
ORIGINAL ARTICLE

Occipital Nerve Stimulator Lead Pathway Length Changes with Volunteer Movement: An In Vitro Study

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■ Abstract

Background: Occipital nerve stimulation is a modality reserved for refractory headache disorders. Leads (wires) are inserted subcutaneously in the occipital region to stimulate the distal C1-3 nerves; lead migration may result from repeated mechanical forces on the lead associated with patient movement. The primary aim of this study was to determine implantation pathways associated with the least pathway length change secondary to body movement in an in vitro model of an occipital stimulator system.

Methods: After institutional review board approval, 10 volunteers were recruited. The expected pathway of an occipital stimulator system was identified and measured externally, and then changes in pathway length were measured during various volunteer movements, including neck and low back flexion, extension, rotation, and lateral flexion. The pathways studied included those that connect internal pulse

generators in the gluteal, low abdominal, and infraclavicular regions to occipital leads inserted via a cervical or retromastoid approach.

Results: The flexion/extension pathway length changes associated with midline occipital and retromastoid sites to the infraclavicular site were significantly less than those pathways to the periscapular site. Also, the abdominal site was associated with less pathway length change during flexion/extension than the gluteal site.

Conclusions: Internal pulse generators in sites other than the buttock, including infraclavicular or low abdomen, may be associated with lower lead migration risk.

There are many considerations when selecting insertion sites and lead pathways for occipital nerve stimulation. Implanters and patients may consider these results when contemplating surgical approaches to this challenging form of peripheral nerve stimulation. ■

Key Words: headache, complications, implantation, occipital nerve stimulation

INTRODUCTION

In 1999, Weiner reintroduced the technique of peripheral nerve stimulation to treat headache disorders.¹ In occipital nerve stimulation (ONS), a lead (inert polyurethane covered wire) is inserted subcutaneously in the occipital region to stimulate the distal branches of the C1-3 nerves. The wire is connected to a remotely

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implanted internal pulse generator (IPG). The lead and an extension cable (if needed) are tunneled subcutaneously from the occiput to the IPG. Small, uncontrolled case series describe both benefits of ONS and complications such as lead migration and erosion.¹⁻⁶

Lead migration (wire movement resulting in loss of effective stimulation) can be a distressing complication of ONS, especially for previously refractory patients experiencing excellent headache relief. Mechanical stress on the ONS components can be significant because the lead and extension may traverse highly mobile body regions, including the neck, thorax, and abdomen. The specific mechanism(s) of lead migration are not fully understood; in our practice, lead migration has been observed despite the use of stress relief loops, medical grade silicone glue at the lead anchor interface, and the use of different IPG implantation sites including the low abdomen (A), buttock, and infraclavicular region.

At least two techniques have been described for occipital lead placement and anchoring: via a vertical C1 level midline cervical incision^{3,7-11} or a retromastoid (R) incision.^{1,2,12-14} From either the midline or retromastoid incision, the subcutaneous lead(s) are tunneled to an IPG. For tunneling to remote IPGs in the buttock or low abdomen, a periscapular (P) connection site may be needed to join the lead to an extension. At the cervical, retromastoid, and periscapular sites, the lead and/or extension connector is anchored to the fascia, and stress relief loops are added. Stress relief loops can also be placed at the IPG site.

The primary aim of this study is to determine implantation pathways associated with the least pathway length change secondary to body movement in an *in vitro* model of an ONS system. Infraclavicular, buttock, and low abdominal IPG implantation sites will be compared during a number of volunteer physical maneuvers, including neck and low back flexion, extension, rotation, and lateral flexion.

METHODS

After institutional review board approval, 10 healthy adult volunteers (5 men, 5 women, age > 18 years) were recruited. The participants were solicited with advertisement on posters and our institutional intranet site. The first 5 women and 5 men who responded and met the criteria were included. Exclusion criteria included any spinal abnormalities such as scoliosis or degenerative changes that would preclude flexing, extending or rotating the neck, thorax, and lower back. Each volunteer signed an informed consent before the study began.

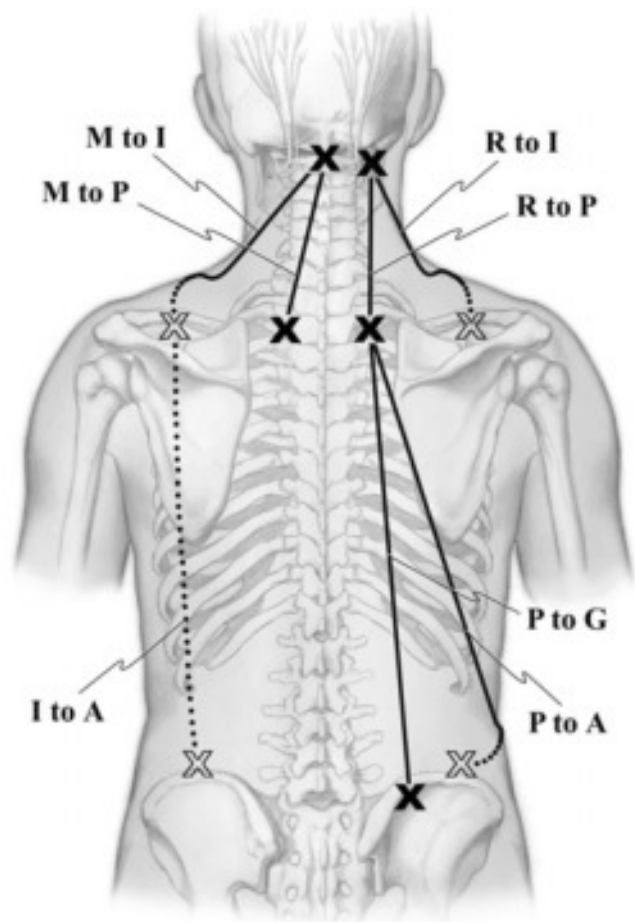


Figure 1. Pathways measured. M to P = midline occipital to periscapular pathway; R to P = retromastoid to periscapular pathway; M to I = midline occipital to infraclavicular pathway; P to G = periscapular to gluteal pathway; P to A = periscapular to low abdomen pathway; I to A = infraclavicular to low abdomen pathway. Dotted lines and open Xs represent anterior pathways and attachment sites. Solid lines and Xs are posterior.

For each volunteer, the expected path of a subcutaneous occipital stimulator system was externally approximated based upon our surgical practice and descriptions in the literature. Figure 1 illustrates the pathways measured. The standardized locations of the attachment sites were as follows: midline occipital (M)—at the C1 level, approximated by bisecting the intermastoid line (mastoid process tip to tip); retromastoid—1 cm medial and 1 cm inferior to tip of mastoid process; periscapular—at level of the superior aspect of the scapula, midway between the medial edge of the scapula and the adjacent spinous process at that level; infraclavicular—2 cm inferior to the midpoint of the clavicle; gluteal (G)—2 cm lateral and 2 cm inferior

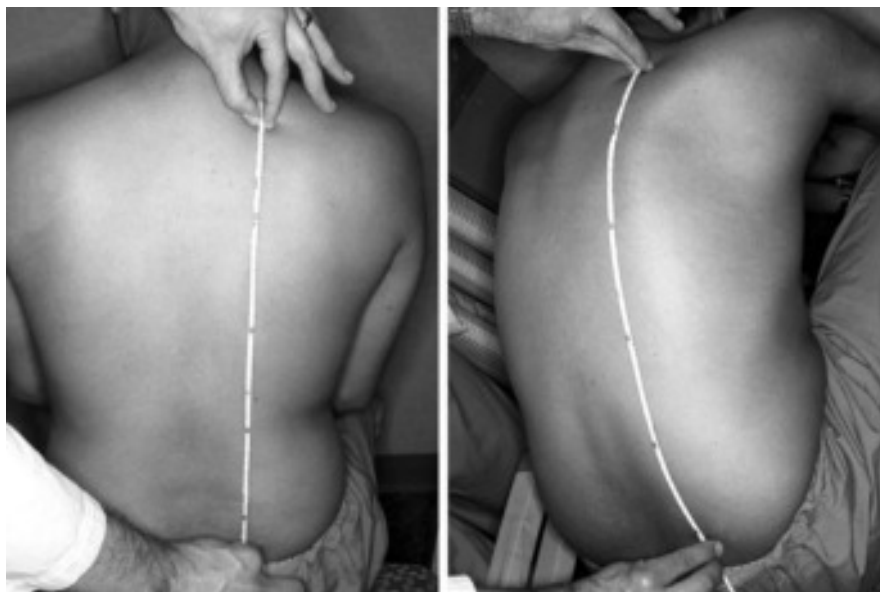


Figure 2. Cable ruler held to the skin, from the periscapular site to gluteal site (P to G pathway), at baseline (picture, left) and during flexion at the waist (picture, right) from the sitting position.

Table 1. Pathways and Volunteer Movements

Attachment Sites	Midline Occipital	Retromastoid	Periscapular Total	Periscapular Segment 1	Periscapular Segment 2	Periscapular Segment 3	Infraclavicular
Periscapular	M to P [†]	R to P [†]					
Infraclavicular	M to I [†]	R to I [†]					
Gluteal			P to G [‡]	P to G [*]	P to G [*]	P to G [*]	
Low Abdomen			P to A [‡]	P to A [*]	P to A [*]	P to A [*]	I to A [‡]

* Flexion at the waist only.

† Volunteer movements measured: M to P and R to P, and M to P and M to I.

1. Neck flexion
2. Neck extension
3. Neck lateral rotation right
4. Neck lateral rotation left
5. Neck lateral bend right
6. Neck lateral bend left

‡ Volunteer movements measured: P to G and P to A, and I to A.

1. Scapular protraction (arms straight out in front, fingers touching, pull shoulders anterior)
2. Scapular retraction (elbows bent and arms pulled back, as if to "pinch a quarter between the scapulas")
3. Flexion at the waist
4. Extension at the waist
5. Waist lateral rotation—right
6. Waist lateral rotation—left
7. Waist lateral bend—right
8. Waist lateral bend—left
9. Sit and flexion at the waist

M to P, midline occipital to periscapular pathway; R to P, retromastoid to periscapular pathway; M to I, midline occipital to infraclavicular pathway; P to G, periscapular to gluteal pathway; P to A, periscapular to low abdomen pathway; I to A, infraclavicular to low abdomen pathway.

to the posterior superior iliac spine; and low abdomen—2 cm medial and 2 cm superior to the anterior superior iliac spine.

A cable model (with millimeter ruler markings) was used to mimic a lead/extension combination in an ONS system. The cable was placed externally along the expected path of a surgical implant. It was manually held adjacent to the skin (Figure 2), and fixed at the simulated proximal attachment site. However, cable movement was not inhibited, ie, the cable was allowed to slide under the investigator's fingers along the

pathway. After obtaining baseline pathway length measurements, cable pathway length change was measured at the distal attachment site with various body movements. The volunteer performed each movement five times, and the mean value was used for analysis. From half of the volunteers, the measurements were obtained on the left side of the body. All movements were assumed to represent maximal volunteer movement without discomfort.

Table 1 summarizes the pathways and volunteer movements. For all baseline measurements, the volun-

teer was asked to assume a comfortable, neutral position. Because we anticipated that flexion at the waist would be associated with the greatest cable movement, the P to G and P to A pathways were divided into thirds (segments) in an effort to understand which segment is associated with the greatest movement. Segment 1 is the most proximal (to the P attachment site), segment 2 represents the middle third, and segment 3 is the most distal.

The overall differences between pathways were analyzed using repeated measures analysis of variance. For significant differences ($P < 0.05$), pairwise comparisons were evaluated using paired t -tests. P values less than 0.05 were considered statistically significant. All computations were performed using SAS software version 9 (SAS Institute, Inc., Cary, NC).

RESULTS

Table 2 provides baseline characteristics of the healthy volunteers. Table 3a documents pathway length changes for the series of neck movements. A significant P value in Table 3a indicates that it is likely that an overall difference exists between pathway length changes for the given volunteer maneuver. There were no significant differences in the pathway length changes when the volunteers performed neck lateral rotations and bends. However, neck flexion and extension produced statistically significant pathway length changes.

Pairwise comparisons for the various pathways are provided in Table 3b, showing which pathway length

Table 2. Baseline Characteristics of Healthy Volunteers (N = 10)

	n (%)	Mean (SD)	Median	Range
Height (cm)		173 (10)	174	152–189
Weight (kg)		82 (17)	83	54–106
Age (years)		33.6 (8.3)	33.4	23–50
Female	5 (50)			
Left side	5 (50)			

SD, standard deviation.

Table 3b. Pairwise Comparisons for Neck Movements

	M to P vs. M to I	M to P vs. R to P	M to I vs. R to I	R to P vs. R to I
Neck flexion	< 0.001	0.009	0.35	< 0.001
Neck extension	0.009	0.23	0.96	< 0.001

M to P, midline occipital to periscapular; M to I, midline occipital to infraclavicular; R to P, retromastoid to periscapular; R to I, retromastoid to infraclavicular.

Table 3a. Neck Movements for Healthy Volunteers (N = 10; All Measurements Are in Millimeters)

	M to I			R to P			R to I			P value
	Mean (SD)	Median (range)	Mean (SD)	Median (range)	Mean (SD)	Median (range)	Mean (SD)	Median (Range)		
Total pathway measurement	163 (18)	160 (120, 185)	249 (26)	250 (190, 280)	159 (17)	160.0 (130, 190)	194 (25)	250 (160, 250)		
Neck flexion	24.3 (7.9)	22.7 (12.8, 37.6)	-1.3 (2.4)	-0.1 (-5.4, 0.8)	19.6 (6.3)	17.9 (12.2, 31.4)	-2.7 (3.7)	0.6 (-9.4, 0.6)	< 0.001	
Neck extension	-32 (21)	-25 (-91, -19)	-8.1 (4.5)	-8.4 (-16.4, -1.4)	-23.0 (7.0)	-23.9 (-33.6, -11.0)	-8.0 (4.6)	-0.4 (-17.2, -0.4)	0.003	
Neck lateral rotation right	4.2 (4.0)	3.4 (0.0, 11.2)	-3 (15)	-3 (-22, 17)	5 (21)	1.0 (-20, 40)	3.3 (8.4)	19.2 (-8.0, 19.2)	0.48	
Neck lateral rotation left	1.1 (5.0)	1.9 (-11.2, 5.6)	-2 (13)	3 (-22, 13)	8 (19)	4 (-13, 36)	1.8 (6.2)	10.2 (-7.8, 10.2)	0.37	
Neck lateral bend right	-2.1 (4.8)	-1.2 (-13.4, 4.0)	-4.0 (8.2)	-0.2 (-20.2, 2.8)	2 (15)	4 (-15, 18)	-3 (14)	15 (-26, 15)	0.32	
Neck lateral bend left	0.5 (4.3)	0.0 (-5.2, 9.4)	1.7 (5.4)	-0.7 (-4.8, 11.2)	6 (15)	4 (-14, 26)	0 (12)	16 (-17, 16)	0.22	

M to P, midline occipital to periscapular; M to I, midline occipital to infraclavicular; R to P, retromastoid to periscapular; R to I, retromastoid to infraclavicular.

Table 4a. Scapular and Waist Movements for Healthy Volunteers (N = 10; All Measurements Are in Millimeters)

	P to G		P to A		I to A		P value
	Mean (SD)	Median (range)	Mean (SD)	Median (range)	Mean (SD)	Median (range)	
Pathway measurement	448 (25)	450 (405, 490)	563 (47)	560 (480, 640)	467 (54)	465 (400, 590)	
Scapular protraction	16.7 (8.6)	16.7 (1.2, 31.8)	5.2 (4.1)	5.1 (-0.6, 12.4)	-2.3 (3.6)	-2.0 (-9.4, 1.8)	< 0.001
Scapular retraction	6.1 (5.3)	4.8 (-0.2, 18.4)	-5.1 (3.5)	-4.8 (-10.6, 0.00)	10.4 (4.5)	9.6 (5.4, 18.8)	< 0.001
Flexion at waist	89 (12)	90 (66, 108)	-3 (10)	0 (-31, 3)	-37 (15)	-35 (-69, -14)	< 0.001
Extension at waist	-44 (21)	-47 (-88, -15)	-6.2 (4.7)	-5.8 (-13.4, 0.4)	24 (11)	22 (12, 48)	< 0.001
Waist lateral rotation right	-1.2 (4.8)	0.0 (-9.0, 4.0)	-6 (21)	-2 (-59, 16)	5 (9.5)	7.1 (-7.8, 18.6)	0.31
Waist lateral rotation left	5.5 (5.5)	4.9 (-3.2, 14.4)	-2 (12)	-2 (-18, 17)	5 (13)	2 (-14, 25)	0.34
Waist lateral bend right	3 (34)	-1 (-32, 50)	2 (15)	1 (-15, 30)	-1 (12)	1 (-25, 11)	0.70
Waist lateral bend left	9 (32)	7 (-30, 74)	1.6 (8.7)	-0.1 (-8.0, 18.2)	3 (16)	4 (-26, 23)	0.55
Sit and flexion at waist	73 (21)	73 (-45, 110)	-4.6 (7.3)	-1.7 (-22.8, 0.4)			< 0.001
Flexion at waist—segment 1	-0.9 (8.3)	0.0 (-18.0, 9.8)	-0.4 (7.0)	1.0 (-19.0, 5.6)			0.82
Flexion at waist—segment 2	18 (10)	17 (1, 35)	9.7 (6.7)	10.7 (0.0, 21.2)			0.03
Flexion at waist—segment 3	82 (14)	81 (60, 105)	-5 (12)	-1 (-37, 2)			< 0.001

P to G, periscapular to gluteal site; P to A, periscapular to low abdomen; I to A, infraclavicular to low abdomen; SD, standard deviation.

Table 4b. Pairwise Comparisons for Scapular and Waist Movements

	P to G vs. P to A	P to G vs. I to A	P to A vs. I to A
Scapular protraction	0.001	< 0.001	< 0.001
Scapular retraction	< 0.001	0.049	< 0.001
Flexion at waist	< 0.001	< 0.001	< 0.001
Extension at waist	< 0.001	< 0.001	< 0.001

P to G, periscapular to gluteal site; P to A, periscapular to low abdomen; I to A, infraclavicular to low abdomen.

changes were significantly different when comparing one pathway to another during neck flexion and extension. Most notable is the large absolute (Table 3a) and relative (Table 3b) differences in pathway length changes when comparing the periscapular site with the infraclavicular site during neck flexion. Both the M to P pathway and retromastoid to P pathway were associated with significantly greater pathway length changes during neck flexion when compared with the M to I or R to I pathways. There was no significant difference when comparing the M to I and R to I pathways. Although there were also significant differences in pathway length changes during neck extension (Table 3a), the values were all negative (ie, the pathways shortened). The magnitude of pathway length shortening was significantly greater for the periscapular sites vs. the infraclavicular sites.

Table 4a documents pathway length changes for the series of scapular and waist movements, with the cable attached at the periscapular or infraclavicular sites. Similar to Table 3a, a significant *P* value in Table 4a indicates it is likely that an overall difference exists

between pathway length changes for the given volunteer maneuver. There were no significant differences in the pathway length changes when the volunteers performed waist lateral rotations and bends. However, scapular movements, waist flexion and extension, and sitting and flexing at the waist produced significant pathway length differences, as did flexion at the waist in segments 2 and 3.

For scapular movements and flexion/extension at the waist, pairwise comparisons for the various pathways are provided in Table 4b, showing which pathway length changes were significantly different when comparing one pathway with another. Most notable were the large (Table 4a) and relative (Table 4b) positive pathway length change differences between the P to G and P to A pathways during flexion. During extension, both of these pathways shortened (ie, the values were negative).

There were significantly different pathway length changes in the middle (segment 2) and lower (segment 3) segments during flexion at the waist for the P to G pathway compared with the P to A pathway. The pathway length differences in segment 3 were the most dramatic, with a mean (SD) of 82 (14) mm in the P to G pathway vs. -5 (12) mm in the P to A site, *P* < 0.001. Sitting and flexing at the waist also produced significant pathway length differences, P to G vs. P to A.

Finally, when the P to G and P to A pathways were compared with the I to A pathway, the results were mixed. In flexion, the I to A pathway was shortened significantly compared with the P to G and P to A pathways, while I to A was significantly lengthened in extension. The magnitude of change was the smallest in

the P to A pathway during both maneuvers compared with the P to G and I to A pathways.

DISCUSSION

This study allowed us to test during volunteer movement, various simulated lead pathway length changes in an in vitro model of an occipital nerve stimulator system. The primary findings of this study are that the flexion/extension pathway length changes associated with the M and retromastoid sites to the infraclavicular site were significantly less than those pathways to the periscapular site. Also, the low abdomen site was associated with significantly less pathway length change during flexion/extension when compared with the gluteal site (P to A vs. P to G). Therefore, IPG insertion in sites other than the buttock, including infraclavicular or low abdomen, may be associated with lower lead migration risk. Implanters may consider these results when counseling patients regarding options for IPG implantation in ONS.

There are a number of considerations when selecting insertion sites and lead pathways for ONS. Patients may prefer IPG sites other than the infraclavicular site for cosmetic reasons. Implanters may prefer the gluteal region because the patient can be in the prone position for insertion (no need for lateral decubitus position and easy access to the occiput compared with lateral decubitus where one side is dependent). However, the retromastoid approach, with the leads tunneled to an infraclavicular IPG, may be advantageous for two reasons: first, our study showed that pathway length changes were minimal in this pathway; and second, the tunneling distance required is short and direct (compared with the midline occipital approach).

If a non-infraclavicular IPG site is selected, our data suggest that the low abdominal site is superior to the gluteal site, although the abdominal site will require the patient to be in the lateral decubitus position for insertion unless a two-stage procedure with intra-operative repositioning is used. Potentially, an “anterior approach” could be used, from retromastoid or midline cervical to the low abdomen via the anterior chest. The anterior approach is analogous to the pathway used for tunneling a ventriculoperitoneal shunt. However, the P to A pathway was associated with less absolute pathway length change than I to A in both flexion and extension, suggesting it is a superior route for low abdominal IPGs.

Of interest is the pathway length changes associated with segment 3 during flexion. The G site (P to G) was associated with significantly greater pathway length

change in segment 3 compared with the low abdomen (P to A). This result may be useful for decision-making not only in ONS, but also in spinal cord stimulator implantation where the IPG can be inserted in either gluteal or low abdominal sites.

Any implanted stimulator system is subject to complications such as infection, lead fracture, erosion, or migration of components.^{15,16} Lead migration is not unique to ONS systems; spinal cord stimulators have also been reported to migrate within the epidural space leading to loss of stimulation.¹⁷ However, subcutaneous implantation of ONS components may place excessive mechanical stress on the components because the lead may be tunneled across highly mobile body regions for distances of a meter or more.

It is unknown if the use of stress relief loops, silicone glue at connection sites, or specific anchor types reduce the incidence of lead migration. Further, it is unknown if pathway length shortening is a risk factor for lead migration, and how pathway shortening might compare with lengthening in terms of mechanical fatigue. During both neck and low back extension, we noted negative pathway length changes. Our conclusions are based on the assumption that pathway length increases are a greater risk factor than pathway length decreases, but we are unaware of any in vivo data to support our intuition.

There are a number of limitations of our study. Although we were able to measure the pathway length change of the cable ruler with volunteer movement, this does not necessarily correlate with the mechanical stress on an in vivo ONS system. We are unaware of any published data demonstrating a correlation between lead migration and IPG sites. Furthermore, an important factor in lead migration is anchoring at both the insertion and IPG sites. Our study only examines the impact of movement on pathway length; anchoring failure was not studied. The process of manually holding the cable to the estimated lead pathway was imperfect, ie, our model probably did not perfectly mimic how a subcutaneous lead behaves. In particular, we do not know how an in vivo lead behaves during patient extension. Does the lead “curl up” or otherwise slide in tissue planes when the pathway shortens? Also, it is unknown how scar formation or encapsulation of components with reactive tissue might affect their ability to move in vivo.

In addition to the aforementioned limitations, we did not test an IPG implantation site in the infra-axillary region (midaxillary line), although anecdotally, this site

has been used by implanters. We suspect the lead pathway length change associated with this site would be similar to the low abdominal site. Finally, our volunteers were “young and healthy.” The lead migration we documented should be considered maximal compared with older, less flexible patients.

In conclusion, our data suggest that IPG implantation sites other than the gluteal region may be associated with less pathway length change during patient movement. The lesser pathway length change associated with infraclavicular and low abdomen IPG sites may result in less lead migration. Further studies may better characterize the causes of lead migration, ultimately contributing to fewer mechanical complications and better patient outcomes.

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REFERENCES

1. Weiner RL, Reed KL. Peripheral neurostimulation for the control of intractable occipital neuralgia. *Neuromodulation*. 1999;2:217–221.
2. Oh MY, Ortega J, Bellotte JB, Whiting DM, Alo KM. Peripheral nerve stimulation for the treatment of occipital neuralgia and transformed migraine using a C1-2-3 subcutaneous paddle style electrode: a technical report. *Neuromodulation*. 2004;7:103–112.
3. Kapural L, Mekhail N, Hayek SM, Stanton-Hicks M, Malak O. Occipital nerve electrical stimulation via the midline approach and subcutaneous surgical leads for treatment of severe occipital neuralgia: a pilot study. *Anesth Analg*. 2005;101:171–174.
4. Schwedt TJ, Dodick DW, Hentz J, Trentman TL, Zimmerman RS. Occipital nerve stimulation for chronic headache—long-term safety and efficacy. *Cephalalgia*. 2007;27:153–157.
5. Trentman TL, Dodick DW, Zimmerman RS, Birch BD. Percutaneous occipital stimulator lead tip erosion: report of two cases. *Pain Physician*. 2008;11:253–256.
6. Jasper JF, Hayek SM. Implanted occipital nerve stimulators. *Pain Physician*. 2008;11:187–200.
7. Popeney CA, Alo KM. Peripheral neurostimulation for the treatment of chronic, disabling transformed migraine. *Headache*. 2003;43:369–375.
8. Rodrigo-Royo MD, Azcona JM, Quero J, Lorente MC, Acin P, Azcona J. Peripheral neurostimulation in the management of cervicogenic headache: four case reports. *Neuromodulation*. 2005;8:241–248.
9. Johnstone CS, Sundaraj R. Occipital nerve stimulation for the treatment of occipital neuralgia—eight case studies. *Neuromodulation*. 2006;9:41–47.
10. Schwedt TJ, Dodick DW, Trentman TL, Zimmerman RS. Occipital nerve stimulation for chronic cluster headache and hemicrania continua: pain relief and persistence of autonomic features. *Cephalalgia*. 2006;26:1025–1027.
11. Burns B, Watkins L, Goadsby PJ. Treatment of medically intractable cluster headache by occipital nerve stimulation: long-term follow-up of eight patients. *Lancet*. 2007;369:1099–1106.
12. Slavin KV, Colpan ME, Munawar N, Wess C, Nersesyan H. Trigeminal and occipital peripheral nerve stimulation for craniofacial pain: a single-institution experience and review of the literature. *Neurosurg Focus*. 2006;21:1–5.
13. Slavin KV, Nersesyan H, Wess C. Peripheral neurostimulation for treatment of intractable occipital neuralgia. *Neurosurgery*. 2006;58:112–119. Discussion 112–119.
14. Magis D, Allena M, Bolla M, De Pasqua V, Remacle JM, Schoenen J. Occipital nerve stimulation for drug-resistant chronic cluster headache: a prospective pilot study. *Lancet Neurol*. 2007;6:314–321.
15. Hamani C, Lozano AM. Hardware-related complications of deep brain stimulation: a review of the published literature. *Stereotact Funct Neurosurg*. 2006;84:248–251.
16. Rainov NG, Heidecke V. Hardware failures in spinal cord stimulation (SCS) for chronic benign pain of spinal origin. *Acta Neurochir Suppl*. 2007;97:101–104.
17. Cameron T. Safety and efficacy of spinal cord stimulation for the treatment of chronic pain: a 20-year literature review. *J Neurosurg*. 2004;100:254–267.