

Comment on ‘Treatment of Painful Diabetic Neuropathy with 10 kHz Spinal Cord Stimulation: Long-Term Improvements in Hemoglobin A1c, Weight, and Sleep Accompany Pain Relief for People with Type 2 Diabetes’ [Letter]

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Dear editor

We read with great interest the recent article by Klonoff et al, titled

Treatment of Painful Diabetic Neuropathy with 10 kHz Spinal Cord Stimulation: Long-Term Improvements in Hemoglobin A1c, Weight, and Sleep Accompany Pain Relief for People with Type 2 Diabetes.¹

This study provides compelling evidence that high-frequency (10 kHz) spinal cord stimulation (SCS) not only offers significant pain relief for individuals with painful diabetic neuropathy (PDN) but also results in long-term improvements in glycemic control and weight management. These findings are particularly relevant given the growing prevalence of type 2 diabetes (T2D) and the associated burden of neuropathic pain.

While the study presents promising results, several limitations and areas for further inquiry should be considered. First, the study's post hoc nature and the lack of a control group for the full 24-month period limit the ability to draw definitive conclusions about the causality of the observed improvements in HbA1c and weight. Future studies should consider randomized controlled trials with longer follow-up periods to confirm these findings.

Second, the study acknowledges the potential for bias due to the lack of blinding, which may affect patient-reported outcomes such as pain intensity and sleep quality. Objective measures of pain and sleep, such as actigraphy or polysomnography, could provide additional validation of these subjective reports.

Third, the study's findings are based on a specific patient population with refractory PDN, which may limit the generalizability of the results to other patient groups. Future research should explore the effectiveness of 10 kHz SCS in a broader range of patients with PDN, including those with less severe or earlier-stage disease.

Fourth, while the study suggests potential metabolic benefits of 10 kHz SCS, the underlying mechanisms remain unclear. Future studies should investigate the potential pathways through which SCS may influence glycemic control and weight, such as changes in inflammatory markers, insulin sensitivity, or sympathetic tone.²

Lastly, the study's reliance on standard clinical care for glycemic management introduces potential confounding factors. Future studies should include detailed assessments of diabetes medication changes and their impact on HbA1c and weight to better understand the independent effects of SCS.

In conclusion the study by Klonoff et al provides valuable insights into the potential benefits of 10 kHz SCS for managing PDN and improving metabolic outcomes in individuals with T2D. The findings suggest that 10 kHz SCS may offer a durable and multifaceted treatment option for PDN, addressing both pain relief and metabolic control. Future research should address the limitations identified here and further explore the mechanisms underlying the observed improvements. Such studies will be crucial for establishing 10 kHz SCS as a standard treatment option for PDN and informing clinical practice guidelines.

Disclosure

The author(s) report no conflicts of interest in this communication.

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