Peripheral Diabetic Neuropathy: Current Concepts in Treatment
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Setting the scene:
To review pathophysiology and current concepts in the treatment of diabetic peripheral neuropathy (PN)

What did they do?
The exact etiology and pathogenesis of PN is unclear. PN may be the result of insulin deficiency or hyperglycemia coupled with poorly defined genetic or environmental factors. Several prominent theories have emerged to explain the pathogenesis of PN. These include (1) accumulation of sorbitolin the nerve cells,(2) decrease in nerve free myoinositol and decreased activity of nerve sodium potassium adenosine triphosphatase, (3)increased non-enzymatic peripheral nerve glycosylation, and (4)nerve hypoxia. PN affects 5-50% of people with diabetes and most commonly is characterized by tingling or burning sensations, particularly in the calves, ankles, and feet, with a loss of vibratory sense. Treatment of PN, for them most part, has been unsatisfactory. Therapy has been directed toward either improving nerve function or alleviating symptoms of PN, including pain and paresthesia. Glycemic control may slow the progression of PN. Hyperglycemia also is associated with decreased pain threshold in patients with diabetes mellitus. The aldose reductase inhibitors, particularly tolrestat, have been shown to improve objective and subjective neurologic function. Pain or paresthesia has been treated effectively with antidepressants, lidocaine, mexiletine, and capsaicin. The anticonvulsants phenytoin and carbamazepine may be effective, but are associated with a greater degree of adverse effects. Experimental treatments, such as gamma-linolenic acid, gangliosides, uridine, and the corticotropinz, analogORG2766, have been effective in improving neurologic function.

Takeaway message:
Treatment of PN remains unsatisfactory. Therapy should be directed toward prevention with glycemic control and symptomatic treatment of existing PN.