Peripheral median nerve stimulation for the treatment of iatrogenic complex regional pain syndrome (CRPS) type II after carpal tunnel surgery

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A B S T R A C T

We report on the use and follow-up of direct peripheral nerve stimulation of the median nerve for the treatment of iatrogenic complex regional pain syndrome (CRPS). A 56-year-old woman presented with CRPS type II in the right forearm and hand, which had started after multiple carpal tunnel surgeries and had lasted for 2 years. The visual analogue scale (VAS) score was 8–10 out of 10. After a successful 15-day trial of median nerve peripheral nerve stimulation via a quadripolar lead in the right carpal tunnel space, an implantable pulse generator was inserted in the right infracavicular space. The VAS score decreased to 1–2 out of 10 and the patient regained the ability to sleep. After 36 months of follow-up, the patient was still experiencing good pain relief without other treatment. We conclude that peripheral nerve stimulation is easy to use in pain management and could offer a valid treatment option for iatrogenic CRPS type II.

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1. Introduction

Peripheral nerve stimulation (PNS) is effective in the management of pain. It requires a surgical procedure to implant the surgical lead near the damaged nerve.

We describe direct peripheral stimulation of the median nerve in the successful treatment of chronic pain caused by iatrogenic complex regional pain syndrome (CRPS) type II after multiple carpal tunnel surgeries.

2. Case report

2.1. History and clinical presentation

A 56-year-old woman was referred to our department from a multidisciplinary pain clinic with documented CRPS type II (according to the International Association for the Study of Pain [IASP] revised criteria) in the right forearm and hand. The pain, which had lasted for 2 years, started after failed endoscopic carpal tunnel release and then open surgery performed in another hospital. Electromiographic studies confirmed severe right median nerve dysfunction at the wrist with markedly prolonged motor and sensory distal latencies. Hyperesthesia, allodynia, and hyperpathia were found mostly at the wrist and in the area of the medial nerve. Pain intensity during forearm movement and standing was measured using a visual analogue scale (VAS) (0–10-point) scale and a verbal examination scale (no pain, low, annoying, strong, and not tolerable). The average VAS score reported by this patient was 8 out of 10, with a maximum close to 10 during the night and a pain intensity characterized as “strong”. The McGill questionnaire score was 48 and the Hospital Anxiety Depression (HAD) scales indicated anxiety within the clinical range (12 out of 21), but no depression (6 out of 21). Physical examination revealed severe limitations of right wrist and hand motion. The skin was pale with signs of atrophy and an enlarged scar (Fig. 1A). External transcutaneous electrical nerve stimulation (TENS) therapy, along with pharmacotherapy with amitriptyline (up to 150 mg at night), gabapentin (up to 3600 mg per day), and an opioid trial failed to provide substantial pain relief. The patient also refused surgical sympathectomy after sympathetic block.

2.2. Two-stage surgical procedure

2.2.1. Stage 1

We performed the carpal tunnel exploration under local anaesthesia (6 mL 1% lidocaine without epinephrine) using a curvilinear, longitudinal incision that paralleled the thenar crease and reached the forearm. The antebrachial fascia was isolated, divided longitudinally and the median nerve was identified in the proximal portion of the surgical incision. The thickened transverse carpal ligament was found intact in its distal portion and divided longitudinally along the ulnar aspect of the median nerve until the fat that enveloped the superficial palmar arch. The intracanalar median nerve was pale and flattened (Fig. 1B). After freeing the medial nerve from inflammatory adhesions by gentle dissection and
external neurolysis, we inserted a paddle-style quadripolar electrode (Medtronic; Minneapolis, MN, USA) into the carpal tunnel space (between the vascular/median nerve bundle and the fascia) (Fig. 1C). Intraoperative testing was performed to assess whether stimulation produced paresthesias over the entire area of the patient’s pain.

We then secured the electrode with a suture to the subcutaneous tissue and underlying fascia (Fig. 1D) to avoid migration. Extension wiring was tunneled from the incision site to the exit site in the forearm skin so that the extended trial could be performed.

2.2.2. Extended Stimulation Trial

The patient was instructed on how to adjust the hand-held pulse generator and given a pain diary to record the results of trial stimulation. The patient reported pain relief during the 15-day stimulation trial. The stimulation amplitude ranged between 1.5 V and 2.2 V, achieving complete paresthesia of the right forearm and hand. The VAS score decreased to 2 out of 10 and the patient regained the ability to sleep.

2.2.3. Stage 2

A subcutaneous pocket was created in the right infraclavicular space. The extension cable was tunneled from the forearm incision to the pocket through a clean tunnel and connected to the electrode. The distal end of the extension wire was connected to an implantable neurostimulator (Synergy, Medtronic) that was secured in the infraclavicular subcutaneous pocket.

2.3. Follow-up

At the 6-month follow-up examination, the patient had stopped use of all pain medication and reported improvement of her sleep and functional status. Daily activities were now possible with little effort. At clinical examination signs of atrophy were clearly reduced. The VAS score has decreased to 2 out of 10 and the verbal pain intensity score had decreased from “strong” to “low” pain. The McGill questionnaire score was 17 and HAD score for anxiety improved to within the normal range (6 out of 21). At 36 months of follow-up, the patient reported continuing good results.

3. Discussion

CRPS, previously referred to as reflex sympathetic dystrophy, causalgia and algodystrophy, is an uncommon and poorly understood neuropathy that normally affects the limbs.

CRPS type I occurs after minor injury that may be unnoticed by the patient because it partially or predominantly affects unmyelinated
CRPS type II is associated with an identifiable nerve injury, often following trauma or surgery. A validation study of the IASP criteria for CRPS indicated significant overdiagnosis and proposed a set of more specific diagnostic criteria as follows:

1. continuing pain that is disproportionate to any inciting event 
2. the patient must report at least one symptom in each of the four following categories (a–d) 
3. the patient must display at least one sign in two or more of the following four categories: 
   (a) sensory: hyperalgesia and/or allodynia 
   (b) vasomotor: temperature asymmetry; and/or skin color changes and/or asymmetry 
   (c) sudomotor/edema: edema and/or sweating changes and/or sweating asymmetry 
   (d) motor: decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin). 

CRPS has been reported as a complication of surgical carpal tunnel release in 2% to 5% of patients. Its management presents a challenge: multiple drug therapy, nerve blocks, and physical therapy (including TENS and biofeedback) and also surgical sympathectomy have been proposed. There are diverse experiences and mainly anecdotal outcomes reported for spinal cord stimulation, intrathecal infusion, and PNS for the management of CRPS type I and II. Spinal cord stimulation (SCS) is a well-established treatment, even if the results are controversial, in particular for CRPS type II. SCS does not produce a durable and statistically significant improvement in the pain from chronic CRPS-I. Nevertheless, patient satisfaction at the 5-year follow-up remains high.

PNS can be explained by the “gate-control” theory of pain. PNS is likely to recruit more nerve fibers for activating inhibitory interneurons than SCS, whose direct electrical effects at the spinal cord level are “buffered” by the cerebrospinal fluid, which is an excellent conductor of electrical field energy, and is influenced by the patient’s spinal anatomy. PNS also permits recruitment of primary afferent A-β fibers, which project to the spinothalamic tract and not only to the dorsal columns as occurs in SCS. Waisbrod et al. described 11 cases of painful neuropathies managed with PNS. The pain relief was 58% complete and 21% partial with an average of 11.5 months follow-up. Picaza et al. reported on 37 patients treated by PNS and followed for greater than 1 year, stating 50% long-term success in nerve injury after trauma. In a review by Long, PNS for painful neuropathies of nerve injury origin was reported to have good effect in 82.5%, whereas the response rates in other kinds of pain were 25% to 50%.

Weiner and Reed have also reported on the use of PNS via a percutaneous approach mostly for occipital neuralgia. Electrode migration has been a significant complication of using cylindrical electrodes. In Weiner and Reed’s original study, 13 of 35 patients required surgical revision for cylindrical electrode migration. Percutaneous peripheral stimulation of the median nerve into the interscalene space after injury due to occlusion of the right omeral artery has also been reported. Electrode migration occurred after 2 weeks and surgical revision was necessary. Use of a paddle style electrode rather than the cylindrical style may have advantages in this region in reducing migration from muscular tension or anchor dislodgment. This electrode platform has a larger profile and can be secured by a suture to the subcutaneous tissue or underlying fascia, with or without an anchor. It is also able to direct the delivery of electrical current toward the nerve “anteriorly” with less posterior spread. The anticipated benefit from this “anteriorly” directed current would be lower perception and usage ranges, and subsequent increased battery longevity.

4. Conclusions

Peripheral nerve stimulation is a valid treatment option for pain resulting from nerve injury such as in CRPS type II. Because these injuries are relatively rare, there is little application for PNS. Nevertheless our data indicate that this could be a very successful technique for the few patients who require it.

References