Chronic stimulation of the posterior hypothalamic region for cluster headache: technique and 1-year results in four patients

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Object. Cluster headache (CH) is the most severe of the primary headache disorders. Based on the finding that regional cerebral blood flow is increased in the ipsilateral posterior hypothalamic region during a CH attack, a novel neurosurgical procedure for CH was recently introduced: hypothalamic deep brain stimulation (DBS). Two small case series have been described. Here, the authors report their technical approach, intraoperative physiological observations, and 1-year outcomes after hypothalamic DBS in four patients with medically intractable CHs.

Methods. Patients underwent unilateral magnetic resonance (MR) imaging–guided stereotactic implantation of a Medtronic DBS (model 3387) lead and Sotrela pulse generator system. Intended tip coordinates were 3 mm posterior, 5 mm inferior, and 2 mm lateral to the midcommissural point. Microelectrode recording and intraoperative test stimulation were performed. Lead locations were measured on postoperative MR images. The intensity, frequency, and severity of headaches throughout a 1-week period were tracked in patient diaries immediately prior to surgery and after 1 year of continuous stimulation.

At the 1-year follow-up examination, DBS had produced a greater than 50% reduction in headache intensity or frequency in two of four cases. Active contacts were located 3 to 6 mm posterior to the mammillothalamic tract. Neurons in the target region showed low-frequency tonic discharge.

Conclusions. In two previously published case series, headache relief was obtained in many but not all patients. The results of these open-label studies justify a larger, prospective trial but do not yet justify widespread clinical application of this technique.

KEY WORDS • cluster headache • hypothalamus • neuronal recording • deep brain stimulation

Cluster headache is a primary headache disorder characterized by attacks of excruciating unilateral peri-orbital pain, usually with evidence of ipsilateral cranial autonomic disturbance. The attacks tend to occur at specific times of the day and may disappear completely during certain months of the year. Approximately 10 to 20% of patients with CH have debilitating headaches, despite the provision of optimum medical therapy, or can only control attacks with long-term use of high-dose corticosteroid agents. In these medically intractable cases, trigeminal nerve ablation procedures have been performed, with little benefit. Positron emission tomography studies, in which 15O-H2O is used as the tracer, have shown a focal increase in cerebral blood flow in the ipsilateral posterior hypothalamic region during a CH attack. Based on this finding, Leone et al. introduced a promising new surgical procedure for severe CH: chronic DBS of the posterior hypothalamic region. Short-term outcomes have been reported for only 12 patients by authors of two small clinical series. Here, we report our technical approach, intraoperative physiological observations, and 1-year outcomes for DBS of the posterior hypothalamic region in four patients with medically intractable CH.

Clinical Material and Methods

Preoperative Screening

Patients with CH referred for possible surgical treatment were screened by a neurologist (N.H.R. or J.L.O.). Criteria for offering surgical therapy were as follows: 1) Patients must have met the International Headache Society diagnostic criteria for CH. 2) Patients must have had chronic CH, or severe episodic CH occurring for at least 6 months of the year, for the last 2 years preceding surgery. 3) Patients must...
have had at least seven debilitating headaches per week, reaching a severity of at least 6 on a visual analog scale with scores ranging from 1 to 10 during their severe headache season. 4) Patients must have tried and had inadequate relief from prophylactic therapy, including treatment with verapamil, lithium, divalprox sodium, methysergide, and short-term corticosteroid therapy. 5) Patients must have had inadequate relief from abortive therapy, including the use of oxygen, sumatriptan, and opiates. These criteria are consistent with recently proposed guidelines for DBS therapy in CH.\(^\text{13}\) The University of California at San Francisco institutional review board granted approval for this retrospective study.

**Headache Assessment**

Prior to surgery, the patients were asked to fill out headache diaries for at least 7 consecutive days during the month prior to surgery, as well as during the 12th month following surgery. The diaries asked patients to score each day: 1) the time of day each headache episode occurred, 2) the duration of the headache, 3) the intensity of the headache on a visual analog scale (scores from 1 [no pain] to 10 [worst imaginable pain]), and 4) the use of abortive and prophylactic medications. Headache characteristics were averaged from headache diaries over the 1-week period. Patients were considered “responders” to DBS therapy if at the 1-year interval, there was a greater than 50% reduction in headache frequency, intensity, or both, compared with the preoperative status.

**Stereotactic Localization of the Target**

The surgical technique included MR imaging–based stereotaxy, microelectrode recording in the region of the MR imaging–defined target, and intraoperative test stimulation to define voltage thresholds for stimulation-induced adverse effects.

Following placement of a Leksell stereotactic headframe, two MR image sets were obtained on a Phillips Intera 1.5-tesla MR imager: 1) a volumetric Gd-enhanced 3D gradient echo MR image covering the entire brain in 1.5-mm axial slices, for trajectory planning and visualization of the AC–PC line (TR 20 msec, TE 2.9 msec, matrix 256 \(\times\) 192, flip angle 3, number of excitations 1); 2) a T\(_2\)-weighted FSE sequence, limited to the diencephalon and midbrain, in the axial plane at 2-mm slice thickness. The T\(_1\)-weighted FSE image was mainly used for visualizing structural detail in the immediate vicinity of the brain target, the MTT and the red nucleus (TR 3000 msec, TE 90 msec, matrix 268 \(\times\) 512, number of excitations 6, bandwidth 183 Hz/pixel, interleaved). Images were imported into a stereotactic surgical planning software package (Framelink version 4.1, Medtronic-SNT), computationally fused, and reformatted to produce images orthogonal to the AC–PC line and mid-sagittal planes.

The brain target was selected using the coordinates provided by Franzini et al.\(^\text{3}\)—that is, 2 mm lateral, 3 mm posterior, and 5 mm inferior to the midcommissural point. The T\(_1\)-weighted FSE sequence showed that the intended target was 3 to 5 mm posterior to the MTT and was medial to the anterior border of the red nucleus. The intended target was superimposed on the appropriate axial section from the Schaltenbrand and Warren human brain atlas and is shown in Fig. 1. A “default” trajectory through the brain was set at 60° from the AC–PC line in the sagittal projection and 10° from the vertical in the coronal projection. This trajectory was visualized on the volumetric MR imaging study using “navigation” views. Small adjustments in the arc and ring angles were made to avoid traversing the sulci, lateral ventricle, cortical veins, and dural venous lakes (easily seen on Gd-enhanced images).

**Single Unit Recording**

A single microelectrode penetration was made to the stereotactic target. Single-unit discharge was recorded with glass-coated platinum/iridium microelectrodes, with an impedance of 0.4 to 1.0 Mohm at 1000 Hz (FHC, Inc.). Microelectrodes were advanced into the brain using a motorized microdrive (FHC, Inc.). Recordings were filtered (300 Hz–5 KHz), amplified, played on an audio monitor, and digitized (20-kHz sampling rate) using the Guideline System 3000 (FHC, Inc.). Because the “electrophysiological signature” of the targeted region is not well understood, we made no attempt to utilize microelectrode recording to modify the final brain target. Recordings were made only to document the physiology along the trajectory to the anatomical target.

**Intraoperative Test Stimulation and Permanent Hardware Placement**

The Medtronic model 3387 quadripolar DBS lead was placed with its distal end at the anatomic target. Test stimulation was performed in bipolar mode using contacts 0–3, +185 Hz, and 60-\(\mu\)sec pulse width (model 3625 external tester, Medtronic, Inc.). Voltage was increased at 0.5 V/second up to 10 V during continuous examination of the patient’s cranial nerve function. Voltage threshold for oculomotor disturbance or subjective phenomena, such as mood
change or vertigo, were noted. The patient’s blood pressure and pulse were carefully monitored during the test stimulation. Leads were anchored to the skull with a lead-anchoring device (Stim-lock, Image Guided Neurologics). After scalp closure and headframe removal, general anesthesia was induced to allow placement of the lead extender and pulse generator (Soleta, Medtronic, Inc.).

**Programming Parameters**

Programming parameters were based on the two published case series\(^2\),\(^3\) monopolar stimulation of 60-μsec pulse width, 185-Hz frequency, and 1 to 3 V (stopping short of the threshold for persistent stimulation-induced adverse effects). Devices were kept activated at all times postoperatively.

**Measurement of Electrode Locations**

Postoperative MR imaging to document the location of the electrode tip was performed in all cases, according to the published safety guidelines for brain MR imaging in patients with implanted DBS systems.\(^4\) The postoperative MR imaging protocol was identical to the aforementioned preoperative stereotactic protocol. The images were imported into an image-processing software program (Framelink 4.1, Medtronic-SNT) and computationally reformatted into standard anatomical planes orthogonal to the midsagittal plane (contacts 1.5 mm long, spaced 3 mm center-to-center), and the coordinates of the tip and entry point. The formulas for these calculations have been previously published.\(^5\)

**Results**

**Patient Characteristics and 1-Year Results**

Patient characteristics determined at the baseline preoperative visit are summarized in Table 1. All patients exhibited unilateral left-sided symptoms and received left-sided implants. Changes in the frequency, duration, and intensity of headaches after 1 year of DBS therapy, along with postoperative medications, are summarized in Table 2. Two of four patients (Cases 1 and 2) were considered responders to DBS therapy based on a greater than 50% reduction in headache frequency or intensity. One of the two patients (Case 2) in whom there was a positive response had been using sumatriptan injections to reduce the intensity and duration of each headache and has not required any abortive therapy postoperatively due to significantly lower intensity and frequency of headaches. This may explain the increase in duration of the headache events that remained. One of the patients (Case 3) has had transient complete suppression of headaches for 1 to 2 weeks following each reprogramming session but no persistent improvement in headaches, or reduction in abortive therapy, in the intervals between programming changes. This patient is considered a nonresponder, and he no longer uses the device. One patient (Case 4) experienced a modest reduction in headache intensity, but the reduction did not reach the predefined 50% needed in headache intensity or frequency and hence is also considered a nonresponder.

**Surgical Complications**

There was a single surgical complication: an intraoperative TIA in Case 1. This occurred 5 minutes after intraoperative test stimulation (using the deepest contact as the anode [60-μsec pulse width, 185 Hz, \( \leq 10 \) V]). The patient was noted to be hemiplegic on the side ipsilateral to the implant, as well as drowsy. The deficits resolved completely in 5

### Table 1

Summary of baseline characteristics\(^*\)

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Headache Onset</th>
<th>Headache Surgery</th>
<th>Medication (mg/day)</th>
<th>Prophylactic</th>
<th>Abortive</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>21</td>
<td>58</td>
<td></td>
<td>hydrocodone (45)</td>
<td>none</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>40</td>
<td>54</td>
<td></td>
<td>levetiracetam (1000)</td>
<td>none</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>29</td>
<td>41</td>
<td></td>
<td>prednisone (10–60),</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>50</td>
<td>66</td>
<td></td>
<td>verapamil (1200),</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* sq = subcutaneous.
† The patient in Case 2 underwent headache evaluation and surgery during the less intense time of his seasonal headache cycle.
‡ A 5-day course three times/year.
minutes. Pulse and blood pressure were unchanged during the episode. Emergency head computed tomography scanning revealed no hemorrhage, and subsequent MR imaging/MR angiography showed no diffusion abnormalities or abnormalities in intracranial vessels. The DBS tip, however, was noted to be slightly deep to the target, having exited the floor of the third ventricle, and terminated within the interpeduncular cistern near midline. We hypothesize that high voltage stimulation in the setting of the interpeduncular tip placement may have induced a contralateral thalamoperforator spasm, resulting in transient capsular ischemia and ipsilateral motor dysfunction. Contact 0 was not used in subsequent programming. The TIAs did not recur in this case and did not occur in the subsequent three cases.

**Single-Unit Physiological Parameters**

Microelectrode recordings in the region of the anatomical target showed low-amplitude, low-frequency, spontaneously active cells. Neuronal action potentials were present at the most ventral extent of the target region, except in Case 1. In this case, the last 2 mm of the recording was electrically silent, and postoperative imaging thereafter demonstrated that the lowest contact was in the interpeduncular cistern (previously described). Stable recordings amenable to formal analysis were relatively infrequent due to significant pulsation artifact (variation in amplitude of the action potential in synchrony with the cardiac cycle). A sample recording is shown in Fig. 2. Seven units recorded within 5 mm of the anatomical target were amenable to analysis based on the identification of well-isolated single unit recorded for at least 10 seconds. The mean (± standard deviation) spontaneous discharge rate was 20 ± 17 Hz. Cells with a higher discharge rate (20–40 Hz) tended to occur 3 to 5 mm dorsal to the target, which probably corresponds to the ventromedial thalamus. Cells within 3 mm of the target area had discharge rates less than 20 Hz.

**Test Stimulation Thresholds**

Intraoperative test stimulation thresholds are shown in Table 3. Oculomotor effects were observed in all patients and were accompanied by vertigo. Reversible, reproducible, stimulation-induced mood changes, described as dysphoric, were noted in two of the four patients. Additionally, at stimulation higher than 4 V (60-μsec pulse width, 185 Hz), all patients noted a vague sense of dizziness or warmth, but these sensations were too vague and transient to determine a precise voltage threshold. No changes in pulse rate or blood pressure were observed during test stimulation.

**Electrode Locations**

Figure 3 shows the location of the electrode tip on axial MR imaging, 5 mm inferior to the intercommissural line, in the patient in Case 4. The lead tip is 6 mm posterior to the MTT. Individual and mean electrode locations for the four patients are provided in Table 4.

**Discussion**

**Clinical Outcomes of DBS for CH**

This report adds to the small but growing world experience in medically intractable CH treated with chronic DBS of the posterior hypothalamic region. The group from Milan, Italy that pioneered this procedure has published clinical outcomes on their initial eight patients. In this open-label study, with follow-up times of 1 to 48 months, three of eight patients were reported to be pain free without the need for medication, whereas the remaining five patients were reported to be pain free while receiving continued prophylactic therapy (verapamil or methysergide). The first patient treated remained free of headache for 4 years.

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They reported clinical follow up in four of their six patients; a second open-label clinical study of six patients who received implants the authors found greater variability in outcomes. They reported clinical follow up in four of their six implant-treated patients for whom the mean follow-up period was 14.5 months. Two of the four were free of headaches at the end of the follow-up period, but the follow-up period was very short.

We chose to report outcomes at 1 year because comparing baseline values with outcomes measured at different times of year could introduce a confounder related to the seasonal nature of the attacks. Two of our four patients were considered responders to DBS therapy, having had a greater than 50% decrease in headache frequency, intensity, or both. One patient, however, had only a slight decrease in headache intensity, and a fourth had no persistent benefit and no longer uses the device. Our results are less uniformly successful than those reported by Franzini et al., but they are similar to those reported by Schoenen et al. Taken together, the results of these small open-label studies suggest that many but not all patients may derive significant benefit from this procedure. In the 4-year follow-up case report of Leone et al., the return of headache due to inadvertent DBS inactivation suggests that the effect is not entirely due to placebo. A placebo-controlled trial would be needed to determine the true placebo effect of this treatment.

The results of the present series appear to justify a larger clinical trial of DBS stimulation. The surgical team must be mindful of potential vascular complications of this procedure, as underscored by the postoperative death due to intracerebral and intraventricular hemorrhage in a previously published series, as well as the TIA observed in one of our patients immediately after intraoperative test stimulation. Given that up to 50% of otherwise typical patients with CH could be nonresponders to this therapy, a critical issue for future studies will be to identify predictors of outcome that can prospectively distinguish responders from nonresponders. The presence or absence of ipsilateral posterior hypothalamic activation by H$_2$O-based PET during a CH attack, which provided the initial motivation for the introduction of this procedure, could prove useful in this regard.

**Brain Target for DBS in CH**

The brain target used in this study was that proposed by Franzini et al. —2 mm lateral, 5 mm inferior, and 3 mm posterior to the midcommissural point. While this target was based on the activation by H$_2$O-based PET scanning during a CH attack, it does not correspond to a specific anatomical entity. The continuous rim of gray matter lining the inferior wall of the third ventricle and the upper Sylvian aqueduct includes the hypothalamus proper, the PVGM, and the PAGM. The targeted point in this and prior series is immediately anteromedial to the dorsal part of the red nucleus, and 4 mm posterior to the MTT. Some anatomists consider the MTT to be the posterior border of the hypothalamus, but in some human brain atlases the hypothalamus is depicted as extending several millimeters posterior to the MTT. Therefore, the “hypothalamic” DBS target

**TABLE 4**

<table>
<thead>
<tr>
<th>Case</th>
<th>AP Dist MTT to Lead</th>
<th>Lead Tip Coordinates (mm from MCP)</th>
<th>Approach Angle (° from vert)</th>
<th>Active Contact Coordinates (mm from MCP)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Lat</td>
<td>AP</td>
<td>Vert</td>
</tr>
<tr>
<td>1</td>
<td>3.5</td>
<td>1.70</td>
<td>0.00</td>
<td>−5.20</td>
</tr>
<tr>
<td>2</td>
<td>4.7</td>
<td>1.30</td>
<td>−2.70</td>
<td>−4.30</td>
</tr>
<tr>
<td>3</td>
<td>5.3</td>
<td>1.80</td>
<td>−3.50</td>
<td>−5.40</td>
</tr>
<tr>
<td>4</td>
<td>6</td>
<td>2.90</td>
<td>−3.30</td>
<td>−5.00</td>
</tr>
<tr>
<td>mean ± SD</td>
<td>4.9 ± 0.5</td>
<td>1.9 ± 0.7</td>
<td>−2.3 ± 1.6</td>
<td>−4.9 ± 0.5</td>
</tr>
</tbody>
</table>

* All measurements were made on postoperative MR images. Abbreviations: dist = distance; MCP = midcommissural point; SD = standard deviation; vert = vertical.
proposed by Franzini et al., and used in the present study, could be considered to be either in the posterior hypothalamus or within the anterior PVGM.

Possible Mechanisms of Action

In general, the mechanism of the therapeutic effect could be related to either activation or inhibition of neuronal cells or fibers. The posterior hypothalamic/anterior PAGM region harbors cells containing melatonin as well as various opiate peptides. The median forebrain bundle, containing fiber tracts involved in all major ascending catecholaminergic systems, as well as hypothalamic efferent projections to the brainstem and spinal cord, also traverse this area. Schoenen et al. addressed the possible hormonal effects of DBS in their series of patients with CH. Chronic DBS had no effect on urinary excretion of cortisol or melatonin and no effect on plasma levels of oxytocin and vasopressin. There was no long-term effect on pressure pain thresholds in the supraorbital area, indicating that a general analgesic effect could not explain the headache benefit. It is possible that DBS counteracts the focal increase in regional cerebral blood flow seen on PET during a CH attack, but this has yet to be confirmed.

Resurrection of an Old Procedure?

During the past 50 years, lesioning and DBS of the hypothalamus, PVGM, and PAGM have been performed for various types of pain and other neurological conditions. Lesioning of the hypothalamus was introduced by Sano and colleagues primarily as a treatment for aggressive behavior, but some procedures were also undertaken for neuropathic pain. The most frequently reported target coordinate was 2 mm inferior to the midsagittal plane and 2 mm lateral to the wall of the third ventricle, placing the target 4.5 mm anterosuperior to the present DBS target in CH conditions (reviewed by Hunter). Medial thalatomy has been performed for intractable pain. Lesions were usually placed in the centromedian or parafascicular nuclei in an attempt to interrupt the “nonspecific” part of the spinothalamic system, thought to innervate these medial thalamic nuclei after synapsing in the midbrain reticular formation. In most reports, the medial thalamic target was 6 to 8 mm superior and lateral to the present CH DBS target. Finally, many authors have described chronic DBS of the PVGM in the treatment of neuropathic pain. Young et al. reported the PVGM target to be near the PC, 3 to 4 mm lateral to the midline. The authors of an autopsy series have confirmed that clinically effective contacts in PVGM DBS were placed near the PC, and much more posterior than the CH DBS target. Thus, the CH DBS target presently under study appears to be 3 to 10 mm posterior, inferior, or anterior to that used in stereotactic interventions in the hypothalamus, medial thalamus, and PVGM previously performed for neuropathic pain or other conditions.

Electrophysiology of the Target Region

In both previously published cases series of DBS for CH, microelectrode recordings in the region of the stereotactic target were performed. Quantitative analysis of electrical activity was not conducted, however, and a neuronal “signature” for the cells in the target region has not been described. Our extracellular single-unit neuronal recordings indicate that the target region is characterized by neurons tonically discharging at a rate less than 20 Hz, but more recordings would be necessary to confirm this. The low amplitudes of the recorded action potentials, compared with basal ganglia recordings performed using identical methods, suggest that the neuronal cell bodies and/or transmembrane current flows are small. The pulsation artifact in the neuronal recordings is more significant in the CH target than basal ganglia recordings, presumably due to the proximity to the third ventricle and the basilar artery. Recording deep to the location of the intended target is not recommended because of the risk of vascular injury associated with penetration of the interpeduncular cistern. Thus, clear physiological boundaries for this region are not easily determined.

Responses to acute intraoperative test stimulation may help to confirm the appropriate target localization. Similar to the other case series of DBS in cases of CH, we found oculomotor disturbances in the target region in voltage ranges only slightly higher than those used for therapeutic benefit. Ophthalmoplegia or skew deviation was the most consistent finding. Test stimulation in the present series did not evoke sympathomimetic responses (elevated blood pressure and pulse rate), unlike acute stimulation several millimeters more anterior at the Sano hypothalamic target. Test stimulation more posteriorly in the PVGM has been reported to produce nystagmus and paralysis of upward gaze.

Conclusions

Cluster headache is the most severe known primary headache disorder. In 10 to 20% of cases symptoms are medically intractable. Based on the finding of increased blood flow in the posterior hypothalamic area on H15O-based PET scanning during spontaneous and nitroglycerin-induced CH attacks, Franzini et al. introduced DBS of the posterior hypothalamic region for medically intractable CH. The target point selected, 3 to 5 mm posterior to the MTT, is in the border zone between the posterior hypothalamus and anterior PVGM. Deep brain stimulation alleviated CH in many, but not all, of the 12 reported cases from two European centers, and in two of four cases in the present study. The results of these small open-label studies justify the undertaking of a larger prospective trial but do not yet justify widespread clinical application.

Disclosure

We thank Monica Volz, R.N., and Susan Heath, R.N., for assistance with DBS programming, and Paul House, M.D., Nadja Levesque, R.N., and Jeffrey Myers, R.N., for assistance with surgical procedures.

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References

Hypothalamic deep brain stimulation for cluster headache


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