Deep Brain Stimulation of the Ventroposteromedial (VPM) Thalamus 10 Years after VPM Thalamotomy to Treat a Recurrent Facial Pain

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Abstract
We report the successful treatment of recurrent facial pain by deep brain stimulation (DBS) of the ventroposteromedial thalamic nucleus (VPM-DBS), 10 years after VPM thalamotomy. A 62-year-old woman who suffered from an atypical right-sided trigeminal neuralgia of the V1 and V2 branches was successfully treated a decade ago with a radiofrequency VPM thermocoagulation. Ten years later, the same burning right-sided trigeminal pain progressively recurred and was resistant to medical treatments. A DBS procedure was proposed to the patient aiming to stimulate the vicinity of the preexisting stereotactic lesion. Intraoperatively, the pain relief was immediate at low stimulation intensities. Eleven months later, the patient remains pain free. This case report suggests that DBS targeting an area of the VPM close to the previous stereotactic lesion is possible as a salvage therapy, and can successfully achieve relief of facial pain 10 years after VPM thalamotomy.
anatomy resulting in a long-lasting period free of pain, she presented with progressive recurrent burning facial pain in the right V1 and V2 hemiface, which was resistant to medical treatment. The symptomatology was similar to the initial presentation before the thalamotomy 10 years previously. Hypoesthesia to light touch and pain were felt in the right V1 and V2 regions; no allodynia was observed. Corneal reflexes were symmetric. The skin examination did not reveal any autonomic asymmetry.

A DBS procedure was proposed to the patient aiming to stimulate the vicinity of the preexisting thalamotomy lesion located in the VPM thalamic nucleus. The stereotactic targeting was performed on the Stereotact & Funct. Neurosurg. The surgery was performed under local anesthesia. A microelectrode was inserted through the planned trajectory. We performed recording during the insertion of the temporary electrode within the trajectory, targeting the anterolateral region of the preexisting thalamotomy. Single-unit recordings were performed using a LeadPoint 3.0 system, which recorded 12 firing units from 7 mm to the target. In order to detect any effect of the previous thalamotomy on the unit characteristics, the upper part of the trajectory (–7 to –4 mm) was compared to the lower part of the trajectory (–4 to 0 mm). The upper trajectory single-unit mean amplitude (220 μV, n = 7) was significantly higher (p < 0.01) compared to the lower part (107 μV, n = 5). On the other hand, the signal root mean square and the single-unit frequency were comparable throughout the trajectory. Hence, these cellular firing rates were compatible with thalamic cell activity recorded in an awake patient. Figure 2 shows an example of a 1-second recording of neuronal activity 4 mm above the target.

Macrostimulation was performed every 2 mm with incremental intensities from 7 mm above the target to the target level, at a pulse width of 90 μs. At each stimulation level, 3 different frequencies were tested (2, 50 and 200 Hz). The electrode location was confirmed with the intraoperative O-arm® image acquisition.

Intraoperatively, pain relief was clearly achieved 7 mm above the target at 1 mA and 200 Hz. Paresthesia in the face and arm were reported by the patient at an intensity >2 mA. Since the pain did not recur after the first stimulation track and even after having stopped the stimulation, no further exploration was performed and a 3387 Medtronic® electrode was inserted into the target. The electrode was connected to a tunneled extension without stimulation and the patient was transferred to the intermediate care unit for postoperative management.

The radiological control of the electrode placement showed that contacts 0, 1 and 2 (fig. 3a–c) were located in the VPM, whereas contact 3 (fig. 3d) was just above it. External stimulation was started the following day, when the patient complained of recurrent left facial pain. After a week of continuous external bipolar stimulation (contacts 0–1–2+/3+, pulse width 120 μs, frequency 130 Hz, intensity 2 mA) providing consistent pain relief, the electrode was internalized and connected to a Medtronic Activa™ SC 603 internal pulse generator. The patient was discharged and used stimulation 24 h per day at the same settings. Four months later, due to recurring pain in the cheek, new stimulation parameters were tested. The best pain relief was obtained with a continuous bipolar stimulation (contacts 0–1–2+/3+, pulse width 90 μs, frequency 50 Hz). There was no recurrence of the neuropathic facial pain 11 months after the intervention.

**Discussion**

We report a patient who suffered from recurrence of facial pain 10 years after a previous VPM thalamotomy. An additional DBS in the vicinity of the thalamic lesion was performed. This resulted in pain relief lasting so far for 11 months.

While the strategy of performing a thalamotomy following a DBS has already been described in a few cases of tremor [9, 10], the reverse procedure, namely a DBS procedure following a thalamotomy, has rarely been reported. We found the description of a ventralis intermedius gamma thalamotomy following a case of insufficient tremor reduction [11]. However, to the best of our knowledge, thalamic VPM-DBS following an ipsilateral thalamotomy after a long pain-free interval has never been described previously.

To understand the unusual recurrence of neuropathic pain in this patient, we will discuss two hypotheses. The first one is related to the size of the lesion, which may have been too small or not precisely located to provide com-
Fig. 2. Example of a 1-second recording of neuronal activity 4 mm above the target. The average firing rate was 29 Hz. Bursts of cellular firing (arrows) were recorded during the final pathway of our DBS trajectory in our report from 7 to 1.5 mm above the target (cartesian coordinates of the target: x = –10.09 mm, y = –8.48 mm, z = +1.5 mm). These bursts of cellular firing are compatible with thalamic cell activity.

Fig. 3. Axial view of image fusion of the preoperative MRI and the perioperative axial image (O-arm), showing the position of DBS contact 0 (a, thick arrow), contact 1 (b, thick arrow) and contact 2 (c, thick arrow) located within the VPM, in relation to the preexisting VPM thalamotomy (thin arrows). Contact 3 was located outside of the VPM. x = Laterality, y = anteroposteriority, z = verticality. a Contact 0 cartesian coordinates from MC: x = –10.09 mm; y = –8.06 mm; z = 1.5 mm. b Contact 1 cartesian coordinates from MC: x = –11.04 mm; y = –6.62 mm; z = 3.58 mm. c Contact 2 cartesian coordinates from MC: x = –12.09 mm; y = –5.02 mm; z = 5.89 mm. d Contact 3 cartesian coordinates from MC: x = –13.18 mm; y = –3.36 mm; z = 8.27 mm.
plete and sustained pain relief. However, the fact that the patient was pain free during 10 years may not corroborate this hypothesis. The second one favors the regrowth or late sprouting of fibers responsible for pain recurrence.

Even if the reason for the late recurrence of the neuropathic pain in our patient could not clearly be established, a new treatment aiming at the vicinity of the previous lesion was attempted. In our case, knowing that the entire volume of the VPM was not lesioned during the first procedure, we could propose performing either a new lesion or a DBS procedure in the vicinity of the previous lesion. We chose the second option in order to avoid adverse effects (such as internal capsule lesioning or the occurrence of anesthesia dolorosa and other sensory disturbances) due to a too large lesion.

In order to plan the best electrode targeting, we first needed to localize the lesion within the thalamus and to estimate its size. According to the theory of Head and Holmes [12], the thalamus, and specifically the VPM nucleus, is the chief organ responsible for the integration of nociceptive stimuli, and hence, the perception of pain in the face. The average dimensions of the human thalamus are 30–36 mm rostrocaudally, 20–24 mm in width and 14–20 mm in height [13, 14]. The cartesian dimensions of the VPM measured in the Schaltenbrand atlas from the midcommissural point (MC) are 9–14.5 mm in the x-axis, –4 to –10 mm in the y-axis and +1.5 to +7 mm in the z-axis, for a total volume of 181.5 mm³.

According to the literature, the size of a thalamotomy lesion differs from one individual to other and is unpredictable, even when performed under constant thermo-coagulation parameters [15]. It ranges from no lesion [16] to a 177-mm³ lesion, with mean values varying from 26 to 74.5 mm³ [15–17]. The volume of the thalamotomy lesion, measured in our patient with a fine-cut CT scan and MRI 10 years after the thalamotomy procedure, was about 269.6 mm³ (4.65 mm in length, 5.73 mm in width, 10.12 mm in height), which is larger than the maximal estimated anatomical volume of the VPM nucleus. However, the lesion was not centered on the VPM. Its cartesian relation to the MC point was 5.39–11.12 mm in the x-axis, 7.67–12.32 mm in the y-axis and 0.81–10.93 mm in the z-axis. Therefore, the lesion covered the VPM nucleus completely in the inferosuperior distribution and partially in the anteroposterior and mediolateral directions. This information guided us to plan the trajectory of the DBS electrode, which was inserted in the anterolateral region of the thalamotomy lesion in the residual VPM, which yielded immediate and so far persisting pain relief.

To set up the stimulation parameters, we were guided by the surgical results, showing complete pain relief at 200 Hz during the first stimulation track. This frequency is unusually high to obtain pain relief. Indeed, most of the relevant literature in the field of pain management reports that low frequency stimulation (≤50 Hz) seems to have an analgesic effect, whereas higher frequency stimulations are described as causing hyperalgesia [18–20]. We do not have any clear explanation why our patient first responded better to higher frequencies and 4 months after surgery had more benefit with stimulation at 50 Hz. However, the subsequent adjustments of the parameters were guided by the patient’s pain response to stimulation, and the effect is still maintained 12 months after DBS.

Conclusion

We report the successful treatment of recurrent facial pain by VPM-DBS 10 years after VPM thalamotomy. The cause of a delayed recurrence of nociceptive facial pain that was controlled for 10 years following a previous VPM thalamotomy is difficult to understand. We believe that this report of a successful case of DBS as a second-step procedure after a VPM thalamotomy is fundamental as a salvage therapy, as this strategy has never been reported previously and has to be considered in the arsenal of therapeutic tools proposed in the case of pain recurrence after thalamotomy. Our patient is still pain free 11 months after surgery. It remains to be seen whether this effect of DBS will be maintained in the longer term.

References


