Rationale, Science, and Economics of Surgical Nerve Decompression for Diabetic Neuropathy Foot Complications

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KEYWORDS

- Diabetic neuropathy • Diabetes foot complications • DSPN pain
- Diabetes nerve compression • Nerve decompression
- Diabetes neuropathy economics

KEY POINTS

- Diabetic symmetric peripheral neuropathy (DSPN), also distal sensorimotor polyneuropathy, is responsible for a sizable percentage of complaints seen by podiatrists in North America.
- Neuropathy patients may have pain, numbness, deformities, and restriction of motion, which can lead to ulceration, local infection, sepsis, amputation, and death.
- Current treatments are directed at the control of those signs and symptoms rather than minimizing the precipitating neuropathy that is universally present.
- The current paradigm of etiopathogenesis, length-dependent axonopathy (LDA), overlooks evidence suggesting that peripheral nerve compression and entrapment, which is amenable to surgical decompression, is significantly contributory to DSPN.
- Peer-reviewed literature reports subjective and objective benefits after peripheral nerve decompression (ND) surgery. Reduction of pain, increased sensibility, including 2-point discrimination, improved balance, healing of initial diabetic foot ulceration (DFU), reduction in DFU recurrence and subsequent amputation, improved vascularity measured by transcutaneous Po_2, and improvement of nerve conduction velocity and motor evoked potentials electromyography have all been documented.

INTRODUCTION

Addressing podiatric complications of diabetes mellitus is a large and complicated prospect in which important strides have been made; however, it is still a very great challenge to patients, physicians, insurers, and society as a whole. This article examines the magnitude and parameters of the problem and ventures into the topic of

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Clin Podiatr Med Surg 33 (2016) 267–282
http://dx.doi.org/10.1016/j.cpm.2015.12.004 **podiatric.theclinics.com**
0891-8422/16/$ – see front matter

surgical nerve decompression (ND) in diabetes. Surgical ND is an effective generation-old method, which, regrettably, is still seriously in the academic wilderness. This article reviews the initial clinical insight, laboratory, anatomic and clinical evidence, and germane recent science suggesting the involvement of physical compression in diabetic symmetric peripheral neuropathy (DSPN). This article offers some thoughts on the deep academic skepticism of ND, and the splendid opportunity that ND seems to offer for neuropathic pain relief, avoidance of diabetic foot complications, and reduction of medical expense and societal costs.

The most common nerve problem encountered in foot and ankle practice in the developed world is diabetic neuropathy. Diabetes mellitus is a disease notable for disturbances of glucose metabolism. Neuropathy is frequently present either initially or eventually in the course of this disease, with an incidence estimated at 30% to 70%.[1,2] The most frequent presentation, to which this article is limited, is diabetic sensorimotor polyneuropathy, often termed DSPN, and described as a progressive, symmetric, distal peripheral neuropathy. Stocking-and-glove anesthesia is a standard clinical description.

PATHOGENESIS

DSPN is often explained as a "dying back" of axons. The classic understanding presumes DSPN to be an irreversible condition beginning usually in the feet, appearing later in the upper extremity, and progressing in severity and proximal involvement with time. The theoretic pathologic cause is termed length-dependent axonopathy (LDA), implying that the metabolic disturbances initially and most severely affect the longest axons, and progressively involve shorter axons as disease persists. At the molecular level, how diabetes produces differing physiologic effects based on axon length has not been explained.

DIABETIC SYMMETRIC PERIPHERAL NEUROPATHY COMPLICATIONS

In any case, DSPN clinically leads to leg and foot symptoms, signs, and a cascade of increasingly severe complications, which often lead to progressive debility and life changes resulting in early demise. Early complaints may be tingling, burning, dry skin, lancinating pains, and numbness. Physical changes of calluses, clawed or hammer toes, and nail alteration are frequently accompanied by joint contracture, atrophic skin, local concentrations of increased tissue pressure, intrinsic muscle atrophy, loss of ankle motion, and deep tendon reflex attenuation. With numbness comes increased risk of foot injury and delayed recognition of tissue damage or wounds, allowing progression of deformity or local infection. The infected wound can progress to sepsis, gangrene, minor or major amputations, and even death. The hazards of this dreadful state of affairs have led to efforts at abatement such as the federally sponsored Lower Extremity Amputation Prevention (LEAP) program. LEAP counsels 5 relatively simple activities for diabetes patients, listed in **Box 1**. Primary care providers should attend to initial diabetic foot ulceration (DFU) prevention activities summarized in **Box 2**.

TREATMENTS AND OUTCOMES

Recommended interventions for avoiding the cascade of DSPN complications are based on an understanding that neuropathy is a universal constant; however, its sequelae can be recognized and minimized, or prevented. Repetitive stress on the skin in the presence or absence of deformity and loss of joint mobility can lead to skin inflammation and eventual tissue breakdown generating a wound, with faulty healing resulting in a chronic ulcer. Sensibility loss complicates the situation with late wound recognition and anesthesia allowing tolerance of continued ambulation