$) than for those with a negative Tinel sign.

In the patient with idiopathic neuropathy, the prognostic ability of the pre-operative positive Tinel sign is given in **Table 4**. The most important implication of this result is that in the patient with symptoms of diabetic neuropathy in the foot, the presence of a positive Tinel sign demonstrates that 93% of the patients can expect a good to excellent outcome, and that, conversely, if the pre-operative Tinel sign is negative, just 23% of the patients can expect a good to excellent outcome. The difference in the percentage of patients who achieve an excellent result with a positive Tinel sign is significantly better ($P < .002$) than for those with a negative Tinel sign.

Table 4
Results in idiopathic neuropathy

Tinel Sign	Good/Excellent	Failure/Poor
Positive, n	25	6
Negative, n	2	7

Sensitivity = 93%
Specificity = 54%
Positive Predictive Value = 81%
Negative Predictive Value = 77%

DISCUSSION

The physical finding of a distally radiating buzzing sensation when a site of suspected nerve problem is percussed, a "positive Tinel sign", is used clinically today to identify a site of nerve entrapment or injury, such as a neuroma. The original description by Tinel described something different; a "tingling" that radiated distally in the territory of the injured nerve, and this site splitting into two, one that stayed where it originally was and one that travelled distally in the patient in whom the nerve was regenerating.[22] It is worth nothing however, that in Kaplan's translation of Tinel's paper, the word "entrapment" does appear: "In complete interruptions of the nerve produced by very tight entrapment [the tingling sign is present]". Tinel wrote that "tingling reveals the presence of regenerating axons". Today, while we still use the original method of Tinel to identify neural regeneration, the presence of a "positive Tinel sign" is most often utilized to identify nerve entrapments, with the most common use being palpation of the median nerve over the carpal tunnel, and the ulnar nerve in the post-condylar groove. While it is appropriate to state that the underlying neurophysiologic or pathophysiologic process that results in the positive Tinel sign is unknown, it is likely that the demyelination and partial remyelination, accompanied by axonal degeneration and regeneration that is present in chronic nerve entrapment render the peripheral nerve mechanosensitive.[23-28] It is clear from clinical experience that the presence of a positive Tinel sign changes during the course of nerve compression,[29] and this is probably related to the degree of neuropathology present at the time of the examination. It follows, then, that a time will occur in the course of chronic nerve compression when a Tinel sign will become positive, and then, with further progression of the neuropathology, the Tinel sign will become negative. It is against this background the results of the present study must be interpreted. The results of this study demonstrated that the probability of obtaining a good to excellent result is better when the Tinel sign is present compared to when it is absent.

In patients with diabetic neuropathy, there is an increased incidence of peripheral nerve compression. The most recent report related to this observation,[30] which was for median nerve compression in the carpal tunnel, demonstrated an incidence of carpal tunnel syndrome of 2% in the non-diabetic population, of 14% in the diabetic population without neuropathy, and of 30% in the diabetic population with neuropathy. Importantly, that study [30] concluded that "electrodiagnostic parameters are not significant predictors of clinical carpal tunnel syndrome in diabetics," and that "therapeutic decisions for carpal tunnel syndrome can be made independently of electrodiagnostic findings." A similar study has not been done for lower extremity peripheral nerve entrapments in the diabetic population, nor for the population of patients with neuropathy of unknown etiology. In the absence of electrodiagnostic testing being able to identify reliably a site of superimposed nerve entrapment in the population of patients with neuropathy, the presence or absence of a positive Tinel sign at a known anatomic site of nerve compression becomes clinically important in patient care/management decisions.

The results of the present study demonstrate that in the patient with diabetes, who has symptoms of neuropathy, in terms of numbness, paresthesias, or pain, the Tinel sign is of prognostic significance. The patient with a positive Tinel sign can expect a high probability of a successful outcome in terms of restoration of sensation and relief of pain. The 95% positive predictive value for this group of 46 diabetics is the highest reported (see **Table 1**). This is most likely due to the fact the patients are now being referred earlier in the course of their neuropathy for surgical decompression. In contrast, if the Tinel sign is negative, the patient has only about one chance in three of having such a successful outcome. This smaller likelihood of success is best interpreted as a poorer success rate in the patient with a more advanced degree of neuropathy. This knowledge will permit the surgeon to counsel the patient more appropriately in terms of what to expect from decompression of the tibial nerve and its distal branches.

This study reports for the first time an experience with decompression of lower extremity nerves in the patient with idiopathic neuropathy. The symptoms of numbness, paresthesias or pain are the same in these patients as they are for the patients with diabetic neuropathy. In the patient with diabetic neuropathy, it is known that the increased susceptibility for chronic nerve compression is related to the increased endoneurial water content of the nerve, due to conversion of glucose to sorbitol (increased aldose reductase activity), and to the decrease in the slow component of axoplasmic transport.[31] The mechanism of increased susceptibility for chronic nerve compression is also understood for chemotherapy-induced neuropathy, where, for example, cisplatin and taxol bind to tubulin in the axoplasm, resulting in a decrease in the slow component of axoplasmic transport.[32] With a neuropathy of unknown etiology, however, by definition, the cause of the neuropathy is not known, and therefore it is not clear if the peripheral nerve is susceptible to chronic compression or not. It is also not known if the underlying neuropathology will prevent neural regeneration if the nerve is decompressed. Therefore, the results of this study demonstrating a positive predictive value of 93% for a positive Tinel sign in patients with idiopathic neuropathy is a critical finding in the extension of the concept of decompression of peripheral nerves for the treatment of the symptoms of this indeterminate neuropathy. The results of this study as applied to the group of patients with idiopathic neuropathy, that the probability of success in the absence of a Tinel sign is only about one in five, suggests that surgical decompression of a peripheral nerve should not be offered to the patient with a neuropathy of unknown etiology unless a positive Tinel sign is present.

Finally, the results of this study demonstrate significantly better probability of achieving a good to excellent outcome for a patient with either diabetic ($P < .003$) or idiopathic ($P < .002$) neuropathy if their Tinel sign is positive compared with a pre-operative absence of a Tinel sign. This confirms the value of this physical finding in identifying an underlying, superimposed chronic nerve compression in the patient with symptoms of pain or numbness in the patient with neuropathy.

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Chapter 7

Recovery of sensation and relief of pain

Based on

Valdivia JMV, Dellon AL, Weinand ME, Maloney CT Jr, Surgical treatment of peripheral neuropathy: outcomes from 100 consecutive decompressions.
J Amer Podiatric Med Association 95:451-454, 2005.

ABSTRACT

Objective

We report the outcome of 100 patients with peripheral neuropathy treated surgically by multiple nerve decompressions of the peroneal and tibial system. This is a promising approach for the treatment of pain, numbness and balance problems from known causes of nerve compression in the lower extremity.

Methods

Records of 100 consecutive patients with diabetic and non-diabetic neuropathy, treated surgically by multiple nerve decompression, were reviewed to document changes in the visual analog scale, sensation improvement, reduction in pain medication requirement, and complication rate. All patients underwent tarsal tunnel release and neurolysis of multiple lower extremity nerves of the tibial and peroneal system as a concomitant part of the procedure. Patients offered surgical intervention had documented sensory abnormalities using neurosensory testing by the Pressure-Specified Sensory Device (PSSD) and a positive Tinel's sign on exam over the involved nerve. Patients were contacted by phone to confirm the long-standing symptom relief.

Results

Eighty-seven percent of the patients with preoperative numbness reported sensation improvement. Ninety-two percent of patients with balance problems reported improved balance after the procedure. From those patients that underwent the procedure mainly for pain relief, 85.2% reported an improvement in the visual analog scale in more than 50%.

Conclusion

Similar to experiences found in the upper extremity, nerve decompression in the lower extremity is a safe and affective procedure to improve the quality of life of patients with peripheral neuropathy secondary to nerve compression. Documentation and staging of the severity of neuropathy with neurosensory testing and the presence of Tinel's sign determines surgical candidates. Decompression and neurolysis of compressed lower extremity nerves improves sensation, and decrease pain much like results of nerve decompression in the upper extremity and other areas. The great majority is very satisfied with the results.

INTRODUCTION

The propensity of peripheral nerves in diabetics to be compressed by normal anatomic structures has been well recognized and studied [1-12]. Surgical decompression for treatment of entrapment syndromes in diabetics has been described with good clinical outcomes [5,13-15].

This case series results reflect the promising alternative for patients with peripheral entrapment syndromes, mostly due to diabetes, who were told at some point that nothing could be done to relieve their pain and numbness, besides tight glycemic control. The clinical improvement in pain and sensation is very dramatic. The patients even show immediate signs of improvement in the recovery room right after the procedures.

CLINICAL MATERIAL AND METHODS

A prospective study of the senior authors first one hundred consecutive patients with neuropathy treated surgically was performed to evaluate the efficacy of nerve decompression for the symptomatic treatment of neuropathy. The majority of these patients were diabetics. All patients were evaluated previously for other conditions that may present with peripheral neuropathy. All were evaluated with the Pressure-Specified Sensory Device ‰ (PSSD)[16-20] to confirm the physical findings and evaluate the severity of neuropathy. Patients underwent tarsal tunnel release and decompression of the common peroneal, deep peroneal, tibial, calcaneal, medial plantar and lateral plantar nerves in the lower extremities, in an outpatient setting and wit. An unbiased observer recorded variables for each patient, including pain in the visual analog scale (VAS) before and after the procedures. Other variables analyzed included associated conditions, numbers of years with diabetes, number of years with symptoms, two-point discrimination, previous diagnosis of depression and previous diagnosis of fibromyalgia. The post-operative variables analyzed were: pain in visual analog scale, complication rate, subjective sensation improvement, subjective balance improvement, and reduction in pain medication requirement.

The data was stored and analyzed by an unbiased observer using the SPSS statistical program, and in collaboration with the Epidemiology Department of the University of Arizona.

Each chart was reviewed by the investigator (JMVV), without further reference regarding the outcomes by the surgeon (CTM). Each patient was also contacted by phone to confirm the results and record any change in the surgical outcomes.

RESULTS

A total of one hundred records were reviewed. One hundred and thirty-four lower extremities were operated. Sixty percent had the previous diagnosis of diabetes, while forty percent had the diagnosis of idiopathic peripheral neuropathy, confirmed previously by a neurologist. All patients were ruled out as having peripheral vascular disease responsible for their symptoms by the presence of a palpable pedal pulses or acceptable Doppler studies of the lower extremity. Ninety-one percent of the patients had only lower extremity symptoms, without any upper extremity symptoms. Fifty-eight percent had bilateral lower extremity symptoms.

Most of our patients (81%) were over 50 years old when they underwent surgery, with a mean age of 63. Fifty-six percent were males, and forty-four percent were females. The mean number of years with the diagnosis of diabetes was 12, and the mean number of years with symptoms for the group was 6 (**Table 1**).

In the pre-operative period, most of the patients were in severe pain. The mean rate in the visual analog scale before surgery was 8.4 points over 10. Forty-four percent had 10 out of 10 of pain rate, ninety-five percent had 6 or more points in the visual analog scale (**Table 2**). Their pain was mostly in the plantar surface (posterior tibial nerve), dorsum of the foot and first web space (deep peroneal nerve), and lateral calf (common peroneal nerve). Most of them reported to have severe impairment in the daily activities due to pain. All of the patients had a positive Tinel's sign over the compressed nerves on exam.

Table 1

	Age	Visual Analog Scale	Years with Diabetes	Years with Symptoms
Mean	62.97	8.48	12.1	6.36
Minimum	30	0	1	1
Maximum	84	10	30	23

Table 2

V.A.S	Frequency	Valid Percent
0	1	1%
4	2	2%
5	2	2%
6	10	10%
7	4	4%
8	31	31%
9	6	6%
10	44	44%
Total	100	100

All patients underwent neurolysis of the common peroneal nerve at the fibular head, the deep peroneal nerve on the dorsum of the foot, tarsal tunnel release and decompression of the medial and lateral plantar, and calcaneal nerves. A total of 134 lower extremities underwent surgery. None of the patients reported worsening of pain or previous symptoms after surgery. All surgeries were performed under general anesthesia, with a tourniquet to minimize blood loss, in an outpatient setting.

Pain

Ninety-nine patients had pain before the procedure, from these, 85 (85.8%) reported clinical improvement in pain, measured by a decrease in pain in more than 50 % from the preoperative pain rate. Also, in 71 of these 99 patients (71.7%) the pain improved in more than 5 points in the visual analog scale. The group improved their pain after surgery in an average of 6.4 points in the visual analog scale. Remarkably, the percentage of patients with pain rate of 10 out of 10 decreased from a 44% preoperatively to a 2% after the procedure. Moreover, 36 patients (36%) had no pain at all (0/10) after the lower extremity nerve decompressions. These patients reported verbally that the felt very satisfied and happy with the results, which were confirmed in most of them as soon as in the immediate post-operative period, in the recovery room.

Statistical analysis revealed a significant difference in the number of patients with 6 or more points in the VAS between the pre (95%) and the post-operative period (10%) ($P \leq 0.001$) (**Table 3**). Only one patient had a pain rate of 2 or less in the pre-operative period, while 66 patients fell under this category in the post-operative period; this difference was statistically significant ($P \leq 0.001$) (**Table 4**).
As a group, we compared the pain scales in the VAS between the pre and post-operative period. Analysis of the distribution of pain using the Wilcoxon signed-rank test and the t-test reveled a statistically significant difference between the 2 periods ($P < 0.001$) (**Figure 1**).

Since the indications for surgery were both pain and numbness in the lower extremities, in order to evaluate the efficacy of the procedure to improve each symptom, we assess pain improvement in those patients where the procedure was performed mainly to treat severe pain. Eighty-one of 100 patients had 8 or more points in the visual analog scale at presentation, which objectively reflects high levels of pain. In this group, the procedure was successful in reducing pain in more than 50% from the pain rate before surgery in 85.2% (69 of 81) of patients (**Figure 2**).

Table 3

VAS	Preop	Postop
≥6	95	10
<6	5	90
Total	100	100

Table 4

VAS	Preop	Postop
≤2	1	66
>2	99	34
Total	100	100

Figure 1

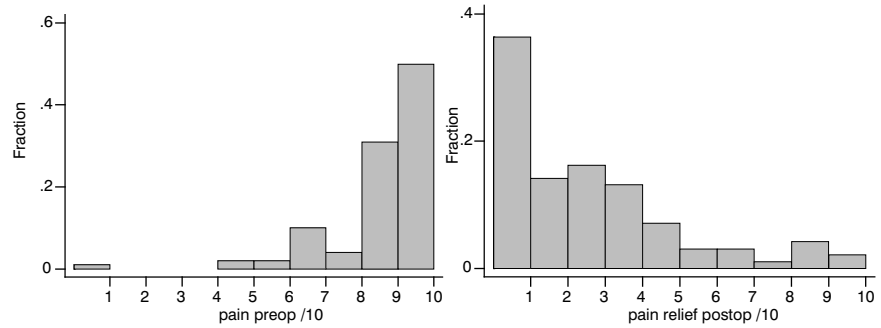
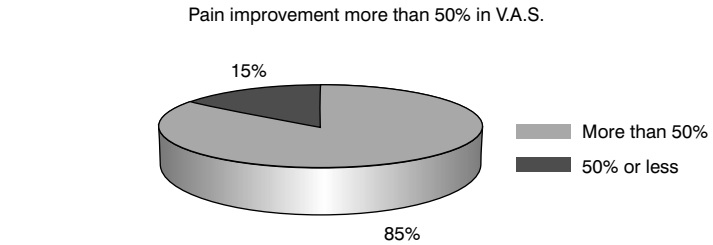


Figure 2



Also, pain was reduced in more than 5 points in the VAS in 61 patients (75.3%). The mean pain improvement in this group was 6.8 ± 2.6 (0-10). 87.3% of these patients either stopped or decreased their dose of pain medication required. In this group, balance improvement was reported by 48 (92.3%) of 52 patients who reported balance and station problems due to pain while walking or prolonged standing position.

Sensation

Ninety-three percent had decreased 2-point discrimination as evaluated by the Pressure-Specified Sensory Device (PSSD) (Sensory Management Services L.L.C., Baltimore, MD). All the patients had increased pressure threshold by the

PSSD. Analysis of the patients in which surgery was performed mainly to improve sensation (95), revealed that sensation was improved in 83 (87.4%) (**Figure 3**). Balance was reported to improve in 58 (92.1%) of 63 patients who reported balance and station problems due to numbness in the lower extremities. No patient had normal sensation prior to surgery, while 83 (83%) either recovered or improved their sensation in the lower extremities. This was also statistically significant ($P \leq 0.001$).

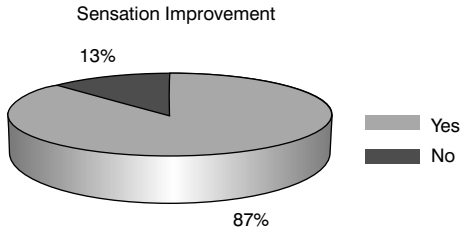
Pain medication requirement

Ninety-nine patients in the pre-operative period required pain medication, while only 22 continued the same dose of pain medication after the surgery. Seventy-seven (78%) stopped or decreased their pain medication requirement; this difference between the pre and postoperative periods was statistically significant ($P \leq 0.001$).

Diabetics vs. Non-diabetics

There was no difference in improvement of pain in more than 5 points or more than 50 % between diabetics and non-diabetics ($P = 0.7$). Also, there was no difference in pain improvement in the VAS between age groups (< 50, 50-60, 61-70, 71-80, 81-90, > 90 years old) ($P = 0.5$). Improvement in more than 5 points in the VAS was more prevalent in females than in males ($P=0.02$).

Figure 3



DISCUSSION

Entrapment neuropathies are distinctive clinical neuropathic sensory and motor syndromes caused by compression or irritation of a peripheral nerve [21-23]. The factors involved in the pathogenesis of nerve damage secondary to compression include demyelination, wallerian degeneration [6-7, 24-26], compression of the vasa nervora, impairment of axonal transport [9, 27, 28] and intraneural connective tissue formation [29]. Moreover, the double crush hypothesis describes the susceptibility of a nerve to injury when it is compressed at more than one site [7,23,30-32].

Peripheral nerve entrapment is frequent in patients with diabetes mellitus [33,34]. Excess of intracellular sorbitol alters the Na-K ATPase activity producing the axonal swelling due to osmotic gradient, making nerves more prone to entrapment than in non diabetics [1,4,35-37], and impairing axonal transport [38].

We analyze patients with peripheral nerve entrapment syndrome, most of them having diabetes acting as one the “crush” in the neuropathy according to the double crush phenomenon theory [7,30]. The study aims to describe the clinical results of patients with disabling lower extremity neurological symptoms, after undergoing decompression of the tibial nerve at the tarsal tunnel, and the common and deep peroneal nerve [39]. Most of our patients were told at some point that nothing could be done for their pain and loss of sensation besides tight glucose control. What is amazing is the dramatic recovery when the nerves are decompressed. Patients start walking again, recovering sensation, and regaining their capacity to work in society. It is surprising how fast these changes take place. One can even test the success of sensation improvement in the recovery room.

Several inclusion criteria were used to define surgical candidates. These include: **1)** history of pain or decreased sensation in the distribution of the posterior tibial (medial and lateral plantar surface), common and superficial peroneal (lateral calf and dorsum of the foot), and deep peroneal (dorsum of the foot, first web space); **2)** Abnormal neurosensory testing by the Pressure-Specified Sensory Device (PSSD); **3)** Positive Tinel’s sign on exam; **4)** Muscle weakness in those innervated by the mentioned nerves; **5)** Absence of severe peripheral vascular disease or radiculopathy that might explain the patient’s symptoms.

As an alternative to the electrodiagnotic testing (EDT), the Pressure-Specified Sensory Device can be used with great sensitivity. It is a valid and reliable instrument when compared with the gold standard [16-20]. Also, the Tinel’s sign was used as an inclusion criteria. The presence of this sign has a sensitivity of 88%, specific-

ity of 50%, and a positive predictive value of 88% in predicting which patient would have a good or excellent outcome [40].

As we see in the series results, the great majority of the patients were satisfied with the procedures. The clinical improvement was analyzed for each individual group where surgery aimed primarily to improve either pain or sensation. Symptoms of a group of patients with peripheral entrapment syndrome can be viewed as Gaussian distribution for pain and another for numbness, both overlapping at the center. When evaluating the patients that underwent the procedure primarily for pain relief, 85.2% reported pain improvement and indeed improved more than 50% in the visual analog scale. Also most of them decreased their pain medication requirement, which further prevents side effects and dependency to these drugs.

Regarding those patients presenting mostly with sensation problems, 87.4% reported a good clinical improvement. Moreover, from those who reported balance problems, 92.1% improved their balance. Balance in these patients was mainly affected because they could not feel their legs, could not sense their position in space.

These results show a significant benefit due to surgical decompression of entrapment neuropathy in this group, and the data is comparable with previous reports of human surgical results [13-15,41, 42].

Nevertheless peripheral entrapment syndromes is considered a rare entity, it is a very common and important pathology that causes emotional distress and poor quality of life. It is a condition that is often misdiagnosed and mistreated [21].

CONCLUSIONS

Decompression of the posterior tibial nerve together with decompression of the deep and common peroneal nerves in the lower extremity decreases pain in patients with peripheral entrapment neuropathy. This approach to peripheral entrapment neuropathy opens a new door for patients under severe stress and handicap due to this condition. The dramatic clinical improvement and enhanced quality of life is the main objective of these surgical interventions. In the future, we predict surgical nerve decompression of the extremities, when indicated, will be the standard of care in patients with debilitating symptoms of peripheral nerve entrapment, due to diabetes or other causes. To compare which alternative is better for the patient, we are developing a prospective-randomized study to compare surgical decompression of peripheral nerves versus medical treatment and tight glycemic control in patients with diabetic neuropathy.

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Chapter 8

Recovery of balance

Based on

Ducis I, Short KW, Delon AL, Relationship Between Loss of Pedal Sensibility, Balance and Falls in Patients with Peripheral Neuropathy.
Ann Plast Surg 52:535-540, 2004

ABSTRACT

The purpose of this study was to describe the relationship between balance and foot sensibility in a population of patients with impaired lower extremity sensation. The hypothesis was that increasing impairment of sensation correlates with impaired balance. To date, no report has investigated the relationship between loss of balance with the degree of sensibility in the foot in a population with neuropathy. Ten control subjects and 35 patients with sensory abnormalities and balance problems related to a neuropathy were evaluated. The MatScan Measurement System was used to measure their ability to stand still, maintaining their balance with their eyes open and then with their eyes shut. The degree to which the person moves while attempting to stand still is defined as "sway," which was recorded for normal and neuropathy patients. Sensibility of the foot was measured with the Pressure-Specified Sensory Device, which is noninvasive and nonpainful. The 1- and 2-point static touch thresholds are measured for the pulp of the big toe, medial heel, and the dorsum of the foot. Loss of 2- or 1-point sensation was recorded as sensibility score and compared with controls. Statistical analysis of data and their comparisons for the 2 groups was completed. There were 55% females in control and 64% in neuropathy patients, whereas average age was 50 and 62 years, respectively. Neuropathy was the result of diabetes in 64.5%, hypothyroidism in 19.3%, their combination in 13%, and of unknown etiology in the remaining 19% of patients. Controls had significantly lower mean sway than neuropathy patients (22.9 ± 9% *v*s. 189.5 ± 180%, $P = 0.006$). Likewise, sensibility score for normal and neuropathy patients was also significantly different (31.4 ± 9% *v*s. 232.8 ± 59%, $P < 0.0001$). When compared with the controls, 99% upper limit of confidence, sensibility in the neuropathy group at the hallux pulp was abnormal at a level consistent with axonal loss in 52% and was completely absent in the remaining 48%. Similarly, at the heel, sensibility was normal in 6.5%, abnormal at a level consistent with axonal loss in 71%, and absent in the remaining 22.5%. The correlation coefficient between sway and sensibility score was 0.36. The results of this investigation for the first time document the intuitive relationship between increasing loss of foot sensibility and increasing loss of balance. These measurements can now be used prospectively to evaluate whether restoration of sensation to patients with neuropathy, through peripheral nerve decompression, can improve balance and reduce falls/fractures in this patient population.

INTRODUCTION

Falls and fall-related fractures are a source of enormous morbidity with high resultant healthcare and disability costs.[1,2] Falls are the most common cause of injury and hospital admissions for trauma account for 87% of fractures in individuals older than 65.[3] Although sensory loss is among many risk factors associated with falls, neuropathy is a known cause of decreased sensibility in the feet.[3,4] Some of the first evidences of peripheral neuropathy in patients with diabetes is seen in toes.[5] The most likely explanation is that the nerve injury, both longitudinal and segmental, occurs randomly at many sites and increases proportionally with the length of the axon. This sensory neuropathy results in a reduction of afferent sensory perception and predisposes neuropathy patients, which often have altered biomechanics, to ulcerations, because no pain can be felt at the sites of increased pressure.[6]

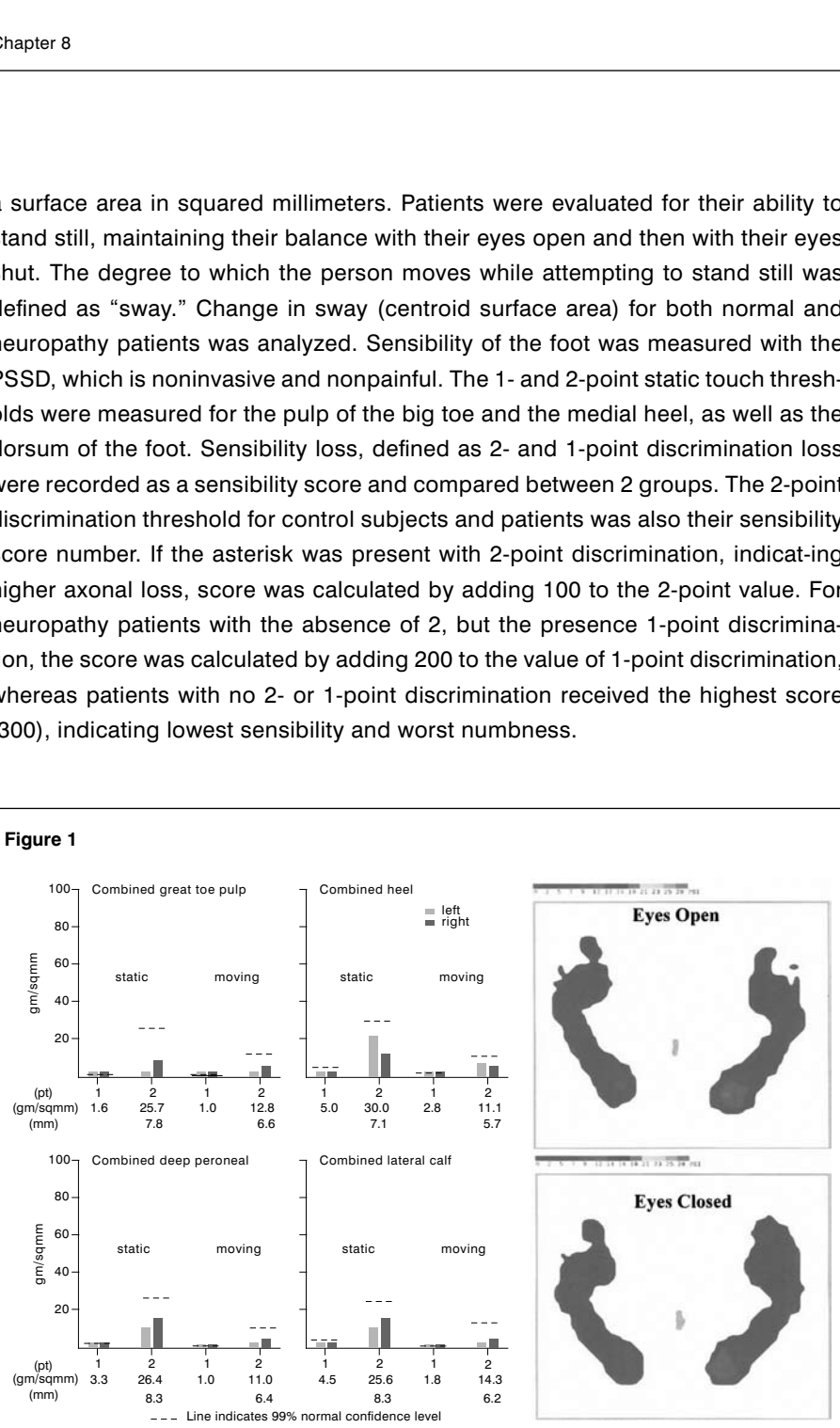
Balance and gait require the central integration of afferent information arising from 3 distinct yet interwoven peripheral sensory systems: the vestibular, visual, and somatosensory skin receptors.[7] Although it is known that postural stability in neuropathy patients is greatly affected by age and diabetes [6,8] to date, no report has quantified the relationship between loss of balance with the degree of sensibility, measured by the Pressure-Specified Sensory Device (PSSD) (Sensory Management Services, LLC, Baltimore, MD) in the foot in a neuropathy population.

MATERIALS AND METHODS

Balance and sensibility in 10 control subjects and 35 patients with complaints of sensory abnormalities and balance problems related to a neuropathy were compared. Neuropathy patients were those with a known diagnosis such as diabetes, hypothyroidism, chemotherapy-induced neuropathy, as well as patients in whom the etiology is unknown. All patients and controls with a history of clinical neurologic dysfunction (cerebrovascular accident, ataxia, Parkinsonism, multiple sclerosis, history of acoustic neuroma or vestibular problems) were excluded from the study. Balance was measured using the noninvasive MatScan Measurement System (Tekscan Inc., Boston, MA) that has a mat on which the patient stands. Embedded within the mat are force transducers that detect changes in the pressure of the surface of the foot that is in contact with the mat, transmitting this information in real-time to the computer. The computer records 30 seconds of data and displays this to show a pattern of the centroid of movement, which is described as

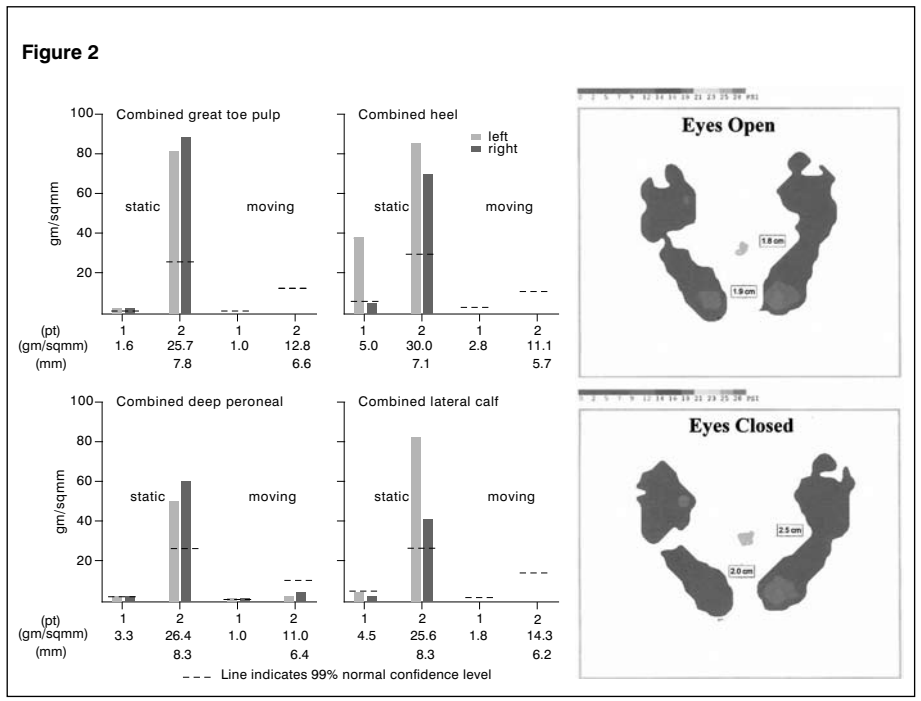
a surface area in squared millimeters. Patients were evaluated for their ability to stand still, maintaining their balance with their eyes open and then with their eyes shut. The degree to which the person moves while attempting to stand still was defined as “sway.” Change in sway (centroid surface area) for both normal and neuropathy patients was analyzed. Sensibility of the foot was measured with the PSSD, which is noninvasive and nonpainful. The 1- and 2-point static touch thresholds were measured for the pulp of the big toe and the medial heel, as well as the dorsum of the foot. Sensibility loss, defined as 2- and 1-point discrimination loss were recorded as a sensibility score and compared between 2 groups. The 2-point discrimination threshold for control subjects and patients was also their sensibility score number. If the asterisk was present with 2-point discrimination, indicat-ing higher axonal loss, score was calculated by adding 100 to the 2-point value. For neuropathy patients with the absence of 2, but the presence 1-point discrimination, the score was calculated by adding 200 to the value of 1-point discrimination, whereas patients with no 2- or 1-point discrimination received the highest score (300), indicating lowest sensibility and worst numbness.

Figure 1



Results of sensibility (left) and sway testing (right) in a normal patient. Note all pressure thresholds for 1- and 2-point discrimination are within 99% confidence interval. Change in sway centroid surface area between open and closed eyes is minimal.

Figure 2



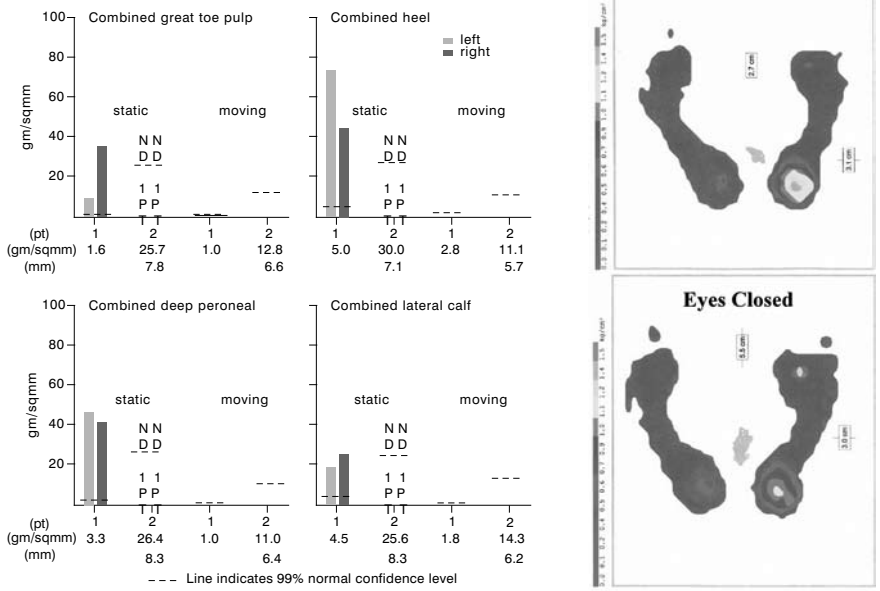
Results of sensibility (left) and sway testing (right) in a patient with moderate neuropathy. Note all pressure thresholds for 1- and 2-point discrimination

RESULTS

Normal patients had sensibility scores of 2-point discrimination with pressure thresholds within 99% confidence interval, whereas their sway surface area minimally changed with closed eyes (**Figure 1**). The mean sensibility score for toe and heel in control patients was 31.4 ± 9%, whereas sway surface area with eyes open was 27.3 ± 6 mm^2 and 33.4 ± 7 mm^2 with eyes closed (nonsignificant increase in surface area of 22.9 ± 9%, $P = 0.07$) (**Figure 6**).

Neuropathy patients had increased 2-point discrimination pressure thresholds when moderate neuropathy was present (**Figure 2**). With more advanced (moderate–severe) neuropathy, 1-point was preserved, whereas 2-point discrimination was lost (**Figure 3**). In patients with severe neuropathy, both 1- and 2-point discrimination were lost (**Figure 4**). The sway surface area visibly increased when tested with eyes closed compared with open. Its surface area proportionally increased with the severity of the neuropathy (**Figures 2–4**). **Figure 2** depicts sensi-

Figure 3

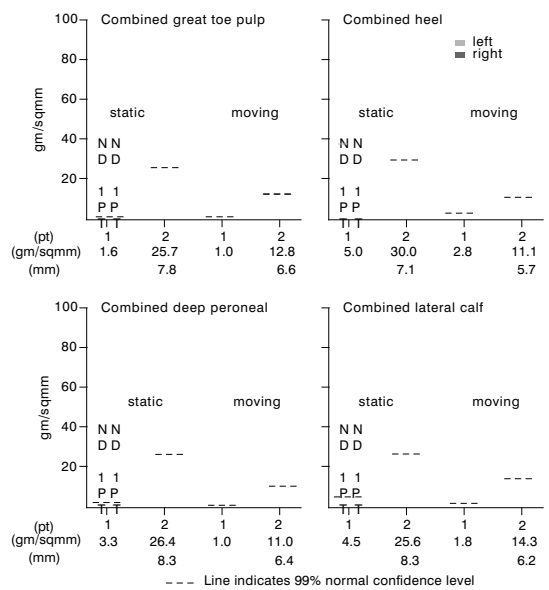


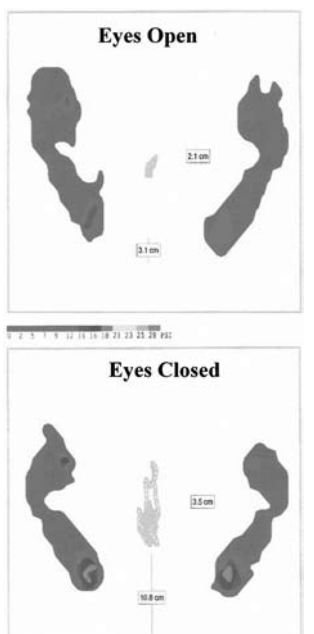
Results of sensibility (left) and sway testing (right) in a patient with moderate–severe neuropathy. Note loss of all 2-point discrimination and increased pressure thresholds for 1-point discrimination. Change in centroid surface area between open and closed eyes is significant.

bility and sway result in a patient with moderate neuropathy, **Figure 3** in a patient with moderate–severe neuropathy, whereas **Figure 4** is for a patient with severe neuropathy.

The mean sensibility scores in neuropathy patients for the toe was 248.7 ± 64% and for the heel it was 216.4 ± 59%. When mean sensibility score results between control and neuropathy groups were compared, a significant difference was found ($P < 0.0001$; **Figure 5**). For neuropathy patients, sway results with eyes open were 52.2 ± 31 mm^2 and 158.5 ± 150 mm^2 with eyes closed, a significant increase in surface area of 189.4 ± 180% ($P = 0.006$; **Figure 6**). The significant difference was also found between control and neuropathy groups for percent sway surface area change ($P = 0.006$). The correlation coefficient between sway (surface area) and sensibility was 0.36.

Figure 4



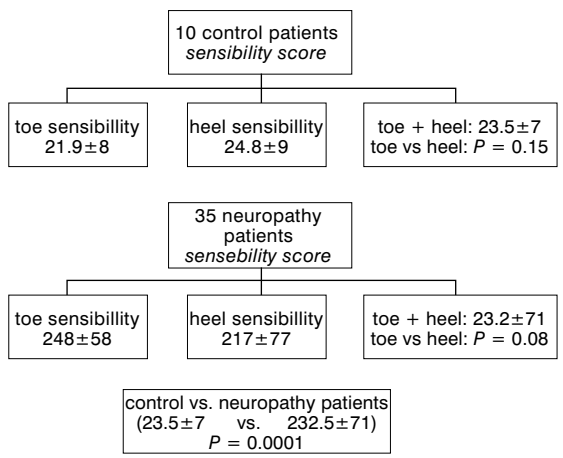


Results of sensibility (left) and sway testing (right) in a patient with severe neuropathy. Note loss of 1- and 2-point discrimination. Change in centroid surface area between open and closed eyes is significant.

DISCUSSION

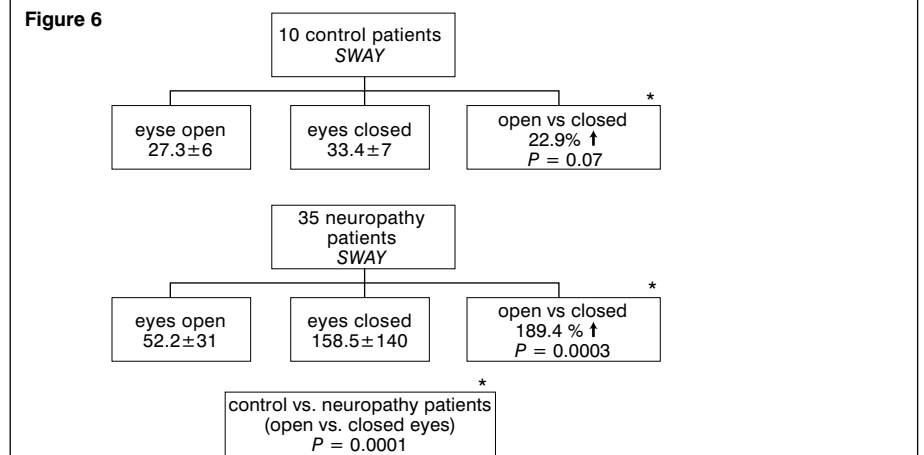
The results of this investigation for the first time document the intuitive relationship between increasing loss of foot sensibility, measured by PSSD and increasing loss of balance. Both of these evaluations were significantly different in neuropathy patients. Patients affected with peripheral neuropathy first lose 2-point static, then 2-point moving discrimination, followed by moving 1-point, and lastly static 1-point discrimination. It is important to understand this sequence to appropriately evaluate sensibility score in neuropathy patients. Unfortunately, many currently used devices used for testing neuropathy, including Semmes-Weinstein monofilaments, fail to detect neuropathy in early enough stages allowing preventable steps toward ulcerations or fractures resulting from falls.[9] Data presented using PSSD enable quantification of the sensibility score as it worsens, and thus detection of neuropathy in its early stages, enabling an appropriate intervention.

Figure 5



Results for sensibility score for control and neuropathy patients. Note significant difference when control and neuropathy patients are compared.

Figure 6



Results for sway surface area for control and neuropathy patients. Note significant difference when control and neuropathy patients are compared.

Balance, when tested in control individuals, was characterized with uniform similarity with only 23% difference between open and closed eyes (**Figure 6**). Unlike controls, neuropathy patients had surface area that greatly varied in size and shape with 189% increase in surface area between open and closed eyes. It was interesting to observe that the 2 groups also significantly differed when sway surface area for open eyes was compared ($P = 0.01$), indicating that peripheral neuropathy rather than ocular changes is responsible for gait problems in diabetic patients. Likewise, balance disturbances with diabetes can also be the result of vestibular system dysfunction. This is explained by vascular involvement of vestibular artery in diabetic patients that greatly diminish the number of cells in the vestibular nerve.[10] Both factors indicate that in patients with peripheral neuropathy, gait and balance disturbances are a result of a systematic dysfunction of peripheral nerves. This can explain our findings that although significant differences in sway surface area and sensibility score were observed between control and neuropathy patients, correlation coefficient linking 2 of them was only 0.36. When nondiabetic neuropathy patients were excluded, the correlation coefficient was 0.46. Peripheral plantar foot nerves are responsible for transmitting the proprioception information back through a posterior column of the spine into a sensory cortex. This path is interrupted when the compression of the tibial nerve at the inner ankle causes the symptomatic peripheral neuropathy. The surgical decompression of peripheral nerves in these patients with symptomatic diabetic neuropathy can restore sensibility.[11] These measurements can now be used prospectively to evaluate whether restoration of sensation to patients with neuropathy, through peripheral nerve decompression, can improve balance and reduce falls/fractures in this patient population.

ACKNOWLEDGMENTS

This project was approved by the Medstar Health Internal Review Board. Dr. Dellon has a proprietary interest in the Pressure Specified Sensory Device. Presented at the Northeastern Society of Plastic Surgeons Annual Meeting, Baltimore, MD, October 2003.

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Chapter 9

Prevention of ulcers and amputations

Based on

Aszmann O, Tassler PL, Dellon AL, Changing the Natural History of Diabetic Neuropathy: Incidence of Ulcer/Amputation in the Contralateral Limb of Patients with a Unilateral Nerve Decompression Procedure.
Ann Plast Surg 53:517-522, 2004.

ABSTRACT

The natural history of diabetic neuropathy is progressive and irreversible loss of sensibility in the feet, leading to ulceration and/or amputation in 15% of patients. The prevalence of neuropathy is more than 50% in those who have been diabetic for 20 years. Decompression of the tibial and peroneal nerves in those with diabetic neuropathy improves sensation in 70% of patients. The impact of this surgery on the development of ulcers and amputations in both the operated and the contralateral, non-operated limb was evaluated in a retrospective analysis of 50 diabetics a mean of 4.5 years (range 2 to 7 years) from the date of surgery. No ulcers or amputations occurred in the index limb of these patients. In contrast, there were 12 ulcers and 3 amputations in 15 different patients in contralateral limbs. This difference was significant at the $p < .001$ level. It is concluded that decompression of lower extremity nerves in diabetic neuropathy changes the natural history of this disease, representing a paradigm shift in health care costs.

INTRODUCTION

Within diabetes mellitus, there are many forms of neuropathy. The distal, large fiber, symmetrical polyneuropathy is the most common form.[1,2] The natural history of this form of diabetic neuropathy is well described, and has remained unchanged for more than half a century in the Western World, in studies including more than 30,000 diabetics.[3-12] For example, in the study of 4400 diabetics reported by Pirart in 1944, neuropathy was present in 12% at the time of diagnosis of diabetes and increased to 50% by the time diabetes had been present for 25 years.[12] Loss of sensibility leads to infection, ulceration, and amputation, which is independent from the amputations due to large vessel disease.[13] The incidence of ulceration is 2.5% per year and occurs in 1 in 6 diabetics in their lifetime.[14-17] Even the Diabetic Control and Complication Trail, whose goal was euglycemia, did not prevent the occurrence of diabetic neuropathy, although it reduced its incidence.[18] The loss of sensibility also results in problems with balance, leading to falls with hip and wrist fracture.[17-20] Those with a painful component to their neuropathy require neuropathic pain medication, often to the point where there are such cognitive changes that they become disabled related to the pain component of the neuropathy alone.[21-28] Eighty to eighty-five percent of amputations are preceded by non-healing ulcers in patients with neuropathy.[29,30] Despite attempts to decrease the number of amputations in the United States of America by various strategies from better glucose control, to monitoring screening exams for impaired sensibility, the number of amputations has continued to increase from 54,000 in 1990,[31] to 92,000 in 1999.[32] The average cost of an ulceration is $27,500 in 1997 and the cost of an amputation ranges from $22,702 for a toe, to $51,281 for a leg, with the annual cost for diabetic neuropathy and its complications in the United States of America being between $4.6 and 13.7 billion dollars.[33, 34] It is estimated that up to 27% of the direct medical costs of diabetes mellitus is related to diabetic neuropathy.[34] There are estimated to be 16 million diabetics in the U.S.A, with this number expected to double by 2030.[35] This number is increasing in epidemic proportions as the overweight population develops insulin resistance.[36-38] "During 2000-2002, an estimated 11.7% of U.S. adults with diabetes had a history fo foot ulcer."[39] As demonstrated by the above review, the natural history of diabetic neuropathy is well-documented: the natural history of diabetic neuropathy is to be progressive and irreversible.

While animal models of early, streptozotocin-induced, diabetic neuropathy have been shown to improve with pharmacologic management, such as aldose reductase inhibitors, or nerve growth factors, randomized prospective trials continue to fail to improve sensibility and relieve pain in patients with symptomatic diabetic neuropathy.[40-42] In contrast, a new optimism was introduced in the 1980's with the realization that the peripheral nerve in diabetes is susceptible to compression, and that this superimposed compression might be the source of the symptoms, rather than the metabolic abnormalities themselves.[43] This was proven in a study where two groups of diabetic rats, each with a serum glucose level > 400 (normal 90-100) were compared; one group had the tibial nerve and its branches in the tarsal tunnel decompressed at the start of the study and the other group had the tarsal tunnel remain intact.[44,45] Diabetes was induced by intra-peritoneal streptozotocin injection, and was not treated with insulin. At the end of one year, approximately half the lifetime for this animal model, the group with the intact tarsal tunnel had the expected progressive neuropathic walking track pattern, while the group without a site of anatomic narrowing over the tibial nerve had a walking track pattern that was not significantly different from the normal, non-diabetic, control rats. Recently this study was repeated with the same result.[46] The first group of patients to have decompression of upper and lower extremity peripheral nerves was reported in 1992.[47] Subsequently there have been five studies confirming that decompression of the tibial nerve and its branches in the four medial ankle tunnels can relieve

pain in up to 90% of patients with painful diabetic neuropathy and restore sensation in up to 80% of patients with impaired sensibility.[48-52]

Outcomes related to ulceration and amputation in patients with diabetic neuropathy who have had decompression of lower extremity nerves have been reported. In the 1992, study, which had a mean of 3.6 years with a range of 1 to 7 years follow-up, no patient developed an ulcer or had an amputation post-operatively.[47] This group of patients contained no one who pre-operatively had an ulceration or amputation. Among the two reports of this surgery whose patient cohorts contained a previous ulcers/amputation history, one study reported no recurrences of ulcerations, despite the fact that its patient population contained 11 patients with a history of previous ulceration and 6 with a history of previous amputation from a total population of 36 patients.[48] The other study reported that one of its 13 patients with a previous history of ulceration, from a total population of 26 patients, did develop a recurrent ulceration.[49] To date, no new ulcerations or amputations have been reported in any patient who has had decompression of peripheral nerves to treat the symptoms of diabetic neuropathy.

Theoretically, restoration of sensibility to the feet should be effective in preventing ulceration and amputation, and, thereby, changing the natural history of diabetic neuropathy. It might be argued however, that the patient who has had surgical decompression of peripheral nerves is now much more aware of the potential complications of diabetes, and that any improvement in incidence of ulcer/amputation is due to either improved glycemic control, or improved foot care, or both. The purpose of the present study was to evaluate the incidence of ulcer/amputation bilaterally in patients who had a unilateral decompression of lower extremity peripheral nerves. The study assumed that glycemic levels would be the same in each lower extremity and that foot care would be given equally to both feet. The hypothesis to be tested was that outcomes in terms of ulceration or amputation would be equally likely to occur in each foot, following a unilateral peripheral nerve decompression using Dellon's surgical technique.[47,50,53-57]

METHODS AND MATERIALS

A retrospective analysis of the patient population that had peripheral nerve decompression for the treatment of symptomatic diabetic neuropathy was initiated by questionnaire, and then with a follow-up telephone interview. A total of 50 patients were identified who fit the inclusion criteria of having had neurolysis of the peroneal nerve at the knee, neurolysis of the deep peroneal nerve over the dorsum of the foot, and decompression of the four medial ankle tunnels, with these surgical procedures having been done on just one limb. The outcomes of ulceration and or amputation were chosen to be such that they could be identified unambiguously by questionnaire or telephone interview. No patients were excluded from this process. Reasons for not having decompression of their contralateral side were related most commonly to changes in overall health status (heart attack), travel distance and travel considerations, and not obtaining full relief of pain or recovery of sensibility from the initial operation. From this process, a cohort of 50 patients was identified.

Statistical analysis was done using the SPSS 9.0 software and Fisher's exact test. The hypothesis tested was that the operated extremity had an equal likelihood to develop an unfavorable outcome (ulceration and/or amputation) as a non-operated extremity. If a patient had an ulceration precede an amputation, for the calculation, this patient was counted just as one amputation. If a patient had more than one toe amputated, the patient was counted as just one patient. If there were multiple ulcerations on the foot, or one on the dorsum and one on the plantar surface, the patient was just counted as one ulceration for statistical purposes.

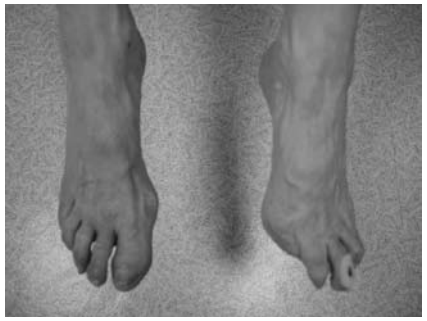
Just one of the 50 patients had an ulcer/amputation of the contralateral foot prior to the index surgery being done. This patient had the left big toe amputated prior to having the surgery on the right foot, not previously ulcerated, foot.

RESULTS

Among the 50 patients, there were no ulcerations and no amputations on the foot that had the peripheral nerve decompressions. In contrast, there were twelve patients with ulcerations and another three patients with amputations that occurred on the contralateral, or non-operated foot. This difference was significant at the $P < 0.001$ level.

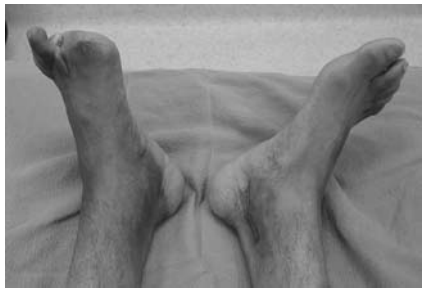
Two patients with examples of these problems are illustrated in **Figures 1 and 2**. The patient in **Figure 1** is the one patient who had the big toe amputated on the left foot at a time distant from the time of the surgery on the right foot, and then developed the dorsal ulceration and new 2nd metatarsal ulceration on the left foot. In **Figure 2** is a patient who had the right foot operated on 7 years before developing an ulcer and subsequent amputation of first and second toes on the contralateral foot.

Figure 1



The left foot of this patient with diabetes had an amputation of the big toe prior to the decompression of the peripheral nerves in the right foot. Subsequent to the nerve decompression surgery, the left foot developed the dorsal and plantar ulcers demonstrated.

Figure 2



The right foot had surgery to decompress peripheral nerves seven years before the patient developed the ulcers and amputations in the contralateral left foot.

DISCUSSION

The results of this study suggest that the natural history of the most common form of diabetic neuropathy, the distal, bilaterally symmetrical, polyneuropathy, can be changed in terms of the impact of improved sensibility upon the development of ulcer and amputation.

The health care cost savings that can be anticipated based upon the results of this study will require actuarial analysis to calculate the full magnitude. Included in these savings will be reduced cost of medication for neuropathic pain, reduced costs for admission to the hospital for foot infection, reduced costs for treatment of foot ulcers, reduced costs for amputation and provision of prosthesis, crutches, and wheel chairs. The reduction in health care costs can be extrapolated to a reduction in treatment of falls, as with improved sensibility should come improved balance and a measurable decrease in fractures from falls. Similar health care cost analyses for diabetes have been done, and that methodology could be applied to the outcomes just described.[34, 58-61] Restoration of sensation to the feet of patients with symptoms of diabetic neuropathy will provide a paradigm shift in health care costs related to the estimated care of about 8 million Americans with this complication.

A further comment is warranted with regard to prevention of recurrent ulceration/amputation. In the two studies reported in which thirty patients had ulceration/amputation prior to the surgical decompression of their peripheral nerves,[48,49] just one of the thirty patients had a recurrence of the ulceration (3.3%). Recurrent ulceration after wound healing was reported a decade ago to be 70% [62] and, sadly, the most contemporary reports, using current foot wear and methodology, report the recurrence rate to be still in that same range.[63-65] These historical observations support, theoretically, permit the extension of the observations of the present study to patients having nerve decompressions who have a history of a previous ulcer/amputation in the foot having this decompression surgery. Although patients having the surgery in the present study did not have a previous ulcer/amputation in the operated extremity, it is clear from this historical review that a reduction in recurrent ulcer/amputation from a 70% to a 3% level again would impact heavily in the health care cost paradigm shift related to the concept of decompression of peripheral nerves for the symptoms of diabetic neuropathy.

The Plastic Surgery community is probably not aware, in general, of the world goal of decreasing the rate of amputation in patients with diabetes by 50%. In 1990, the European community set this as a goal to be achieved within a decade.66 In the United States, the Healthy People 2000, National Health Promotion and Disease Prevention Objectives, published in 1990, gave as Objective 17.10, the reduction of lower extremity amputations from 8.2/1000 (1987 baseline) to 4.9/1000 by the year 2000, a targeted 40% reduction.[67] The methodology to achieve this goal was “proper foot care, and reducing risk factors such as hyperglycemia, cigarette smoking and high blood pressure.” The 1995 “Midcourse Review” simply restated

the above, without giving any data on the status of this objective.[68] The Healthy People 2010 Objectives, submitted for public comment in September of 1998, simply repeated the same objectives of the 1990 initiative, suggesting that either no progress was made or that no data was available on the subject.[69] Boulton, Connor, and Cavanagh, in the conclusion of the third edition of their book, The Foot in Diabetes, published in 2000, conclude that these goals are not being achieved.[69] Indeed, their review of the United Kingdom, European, and Scandinavian data demonstrate the amputation rate is either static or increasing, and the absolute number of amputations is increasing. These experienced diabeticians and epidemiologists continue to champion improved "systems of organization…at the clinic and district level" to achieve patient education and appropriate use of footwear. It is precisely here that we may observe where the impact of peripheral nerve decompression can be of enormous value. When the primary care doctors for the foot, regardless of which specialization that may be, begin to implement standardized measurements to assess impairment of sensibility, then, in addition to the usual educational process for foot protection, it would appear, based upon the results of the study presented here, that referral of appropriate patients for restoration of sensibility by nerve decompression would be appropriate. Adding the surgical intervention to the system of medical care holds the promise of reversing the most significant etiologic factor in the pathogenesis of the historic progressive and irreversible natural neuropathy of diabetes, thereby effectively preventing ulceration and amputation.

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Chapter 10

Discussion:
Global confirmation

DISCUSSION: GLOBAL CONFIRMATION

The sustained, quarter-century of research reported in the preceding chapters has brought to fruition the concept that decompression of multiple peripheral nerves in the lower extremity can restore sensation to the foot, can relieve pain in the foot, and, as a result of restored sensation, can prevent ulceration and amputation in patients with diabetic neuropathy. The health care consequences of these results are of major impact to the peoples of the world.

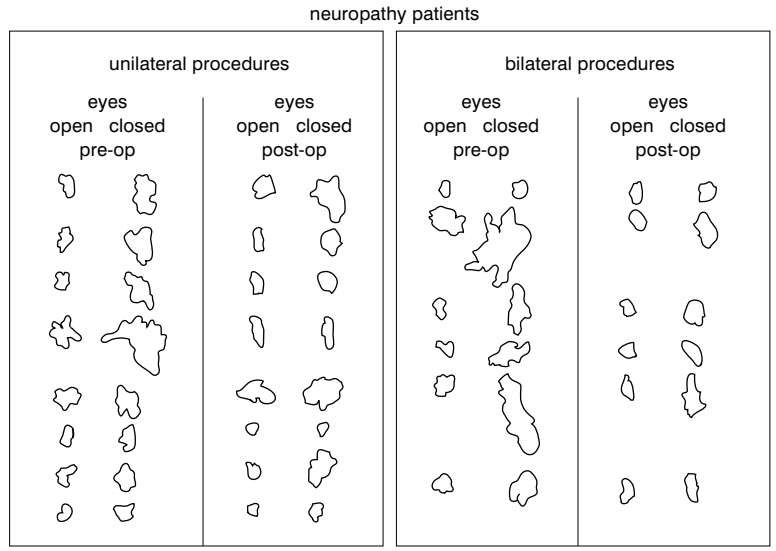
Each preceding chapter has a discussion of its own particular implications. It seems most important, therefore, for this general discussion to be more forward looking and to attempt to answer several questions raised by the preceding body of work. Such questions include: 1) What is the logical conclusion related to the balance study reported in Chapter 8? 2) Have other surgeons been able to duplicate my work with respect to the basic science of neuropathy and the clinical success? 3) Does the success with diabetic patients translate into success for other types of neuropathy? 4) How can doctors and patients be educated about these observations and results?

RESTORATION OF BALANCE: CONCLUSION

In Chapter 8, a method of documenting balance related to the sway of a patient with eyes closed versus eyes open was described. Loss of balance was correlated with the progressive loss of sensation in patients with neuropathy. The logical next question to ask is “will balance be restored to patients with neuropathy after the lower extremity peripheral nerve decompression surgery” that was described previously in Chapter 4. In a follow-up study that has just been published,[1] 14 patients with neuropathy who had peripheral nerve decompression were re-evaluated with the sway test to determine if the changes in post-operative sensibility in the foot resulted in an improvement in their balance. Of these 14 patients, 8 had just one side decompressed, while 6 patients had bilateral decompressions. The raw data from their sway testing is given in **Figure 1**.

Using data from the Pressure-Specified Sensory Device™, that was used to measure sensibility before and after the nerve decompression procedures, the sensation was significantly ($P < .001$) improved following the surgery. This sway data shows that the majority of the patients have a decrease in sway, meaning an improvement in their balance (note the area of the sway pattern is decreased with eyes closed after surgery.)

Figure 1



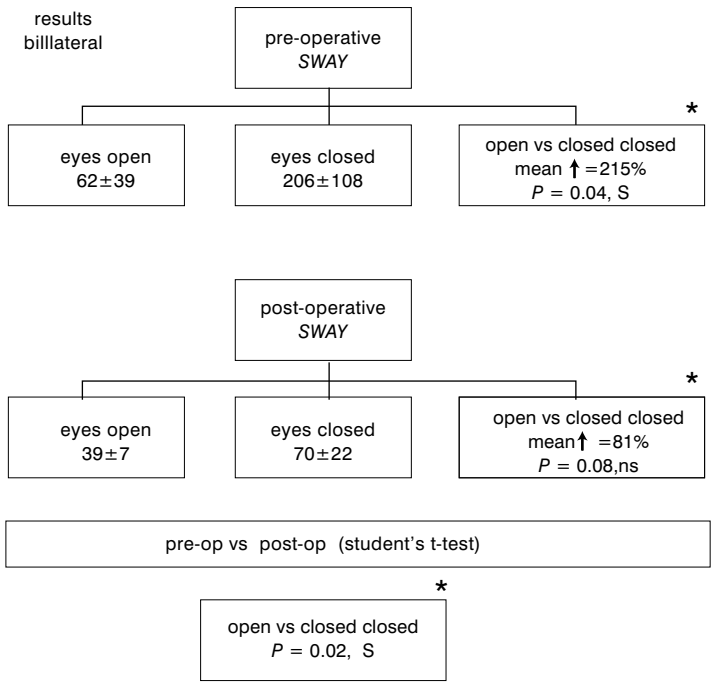
Improvement in sway following decompression of lower extremity peripheral nerves in patients with neuropathy. The closer the “eyes open” and “eyes closed” surface area tracings are to each other, the better is the balance. Patients with bilateral decompressions achieved better balance than those with unilateral surgery.[1]

The results statistically for the group with bilateral decompressions is given in **Figure 2**. It is clear from this work that, as is logical, if sensation is restored, balance is restored as well, and that if this can be achieved for both feet, the improvement in balance should be better than if it were just achieved in one foot. And that is what this study demonstrated.[1] Improved balance will translate into less falls with less hip and wrist fractures. These falls are a significant source of morbidity for the patient population with neuropathy. This study now suggests that rehabilitation in terms of gait and balance training is something to be added to the post-operative regimen of these patients.

GLOBAL CONFIRMATION OF THIS WORK.

Confirmation of the **basic science** work done on diabetic rats described in Chapter 5 was reported by Yuksel’s group from Turkey[2], who extended my observations to include adding an internal neurolysis to the decompression of the diabetic rat’s

Figure 2



Results of the study demonstrating that decompression of peripheral nerves bilaterally will improve balance significantly. This prevents falls, preventing fractures.

sciatic nerve. The internal neurolysis relieved the intraneural pressure and significantly improved the rat's ability to walk when compared to simple decompression alone. This basic diabetic rat compression/decompression model was utilized by Siemionow's group at the Cleveland Clinic not only to confirm the improved gait after decompression but also to demonstrate, for the first time, improved motor function by direct measurement of muscle strength.[3] Thus, the basic principles first described by our work in 1991[4] and 1994[5] has been confirmed by two independent investigators more than a decade later.

Table 1 gives a list of the current publications that have **clinically confirmed** the results of my first study published in 1992[6] documenting improvement in sensory function after peripheral nerve decompression. Some of the studies are subse-

Table 1
Diabetic neuropathy: Results of posterior tibial nerve decompression

	number of		Improvement	
Study	**Patients**	**Nerves**	**Pain**	**Sensibility**
1992, Dellon[6]	31	22	85%	72%
1995, Wieman & Patel[10]	33	26	92%	72%
2000, Caffee[11]	58	36	86%	50%
2000, Aszmann, Kress & Dellon[7]	16	12	n.a.%	69%
2001, Tambwekr[12]	10	10	80%	70%
2003, Wood & Wood[13]	33	33	90%	70%
2004, Biddinger & Amend[14]	15	22	86%	80%
2004, Valdivia, Weinand & Maloney[8]	60	60	85%	85%
2004, Lee & Dellon[9]	46	46	92%	92%
2005, Nelson & Little[15]	6	6	86%	86%
2005, Steck[16]	25	25	84%	72%
2005, Rader[17]	49	49	90%	75%
2005, DiNucci[18]	36	36	80%	80%
2005, Yao[19]	70	70	95%	95%
2006, Siemionow, et al[20]	37	37	90%	90%
Totals	**516**	**464**	**88%**	**79%**

quent reports of patients that I have operated on, such as the one listed as Aszmann, et al, in 2000.[7] That study was critically different from my 1992 study, in that, although I did the surgery, the post-operative measurements were made by a second author, Kress, blinded as to which side had the surgery, and the results were tabulated by someone other than the surgeon, Aszmann. For the study listed as Valdivia, et al [8] although my name is on the paper, the surgery was not done by me, but by another Plastic Surgeon, Maloney, and the data was analyzed by Valdivia. The study by Lee and Dellon, is a group of patients that I operated on, but the results were determined by an independent person, Lee, evaluating my patients post-operatively.[9] Therefore, I believe it is legitimate to include these two

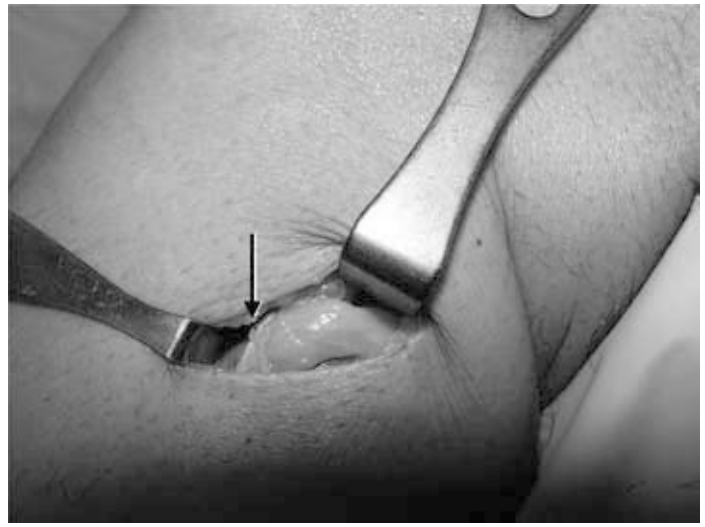
studies in this list of studies that do confirm the efficacy of the surgery that I introduced.

An important aspect of this review of confirmatory publications is that the surgeons doing the decompressions include Plastic Surgery,[6-9,11,12,20] Orthopedic Surgery,[14] Neurosurgery,[19] General Surgery,[10] Podiatric Foot and Ankle Surgery,[13,15-18] and the countries reporting results so far include China[19] as well as the United States.

EXTENSION OF THIS CONCEPT TO OTHER FORMS OF NEUROPATHY

While diabetes is the most common form of diabetes, the American Neuropathy Association estimates that there are as many people in the United States with neuropathy of unknown etiology as there are with diabetic neuropathy. This form is called **idiopathic neuropathy**. When I began this work, patients with painful neuropathy would seek help. If they had abnormal large fiber function, as determined by the Pressure-Specified Sensory Device, to document that they had a mixed, rather than a pure small fiber neuropathy, then I would consider them for surgery. In December of 2004, my work on the relationship of a positive Tinel sign to a successful outcome from decompression surgery in the lower extremity included patients with idiopathic neuropathy.[8] If there was a positive Tinel sign, the success rate in the 40 patients in that series was 80% relief of pain and restoration of sensation. In the large series of patients with neuropathy reported by Valdivia, et al, in September of 2005, the success rate was 90% relief of pain and restoration of sensation in patients with idiopathic neuropathy who had a positive Tinel sign.[9] Again, in the report by Siemionow, et al, in press in 2006, there was a 90% success rate in the 12 patients with idiopathic neuropathy.[20] This group of patients contains many patients who appear to be type II diabetic, but whose serum glucose is within normal range. In my early training in medicine, these people would have been termed “borderline or pre-diabetic. To me this implied they had resistance to glucose entering the cell, and a should have a high insulin level. Investigating this, I found it to be true, and described, in 1999, the first series of patients with hyperinsulinemia and neuropathy.[21] If specifically asked, many of these patients have a positive family history for diabetes, have hypertension and hypercholesterolemia, and have a BMI > 25. Today, these are the criteria for **metabolic syndrome**.[22,23] These people, if studied, will be found to have hyperinsulinemia, and insulin resistance. In time, many of these patients will develop diabetes, but at the time they present with painful neuropathy for decompression (to have a Dellon Triple Procedure), they will be termed idiopathic neuropathic. If they have a positive Tinel sign, they have an excellent chance to be helped (**Figure 3**)

Figure 3



The common peroneal nerve in the left leg of a woman with idiopathic neuropathy who fits the criteria for metabolic syndrome. The fibrous band deep to the peroneus longus muscle is the site of compression (arrow, and see Chapter 2). Note the yellow color of the common peroneal nerve due to fat infiltration, and the swelling of the nerve proximal to the compression.

Another important group of patients who can be helped are those with a **Chemotheraphy-induced neuropathy**. In the last study I did with rats, they received cisplatin chemotherapy.[24] Using the walking track assay, they were shown to develop neuropathy. Decompression of the tarsal tunnel in these rats restored a normal walking track pattern. I then began to apply this technique to patients with neuropathy related to chemotherapy.[25] The drugs known to have neurotoxicity are those in **Table 2**

Table 2
Chemotherapy drugs that induce neuropathy

Vincristine
Cisplatin (platin family)
Taxol
Thalidomide

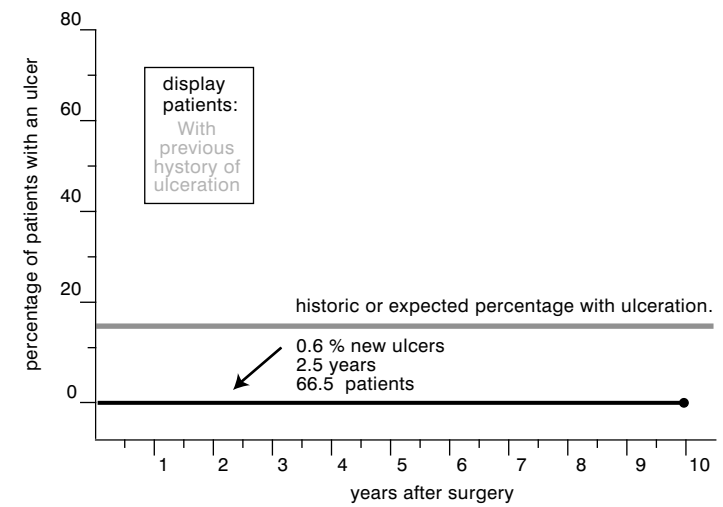
We have found the same thing for chemotherapy-induced neuropathy as we have for diabetic neuropathy; if there is a positive Tinel sign, then there will be a compressed nerve that can benefit from a neurolysis (**Figure 4**). We have now applied this concept to the newest drug for multiple myeloma, thalidomide.[26]

Since the disabling progressive component of **leprosy** is due to the immune reaction to the bacteria within the peripheral nerve, my most recent application the Dellon Triple Procedure was in patients with Hansen's Disease in Ecuador in September of 2004. The follow-up team who visited there in July of 2005 documented improvement in many of these patients. The first publication on this concept, reporting the use of Pressure-Specified Sensory Device to document and stage leprous neuritis, appeared recently.[27]

Figure 4

Left: Note indentation (arrow) in common peroneal nerve of left leg after removal of fibrous compressive band in a woman who had received cisplatin for ovarian cancer. At 4 years post-chemotherapy, she was disabled due to leg pain and weakness. The swelling proximal to the compression is clear. Right: 6 months after the Dellon Triple Decompression had been done on both legs, recovery of function.

Figure 5



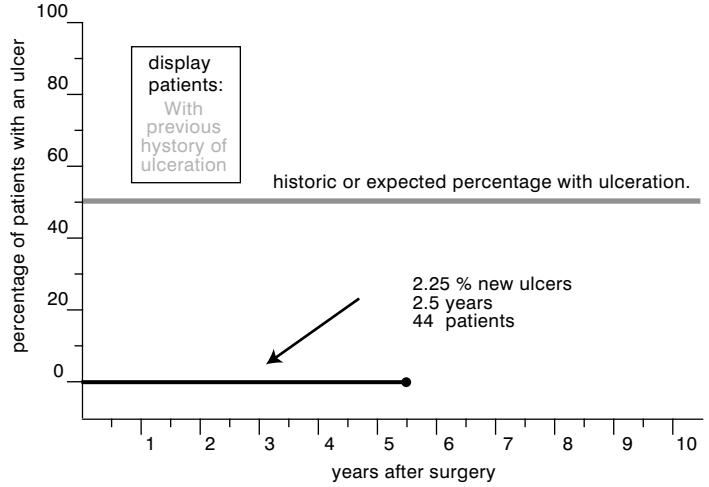
The expected incidence of ulceration in diabetics with neuropathy is 15% (red line). In 665 patients with diabetes who had had the Dellon Triple Procedure, and who do not have a previous history of ulceration, the observed incidence of ulceration at 2.5 years is 0.6%. This is a dramatic change in the natural history of diabetic neuropathy.

EDUCATION OF THE PHYSICIAN AND PATIENT

Finally, I believe I have an obligation to bring these important observations and results to the awareness of physicians, both medical and surgical, and patients. Medicine is conservative, traditional, and slow to change, which is good except where that inertia stands in the way of bringing new hope to patients who have been told for too long by their physicians that there is not treatment available for their neuropathy symptoms. Unfortunately, this is still a problem with the literature on neuropathy today.[28] The internet is probably the best way today to make information available to the public, patient and physician alike. Therefore, in 2003, I initiated a tax-exempt charitable foundation, the Diabetic Neuropathy Foundation of the Southwest (neuroapthysouthwest.org), and through this Foundation began the International Neuropathy Decompression Registry (neuropathyregistry.com). This site is a prospective multicenter trial of the Dellon Triple Procedure for neuropathy. If the visitor to the site clicks on "Statistics", information is now available from 34 surgeons who have entered more than 1300 operations on 800 different

patients on the following outcomes: relief of pain, restoration of sensation, decrease in drugs, recovery of balance appear, prevention of amputation, prevention of ulceration. In this concluding section, I would like to just demonstrate the health care savings shift of this approach to patients with diabetic neuropathy. **Figure 5 and 6** demonstrate the results through January 2006.
It is my goal to continue to educate both the physicians and the patients of the world about the new optimism for this treatment of neuropathy. A “new-ropathy”.

Figure 6



Expected incidence or recurrent ulceration in diabetics with neuropathy is 50% (red line). The observed incidence in this group of patients who have had a Dellon Triple Procedure is 2.25% at 2.5 years. A dramatic health care cost paradigm shift.

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Chapter 11

Summary

Summary in Dutch

NEDERLANDSE SAMENVATTING

Selected bibliography

Summary of curriculum vitae

Acknowledgements

Reviewing committee

SUMMARY

Diabetic neuropathy will affect at least half of all diabetics, at a time when an epidemic in diabetes is occurring world-wide. Diabetic neuropathy predictably causes loss of protective sensation, then ulceration, and then amputation. In the United States, 15% of diabetics develop an ulcer, and about 85,000 amputations occur per year unrelated to vascular occlusion. Health care costs related to neuropathy are enormous.

Over the past 25 years, my research has demonstrated that diabetes makes the peripheral nerve susceptible to chronic compression, and that decompression of nerves at known sites of anatomic narrowing can restore sensory and motor function. Indeed, restoring sensibility to the foot will prevent ulceration and amputation. This work required basic anatomy studies to elucidate the sites in which peripheral nerves were at risk for chronic compression in the upper and lower extremities. This work also required creation of models of chronic nerve compression, double crush injury to nerves, basic human anatomic studies, and study of diabetic rats, monkeys, and humans. This work has been confirmed by investigators in Austria, China, Turkey, and within the United States. There are published now more than 12 human outcome studies confirming that sensation can be recovered, and pain relieved, by decompression of the common peroneal, deep peroneal, and the branches of the tibial nerve in the four medial ankle tunnels, which is the operative approach I first published in 1992.

The approach to the patient is to do non-painful neurosensory testing with the Pressure-Specified Sensory Device (PSSD). This documents the presence of neuropathy, and stages the degree of compression. The traditional nylon 5.07 monofilament only gives an estimate of the latest (most advanced) stage, loss of protective sensation. The PSSD identifies the earliest time of axonal loss. At this time, if there is a positive Tinel sign located over the known site of compression, our data indicate a 90% chance of success for surgical decompression. In a study where only unilateral surgical decompression was done in 50 patients followed an average of 2.5 years, no ulcers or amputations occurred on the decompressed side, whereas 12 ulcers and 3 amputations occurred on the non-operated side ($P < .001$). This study demonstrated that the natural history of diabetic neuropathy can be changed by appropriate surgical intervention.

A prospective multi-centered trial is available on the internet at neuropathyregistry.com. To date, the ulceration rate in 665 patients has been reduced from the expected 15% to the observed 0.60%. The recurrence of ulcers in patients who had a previous ulceration has been reduced from the expected 50% to the observed 2.25%. It is hoped that the Dellon Triple Decompression procedure can be introduced into widespread practice. More details for public information are now available at DellonInstitutes.com.

SUMMARY IN DUTCH (Nederlandse samenvatting)

Neuropathie ten gevolge van diabetes komt bij minstens de helft van alle diabetici voor nu diabetes wereldwijd epidemische vormen heeft aangenomen. Deze diabetische neuropathie veroorzaakt verlies van protectieve sensibiliteit met grote kans op ulceratie en uiteindelijk amputatie van de onderste extremiteit.
In de Verenigde Staten ontwikkelen 15% van de diabetici ulceraties en er vinden jaarlijks ongeveer 85.000 amputaties plaats die niet gerelateerd zijn aan een vaatafsluiting.

Gedurende de afgelopen 25 jaar heb ik met mijn onderzoek aangetoond dat diabetes de perifere zenuw gevoelig maakt voor chronische druk en dat opheffing van deze druk op zenuwen ter plaatse van een anatomische vernauwing, de sensorische en motorische functie kan herstellen. Uiteraard wordt door het herstel van de sensibiliteit van de voet de kans op ulceratie en amputatie verminderd.
Het beschreven onderzoek vergde een uitvoerige studie van de anatomie om die plaatsen te lokaliseren, waar het risico voor chronische compressie van de perifere zenuwen in de onderste extremiteit het grootst is. Voor dit werk was het tevens noodzakelijk experimentele modellen te ontwerpen waarbij zenuwen konden worden blootgesteld aan chronische compressie en "*double crush*" letsel. Daarnaast werden specifieke studies van de menselijke anatomie verricht en diabetes bij ratten, apen en mensen bestudeerd.
De uitkomsten van deze studies werden bevestigd door onderzoekers in Oostenrijk, China, Turkije en in de Verenigde Staten. Inmiddels zijn er meer dan 12 "*human outcome studies*" verricht, welke bevestigen dat de sensibiliteit zich kan herstellen en de pijn verlicht kan worden door decompressie van zowel de nervus peroneus communis, de nervus peroneus profundus en de vertakkingen van nervus tibialis in de vier mediale enkeltunnels, over welke operatieve techniek ik voor het eerst gepubliceerd heb in 1992.

Het onderzoek van de patiënt vindt plaats met behulp van het zogenaamde "Pressure-Specified Sensory Device" (PSSD), dat een niet pijnlijk neuro-sensorisch onderzoek mogelijk maakt. Dit onderzoek documenteert de aanwezigheid van neuropathie en geeft de mate van compressie aan.
Het gebruik van het klassieke nylon 5.07 monofilament geeft slechts een indruk over het laatste (meest gevorderde) stadium, namelijk het verlies van protectieve sensibiliteit. Met het PSSD wordt daarentegen het begin stadium van axonaal verlies aangegeven. Als er een positief Tinel signaal gelocaliseerd wordt op de bekende compressie punten kan op basis van de verkregen data worden gesteld dat door middel van chirurgische decompressie een kans op 90% verbetering van de klachten kan worden verkregen. In een onderzoek bij 50 patiënten, die gemiddeld 2,5 jaar waren gevolgd en waar slechts een eenzijdige chirurgische decompressie had plaatsgevonden, kwamen geen ulcera of amputaties aan de zijde van de decompressie voor, terwijl er aan de niet-geopereerde zijde 12 ulceraties en 3 amputaties voorkwamen ($P < .001$). Dit onderzoek toonde aan dat het ziekteverloop van de diabetische neuropathie veranderd kan worden door een juiste chirurgische interventie.

De resultaten van een prospectief onderzoek in meerdere centra is op internet beschikbaar op www.neuropathyregistry.com. Tot op heden is bij 665 patienten het verwachte optreden van ulceraties verlaagd van 15% naar 0.60%. Het opnieuw optreden van ulceraties bij patiënten waarbij reeds eerder van een ulceratie sprake was, werd teruggebracht van 50 % naar 2.25%. Het is te hopen dat de "*Dellon Triple Decompression*" methode algemeen aanvaard en in praktijk gebracht zal gaan worden. Meer details en informatie is beschikbaar op www.DellonInstitutes.com.

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SUMMARY OF CURRICULUM VITAE

A. Lee Dellon, M.D. graduated from Johns Hopkins University in 1966 and from the Johns Hopkins School of Medicine in 1970. He then completed eight years of additional training, including two years of research at the National Cancer Institute, Surgery Branch, of the National Institutes of Health. He completed a Plastic Surgery Residency at the Johns Hopkins Hospital and a Hand Surgery Fellowship at the Raymond M. Curtis Hand Center, both in Baltimore. Doctor Dellon has received the Certificate of Added Qualifications in Hand Surgery and is Board Certified in Plastic Surgery. He is currently a Professor of Plastic Surgery and a Professor of Neurosurgery at the Johns Hopkins University School of Medicine, Clinical Professor of Plastic Surgery, Neurosurgery and Anatomy at the University of Arizona.

Doctor Dellon’s research interests center on neural regeneration. In the basic research laboratory, his work included models for peripheral nerve compression, neuroma treatment, neural regeneration through absorbable conduits, and diabetic neuropathy. Doctor Dellon’s clinical work is focused on computer-linked devices to measure sensibility, treatment strategies for pain due to neuroma, use of bioabsorbable tubes as a substitute for nerve grafts, treatment of facial pain and of groin pain, and treatment of the symptoms of peripheral neuropathy, whether due to diabetes or unknown causes.

He has won 22 national research awards, including the Radium Society Award in 1974, the Cleft Palate Award in 1977, and the Emanuel Kaplan Hand Surgery Award in 1985. Educational Foundation Awards from the American Society of Plastic and Reconstructive Surgery include those for the immunobiology of skin cancer, prediction of recurrence in non-melanoma skin cancer, partial-thickness skin excision for treatment of benign dyskeratosis, surgical treatment of symptoms of diabetic neuropathy, neurosensory testing, nerve decompression in leprosy, and partial joint denervation. Doctor Dellon is the author of four books, 75 book chapters, and more than 400 articles published in peer-reviewed journals. He is on the Editorial Boards of Journal of Reconstructive Microsurgery, Journal of Clinical and Experimental Plastic Surgery, and The Journal of Hand Surgery. He has been on the Editorial Boards of Plastic and Reconstructive Surgery, Annals of Plastic Surgery, Microsurgery, Peripheral Nerve Regeneration and Repair, Journal of Foot and Ankle Surgery, Journal of Hand Therapy, and Journal of the American Podiatric Medical Association. Doctor Dellon is a founding member and past president of

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