

Prevention of Foot Ulceration and Amputation
By Decompression of Peripheral Nerves
In Patients with Diabetic Neuropathy

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Decompression of Peripheral Nerves; Treatment for Symptoms of Diabetic Neuropathy

Abstract

Diabetic neuropathy occurs in a stocking and glove distribution, consistent with a systemic metabolic disease. This concept has led to the conclusion that there is no role for surgery in this debilitating condition. The classic medical approach to the treatment of diabetic neuropathy is an attempt to achieve a euglycemic state, to obtain regular Podiatric care of the feet, to inspect the feet regularly for the presence of deformity, ulcer and infection, and to help the patient achieve pain control with neuropathic pain medications. Unfortunately, all too often this approach is not successful in relieving the patient's pain, and the loss of sensation provides the basis for infection, ulceration and amputation. The purpose of this article is to review the basic scientific and clinic research that support the statement that "today it is possible to restore sensation and relieve pain in 80% of patients with symptoms of diabetic neuropathy by decompression of multiple peripheral nerves". Furthermore, no one who has had sensation restored with this procedure has developed an ulcer, thereby changing the natural history of diabetic neuropathy.

PATHOPHYSIOLOGIC BASIS FOR SUSCEPTIBILITY TO COMPRESSION OF THE PERIPHERAL NERVE IN DIABETES

There are two metabolic changes in the peripheral nerves of the diabetic that render the nerve susceptible to chronic compression. The most critical is the increased water content within the nerve as the result of glucose being metabolized into sorbitol.¹ This increased water content causes the nerve to have an increased volume. The second metabolic change is a decrease in the slow anterograde component of axoplasmic flow.² This component of axoplasmic flows transports the lipoproteins necessary to maintain and rebuild the nerve. The peripheral nerve, as it crosses a known areas of anatomic narrowing, like the carpal tunnel at the wrist, the cubital tunnel at the elbow, fibular tunnel at the outside of the knee, or the tarsal tunnel at the ankle passes through a region of increased external pressure. As the peripheral nerve in the diabetic has an increased volume, due to its water content, there is an increased pressure on the nerve in each of these anatomic regions.

This increased external pressure creates an increased intraneural pressure which decreases blood flow.³ This results in a relative ischemic condition for the peripheral nerve. The neurophysiologic consequence of decreased blood flow in a peripheral nerve is the perception of paresthesias, interpreted centrally as numbness and tingling. The chronic pathophysiologic result of this area of increased pressure along a peripheral nerve is demyelination. The peripheral nerve with decreased axoplasmic flow, as in the diabetic, cannot transport sufficient protein structures to rebuild itself. Additionally, advanced glycosylation end (AGE) products reduce the normal gliding ability of the peripheral nerve. The non-enzymatic binding of glucose to the collagen within the nerve and in the epineurium are the basis for this decreased elasticity. The combination of this decreased elasticity in the peripheral nerve, combined with the nerve's normal physiologic requirement to stretch as it glides across joints, increases the stress and strain of the peripheral nerve within these areas of known anatomic narrowing. This increasing tension along the nerve further decreases blood flow within the nerve.⁴

The hypothesis that the peripheral nerve in the diabetic has an increased susceptibility to compression has been tested in the rat model.⁵ Rats were made diabetic by being given streptozotocin. Silicone bands were placed about the sciatic nerve in these rats, and also in a group of non-diabetic, age-matched rats. Electrophysiology was tested in both groups after 6 months of banding, as this had been demonstrated to be sufficient time to develop electrophysiologic and histologic changes consistent with chronic nerve compression in this model.⁶ It was found that the diabetic rat had a statistically significant lower conduction velocity and a statistically significant lower amplitude for the sciatic nerve measured across the region of compression than did the non-diabetic banded rat, confirming that the peripheral nerve has an increased susceptibility to chronic nerve compression in the diabetic rat.

RELATIONSHIP BETWEEN CHRONIC NERVE COMPRESSION AND THE SYMPTOMS OF DIABETIC NEUROPATHY.

The patient with diabetic neuropathy has symptoms that include sensory complaints, like numbness and tingling, pain, loss of sensation, and motor complaints, like weakness. The motor complaints extend to the autonomic system as well, and, in the extremities include loss of sudomotor function, so the skin becomes dry and thick. The sensory symptoms occur in a distribution that has been called "stocking and glove", with the symptoms being worse in the lower extremity than the upper extremity. In contrast the patient with a single chronic nerve compression, will have these same symptoms, however they will occur only in the distribution of that particular nerve. For example, the patient with carpal tunnel syndrome, with median nerve compression, will only have the sensory complaints in the palmar aspect of the thumb, index and middle finger, and will only have weakness in the muscles that control part of the thumb's function.⁷ In the upper extremity, chronic compression of the ulnar nerve at the elbow will result in paresthesias in the palmar and dorsal surfaces of the ring and little finger, and weakness of pinch and grip strength. In advanced cases of ulnar nerve compression, there is intrinsic muscle weakness, which creates a "claw" deformity of the hand. Ulnar nerve compression at the elbow can be decompressed surgically.^{8,9} If the radial sensory nerve were compressed in the forearm, there would be numbness over the remaining skin surface of the hand, the dorsoradial skin. The radial sensory nerve can be decompressed surgically, too.¹⁰ If a person were to have chronic compression of the median, ulnar and radial nerves, that person would have a glove distribution of numbness, and have symptoms indistinguishable from the patient with symptomatic diabetic neuropathy of the upper extremity.

This same relationship applies to the lower extremity. Compression of the sciatic nerve's common peroneal nerve at the lateral aspect of the knee occurs in the fibular tunnel. The symptoms include paresthesias or pain from the knee to the top of the foot. The motor component, when complete results in a "drop foot", just as complete compression of the motor branch of the radial nerve, at the elbow, results in a "drop wrist". More commonly, in the leg, there is weakness of the long toe extensor, so this toe is positioned lower than the other toes, and is weak on manual muscle testing.¹¹ Compression of the common peroneal nerve in this location requires a neurolysis by division of the fascial coverings above and below the peroneus longus muscle. Over the dorsum of the foot, the deep peroneal nerve can be entrapped between the extensor digitorum brevis and the junction of the first metatarsal and the cuneiform bone.¹² This entrapment is corrected by excision of the tendon of this small muscle, which is of no functional significance in the foot. Entrapment of the deep peroneal nerve is similar to the radial sensory nerve entrapment in the forearm. The analogy of the carpal tunnel syndrome in the foot is the tarsal tunnel syndrome.^{13,14} However, it must be realized that the tarsal tunnel is really the analogy of the human forearm, and therefore, to achieve restoration of sensation of all toes and the plantar aspect of the foot, the medial and lateral plantar nerves and the calcaneal nerve must each be released in their own separate tunnels, just distal to the tarsal tunnel. It is severe compression of the lateral plantar nerve that creates the hyperextended toes at the metatarsal phalangeal joints. These appear as

"hammer toes" in the diabetic, but are really "clawed toes", due to intrinsic muscle paralysis in the foot, as the "clawed hand" results from intrinsic muscle paralysis in the hand.¹⁵ Relief of paresthesias and pain in the foot, and often correction of the intrinsic muscle wasting, can be accomplished by decompression of the four medial ankle tunnels.¹⁶ If a person were to have chronic compression of the peroneal and tibial nerves, that person would have a stocking distribution of numbness, and have symptoms indistinguishable from the patient with symptomatic diabetic neuropathy of the lower extremity.

How can the physician identify compression of the peripheral nerve? The most reliable clinical finding of a nerve compression is tenderness of the nerve at the site of anatomic narrowing. This sensitivity of the nerve at the site of chronic compression may be manifested simply by the nerve being tender at that site, but most often is manifested by a distally radiating paresthesia in the distribution of the nerve when the nerve is gently percussed. This is referred to as a positive Tinel sign.¹⁷ In a patient with a neuropathy, where a systemic cause exists for the nerve dysfunction, there should be no localizing sign along the course of the peripheral nerve. However, if the neuropathy causes the nerve to be susceptible to nerve compression, then there can be superimposed compression of the peripheral nerve in addition to the underlying neuropathy. Traditionally, electrodiagnostic testing is used to make the diagnosis of peripheral nerve compression, neuropathy, or nerve root compression. There are many times, however, when the peripheral neuropathy is so advantaged that there is no conduction measurable in the peripheral nerve, or the conduction velocity and amplitude are already so reduced, that identification of a superimposed nerve compression in the patient with neuropathy is just not possible technically. In these situations, the physical examination is critical in making this distinction.

TREATMENT OF NEUROPATHY

The treatment of the symptoms of neuropathy presupposes the doctor knows the cause of the neuropathy. If the neuropathy is due to lead poisoning, then chelation of the lead would be the starting point. If the neuropathy is due to a vitamin deficiency, then dietary supplementation with that vitamin is the treatment. If the neuropathy is secondary to hypothyroidism, then thyroid replacement is indicated. The physician can identify the etiology of the neuropathy in each of these cases by appropriate laboratory tests. Treatment of the metabolic problem corrects the cause of the neuropathy and is usually sufficient to relieve the patient of their symptoms.

Laboratory tests can identify the patient with diabetes, and can identify the patient who is not maintaining themselves in glycemic control. Unfortunately, even patients maintaining strict glycemic control, with frequent daily monitoring of their blood glucose, may develop the symptoms of neuropathy. The treatment of the symptoms of neuropathy in the patient with diabetes, in addition to the maintenance of glycemic control, today relies upon a combination of non-narcotic neuropathic pain medications and, when these are insufficient, upon narcotics. The classic triad of neuropathic

medications included Tegretol, Dilantin, and Elavil. Many patients are unable to tolerate the side effects of Tegretol, and Dilantin was often not effective. Because many patients with neuropathy have trouble sleeping, Elavil, whose side effect is most commonly drowsiness, was often the most effective of these three. Currently, Neurontin has become the drug of choice for the treatment of the symptoms of neuropathy. Unfortunately, many patients do not tolerate the doses required of non-narcotic neuropathic pain medications, or simply cannot accept the decrease in cognitive function these drugs induce.

For many patients with neuropathy, it is not so much the pain or the paresthesias that present a problem for them, as the loss of sensibility in their feet. This causes them to have loss of balance, be unable to perceive hot water in the bathtub, be unable to feel the pedals required for driving a car, and make them unsteady when walking down stairs. There is currently no medication available for these symptoms of sensory loss. It is also these symptoms that put the patient at risk for ulcer, infection and amputation. It is this general lack of ability to treat the symptoms of neuropathy in the majority of patients with diabetes that creates the sense of hopelessness in many of these patients, leading to depression and a sense of futility. Against this background, the concept that relief of symptoms of diabetic neuropathy, by decompression of superimposed peripheral nerve compressions, was introduced as a source of optimism for this difficult clinical problem.¹⁸

If the presence of a tight anatomic structure causes the symptoms of neuropathy in the diabetic, then the absence of such a structure should result in the diabetic not developing symptoms of neuropathy. This hypothesis, has, in fact, been tested in the streptozotocin-induced diabetic rat model.¹⁹ In that model, it was first demonstrated that a neuropathic walking track pattern occurred in the rat with diabetes. Then, a group of rats with serum glucose levels of 400 was followed for one year, or about half the rat's lifetime. One half of these rats, with streptozotocin-induced diabetes, had the tarsal tunnel decompressed prior to the onset of diabetes, and the other half were not treated by decompression of the tarsal tunnel, their anatomic site of compression was left intact. Both these groups of rats were then followed for one year with walking track analysis. The group without a tarsal tunnel had walking track patterns that were the same as weight-matched non-diabetic rats, while the diabetic rats with intact tarsal tunnels developed a progressive neuropathic walking track pattern (Figure 1). The results of this study suggested that in the rat model, in the absence of an anatomic site of narrowing, even the poorly controlled diabetic did not develop evidence of diabetic neuropathy in the feet. This study suggested that even if the underlying metabolic neuropathy could not be corrected medically, it might be possible to treat the symptoms that were related to superimposed chronic nerve compressions by decompression of the peripheral nerve. This research model has been applied recently to the neuropathy related to the chemotherapeutic drug cisplatin.²⁰ That study extended the observations made in the diabetic rat model in that decompression of the tarsal tunnel in the rat with cisplatin neuropathy restored a normal walking track pattern.

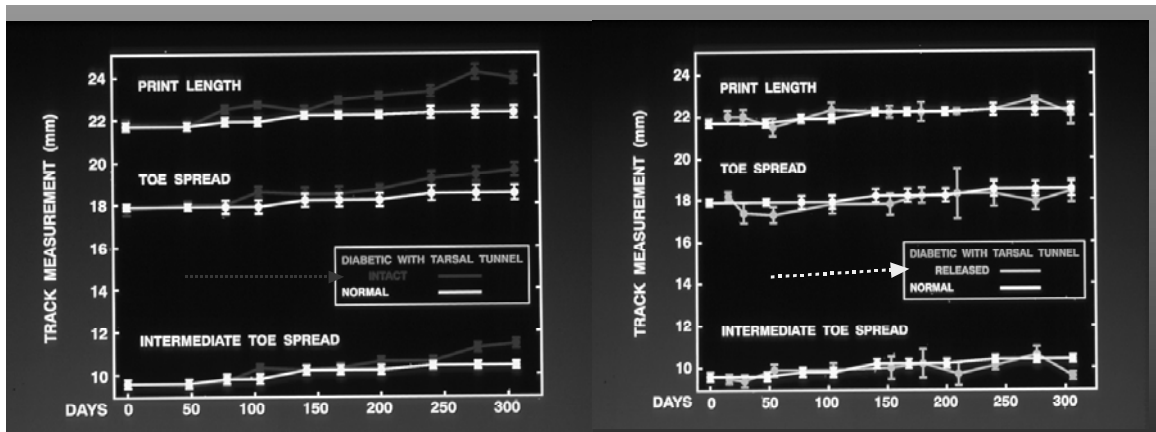


Figure 1. Diabetic rats, with serum glucose levels > 400, have had their print length measured from walking track analysis. An increasing print length occurs with progressive diabetic neuropathy. Control, non-diabetic, weight-matched rats have their print length recorded in both graphs. In A) the diabetic rats have had no surgery on their feet, and therefore have existing tarsal tunnels, which can serve as a site of nerve compression. Note that there is a significant increase in the print length with time, consistent with a progressive neuropathy. In B) the diabetic rats have had tarsal tunnel decompression surgery, so that the potential site for nerve compression no longer exists. In A) with time, there is a progressive increase in print length consistent with a progressive neuropathy. In B), where there is no potential site for nerve compression, the rats walking track pattern is not significantly different from normal.

SELECTION OF THE PATIENT FOR DECOMPRESSION

Over the past twenty years, an approach to selection of the patient for decompression of peripheral nerves has been developed. This approach begins with the measurement of peripheral nerve function in order to stage the degree of nerve impairment.²¹ The model developed for staging the degree of nerve impairment in patients with chronic nerve compression but without a neuropathy has been found to be valid in patients with neuropathy too. Since 1989, the approach to this measurement has been with computer-assisted sensorimotor testing. While vibrometry is useful for evaluating a single patient and comparing that patient to a group of patients, vibrometry does not help the surgeon interested in decompressing a particular nerve. This is because the vibration travels as a wave. If the vibration is not perceived well in the index finger, is it because of a lesion of the median or of the radial nerve? If the vibration is not perceived well in the big toe, is it because of a lesion of the tibial or of the peroneal nerve? While the Semmes-Weinstein monofilament number 5.07 (10 gm of force) may be useful in identifying an individual with diabetes who has lost protective sensation, and is therefore at risk of ulceration in the foot,²²⁻²⁹ this filament represents a cutaneous pressure threshold of greater than 90 gm/mm². At this advanced stage of chronic nerve compression, the patient has lost two-point discrimination, has severe axonal loss, and is most often past the point at which surgical intervention to restore sensation and relieve pain is still possible.³⁰

In contrast, we have found that the Pressure-Specified Sensory Device™ can identify the earliest degree of chronic nerve compression by measuring the pressure required to distinguish one from two points touching the skin. Normative values for the Pressure-Specified Sensory Device™ (PSSD)(Sensory Management Services, LLC, Baltimore, Maryland) have been reported for the upper extremity and for patients with carpal and cubital tunnel syndrome,³¹ and for the lower extremity and for patients with

tarsal tunnel syndrome.³² The PSSD is at least as sensitive as traditional electrodiagnostic studies,³³ and is not invasive and therefore not painful. No electric shocks are used.

The American Diabetes Association has indicated for the past seven years in its annual Standards of Care for the Foot in Diabetes that even the diabetic at low risk for ulceration should have a yearly somatosensory (quantitative sensory testing) measurement of the foot.³⁴⁻³⁶ A guideline, based upon a cross-sectional study of people with diabetes with and without foot ulceration,³⁸ is available for application of measurement with quantitative sensory testing for the diabetic foot. (Figure 2.) As the cutaneous pressure threshold for the big toe increases above the 99% confidence limit for normal (but axonal degeneration has not yet occurred), the patient with diabetes is referred in order first to a diabetes educator and Podiatrist for evaluation of orthotic use, and then to the Podiatrist for fabrication of special shoes. Once the 99% confidence limit is exceeded for the distance at which one from two points can be distinguished, indicating that axonal degeneration has occurred, then a referral to a surgeon knowledgeable in peripheral nerve decompression is appropriate to determine whether the patient would be a candidate for restoration of sensation and relief of pain.

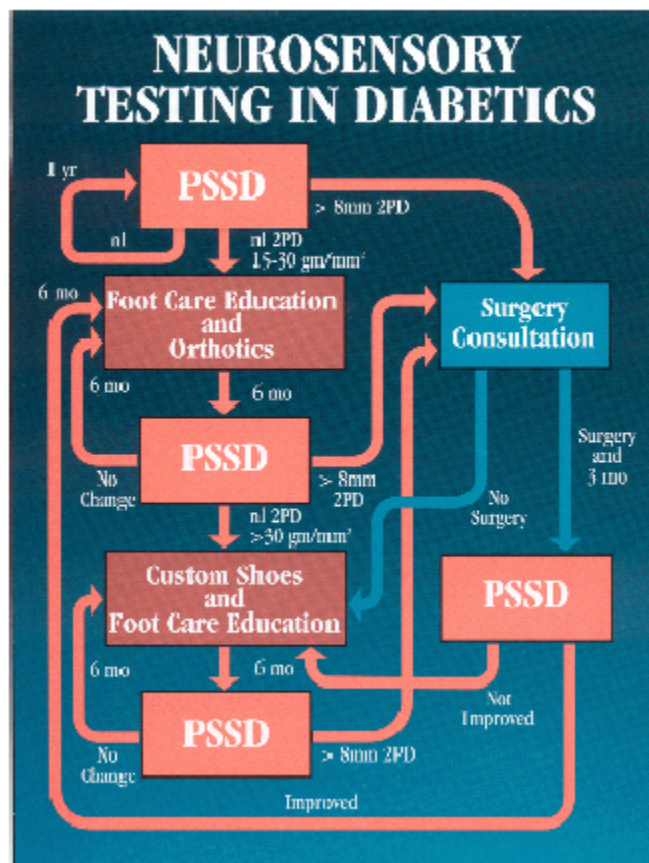


Figure 2: Guidelines, based upon neurosensory repetitive, paired, end-organ stimulation testing with the Pressure Specified Sensory Device™, for the treatment of the foot in the patient with diabetes

The most valid prognostic sign for a good result from decompression of a nerve in the diabetic with symptoms of neuropathy is the presence of a positive “Tinel sign”. This test is done by tapping the region of known anatomic tightness, like the tarsal tunnel, with the examiners finger (not with a percussion hammer). A “positive” test occurs when the patient can feel a radiating sensation, painful or not, into the territory supplied by that nerve, e.g., the arch of the foot, the heel, or the big toe when the percussion is done over the tarsal tunnel. The simple perception by the patient that a thumping occurred is *not* a positive sign. Tapping over several “control” sites, ie. areas of skin without a known anatomic region of compression beneath them, should be done. For the common peroneal nerve at the fibular head, often the nerve is just tender, and a distally radiating perception does not occur. Tenderness of this nerve is sufficient to suggest entrapment at this location. In my experience with patients with diabetic neuropathy, when a superimposed nerve compression is identified by a positive Tinel sign, there is an 80% chance of a good to excellent result, meaning relief of pain and restoration of sensation to the feet.

RESULTS OF DECOMPRESSION OF PERIPHERAL NERVES IN THE DIABETIC

Since 1992, there have been several studies that have evaluated the results of decompression of peripheral nerves in the diabetic. These studies have been reviewed, and their patient populations regrouped to permit comparison of nerve-specific results. These results are presented in Table 1 for carpal tunnel decompression, in Table 2 for cubital tunnel decompression, and in Table 3 for tarsal tunnel decompression.

Table 1 Results of Peripheral Nerve Decompression In Diabetic Neuropathy Median Nerve: Carpal Tunnel Syndrome							
Study	Number of:		Subjective Results:		Two-point discrimination		Ulcer Recurrence
	Nerves	Patients	Excellent	Good	Excellent	Good	
Dellon, 1992 ³⁸	44	34	96%	2%	96%	4%	0%
Aszmann, 2000 ³⁹	8	7	88%	12%	88%	12%	0%

Table 2 Results of Peripheral Nerve Decompression In Diabetic Neuropathy Ulnar Nerve: Cubital Tunnel Syndrome								
Study	Number of:		Subjective Results:		Two-point discrimination		Strength	
	Nerves	Patients	Excellent	Good	Excellent	Good	Excellent	Good
Dellon, 1992 ³⁸	11	8	82%	18%	82%	9%	54%	36%
Aszmann, 2000 ³⁹	7	6	72%	28%	42%	44%	N/A	N/A

N/A = Not available

Table 3 Results of Peripheral Nerve Decompression In Diabetic Neuropathy Posterior Tibial Nerve: Tarsal Tunnel Syndrome							
Study	Number of:		Pre-operative:		Results Improved	Ulcer Recurrence	
	Nerves	Patients	Ulcers	Amput			
Dellon, 1992 ³⁸	31	22	0	0	Pain	85%	0%
Wieman, 1995 ⁴⁰	33	26	13	0	Pain 2PD Ulcer	92% 72% 83%	7%
Chaffe, 2000 ⁴¹	58	36	11	6	Pain Touch Ulcer	86% 50% N/A	0%
Aszmann, 2000 ³⁹	16	12	0	0	2PD	69%	0%

2PD = two point discrimination
N/A = Not available

The results of decompression of the median nerve in the carpal tunnel in the diabetic gives excellent relief of sensory symptoms in about 95% of patients and good results in the remaining 5%, with 95% of the patients recovering useful two-point discrimination.^{38,41} These results are what one would expect in the non-diabetic having carpal tunnel decompression.⁷

The results of anterior submuscular transposition of the ulnar nerve at the elbow, using the musculofascial lengthening technique, in the diabetic, gives excellent relief of sensory symptoms in about 77% of patients and good results in the another 22%, with about 95% of the patients recovering useful two-point discrimination.^{38,41} These results are what one would expect in the non-diabetic having this type of ulnar nerve surgery for moderate to severe degree of ulnar nerve compression.⁷ Recovery of motor function is not as good with just 55% of the patients recovering normal grip strength and 40% recovering normal pinch strength.

The results of decompression of the four medial ankle tunnels, related to the tibial nerve and its medial and lateral plantar and calcaneal branches, is determined by restoration of sensation to the sole of the foot, and relief of pain in the foot. For all four reported groups of patients, each of whom was decompressed using the same surgical technique, pain was relieved in 86% of patients and 72% recovered useful two-point discrimination.³⁸⁻⁴¹ Two studies included patients that had a history of ulceration, and the percentage of patients having relief of pain was the same in these patients, however, many of these patients recovered just protective sensation (no two-point discrimination).^{39,40} Among the 62 patients in this combined series that had never had an

ulcer or amputation, none reported an ulceration or an amputation during the follow-up period of observation. Among the 24 patients in this combined series that had a previous ulcer or amputation, 1 (4%) reported a recurrent ulceration during the follow-up period of observation.

The ability to restore sensation to the feet of a diabetic holds the promise of prevention of ulceration and amputation. Over the period of time that I have been doing this type of nerve decompression in the feet of patients with diabetes, there have been a series we have been able to follow for a mean of 4.5 years who have only had a unilateral set of peripheral nerves decompressed. Figure 3 is an example of such a patient who had the right leg decompressed 7 years prior to this photograph. Sensation had been recovered in this foot. Because of the distance he lived from our office, he never came back to have his opposite foot have the nerve decompression surgery. He developed an ulceration in the contralateral foot, and went on to require amputation of two toes on that foot. Our series to the present includes 43 patients. None of these patients have had an ulcer or an amputation in the side that was decompressed. In contrast, there have been 7 ulcerations and 2 amputations in their contralateral limbs. The statistical significance of the success of peripheral nerve decompression in prevention of ulcer and amputation in this group of 43 patients has a p value of .002.⁴¹



Figure 3. Feet of a diabetic with complications of advanced neuropathy in the left foot. This foot is in contrast with the right foot, which has had surgery to decompress peripheral nerves seven years previously. The patient, who lived at a great distance, was not able to return for surgery to the left foot. It is suggested that decompression of the peripheral nerves in the right leg has altered the natural history of progressive neuropathy.

DISCUSSION

The realization that the peripheral nerve in the patient with diabetes is susceptible to compression can offer the patient, who suffers with unrelieved symptoms of

neuropathy, a new source of optimism.¹⁸ Over the past twenty years, progressing from clinical observations³⁸ to basic science research,¹⁹ and then back to clinical treatment of the diabetic with symptomatic lower extremity neuropathy,⁴² experience has been gained that can now be translated into the regular care of the patient with diabetes. Independent surgical centers have reported essentially the findings; decompression of the tibial nerve and its branches at the ankle and foot level can relieve pain and restore sensibility in about 80% of the patients.^{39,40}

As with the treatment of most diseases, the earlier a patient can be referred for treatment, the better is the chance that the symptoms of the disease can be helped. With regard to diabetic neuropathy, once the patient has developed an ulceration, we know that sufficient sensory axons have degenerated that we may only be able to restore protective sensation by decompression of the peripheral nerve. By contrast, if sensibility can be done earlier in the patient with symptoms of neuropathy in the feet, then the ability to restore sensation can be offered at an earlier stage in the pathophysiology. The reason that the results of decompression of the median nerve in the carpal tunnel has a higher success rate than decompression of the tibial nerve in the tarsal tunnel is that patient's usually present to their physician earlier with hand problems than with feet problems.^{38,41} This earlier presentation of the patient with hand problems than with feet problems may be related to the general pessimism that accompanies the teaching the diabetic neuropathy is "progressive and irreversible".

The observation that patients who have had restoration of sensation to their feet through decompression of peripheral nerves have not developed ulcers or had an amputation suggests that the natural history of diabetic neuropathy may be able to be changed. To affect this change, clinicians responsible for the care of the patient with diabetes will need to measure sensibility in the foot, evaluate the foot for the presence of a Tinel sign over known sites of peripheral nerve compression, and refer the patient to a surgeon trained in lower extremity peripheral nerve decompression techniques. If this concept can be introduced into clinical practice, we should see a significant decrease in foot ulcerations and amputations.

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