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Fully Implantable Peripheral Nerve Stimulation for the Treatment of Hemiplegic Shoulder Pain: A Case Report

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Abstract

This case report describes the first participant treated with a fully-implantable, single-lead peripheral nerve stimulation (PNS) system for refractory hemiplegic shoulder pain (HSP). During the 6-wk trial-stage, a temporary lead was placed percutaneously near the terminal branches of the axillary nerve to the deltoid. The primary outcome measure was the Brief Pain Inventory-Short Form Question 3 (BPI-3), a 0–10 pain numeric rating scale. The participant experienced 75% pain reduction and proceeded to the implant-stage where he received a single-lead, implantable pulse generator. After 3-wks, the participant became pain-free. However, 7-wks after implantation, the system was turned off due to an unrelated acute medical illness. HSP reemerged with BPI-3 of 9. After 11-wks of recovery, PNS was reinitiated and the participant became pain-free through the 9-months follow-up. At 12-months, BPI-3 was a 1. This case report demonstrates the feasibility of a single-lead, fully-implantable PNS system for refractory HSP.

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Keywords

Shoulder Pain; Stroke; Peripheral Nerve Stimulation; Implant

INTRODUCTION

Post-stroke hemiplegic shoulder pain (HSP) is highly prevalent.^{1–3} Of those who suffer from HSP, up to 75% report moderate to severe pain² with a third refractory to available treatments.⁴ Data suggest that surface electrical stimulation (ES) is efficacious for treating HSP.^{5–7} However, it is not well-tolerated and requires skilled personnel to maintain.⁸ A percutaneous peripheral nerve stimulation (PNS) system that stimulates the terminal branches of the axillary nerve to the deltoid muscle was developed to address these limitations.

Clinical trials have confirmed the efficacy of short-term percutaneous PNS in reducing HSP.^{9–14} Stroke survivors who are treated within 18-mo of their stroke experience sustain pain relief after completion of treatment. However, those who are greater than 18-months post-stroke experience significant relief initially, but pain returns within 3-mo.¹⁵ The use of percutaneous PNS beyond 6-wks is not desirable due to the potential increased risk of skin irritation, electrode fracture during removal and electrode-related infections.^{16,17}

For patients who experience only short-term benefit from percutaneous PNS, long-term PNS from a fully implantable system may provide enduring pain relief. A two-stage PNS system has been developed. The trial-stage provides PNS via a temporary, percutaneously placed lead near the terminal branches of the axillary nerve to the deltoid muscle to identify candidates who are likely to benefit from long-term PNS. Patients who qualify receive an implantable pulse generator (IPG) in the anterior chest and an implantable lead near the terminal branches of the axillary nerve to the deltoid via a minimally invasive procedure. This case report describes the first stroke survivor treated with the two-stage PNS system for chronic, refractory HSP.

PREINTERVENTION CLINICAL COURSE

The participant was a 77-year-old male smoker with past medical history of hypertension, hyperlipidemia, prior myocardial infarction (MI), and chronic obstructive pulmonary disease who experienced an ischemic stroke with residual left hemiparesis 10.8-yrs prior to enrollment. He developed HSP shortly after his stroke, which was treated with multiple non-opiate oral analgesics, physical therapy, occupational therapy, and surface ES. These treatments only provided short-term relief.

The participant exhibited left hemiparesis with Medical Research Council muscle grade of 4 for shoulder abduction, and elbow flexion and extension; and 0.5–1 fingerbreadth subluxation. He was cognitively appropriate (Mini-Mental State Exam score 26). His shoulder pain-free passive external rotation ROM was 90° on the affected side and 180° on the unaffected side. He had not received corticosteroid injections, botulinum toxin

injections, acupuncture, or PNS in the prior 3-months. He was instructed to avoid all other treatments during the study.

METHODS

The study protocol was approved by the local Institutional Review Board and conducted under an Investigational Device Exemption from the U.S. Food and Drug Administration. Key inclusion criteria were focused on the adult, stable hemiplegic stroke survivor who either failed or was unable to tolerate at least two conservative interventions for the treatment of shoulder pain. Crucial exclusion criteria were focused on safety and avoidance of subjects with infection, bleeding diathesis, history of arrhythmia with hemodynamic instability, poorly-controlled diabetes, and immune compromise. Also, outcome confounders were minimized with exclusion of subjects taking opioids; having recent shoulder steroid injections or botulinum toxin injection to the affected arm; receiving active therapy; or experiencing disruption to communication capability. Written informed consent was obtained.

Outcome Measures

The primary outcome was the Brief Pain Inventory-Short Form Question 3 (BPI-3), which rates the worst pain in the past week on a 0–10 numeric rating scale (NRS), where 0 indicates “no pain” and 10 indicates “pain as bad as you can imagine.”^{18,19} Secondary outcome measures included BPI-9,^{18,19} Short Form-36 (SF-36v2),²⁰ the Patient Global Impression of Change (PGIC),²¹ and pain-free shoulder passive external rotation ROM. The BPI-9 evaluates pain interference with daily activities during the prior week on a 0–10 NRS, where 0 indicates “does not interfere” and 10 indicates “completely interferes.” The SF-36v2 is a 36-question health survey that evaluates 8 domains: physical functioning, role-physical, bodily pain, general health, vitality, social functioning, role-emotional, and mental health. To facilitate interpretation, norm-based scoring scales each domain to a population mean of 50 and a standard deviation (SD) of 10. The PGIC measures the participant’s impression of changes in quality of life (QoL) since the beginning of the intervention on a 7-point scale from “very much worse” to “very much improved.” The pain-free shoulder ROM was performed in a supine position with shoulder at 0° of abduction. External rotation ROM was measured utilizing a goniometer starting at the fully internally rotated position.

Study Procedures

Trial-stage—The trial-stage consisted of a 3-wk sham period and a 3-wk stimulation period. The external stimulator was a commercially available device (Rehabicare NT2000, Empi, Minneapolis, MN) that generates a biphasic current waveform. The self-anchoring percutaneous lead was previously described.¹²

A physiatrist (V.Q.C.N.) implanted the percutaneous lead using a previously described technique.⁹ The participant was informed that he would receive different levels of stimulation and that he may or may not feel stimulation. The stimulator was programmed to deliver pulse duration of 50 μ sec, amplitude of 20 mA, frequency of 12 Hz, and duty cycle of 50% for 6-hrs daily. These parameters were selected based on prior studies.^{9,10,22,23} Prior

studies demonstrated that stimulation does not need to be on 24-hrs a day due to carry-over.^{9,10,22,23} During the sham period, the stimulator was connected to a nonfunctioning cable such that the stimulator appeared to deliver stimulation, but no stimulation was delivered. At the end of the sham period, the cable was switched to a functional cable to begin treatment. At the end of the stimulation period, the lead was removed with gentle traction.

Primary and secondary outcome measures were administered while the device was off at baseline, at the end of the sham period, and at the end of the stimulation period. The participant met the criterion for entry into the implant-stage, which was defined as 2-point reduction in BPI-3 at the end of the stimulation period relative to the end of the sham period. Previous studies have determined that a 2-point reduction in a 0–10 pain numeric rating scale is the minimum clinically important difference.^{24,25}

Implant-stage—The implant-stage IPG was a small (approximately $8 \times 30 \times 49$ mm), single-channel stimulator (MICROPULSE®, NDI Medical, Cleveland, OH, Figure 1) that outputs a current waveform identical to the external stimulator. The IPG's lithium-ion battery is recharged via a portable, wireless, transcutaneously applied RF magnetic field for approximately 5-hrs every 1–2 weeks, allowing the participant to maintain his daily activities while recharging. The self-anchoring implantable lead was previously described.²⁶

The IPG and lead were placed under local anesthesia and conscious sedation by a cardiologist (W.B.) with experience in placing cardiac pacemakers (Figure 1). Monopolar needle stimulation identified the target location of the lead (position A, Figure 2). The introducer (a tapered needle probe in a sheath) was inserted at least 1 cm deep into the muscle and the needle was removed; the lead was inserted into the introducer sheath, and the sheath was withdrawn leaving the lead anchored in place. The proximal end of the lead was subcutaneously tunneled to position B and then to C with lead slack coiled for tension relief. The IPG was placed in a subcutaneous pocket on the anterior chest wall (position C). The participant was discharged on the same day.

After a week of lead stabilization, the system was programmed and stimulation was initiated. The stimulation parameters were identical to the trial-stage, resulting in a strong, but comfortable contraction of the middle and posterior deltoid muscles. The participant was set to be followed for outcomes assessment with the device off at 3, 6, and 12-wks and 6, 9, and 12-months.

RESULTS

Trial-stage

The participant tolerated the trial-stage procedures without complications. The stimulator recorded compliance rates of 74% and 96% during sham and stimulation periods, respectively. There was no evidence of infection or skin irritation at the lead site. At the end of the trial-stage, the lead was removed intact.

BPI-3 results are shown in Figure 3. The participant experienced 37.5% pain reduction during the sham period and an additional 37.5% pain reduction during the stimulation period. Table 1 shows the results for the secondary measures. In contrast to BPI 3, changes in BPI-9, ROM, and PGIC were significantly greater during the stimulation period than during the sham period. We elected to go forward with the implant stage primarily because the a-priori criterion was satisfied, but also because the secondary measures convincingly discriminated between the placebo and stimulation periods.

Implant-stage

The IPG was placed without complications. By the end of the 3rd week of PNS, the participant was pain-free (Figure 3). Improvements in BPI 9, ROM and PGIC were maintained (Table 1). Six domains of SF-36v2 were now within one SD of the population mean.

At 7-wks, the participant suffered a myocardial infarction (MI), was hospitalized, and the IPG was turned off for safety monitoring. Two cardiologists independently concluded that the event was related to his coronary risk factors and not to the study device. He subsequently underwent coronary artery bypass grafting.

HSP reemerged after the MI with BPI-3 of 3 at 6-wks and 9 at 11-wks. At 9-wks the participant was medically cleared to resume participation in the protocol. The protocol was reinitiated with a new start of stimulation time point. The participant became pain-free within 6-wks (Figure 3). At 6 and 9-months, the participant continued to report no pain, and PNS was reduced to 4 and 3-hrs daily, respectively. At 12-months, BPI 3 was a 1, and PNS was maintained at 3-hrs daily with maintenance of carry-over effect throughout the day.

BPI-9, ROM and PGIC data paralleled that of BPI-3 (Table 1). However, after 6-months, the number of SF-36v2 domains that were within one SD of the population mean progressively declined. Among the domains that had improved to within one SD, only the bodily pain domain remained within one SD at 12-months.

DISCUSSION

This case report describes the first stroke survivor to receive a fully implantable, single-lead PNS system for the treatment of chronic, refractory HSP. The participant experienced significant pain reduction during the trial-stage and proceeded to the implant-stage, where he became pain-free within 3-wks. Due to an MI, the IPG was turned off for 11-wks and shoulder pain returned; however, with the reinitiation of PNS, the participant experienced significant and sustained pain reduction. ROM, BPI-9, and QoL also improved. This case report demonstrates the feasibility of the system.

The results are consistent with prior studies on percutaneous PNS.^{9–13} Significant pain reduction was observed within 3-wks of start of PNS during both stages. Post-hoc analysis of a multi-site RCT^{10,13} suggested that stroke survivors who are beyond 18-months post-stroke experience only short-term pain reduction.¹⁵ Consistent with this prediction, when the IPG was turned off, pain returned within 6-wks. The participant became pain-free when the

PNS was reinitiated. While participants who were beyond 18-months post-stroke in our prior percutaneous RCT experienced only short-term benefit, the IPG allowed this participant to maintain long-term pain relief.

The mechanism of PNS-mediated pain relief is unknown, but may include improvement in biomechanics of the glenohumeral joint,¹² the Gate Theory,²⁷ and reversal of central sensitization.²⁸ Improved biomechanics is unlikely as the RCT that only enrolled participants with glenohumeral subluxation failed to show evidence of improved motor function or reduction of subluxation.^{10,13} Two more recent trials enrolled stroke patients with and without glenohumeral subluxation;^{9,14} however, participants in both groups experienced significant pain reduction, which further suggests that improved biomechanics of the glenohumeral joint is not the mechanism of action.

The Gate Theory postulates that stimulation of low-threshold myelinated primary afferent fibers decreases the response of dorsal horn neurons to unmyelinated nociceptors.²⁷ The theory also predicts that once the non-noxious afferent input is removed pain recurs, which is consistent with clinical experience with TENS.²⁹ Thus, the presence of a long “carry-over” effect in prior studies,^{9–14} and to a lesser degree in the present study, also makes the Gate Theory an unlikely explanation for the observed results.

Finally, PNS may modulate central sensitization, which has been shown to be present in chronic HSP²⁸ as well as in multiple other chronic musculoskeletal pain conditions.³⁰ Data in support of this hypothesis are emerging, but are preliminary. If PNS reduces chronic HSP via modulation of central sensitization, and central sensitization is the convergent mechanism for all chronic musculoskeletal pain, then PNS should also reduce non-stroke related shoulder pain. We first demonstrated that central sensitization was present among neurologically intact patients with chronic subacromial impingement syndrome.³¹ We then treated 10 patients with chronic, refractory subacromial impingement syndrome with percutaneous PNS and demonstrated significant pain reduction with maintenance of pain reduction for 12-wks.^{32,33} The study also demonstrated preliminary evidence of reduction in secondary hyperalgesia, which is consistent with reduction in central sensitization. Additional mechanistic studies are needed to more definitively evaluate these three potential mechanisms.

Depending on the mechanism of action, PNS may have broader implications beyond the treatment of HSP. If the mechanism for pain control is improvement in glenohumeral biomechanics, further research into the utilization of PNS for rotator cuff injury or impingement syndrome is warranted. If the mechanism is associated with the Gate Theory, future research is indicated for the treatment of many neuropathic pain processes. These could include post-amputation phantom pain sensation, complex regional pain syndrome, herpetic neuralgia, or diabetic neuropathy. Finally, if the mechanism is modulation of central sensitization, then it is reasonable to explore the use of PNS for any condition associated with chronic musculoskeletal pain.

Improvements in ROM, BPI-9 and PGIC were maintained for 12-months; however, initial improvements in SF-36v2 subsided by 12-months. A possible explanation for this

discordance is that ROM, BPI-9 and PGIC were shoulder specific, whereas SF-36v2 evaluated overall health-related QoL. With the onset of MI, all SF-36v2 domains deteriorated and never returned to the pre-MI level. Thus, the broad health related impact of his health complications may have contributed to the lower than expected results for the SF-36v2.

In our prior publications, we used the term “intramuscular” electrical stimulation and not PNS. We elected to make this change because the term “intramuscular” suggests direct stimulation of muscle. However, when low levels of current are used to cause muscle contraction, as utilized in this protocol and other functional electrical stimulation protocols, low threshold alpha motor neurons innervating the muscle are being stimulated, not muscle.³⁴ The threshold for causing muscle contraction via direct muscle stimulation is substantially higher than that for causing muscle contraction via motor nerve stimulation.³⁵ Thus, stimulation of denervated muscle requires substantially higher intensity current.³⁶

While case reports are appropriate for introducing novel interventions, they have inherent limitations. Results may not be generalizable to the general stroke population. Since there are no controls, cause and effect cannot be invoked and spontaneous recovery cannot be excluded. Even though a placebo effect was evaluated, it cannot be ruled out in the present case as the slope of BPI-3 reduction during the sham period was identical to that of the stimulation period. Because outcomes were not blinded, assessor bias cannot be ruled out. The optimal dose is not known. It is possible that with longer stimulation afforded by a fully implantable system, a longer carry-over effect might be produced and daily stimulation may not be necessary. The duration of therapeutic effect and adverse effects beyond the 12-mo period are not known. This participant will continue to be followed for both for at least 2 more years. Finally, per the above discussion, without confirming a clear mechanism of action for the pain reduction, implications for further generalization are limited.

CONCLUSIONS

This case report demonstrates the feasibility of a fully implanted PNS system for the treatment of chronic, refractory HSP. Additional studies will evaluate efficacy and safety, elucidate the mechanism of action, define optimal prescriptive parameters, expand clinical indications, and demonstrate long-term effect.

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Figure 1.
Radiograph of IPG and lead.

Figure 2.
Schematic showing the relative positions of IPG and lead.

Figure 3.
BPI-3 scores during trial and implant-stages.

Table 1

Results of secondary outcome measures.

Trial Stage			Implant Stage Pre-acute Illness			Implant Stage Post-acute Illness				
Baseline	End of Sham	End of Treatment	3-wks IPG On	6-wks IPG On	3-wks IPG On	6-wks IPG On	12-wks IPG On	6-mos IPG On	9-mos IPG On	12-mo IPG On
Week	0	6	14	17	32	35	41	55	68	81
BPI-3	8	2	0	0	3	0	0	0	0	1
BPI-9	6.43	1.86	0	0	0.14	0	0	0	0	0
ROM (deg)	90	160	160	-	90	-	130	145	150	150
PGIC	No change	Much improved	Very much improved	-	Very much improved	-	Much improved	Very much improved	Much improved	Very much improved
SF-36v2										
PF	26.4	22.3	30.5	-	28.5	-	30.5	28.5	28.5	32.6
RP	20.8	25.6	28.0	-	24.8	-	28.0	37.5	28.0	25.6
BP	32.1	32.1	60.9*	-	40.9*	-	36.3	50.0*	45.9*	40.1*
GH	38.8	30.8	62.9*	-	37.9	-	37.9	41.2*	38.8	31.8
VT	49.0*	37.0	52.0*	-	49.0*	-	46.0*	57.9*	49.0*	46.0*
SF	40.3*	29.5	56.4*	-	45.7*	-	40.3*	45.7*	40.3*	34.9
RE	33.0	10.3	40.5*	-	40.5*	-	44.3*	44.3*	25.4	33.0
MH	27.4	35.7	52.4*	-	52.4*	-	44.0*	52.4*	38.5	35.7
Within 1 SD**	2	0	6	-	5	-	4	6	3	2

SF-36v2 domains: PF-physical functioning; RP-role physical; BP-bodily pain; GH-general health; VT-vitality; SF-social functioning; RE-role emotional; MH-mental health.

* Within one SD of population norm.

** Number of SF-36v2 domains within one SD of population norm.