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SYMPOSIUM

Reconsidering Nerve Decompression: An Overlooked Opportunity to Limit Diabetic Foot Ulcer Recurrence and Amputation

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Abstract

Nerve decompression for relief of subjective diabetic sensorimotor polyneuropathy pain and numbness has been labeled of "unknown" benefit. Objective outcomes in treatment and prevention of diabetic foot complications are reviewed. There is growing evidence that plantar foot ulceration and recurrence in high-risk feet are minimized with this operation. Avoiding neuropathic and neuroischemic ulcer wounds should theoretically reduce amputations and perhaps mortality risk. Protective effects are hypothesized to act via relief of neurovascular entrapment, thereby improving neurally modulated tissue homeostasis factors. Nerve decompression deserves considerable research attention to understand its role in limiting foot complications. Its apparent benefits challenge the paradigm that diabetic neuropathy is a purely length-dependent axonopathy and may necessitate appreciation of superimposed nerve entrapment as an significant operant factor.

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Introduction

Diabetic sensorimotor peripheral neuropathy (DSPN) continues to be a leading cause of complications of diabetes mellitus. Neuropathy affects an estimated 60% of the 25 million Americans with diabetes and accounts for a disproportionate share of health care expenditures. Diabetic foot ulcer (DFU) treatment and care of wound infections, critical limb ischemia, and sepsis often require costly hospitalization or amputations for resolution and preservation of life. Major efforts to avoid amputations, such as the Lower Extremity Amputation Prevention program have had less effect than hoped.

Reducing DSPN incidence is possible with close attention to maintenance of near-normal glycemia.[1] However, neuropathy, once present, seems recalcitrant to pharmacologic treatment and is considered to be irreversible and progressive.[2] The natural history of DSPN can be viewed (**Figure 1**) as being a complication cascade beginning with metabolic neuropathy and progressing from often painful numbness, through loss of tissue compliance and elasticity, to skin atrophy, tissue or joint rigidity and deformity, and occasionally to Charcot neuro-osteoarthropathy. Progressive stages cause plantar pressure elevation and concentrations with skin breakdown and ulcer formation, frequently progressing to infection, sepsis, and hospitalization. These often resolve only with toe, foot, or life-altering leg or above-knee amputations.

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Abbreviations: (DFU) diabetic foot ulcer, (DSPN) diabetic sensorimotor peripheral neuropathy, (EBM) evidence-based medicine, (IWGDF) International Working Group on the Diabetic Foot, (LDA) length-dependent axonopathy, (NCV) nerve conduction velocity, (ND) nerve decompression

Keywords: diabetic amputations, diabetic foot ulcer, diabetes neuropathy, foot complications, nerve surgery, ulcer recurrence

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Data

Eighty-five percent of amputations in diabetes are preceded by open wounds,[3] the remainder usually by critical limb ischemia. The majority originated as neuropathic or neuroischemic ulcers before entering the complication cascade leading to amputation. Prevention of progression down the cascade could, in theory, forestall the eventual need for amputations. There is no strong scientific evidence that any intervention is effective for *primary prevention* of the initial ulcer wound.[4] Neither patient self-care, physician foot checks, routine podiatric visits, prescription footwear, nor inserts have reduced risk of primary DFU.[5] Most nonischemic initial DFUs can be healed with offloading pressure concentrations via total contact casting or cast boot walkers rendered irremovable.[6] Sadly, such proven treatment is used a minority of the time.[7]

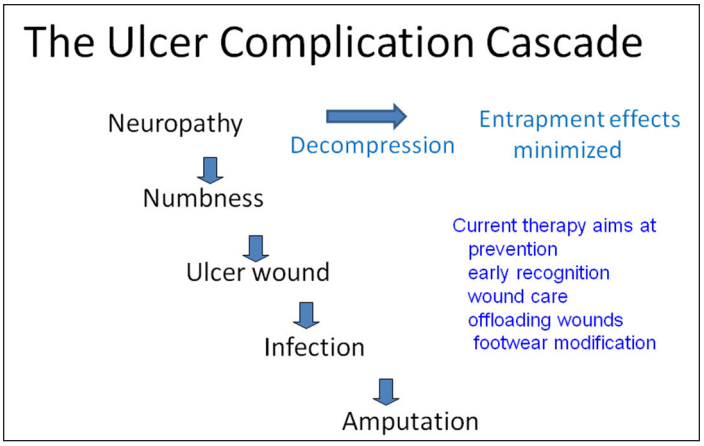

Figure 1. A conceptual illustration of the of the waterfall cascade of foot complications in DSPN. Initial neuropathy can lead to foot wounds, infection, gangrene, or sepsis, then amputation and early mortality. Nerve decompression confronts the neuropathic source of the cascade by minimizing the superimposed contribution of swollen, entrapped nerves.

There is very high risk of recurrence after initial DFU healing, reported to be 25–35% at 1 year, 50% by 3 years, and near 100% by 10 years.[8] Self-monitoring for localized plantar temperature elevation can identify inflammation and allow intervention measures before skin breaks down and an ulceration develops.[9,10] Only this study has presented evidence-based medicine (EBM) level 1 confirmation of protection against ulcer recurrence. However, this method requires a significant supportive infrastructure and lifelong commitment to adherence. Nerve decompression (ND) is another intervention that has shown protection against DFU occurrence and recurrence. Evidence supporting ND is EBM level 2 and below. This commentary reviews evidence that ND protects against DSPN complications and proposes further study protocols to test ND surgery and elucidate how ND might operate.

The Entrapment Hypothesis

Nerve decompression describes operative external neurolysis of peripheral nerves at several anatomic locations where they are anchored in position by fibro-osseous tunnel structures. Tunnels can be considered as stabilizing points, which fix nerve trunks at locations adjacent to joints where the nerve takes a circuitous or angular course. A nerve can thereby tolerate acute changes in direction and angulation and also glide to and fro throughout joint range of motion. Such structures include the wrist's carpal tunnel or the elbow's cubital tunnel, which stabilizes the ulnar nerve. In the leg, similar tunnels include the common peroneal tunnel at fibular neck, the tarsal tunnel at the medial ankle, and the parallel abductor tunnels where medial and lateral plantar nerves plunge into the deep plantar space. Entrapments at these leg and arm tunnels in combination could produce a "stocking-glove anesthesia," the classically described abnormal sensory pattern.

Ideas about similar focal entrapments of nerve in Hansen's disease were developed in the 1950s by such surgical clinicians as Brand[11] and Riordan.[12] They observed swollen nerves constrained in inflexible fibro-osseous passages, correlating this to the variable patterns and combinations of individual nerve dysfunction found in leprosy neuritis. Decompression neurolysis improved motor function, pain, and sensation in such cases.[13] Later, corticosteroids and antibacterial chemotherapy became available to pharmacologically suppress immune responses and inflammation, shrink nerves, and resolve the nerve/tunnel size mismatch. Brand noted many similarities in the clinical pictures of leprosy neuritis and DSPN.[14]

Pertinent Literature and Critique

Dellon[15] developed similar ideas about neuropathic entrapments independently when challenged by his diabetic carpal tunnel patients to help their feet as well. Subsequent anatomic and animal investigations revealed entrapment

sites in human legs as well as arms. Rats rendered diabetic developed enlarged peripheral nerves[15] and consistent gait abnormalities,[16] which could be prevented by prior release of their tarsal tunnel analogue.[17] Dellon's initial retrospective clinical report[18] described ND procedures relieving DSPN pain and improving sensibility in both arms and legs if Tinel's sign and adequate circulation were present.[18,19] A number of other authors confirmed his rat studies[20,21] and success in restoring sensibility and comfort with ND at three leg sites with fibro-osseous tunnels.[22–28] Dellon hypothesized that, in DSPN, the metabolic changes of diabetes can produce enlarged peripheral nerve trunks that superimpose compression and entrapment at inflexible anatomic tunnel structures and can be relieved by surgical external neurolysis to deliver symptomatic relief of pain and recovery of sensibility. Subjective outcomes such as symptomatic pain relief and sensibility recovery are achieved in >80% of cases.[29] Most of the academic foot-care community has not adopted ND, noting that retrospective studies, registry reports, and subjective outcomes are subject to risks of placebo effects and surgeon, patient, or observer bias. Sham surgery or stronger study protocols and objective outcome measures were stipulated before ND could be considered appropriate for DSPN.[2,30,31]

Objective outcome studies and prospective or randomized controlled trial protocols now report beneficial ND effects on balance, tunnel tissue pressures, ulceration risk, DFU recurrence risk, and electrophysiological parameters, as **Figure 2** shows.[32–39]These often include and correlate common subjective measures of DSPN symptoms and signs. Aszmann and coauthors[32] first published the unanticipated finding that *every* ulceration and amputation in 50 DSPN cases that had unilateral ND surgeries for leg pain occurred in the *contralateral*, nonoperated legs. Such International Working Group on the Diabetic Foot (IWGDF) group 1 or 2 cases have an ulcer occurrence risk of 3.3%/year.[40] Aszmann's 50 operated legs developed zero subsequent events of ulceration or amputation in a median 4.5 years (range 2–7), while 30% of the nonoperated contralateral legs developed DFU (12 cases) or toe amputations (3 cases), a scientifically significant result ($p < .001$).

Ducic and coauthors[41] reported bilateral ND improves balance as measured by sway, and Rosson and coauthors[35] found perineural tunnel pressures, so high as to threaten tissue survival, returned to safe levels after ND. In 75 DSPN legs with prior ulcer, of IWGDF group 3, after ND, ipsilateral DFU recurrence risk is >80% *reduced* from historical values.[34] Expected 25–35% historical recurrence risk was compared with 4.6 and 2.3%/year in these legs at 2.5- and 5-year review.[38] This same cohort's 42 intact, nonoperated legs had a hazard ratio of subsequent ulceration of 5.5 (95% confidence interval, 3.6 – 7.0; $p = .048$), during year 2 through year 5 post-ND.[37]

The most robust evidence for use of ND is the prospective work of Zhang and coauthors.[36] Their prospective cohort was 560 subjects, 208 cases with healed DFU (IWGDF group 3) and 352 IWGDF group 1 or 2 DSPN cases with the maximum Toronto Clinical Symptom Score of 19 of 19 points. All legs had palpable foot pulses and positive Tinel's percussion sign. Eighteen months after bilateral ND using Dellon's method,[42] the cohort had developed *zero* DFU, recurrent ulcers, or amputations. Two wound dehiscences and zero clinical infections occurred in the 1120 operated legs. Nerve conduction velocity (NCV) recovered over half of a 30% deficit compared with 40 IWGDF group 0 age-matched controls. Toronto Clinical Symptom Scores declined to lower quintiles in 88%, averaging 12.5/19. Vibration perception recovered half the initial deficit, warm/cold perception recovered to near normal, and two-point discrimination improved from 100% being >9 mm to a mean 6.7 mm; $p = <.05$ for all those subjective outcome measures.

Discussion

Using ND to attack the origin of the neuropathic DFU cascade seems to offer significant protection against complications for at least some DSPN cases. Most reported ND cohorts are selected for pain, palpable pulses, and Tinel's sign, leaving unexamined ND's benefit for painless DSPN or neuroischemic populations. Yet objective evidence is accumulating that nerve entrapment may be playing a significant role in DSPN complications and symptoms, as Dellon hypothesized 25 years ago.[15] Shaper's opinion that "recent studies suggest that nerves play a central role in tissue homeostasis and can orchestrate complex reparative as well as destructive processes in the feet" is supportive.[43] Sympathetic innervation is abundant in the foot[44] and may mediate such processes.

We may speculate that hesitance to adopt Dellon's hypothesis and approach is due to several factors. The origin of these ideas from a surgical source brought with it low credibility for medical practitioners and diabetes specialists.

Posterior Tibial n. Decompression
Tarsal Tunnel
Patient 38179 (March 1, 2013)

EMG Recordings:
Abductor digiti minimi (ADM)
Abductor hallucis (AH)

Pre Release 8:54 AM
ADM: 289µV
AH: 125µV

Post Release 9:00 AM
ADM: 1344µV (465%)
AH: 781µV (625%)

Stimulus Parameters (Pre & Post):
Current (1.5x saturation): 9.0mA
Pulse Width: 100µs

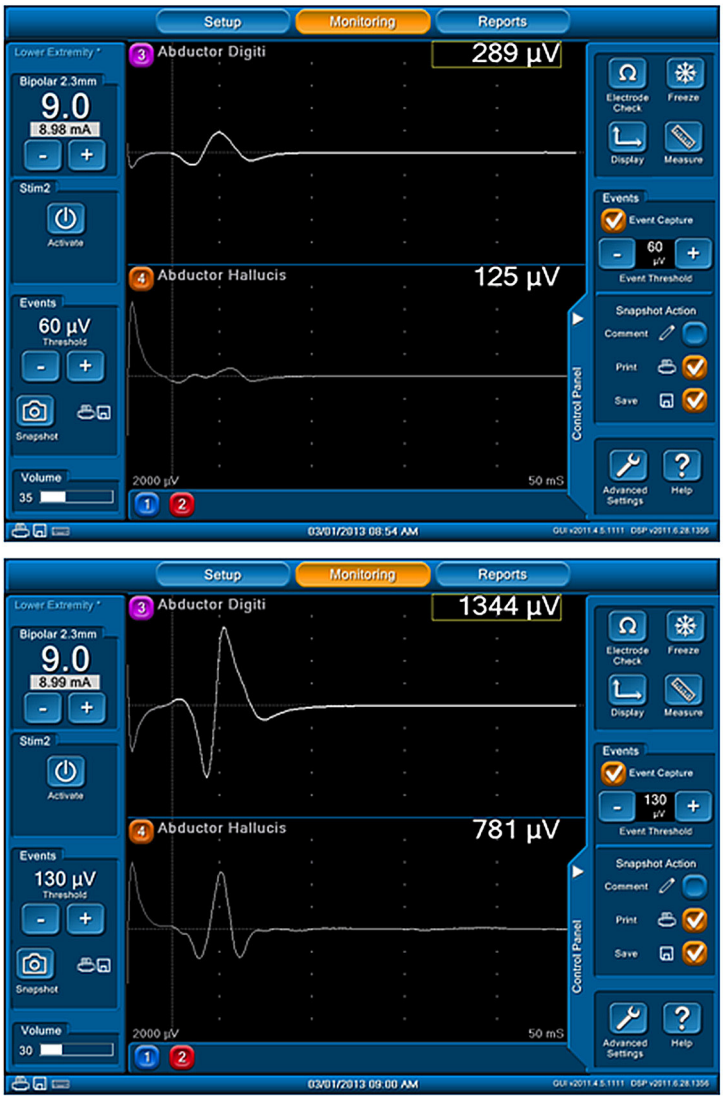

Figure 2. An electromyographic screenshot of foot muscle motor-evoked potential recording before and immediately after external neurolysis of the posterior tibial nerve and its medial and lateral plantar nerve branches. During this 6 min period of tarsal tunnel release, the objective recorded potential values increase by 400%.

Some initially mischaracterized, and still indict,[2] the ND hypothesis as proffering a "cure" for DSPN. Proponents believe instead they are decompressing focal nerve entrapments at anatomic chokepoints. Limited appreciation of variable, asymmetric, and non-global sensory changes[45] prevented recognition of entrapments and contemplation that length-dependent axonopathy (LDA) might fail to adequately explain numbness patterns. The Dellon hypothesis of frequent, metabolically induced, superimposed subclinical entrapment challenges the LDA paradigm as incomplete or inaccurate. Paradigm change can be difficult, challenging, and slow.

Research Directions

A number of investigations could further assess ND safety and benefit or elucidate mechanisms protecting against DSPN wounds:

1. Randomized control protocols should test whether ND is indeed long-term protective for amputation and mortality risk as it appears for ulceration.

2. Protective effects of ND in DFU with negative Tinel's sign and neuroischemia needs prospective testing.
3. Preoperative and postoperative documentation of one- and two-point sensibility, contralateral leg serving as control, with sensitive modalities like the Pressure Specified Sensory Device (Sensory Management Services LLC, Baltimore, MD),[46] need to validate postsurgical sensory improvement.
4. Intraoperative electromyography evaluations with technologies like the Nerve Integrity Monitor (Medtronic, Jacksonville, FL) can show immediate improvement in electromyographic function as in **Figure 2**.
5. Laser Doppler flowmetry, SPY indocyanine green imaging and OxyView hyperspectral oxyhemoglobin/deoxyhemoglobin ratios can examine skin and tissue perfusion changes.
6. Balance improvement[33] with bilateral ND can be correlated to fall risk and gait changes.
7. Nerve enlargement per ultrasound[47,48] needs post-ND evaluation.
8. Reports of rapidly resolving nerve indentations at operation invite correlation to axoplasmic flow or pressure changes.
9. Reports that hammertoes or claw toes resolve post-ND[49] need objective magnetic resonance imaging of plantar muscles.
10. Observations of skin atrophy and ichthyosis resolution lack photographic records.
11. Low operative infection rates reported by Zhang and coauthors,[36] inconsistent with reports of high risk of surgical site infection in DSPN, should be confirmed.[50,51]

Conclusion

Tenets of Dellon's entrapment hypothesis have been demonstrated, i.e., nerve enlargement, relief of pain, and recovery of sensibility after external neurolysis at fibro-osseous tunnels. Newly appreciated ND effects include recovery in objective outcomes such as balance, perineural pressure, NCV, and protection from ulcer occurrence, recurrence, and amputation risk. Oxford EBM evidence levels are

- For the hypothesized nerve enlargement in DSPN by strong objective level I evidence;[47,48]
- Asymmetry and variability of global foot sensibility, Oxford EBM level II-1;[45,52]
- High perineural tissue pressures within inflexible anatomic tunnel structures, and relief of pressure by ND, Level II-1;[35]
- Subjective relief of DSPN pain, recovery of lost sensibility, and improved symptom scores, Level II-2 and II-3 evidence;[36,42]
- Improved objective outcomes, including balance, perineural pressure, NCV, ulcer occurrence, prolonged protection from recurrence, and amputation risk, are supported by Level II-1 to Level II-2 evidence.-

These clinical observations are consistent with the Dellon hypothesis and inconsistent with LDA as a complete etiological hypothesis. Length-dependent axonopathy has no explanation for why these DSPN phenomena might be improved by ND. The Dellon hypothesis of frequent superimposed, metabolically induced, focal nerve entrapment clearly allows one to comprehend this otherwise mystifying data. The prospect that ND minimizes ulceration or reulceration and might protect against progression to amputation risks should be under consideration and investigation by the entire foot care community.

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