Changing the Natural History of Diabetic Neuropathy: Incidence of Ulcer/Amputation in the Contralateral Limb of Patients With a Unilateral Nerve Decompression Procedure

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Abstract: The natural history of diabetes 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, nonoperated limb was evaluated in a retrospective analysis of 50 patients with diabetes a mean of 4.5 years (range, 2–7 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 $P < 0.001$. 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.

Key words: diabetes, neuropathy, tarsal tunnel, decompression, ulceration

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Within diabetes mellitus, there are many forms of neuropathy. The distal, large-fiber, symmetric 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 patients with diabetes.[3–12]

For example, in the study of 4400 patients with diabetes reported by Pirat[12] 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. Loss of sensibility leads to infection, ulceration, and amputation, which is independent from the amputations resulting from large-vessel disease.[13] The incidence of ulceration is 2.5% per year and occurs in 1 in 6 patients with diabetes in their lifetime.[14–17] Even the Diabetic Control and Complication Trial, with the goal of 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 fractures.[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 85% of amputations are preceded by nonhealing ulcers in patients with neuropathy.[29,30]

Despite attempts to decrease the number of amputations in the United States 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 was $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 between $4.6 and $13.7 billion.[33,34] It is estimated that as much as 27% of the direct medical costs of diabetes mellitus is related to diabetic neuropathy.[34] There are estimated to be 16 million patients with diabetes in the United States, 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 of foot ulcer."[39] As demonstrated by the review, the natural history of diabetic

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neuropathy is well documented: *The natural history of diabetic neuropathy is to be progressive and irreversible.*

Although 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 1980s 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 proved in a study in which 2 groups of diabetic rats, each with a serum glucose level ≥ 400 μg/dL (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 intraperitoneal streptozotocin injection and was not treated with insulin. At the end of 1 year, approximately half the lifetime for this animal model, the group with the intact tarsal tunnel had the expected progressive neuropathic walking track pattern, whereas 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, nondiabetic, control rats. Recently this study was repeated with the same result.[46] The first group of patients to undergo decompression of upper and lower extremity peripheral nerves was reported in 1992.[47] Subsequently there have been 6 studies confirming that decompression of the tibial nerve and its branches in the 4 medial ankle tunnels can relieve pain in as many as 90% of patients with painful diabetic neuropathy, and can restore sensation in as many as 80% of patients with impaired sensibility.[48–52,74]

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 follow-up of 3.6 years (range, 1–7 years), no patient developed an ulcer or had an amputation postoperatively.[47] This group of patients contained no one who preoperatively had an ulceration or amputation. Among the 2 reports of this surgery with patient cohorts that contained patients with a previous ulcers/amputation history, 1 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 1 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 the result of improved glycemic control, improved foot care, or both. The purpose of the current 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 the surgical technique by Dellon.[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. Those patients not responding to the questionnaire had 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 4 medial ankle tunnels, with these surgical procedures having been done on just 1 limb. The outcomes of ulceration and or amputation were chosen to be such that they could be identified unambiguously by questionnaire or by telephone interview. No patients were excluded from this process, with the exception that anyone with a comorbidity that might affect peripheral nerve function, such as alcoholic neuropathy, cerebrovascular accident, or multiple sclerosis, were excluded. At the time of the peripheral nerve decompression, documentation of the neuropathy with neurosensory testing demonstrated a symmetric sensory component to the neuropathy in terms of abnormal cutaneous pressure threshold and abnormal 2-point discrimination, and there was no difference in the circulation to the 2 feet as demonstrated by clinical evaluation of the posterior tibial and dorsalis pedis pulses. 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. No specific identifiable trauma was reported by any of the patients to the contralateral, nondecompressed extremity that may have caused the subsequent ulceration/amputation other than the underlying neuropathy.

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Statistical analysis was done using SPSS 9.0 software and the Fisher 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 nonoperated extremity. If a patient had an ulceration precede an amputation, for the calculation, this patient was counted as just 1 amputation. If a patient had more than 1 toe amputated, the patient was counted as just 1 patient. If there were multiple ulcerations on the foot, or 1 on the dorsum and 1 on the plantar surface, the patient was just counted as 1 ulceration for statistical purposes.

Just 1 of the 50 patients had an ulcer/amputation of the contralateral foot before the index surgery was done. This patient had the left big toe amputated before having the surgery on the right, not previously ulcerated, foot.

RESULTS

The mean follow-up from the date of surgery was 4.5 years (range, 2–7 years). Of the 50 patients, there were no ulcerations and no amputations on the foot that had the peripheral nerve decompression. In contrast, there were 12 patients with ulcerations and another 3 patients with amputations that occurred on the contralateral, or nonoperated, foot. This difference was significant at $P < 0.001$.

Two patients with examples of these problems are illustrated in Figures 1 and 2. The patient in Figure 1 is the 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 second metatarsal ulceration on the left foot. Figure 2 shows a patient who had the right foot operated on 7 years before developing an ulcer and subsequent amputation of the first and second toes on the contralateral foot.

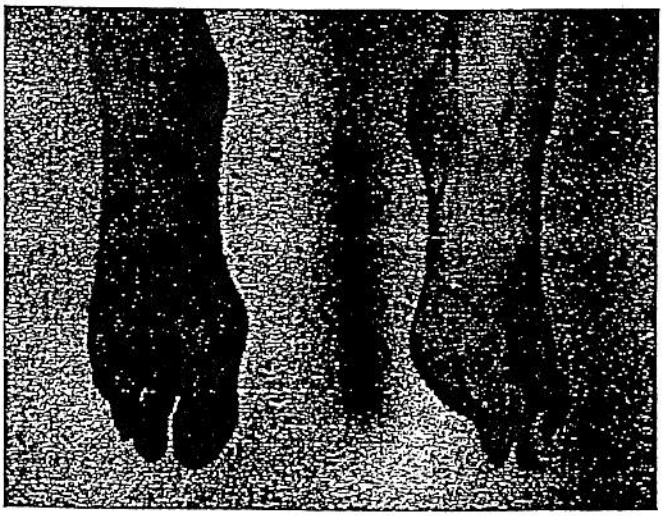

FIGURE 1. The left foot of this patient with diabetes had an amputation of the big toe before the decompression of the peripheral nerves in the right foot. Subsequent to the nerve decompression surgery, the left foot developed the dorsal and plantar ulcers demonstrated.

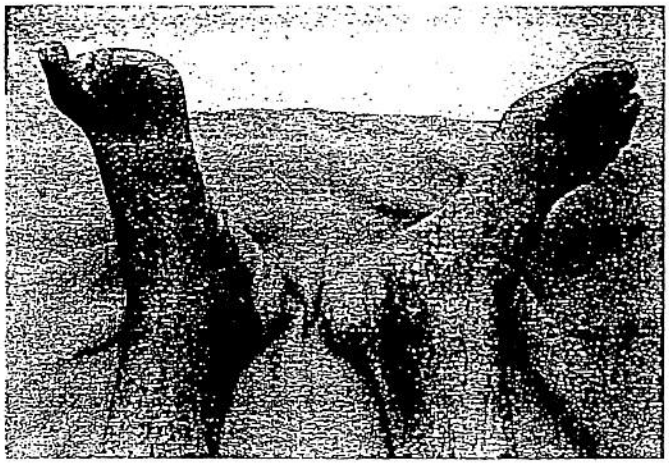

FIGURE 2. The right foot had surgery to decompress peripheral nerves 7 years before the patient developed the ulcers and amputations in the contralateral left foot.

DISCUSSION

The results of this study suggest that the natural history of the most common form of diabetic neuropathy—distal, bilaterally symmetric polyneuropathy—can be changed in terms of the impact of improved sensibility on the development of ulcer and amputation. These results apply to those patients with type I or type II diabetes, because there is no difference in the relationship of ulceration and amputation to either type once neuropathy is present. These results cannot be generalized to patients with neuropathy of unknown etiology or those with a medical condition such as leprosy.

The health care cost savings that can be anticipated based on 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, and reduced costs for amputation and provision of prosthesis, crutches, and wheelchairs. The reduction in health care costs can be extrapolated to a reduction in treatment of falls, because with improved sensibility should come improved balance and a measurable decrease in fractures from falls. The relationship between decreased sensibility in the feet and loss of balance has been documented recently in patients with diabetic neuropathy.[58] Similar health care cost analyses for diabetes have been done, and that methodology could be applied to the outcomes just described.[34,59–62] Restoration of sensation to the feet of patients with symptoms of diabetic neuropathy will provide a paradigm shift in health care costs

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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 2 studies reported in which 30 patients had ulceration/amputation before the surgical decompression of their peripheral nerves,[48,49] just 1 of the 30 patients had a recurrence of the ulceration (3.3%). Recurrent ulceration after wound healing was reported a decade ago to be 70%[63] and, sadly, the most contemporary reports, using current foot wear and methodology, report the recurrence rate to be still in that same range.[64–66] These historical observations theoretically permit the extension of the observations of the current 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 current 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% level 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 to decrease 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.[67] 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.[68] The methodology to achieve this goal was "proper foot care, and reducing risk factors such as hyperglycemia, cigarette smoking and high blood pressure."[68] The 1995, *Midcourse Review* simply restated the previous statements, without giving any data on the status of this objective.[69] The Healthy People 2010 objectives, submitted for public comment in September 1998, simply repeated the same objectives of the 1990 initiative, suggesting that either no progress was made or that no data were available on the subject.[70] Boulton et al,[71] 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. Indeed, their review of the UK, European, and Scandinavian data demonstrate that 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 on the results of the study presented here, that referral of appropriate patients for restoration of sensibility by nerve decompression would be appropriate.

The most recent reviews of diabetic neuropathy (306 references)[72] and treatment/prevention of diabetic foot ulceration (54 references)[73] from the leaders of the medical community unfortunately fail to include even 1 comment on the possibility of relieving pain and restoring sensation to the feet of patients with diabetic neuropathy. In contrast, the most recent review of the role of surgical decompression for diabetic neuropathy, by an orthopedic foot and ankle surgeon, which included the results in his own series of patients, confirmed that "in properly selected patients, surgical releases can decrease pain and improve sensation."[74] It is time for the medical and surgical community to become involved in each other's literature, and unite to provide the clinical trials necessary for evidenced-based decision making. The indications for surgical decompression suggested in this most recent review, with which we would agree, are (1) diabetes as the only known cause of neuropathy, (2) medical and vascular stability, (3) signs of axonal damage (widening of 2-point discrimination), and (4) presence of a Tinel sign over known areas of compression. To these we would add that it is ideal if there is no pedal edema and the patient weighs less than 140 kg, and the presence of an ulceration or past history of an ulceration is not a contraindication to the surgical decompression of peripheral nerves. 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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