Low Long-Term Risk of Foot Ulcer Recurrence After Nerve Decompression in a Diabetes Neuropathy Cohort

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Background: Use of nerve decompression in diabetic sensorimotor polyneuropathy is a controversial treatment characterized as being of unknown scientific effectiveness owing to lack of level I scientific studies.

Methods: Herein, long-term follow-up data have been assembled on 65 diabetic patients with 75 legs having previous neuropathic foot ulcer and subsequent operative decompression of the common peroneal and tibial nerve branches in the anatomical fibro-osseous tunnels.

Results: The cohort's previously reported low recurrence risk of less than 5% annually at a mean of 2.49 years of follow-up has persisted for an additional 3 years, and cumulative risk is now 2.6% per patient-year. Nine of 75 operated legs (12%) have developed an ulcer in 4,218 months (351 patient-years) of follow-up. Of the 53 contralateral legs without decompression, 16 (30%) have ulcerated, of which three have undergone an amputation. Fifty-nine percent of patients are known to be alive with intact feet a mean of 60 months after decompression.

Conclusions: The prospective, objective, statistically significant finding of a large, long-term diminution of diabetic foot ulcer recurrence risk after operative nerve decompression compares very favorably with the historical literature and the contralateral legs of this cohort, which had no decompression. This finding invites prospective randomized controlled studies for validation testing and reconsideration of the frequency and contribution of unrecognized nerve entrapments in diabetic sensorimotor polyneuropathy and diabetic foot complications. (J Am Podiatr Med Assoc 103(5): 380-386, 2013)

Neuropathy continues to be a very serious complication of diabetic metabolic disease. The primary symptoms of pain and numbness frequently lead to a well-known cascade of clinical complications. Sensory loss leads to unrecognized wounds, diabetic foot ulcers (DFUs), local infection, progressive tissue sepsis, life-changing amputations, and early death. National Diabetes Information Clearinghouse statistics indicate that at least 60% of the more than 25 million diabetic patients in the United States have neuropathy. With diabetic sensorimotor polyneuropathy (DSP) comes the risk of DFU of 2.5% to 10% annually[1,2] and a 15% to 25% lifetime prevalence.[3] Foot infection incidence in diabetic patients is 4.6% yearly, and hospitalization for foot complications is required in 1.8% to 2.3% annually.[4,5] The cost of ulcers and their sequelae is an enormous economic problem. Ulcer recurrence is a significant risk in DFUs,[6] and at least 80,000 major leg amputations per year are performed on diabetic patients, nearly all of which are preceded by a foot wound.[7] Five-year mortality after either DFU or major amputation is almost 50%,[8] similar to colon, prostate, and breast cancer malignancy.[9] It is no wonder that foot or leg amputation is the complication most feared by the diabetic patient.

Avoidance and treatment of DFU and recurrence prevention has focused on risk recognition, patient education, foot self-care, appropriate footwear, and reduction of pressure concentrations. None of these medical interventions are supported as yet by robust science demonstrating their effectiveness for primary prevention.[10-13] The review by Bus et al[14] concludes that there are no scientifically proven methods to prevent ulceration. When ulcers occur, healing is regularly accomplished using pressure-relieving total-contact casting or cast walkers rendered irremovable,[15,16] but in clinical practice

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these are used infrequently[17] compared with the less effective method of shoe modification.

Ulcer recurrence continues to be a problem, with "best practices" still seeing recurrence rates of 25% per year[6] and reports in the literature[18] of up to 80% annually. Arad et al[19] reviewed all of the interventions for recurrence avoidance evaluated with evidence-based medicine level I evidence in randomized controlled trials and found that only daily plantar temperature monitoring, with activity restriction until asymmetrical temperature change normalizes, has a significant protective effect. This method, evaluated in "high-risk," previously ulcerated feet[6,20] of the International Working Group on the Diabetic Foot (IWGDF) risk group 3, awaits confirmation by other investigators. Against this disheartening record, the search continues for other effective preventive or therapeutic protocols for DFU and its recurrence.

One prospective intervention that still has promising potential for ulcer and reulceration prevention is nerve decompression (ND). Since 1988, Dellon[21] has been publishing reports on relief of pain and recovery of sensation after nerve releases in most cases of painful DSP with adequate circulation and a positive Tinel percussion test result suggesting nerve entrapment. The Tinel sign is thought to demonstrate viable but irritable compressed nerve tissue.[22] It is sought by gentle percussion of nerve trunks at known fibro-osseous entrapment sites in the upper and lower extremities, such as the carpal, cubital, peroneal, tarsal, and medial and lateral plantar tunnels. Dellon's therapeutic approach is relief of nerve entrapment in DSP with ND via operative external neurolysis.[23,24] Although other authors[25] have confirmed subjective outcomes of pain relief and sensory return, many academics believe that all of this work is weak science.[26,27] The primary criticisms of these reports include the retrospective and uncontrolled case series designs, the lack of randomization and blinding, and the use of subjective (pain and sensation) outcome measures strongly susceptible to the placebo effect and the risks of surgeon, observer, and patient bias. Stronger scientific evidence requires protocols using controls, randomization, a prospective study design, and, in particular, objective outcome measures. A 2004 study by Aszmann et al[28] used a very pertinent objective measure in reporting that of 50 patients who had unilateral lower-extremity nerve releases for painful DSP, none developed a foot ulcer or underwent any amputation on the operated leg during mean follow-up of 4.5 years. The contralateral legs, however, without operative ND, had acquired an ulcer or amputation in 30%. This intriguing objective finding—that ND might protect against ulcer and amputations—has lead to the present study design.

Design

This is an institutional review board–approved level 2b (EBM, Oxford, England), grade B (DynaMed, EBSCO, Ipswich, MA), retrospective, objective outcome, multisite, observational study of ulcer recurrence rates after ND in a high-risk cohort of diabetic patients with previous DFU. An initial retrospective report on this cohort at a minimum 12-month follow-up found the risk of ipsilateral leg ulcer recurrence to be low at 4.3% per year.[29] This article extends the post-ND follow-up prospectively an additional 3 years. An important secondary objective outcome, the contralateral nonoperated leg ulcer occurrence risk, is evaluated as a metabolic and activity control in the 53 unilateral procedures.

Table 1 summarizes pertinent data in the initial report of the cohort. The study participants were 65 self-identified diabetic patients from five private practitioner plastic, orthopedic, and podiatric surgeons. Previous foot ulceration had occurred on 1 to 5 or more occasions in each of the 75 operated feet, and the original indication for surgery was painful DSP. Each patient had ND by external neurolysis of lower-extremity nerves at three leg sites: 1) the common peroneal nerve at the fibular neck in the peroneal tunnel, 2) the dorsal sensory branch of the deep peroneal nerve under the extensor hallucis brevis tendon on the dorsal foot, and 3) the posterior tibial, medial calcaneal, medial plantar, and lateral plantar branches of tibial nerve at the tarsal tunnel area of the medial ankle and foot.

All of the patients were taking medication for diabetes treatment. Each operated foot had a palpable pulse or an ankle-brachial index greater than 0.8 and a positive Tinel sign at one or more fibro-osseous nerve tunnel sites. Shoe inserts, if used, were worn bilaterally. Subjective neuropathy scores or sensory measures and subjective pain changes were not available for analysis. The 53 contralateral nonoperated legs, some of which had prior healed ulcer events, were monitored from the time of the index operation forward, recording ulcer occurrence or further recurrences, amputation events, and revascularization procedures.

Table 1. Initial Demographic Data for 75 Legs of 65 Patients with Diabetes, Previous DFU, Adequate Circulation, and Subsequent Lower-Extremity NDs

Characteristic	Value
Age (mean, range [years])	74.5, 42–86
Diabetes type 1, 2 (%)	13, 87
Duration of diabetes known to patient (mean [years])	13.5
Duration of follow-up after operative ND (mean, range [years])	2.49, 1–13
Open ulcer at time of surgery (No.)	4
Open ulcer healed without recurrence (No.)	4
Postoperative complications, resolved with local care (No.)	3
Delayed wound healing (No.)	3
Treating surgeon's training (No. of operated legs)	
Plastic surgery (n = 2)	18
Orthopedic surgery (n = 1)	4
Podiatric medicine (n = 2)	53

Abbreviations: DFU, diabetic foot ulceration; ND, nerve decompression.

Results

The initial retrospective analysis in 2007 found ulcer recurrence risk to be 4.3% per patient-year.[29] Followed prospectively to 2010, this same cohort now shows an overall 2.6% annual recurrence rate, with a mean ulcer-free interval of 56 months (4.7 years). In total, nine ulcers of 75 operated legs (12%) have recurred in 351 patient-years (4,218 months). Four of these ulcer recurrences are at new foot sites versus five in previous locations. Eight patients with 12 operated legs (16%) died without DFU recurrence after mean survival of 5.2 years. Five-year mortality after ND was 8% (5 of 65 patients). Four operated legs in three patients (5.3%) are now lost to follow-up. The contralateral legs have fared less well, with single or multiple events of ulcer occurrence and recurrence in 30% that required one below-the-knee, one transmetatarsal, and one multiple-toe amputation.

Current cohort results are listed in Table 2. The individual outcomes of the 75 operated legs are displayed in Figure 1 showing the ulcer-free survival time of the entire cohort of operated legs and indicating the status as intact foot, intact at death, lost to follow-up, or recurrence. The Kaplan-Meier analysis shown in Figure 2 illustrates the survival function of all 75 operated legs, the 53 unilateral operation cases, and their 53 contralateral, nonoperated mates. In the 53 unilateral cases, the legs without ND showed markedly poorer ulcer-free survival. One surgeon's 19 unilateral cases included ulcer appearance in ten nonoperated limbs, totaling 22 separate ulcer events requiring three amputations in the 5 to 7.5 years after the operation date. His ND legs, meanwhile, had no amputations.

Statistical comparison of all 75 operated legs with the 53 nonoperated legs of the unilateral surgery group was undertaken. Using the two-sample t test and a null hypothesis that ND will not affect ulcer recurrence risk, statistical analysis rejects the null hypothesis, supporting the conclusion that ND is beneficial in the prevention of ulcer recurrence ($P = .013$).

Discussion

The primary strengths of this study and its findings are the longitudinal design, long-term follow-up, and use of an objective outcome measure, namely the recurrence of DFU. The availability of the nonoperated leg for comparison in most cases provides an important internal control for the degree of neuropathy, metabolic environment, and activity levels. This should allow great confidence that the operated leg and its contralateral mate are functioning in an identical milieu. Metabolically induced length-dependent axonopathy (LDA), the current paradigm hypothesized to be causal physiologically for DSP, ought not to differ between legs, so any changes in outcome can be confidently ascribed to the ND intervention.

One concern expressed by critics of ND is the peril of exposing their patients to a surgery whose justification is yet to be established. To this point, Wukich et al[30] have published since our initial study a report finding a 13% incidence of infection after all foot surgeries on high-risk diabetic patients, which includes those with neuropathy. Our finding (Table 1) of zero wound infections and three cases of benign delayed healing requiring no systemic treatment suggests that these soft-tissue ND foot procedures may have a much smaller hazard, at least in DSP with adequate circulation.

A comparison of these results with other ulcer occurrence and recurrence reports could be misleading unless close attention is given to their cohort composition and previous treatments. Some recurrence studies include patients who have had major amputations whose recurrence risk has become zero. Faglia et al,[16] for example, reported on 115 cases with 196 limbs who were previously hospitalized for treatment of DFU. They calculated a commendable 12.8% rate of new ulcer formation

Table 2. Longitudinal Objective Outcomes in Neuropathic DFU After ND

Outcome	Initial Retrospective Analysis in 2007	Prospective Follow-up Analysis in 2010–2011
Total DFU legs operated	75	NA
Total patients	65	NA
Ulcer-free follow-up (mean, range [months])	NA	56, 1–160
Recurrences of ipsilateral DFU	8 in 187 patient-years; 4.3% annual risk	9 in 351 patient-years; 2.6% annual risk
Recurrence site, same/other (No.)	4/4	5/4
Time to recurrence (median, range, mean [months])	12.5, 2–59	23, 2–59, 19
Death during follow-up without recurrence	2 patients, 3 legs	8 patients, 12 legs at a mean of 62 months (5.2 years)
Lost to current follow-up	NA	4 legs in 3 patients = 5.3% of the cohort
Patients with ulcer/amputation on the contralateral nonoperative foot	9/53 ulcers (17%)	16/53 (30%) have developed ulcers leading to 1 below-the-knee, 1 multiple-toe, and 1 transmetatarsal amputation (6%)
Operated legs confirmed ulcer free, without amputation	NA	50/75 legs (67%) at a median of 56 months; mean, 62 months; range, 45–86
Comparative "best care" recurrence risk in the literature[6] for IWGDF risk group 3 in DSP	NA	25% annually, up to >80% in the literature
Apparent annual reduction of DFU recurrence risk with ND (%)	NA	>85[a]

Abbreviations: DFU, diabetic foot ulceration; DSP, diabetic sensorimotor polyneuropathy; IWGDF, International Working Group on the Diabetic Foot; NA, not applicable; ND, nerve decompression.

[a] 1 – (2.6% / 25%) = 0.896.

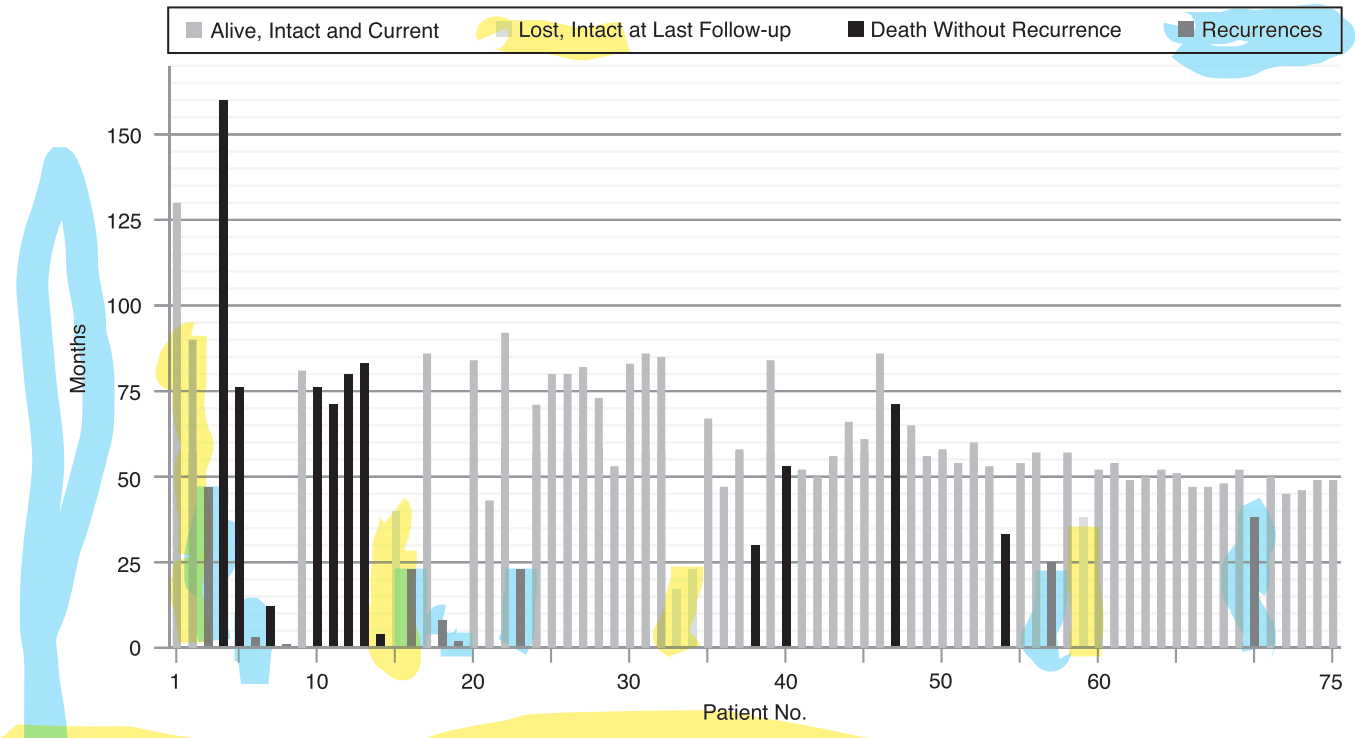

Figure 1. Individual outcomes of operated legs after nerve decompression.

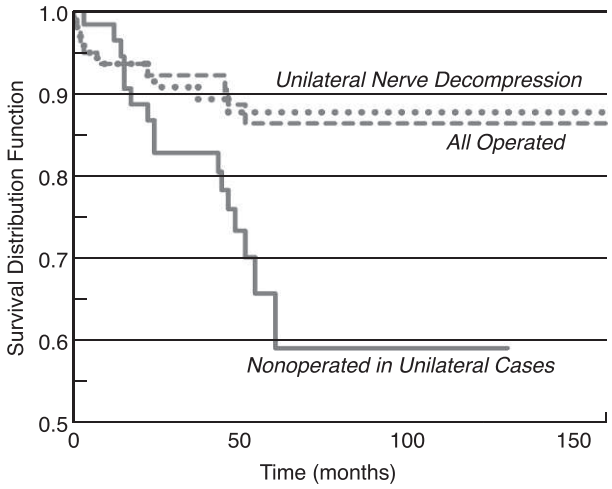

Figure 2. Kaplan-Meier graph of ulcer-free survival of legs.

(25 of 196) on either the index or contralateral limb. However, 30% of index limbs had vascular procedures, and 24% had major and 56% minor amputations incident to their previous hospitalizations. Only 16% were classified as neuropathic, with the balance either neuroischemic or ischemic.

Neuropathic, ischemic, and neuroischemic ulcers may be associated with different outcomes in recurrence and in amputation (11%, 29%, and 25%, respectively) and 5-year mortality (45%, 55%, and 18%, respectively), as Moulik et al[8] found. For comparison of outcomes, we favor Jeffcoate et al's[31] patient-oriented measure of percentage surviving with intact foot and healed ulcer. This, they reported, is only 45% at 12 months for patients with a foot ulcer presenting to a multidisciplinary UK foot clinic. Ghanassia et al,[32] using a similar "global therapeutic success" measure, found that 31.5% of a cohort (28 of 89) in France having had hospital treatment for diabetes and foot ulcer were intact and surviving at a mean of 6.5 years. This study's neuropathic group, with 67% alive and intact at a mean of 5.0 years, doubles that success rate.

The present study cohort best compares with the two control groups of a randomized trial of plantar thermal monitoring reported by Lavery et al.[6] They found success with skin temperature self-monitoring and activity restriction in reducing the incidence of DFU recurrence in International Working Group on the Diabetic Foot risk group 3 cases (healed neuropathic ulcers) to 6.8% per year. Their two control groups experienced 25% per year recurrence rates in the standard patient education group and an enhanced education plus visual self-monitoring group. The present study's overall recurrence risk of 2.6% per year during much longer follow-up is promising compared with the temperature-monitoring intervention and its controls. The result seen in Figure 2 comparing ulcer risk for all operated legs with that for the 53 nonoperated legs is quite favorable for ND, as is the scientifically stronger, self-controlled comparison of the operated and nonoperated control legs in the unilateral cases.

The finding of durable low rates of ulcer recurrence in healed neuropathic DFU cases after ND has interesting implications for a long-running debate about etiology and treatment of DSP and its complication cascade. It is universally accepted that diabetic patients as a group have a high incidence of single-nerve compressions, such as carpal tunnel or cubital tunnel syndromes, and that operative decompression is an appropriate treatment thereof.[33] There is also an understanding that multiple nerve entrapments can occur as mononeuritis multiplex, which may similarly be appropriately treated surgically. But these described nerve entrapments are considered to be outside the umbrella DSP etiology described as LDA. For Dellon's operative approach to make sense, and for surgical decompression to be an effective prevention of DFU recurrence as this report implies, there must be an etiology of DSP other than LDA, or entrapment is an additional aspect augmenting whatever factors LDA contributes to DSP ulcer causation. Nerve decompression could in no way alleviate a purely metabolic LDA neuropathy unless physical entrapment, obvious or occult, is also involved. Nerve decompression would not alter the plantar pressure concentrations, skin callus, tight gastrocnemius-soleus muscle group, foot deformities, smoking, amputation and ulcer history, or peripheral arterial disease, all shown to be important correlates of initial ulcer development. What it may do is restore protective sensation, alter axoplasmic flow, or improve vascular microcirculation. It could also be ameliorating other tissue homeostasis parameters to generate some protection from tissue breakdown. The physiologic biochemical or biomechanical method by which ND accomplishes benefit is presently as cryptic as are the physiologic mechanisms by which LDA is presumed to operate.[34]

The current focus of evidence-based medicine on stronger science and level I randomized controlled studies to guide therapy certainly raises the bar for consideration of unusual and unexpected therapies. This is particularly so when paradigm-challenging information comes from outside the primary academic disciplines involved. So long as level I studies on ND in DSP have not been completed, skeptics

will continue to say, "There is no evidence." Yet, it is more accurate and honest to say, "No level I randomized controlled trial has examined this issue." This seems to describe the case for ND in DSP. Several reviews of the issue[19,26,27] conclude that the evidence for ND therapy is lower-level class IV evidence, is subject to significant design weaknesses, and is "unproven," although the term "ineffective" is never used. The assembly of evidence of ND benefit has now progressed from clinical dilemma through anatomical cadaver studies, laboratory experimentation, and fibro-osseous entrapment hypothesis to clinical application, retrospective studies, prospective subjective outcome studies, and expert opinion that the technique has promise. The use of objective measures of high intratunnel neural pressures,[35] post-ND balance improvement,[36] and the present report of apparent long-term protection against DFU recurrence now set the stage for blinded, prospective, randomized controlled trials.

The results reported herein suggest that ND in DSP with DFU may be a therapy with significant benefit, acceptably small risks, moderate comparative costs, and durable protective effect independent of ongoing patient commitment to action. Further objective technological measures, such as hyperspectral imaging of oxygenated hemoglobin, circulation studies, tissue oxygenation values, and intraepidermal nerve fiber density, may identify quantifiable objective parameters on which ND may operate. Simultaneously, the present clinical findings of low recurrence risk of neuropathic DFU after ND in DSP should be challenged with prospective randomized controlled trial protocols with control/experimental cohorts to see whether this observed greater than 80% reduction in DFU recurrence risk can be confirmed with level I evidence. The expense and effort of such level I scientific studies would be quickly recovered in decreased costs for ulcer care if ND proves effective, and many thousands of people now experiencing DSP and DFU might be spared the current high risks of amputation and early mortality.

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References

1. Boyko EJ, Ahroni JH, Stensel V, et al: A prospective study of risk factors for diabetic foot ulcer: the Seattle Diabetic Foot Study. Diabetes Care **22:** 1036, 1999.
2. Boyko EJ, Ahroni JH, Cohen V, et al: Prediction of diabetic foot ulcer occurrence using commonly available clinical information: the Seattle Diabetic Foot Study. Diabetes Care **29:** 1202, 2006.
3. Singh N, Armstrong DG, Lipsky BA: Preventing foot ulcers in patients with diabetes. JAMA **293:** 217, 2005.
4. Lavery LA, Armstrong DG, Wunderlich RP, et al: Risk factors for foot infections in individuals with diabetes. Diabetes Care **29:** 1288, 2006.
5. Lavery LA, Wunderlich RP, Tredwell JL: Disease management, for the diabetic foot: effectiveness of a diabetic foot prevention program to reduce amputations and hospitalizations. Diabetes Res Clin Pract **70:** 31, 2005.
6. Lavery LA, Higgins KR, Lanctot DR, et al: Preventing diabetic foot ulcer recurrence in high-risk patients: use of temperature monitoring as a self-assessment tool. Diabetes Care **30:** 14, 2007.
7. Pecoraro RE, Reiber GE, Burgess EM: Pathways to diabetic limb amputation: basis for prevention. Diabetes Care **13:** 513, 1990.
8. Moulik PK, Mtonga R, Gill GV: Amputation and mortality in new-onset diabetic foot ulcers stratified by etiology. Diabetes Care **26:** 491, 2003.
9. Armstrong DG, Wrobel J, Robbins JM: Guest Editorial: are diabetes-related wounds and amputations worse than cancer? Int Wound J **4:** 286, 2007.
10. Maciejewski ML, Reiber GE, Smith DG, et al: Effectiveness of diabetic therapeutic footwear in preventing reulceration. Diabetes Care **27:** 1774, 2004.
11. Cavanagh PR, Bus SA: Off-loading the diabetic foot for ulcer prevention and healing. Plast Reconstr Surg **127** (suppl 1)**:** 248S, 2011.
12. Dorresteijn JA, Kriegsman DM, Assendelft WJ, et al: Patient education for preventing diabetic foot ulceration. Cochrane Database Syst Rev **5:** CD001488, 2010.
13. Dorresteijn JA, Kriegsman DM, Valk GD: Complex interventions for preventing diabetic foot ulceration. Cochrane Database Syst Rev **1:** CD007610, 2010.
14. Bus SA, Valk GD, van Deursen RW, et al: Specific guidelines on footwear and offloading. Diabetes Metab Res Rev **24** (suppl 1)**:** S192, 2008.
15. Katz IA, Harlan A, Miranda-Palma B, et al: A randomized trial of two irremovable off-loading devices in the management of plantar neuropathic diabetic foot ulcers. Diabetes Care **28:** 555, 2005.
16. Faglia E, Caravaggi C, Clerici G, et al: Effectiveness of removable walker cast versus nonremovable fiberglass off-bearing cast in the healing of diabetic plantar foot ulcer: a randomized controlled trial. Diabetes Care **33:** 1419, 2010.

17. Wu SC, Jensen JL, Weber AK, et al: Use of pressure offloading devices in diabetic foot ulcers: do we practice what we preach? Diabetes Care **31:** 2118, 2008.
18. Edmonds ME, Blundell MP, Morris ME, et al: Improved survival of the diabetic foot: the role of a specialized foot clinic. Q J Med **60:** 763, 1986.
19. Arad Y, Fonseca V, Peters A, et al: Beyond the monofilament for the insensate diabetic foot: a systematic review of randomized trials to prevent the occurrence of plantar foot ulcers in patients with diabetes. Diabetes Care **34:** 1041, 2011.
20. Armstrong DG, Holtz-Neiderer K, Wendel C, et al: Skin temperature monitoring reduces the risk for diabetic foot ulceration in high-risk patients. Am J Med **120:** 1042, 2007.
21. Dellon AL: A cause for optimism in diabetic neuropathy. Ann Plast Surg **20:** 103, 1988.
22. Lee CH, Dellon AL: Prognostic ability of Tinel sign in determining outcome for decompression surgery in diabetic and nondiabetic neuropathy. Ann Plast Surg **53:** 523, 2004.
23. Dellon AL: Diabetic neuropathy: review of a surgical approach to restore sensation, relieve pain, and prevent ulceration and amputation. Foot Ankle Int **25:** 749, 2004.
24. Dellon AL: Neurosurgical prevention of ulceration and amputation by decompression of lower extremity peripheral nerves in diabetic neuropathy: update 2006. Acta Neurochir Suppl **100:** 149, 2007.
25. Dellon AL: The Dellon approach to neurolysis in the neuropathy patient with chronic nerve compression. Handchir Mikrochir Plast Chir **40:** 1, 2008.
26. Chaudhry V, Stevens JC, Kincaid J, et al: Practice advisory: utility of surgical decompression for treatment of diabetic neuropathy: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. Neurology **66:** 1805, 2006.
27. Chaudhry V, Russell J, Belzberg A: Decompressive surgery of lower limbs for symmetrical diabetic peripheral neuropathy. Cochrane Database Syst Rev **3:** CD006152, 2008.
28. 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, 2004.
29. Nickerson DS: Low recurrence rate of diabetic foot ulcer after nerve decompression. JAPMA **100:** 111, 2010.
30. Wukich DK Lowery NJ, McMillen RL, et al: Postoperative infection rates in foot and ankle surgery: a comparison of patients with and without diabetes mellitus. J Bone Joint Surg Am **92:** 287, 2010.
31. Jeffcoate WJ, Chipchase SY, Ince P, et al: Assessing the outcome of the management of diabetic foot ulcers using ulcer-related and person-related measures. Diabetes Care **29:** 1784, 2006.
32. Ghanassia E, Villon L, Thuan Dit Dieudonné JF, et al: Long-term outcome and disability of diabetic patients hospitalized for diabetic foot ulcers: a 6.5-year follow-up study. Diabetes Care **31:** 1288, 2008.
33. Vinik A, Ullal J, Parson HK, et al: Diabetic neuropathies: clinical manifestations and current treatment options. Nat Clin Pract Endocrinol Metab **2:** 269, 2006.
34. Krishnan ST, Quattrini C, Jeziorska M, et al: Neurovascular factors in wound healing in the foot skin of type 2 diabetic subjects. Diabetes Care **30:** 3058, 2007.
35. Rosson GD, Larson AR, Williams EH, et al: Tibial nerve decompression in patients with tarsal tunnel syndrome: pressures in the tarsal, medial plantar, and lateral plantar tunnels. Plast Reconstr Surg **124:** 1202, 2009.
36. Ducic I, Taylor NS, Dellon AL: Relationship between peripheral nerve decompression and gain of pedal sensibility and balance in patients with peripheral neuropathy. Ann Plast Surg **56:** 145, 2006.