

Using Preimplanted Deep Brain Stimulation Electrodes for Rescue Thalamotomy in a Case of Holmes Tremor: A Case Report and Review of the Literature

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Keywords

Holmes tremor · Rescue thalamotomy · Lesioning · Deep brain stimulation

Abstract

Background: Chronic stimulation of the thalamus is a surgical option in the management of intractable Holmes tremor. Patients with deep brain stimulation (DBS) can encounter infection as a postoperative complication, necessitating explantation of the hardware. Some studies have reported on the technique and the resulting efficacy of therapeutic lesioning through implanted DBS leads before their explantation. **Case Description:** We report the case of a patient with Holmes tremor who had stable control of symptoms with DBS of the nucleus ventralis intermedialis of the thalamus (VIM) but developed localized infection over the extension at the neck, followed by gradual loss of a therapeutic effect as the neurostimulator reached the end of its service life. Three courses of systemic antibiotic therapy failed to control the infection. After careful consideration, we decided to make a rescue lesion through the implanted lead in the right VIM before explanting the complete DBS hardware. The tremor was well controlled after the rescue lesion procedure, and the effect was sustained during a 2-year follow-up period. **Conclusion:** This case and the previously discussed

ones from the literature demonstrate that making a rescue lesion through the DBS lead can be the last plausible option in cases where the DBS system has to be explanted because of an infection and reimplantation is a remote possibility.

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Introduction

Holmes tremor (HT) occurs after a lesion involving the cerebellum, midbrain, or thalamus [1, 2]. Such lesions are usually a result of hemorrhage, trauma, tumors, or infection [3–10]. Lesions affecting specific tracts, such as the cerebellothalamic or nigrostriatal tract, are considered the main cause of HT [2, 11–13]. It is a combination of resting, postural, and intention tremor.

In some cases, the tremor is responsive to different medical treatments. However, for the majority of patients, medication fails to alleviate the tremor, in which case stereotactic interventions are considered [14–16]. Studies have shown the efficacy of ablative and stimulation procedures in the management of HT. Sometimes, thalamotomy may have some short- and long-term complications, which may not be reversible and manageable [8, 17]. Deep brain stimulation (DBS) of the nucleus ventralis intermedialis of the thalamus (VIM) could be care-

fully considered as a treatment option for the management of HT [10, 18–20]. DBS is accompanied by surgical as well as hardware- and stimulation-related complications influencing the outcome.

Hardware infection has been reported to result in unsurmountable challenges during postoperative management of DBS patients [21, 22]. Antibiotic therapy and abscess drainage are the primary options. Complete or partial hardware removal may be necessary, followed by reimplantation of the DBS hardware or rescue ablative surgery.

DBS leads are primarily designed for neuromodulation of brain targets and networks. Since 2001, there have been reports showing the feasibility of using DBS leads for rescue ablative surgeries [23, 24]. The results of this method were comparable to those of DBS in most cases.

In this case report, we present a DBS patient with HT on whom rescue thalamotomy following infection was performed using the DBS lead before explanting the hardware.

Case Presentation

A 37-year-old male without any underlying disease developed severe traumatic brain injury after a car accident in 2005, leading to deep coma for about a month. Computerized tomography (CT) of the brain revealed diffuse axonal injury and focal midbrain contusions. After regaining consciousness, his speech and memory impairment recovered gradually, but a spastic left-sided weakness more prominent in the upper extremity persisted. During the first year after the accident, he had frequent paravertebral and upper- and lower-extremity spasms controlled partially by oral antispasmodic medication.

At the end of the first year, he complained of a gradually increasing tremor on his left side. The tremor was not severe in the beginning. After a while, the severity of the tremor made it relatively impossible for him to cope with his activities of daily living. The distally severe low-frequency tremor had uniform rest, action, and postural components. Brain magnetic resonance imaging (MRI) showed small lacunar lesions in the area of the basal ganglia. Based on a detailed neurological assessment, the patient was diagnosed with HT. Medical treatments had no effect on tremor suppression.

Unilateral VIM DBS was implanted at an overseas center, sponsored by his employer in 2012. The initial programming of the implantable neurostimulator (INS) led to complete resolution of the tremor and frequent muscle spasms. The patient felt a significant improvement in tremor suppression, resulting in improved functioning. At the end of the first year of DBS of the VIM, he developed a pustular eruption over the extension connector roughly under the mastoid eminence, gradually leading to persistent purulent discharge from the wound. Culture showed growth of *Staphylococcus aureus* bacteria. Multiple attempts of outpatient and inpatient systemic antibiotic therapy and local debridement of the wound were made to control the infection. These efforts were



Fig. 1. Nonstereotactic CT scan after deep brain stimulation.

mostly transiently effective, and discharge commenced again. Due to INS depletion, gradual loss of tremor suppression was observed.

Fearing the spread of infection to the intracranial space and the inability of the patient to undergo another DBS operation (because of economic constraints), we offered him the option of rescue thalamotomy through the implanted lead, followed by explantation of the complete INS system. The neurostimulator settings could not be read by the physician programmer as the device had reached the end of its service life when we met the patient and the programming notes were not available. We proposed that a lesioning procedure might help restore a more lasting tremor control for the patient without periodic neurostimulator replacements at the end of service life.

Surgical Approach

Prior to the surgical procedure, we evaluated the feasibility of using electrodes on the DBS lead for lesioning in egg albumin preheated in a water bath to normal body temperature. Twenty coagula between various pairs of electrodes on the DBS lead were made to determine optimum time, temperature, and energy parameters for achieving a suitable size and shape of the intended rescue lesion. Egg white may not exactly reflect the exact lesion shape and size in the nucleus of interest.

After in vitro bipolar lesion testing with a DBS lead (model 3389; Medtronic Inc., Minneapolis, MN, USA) and the radiofrequency generator (model G4; Cosman Medical Inc., Burlington, MA, USA), we decided to choose a setting of 35 mA for 40 s.

A preoperative CT scan (Fig. 1) showed that the DBS lead was in the thalamic region with respect to the mid-commissural point (functional coordinates verified with indirect targeting). The location of each of the electrodes was ascertained. From this analysis, the two inferior electrodes could be considered for lesion making based on intraoperative test stimulation confirmation.

In the operating room, an incision was made in the temporal area under local anesthesia and the lead was disconnected from the extension. Impedance testing of the lead was performed to ensure the connectivity of all the electrodes from the proximal to the distal end. Test stimulation with an external neurostimulator (model

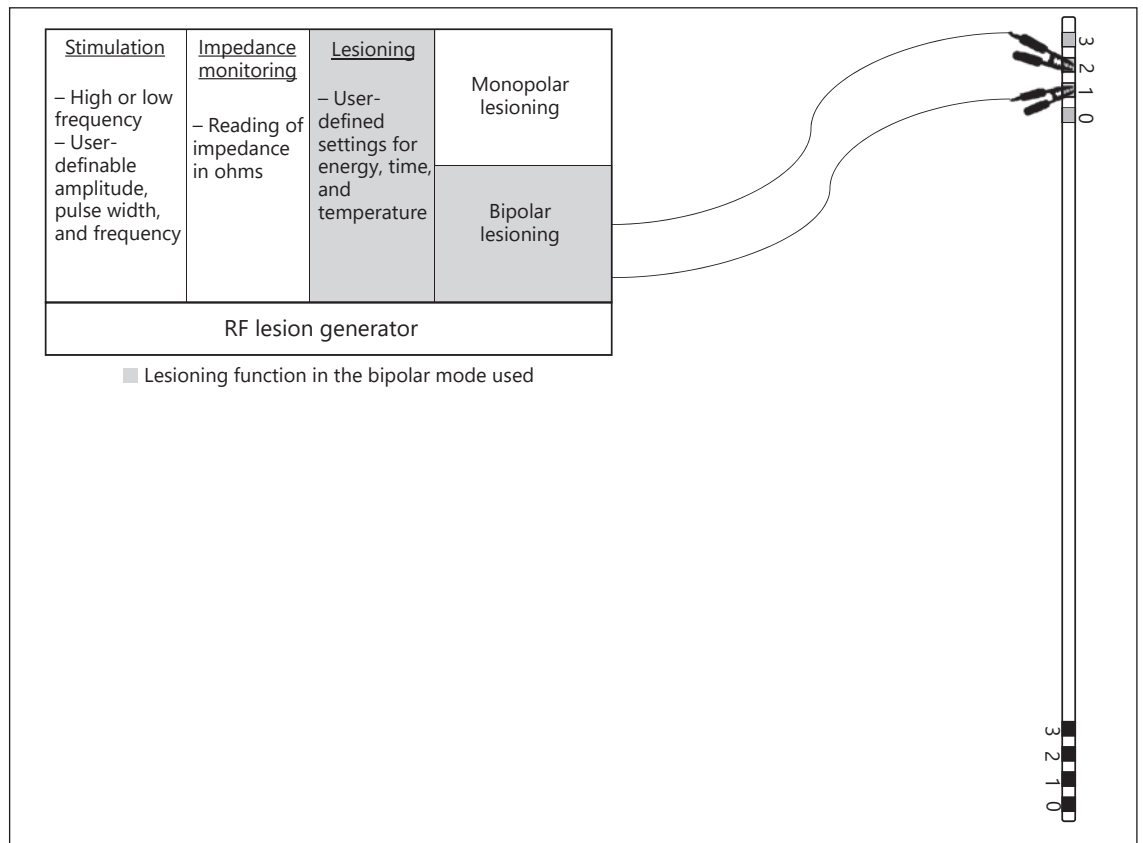


Fig. 2. Schematic connection diagram depicting our setup used for the rescue lesion. RF, radiofrequency.

37022; Medtronic Inc.) in a narrow bipolar configuration was performed between the adjacent pairs of electrodes in order to determine the most efficacious pair. The macrostimulation of every adjacent electrode pair suppressed the tremor significantly, so the central pair was chosen for bipolar lesion making (lesion centered between these two electrodes). We could not perform a reversible test lesion, nor monitor the temperature or impedance. Figure 2 shows the general schematic connection diagram depicting the rescue lesion setup. The lesioning function in the bipolar mode was used. Some features like impedance (lack of a Wheatstone bridge) and temperature (lack of a thermocouple) may not work in this mode. The patient was continuously monitored by the neurologist during the lesion making process. Explantation of the INS system was done thereafter, and the patient was administered intravenous antibiotic therapy. Postoperative MRI scans were used to confirm the location of the rescue lesion (Fig. 3).

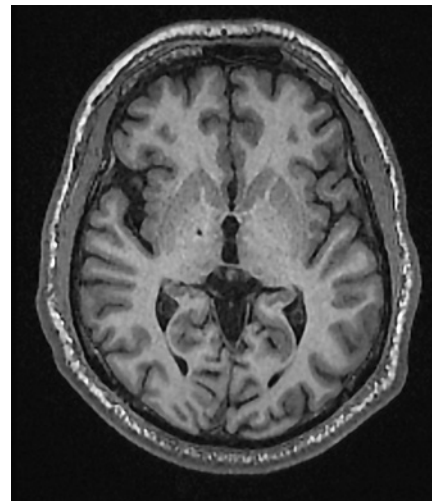


Fig. 3. Lesion after rescue thalamotomy at the level of the AC-PC plane.

Discussion

HT was first described by Gordon Holmes in 1904 [25]. It was previously known as rubral, midbrain, or thalamic tremor and Benedikt syndrome. At a movement

disorder congress held in 1998, the following criteria were accepted for diagnosis of HT: (1) tremor present at action, posture, and rest; (2) slow frequency (usually <4.5 Hz); and (3) tremor occurring with a delay of at least 2 weeks after the causative brain lesion [2, 11].

The cause of the brain lesion could be trauma, hemorrhage, infection, a cerebrovascular accident, multiple sclerosis, radiation, tumors, and neuroleptic drugs [3–10, 26–29]. The lesion usually affects the cerebellum, thalamus, or midbrain. Generally, lesions that involve the cerebellothalamic or nigrostriatal system are the main etiology of HT [2, 11–13, 30]. Neuronal rearrangement and neurogenesis occur after neuronal damage, which might explain the delay in onset of tremor after the pathology [31, 32].

Raina et al. [11] reported on a series of 19 cases of HT, 3 of whom had a massive lesion but with less severe symptoms. On the other hand, a case with a very small thalamic lesion showed more severe symptoms. The authors concluded that the size of the lesion does not have any direct association with the severity of symptoms, whereas the location of the lesion and the tracts involved could predict symptoms [11, 30].

There are reports of HT cases treated with medications. Levodopa, levetiracetam, trihexyphenidyl, clonazepam, and zonisamide are examples of effective drugs, but in the majority of cases, the tremor is not controlled with medications [14–16].

In medically refractory cases, stereotactic intervention could be carefully considered. Thalamotomy can reduce the tremor significantly and in some cases result in complete resolution. Transient or persistent complications like worsening of dysarthria, ataxia, cognitive issues, and hemiballismus can occur after thalamotomy. Online supplementary Table 1 (for all suppl. material, see www.karger.com/doi/10.1159/000506083) shows the results of studies where ablative procedures for HT were performed [8, 11, 17, 33–36].

In the past three decades, DBS has become a standard of care in the treatment of movement disorders such as Parkinson's disease, tremor, and dystonia. The efficacy of DBS of the VIM in HT has been reported in a few articles [10, 18]. There are recent studies selecting other targets, such as the area between the nucleus ventralis oralis anterior and the zona incerta [18].

Infection as a postoperative complication can pose unique challenges for treatment. The rate of infection is usually between 0 and 15% at different centers [37, 38]. Sillay et al. [22] reported on a series of 19 patients who developed an infection after DBS, and the main pathogen

was *Staphylococcus aureus*. In cases affected by *Staphylococcus aureus*, antibiotic therapy failed to treat the infection.

Antibiotic therapy is normally the first line of treatment in DBS hardware infection. When it is not effective, partial or complete explantation of the INS system may be necessary [22]. Reimplantation or a carefully considered ablative procedure could be treatment options in cases where the whole DBS system has been explanted and complete resolution of the infection has been confirmed.

Oh et al. [24] used the implanted DBS leads for a rescue lesion in 2 patients for the first time. Egg albumin was used to examine the feasibility of this method in vitro. Since 2001 there have been reports of this technique being used in select patients, and the results have been comparable to those of classic radiofrequency ablation or stimulation. Online supplementary Table 2 shows the studies on lesioning through implanted DBS leads and their results [23, 24, 39–44].

Rescue lesions performed through DBS electrodes have complications similar to those of radiofrequency thalamotomy, in addition to some specific ones. Tissue adhesion to electrodes can cause hemorrhage and electrode fracture during lead explantation.

With this technique, a coagulum is first shaped between the nearest points of two adjacent electrodes. Thereafter, the current is transformed between two further points because of tissue damage and changes in impedance. This process continues until a coagulum is formed between the furthest points on adjacent electrodes [45, 46]. Raoul et al. [47] showed that the current increases a little when the coagulum starts to form, and then it decreases as the lesion is about to reach its maximum size. The increase in current can also be considered as a starting point of coagulation in the operation room. The size of the coagulum does not change after a specific time period. In cases where there is no significant control of tremor after an episode of lesioning, the power can be increased to create a larger lesion, or a different pair of electrodes on the DBS lead can be chosen [45–47].

Raoul et al. [47] used a single electrode for creating 200 lesions, and then they evaluated the structure of the electrode. Scanning electron microscopy showed no change in the electrode's structure. This shows that DBS leads which have been used for lesioning can deliver stimulation at nonlesioned sites if necessary. Online supplementary Table 3 compares and contrasts classic radiofrequency ablation and rescue lesioning techniques [48, 49].

Conclusions

According to reports, there are three plausible requirements to ensure an optimal location of the implanted DBS lead and a satisfying outcome. We propose the following workflow after careful consideration:

1. Use conditionally safe MRI to create a patient-specific atlas using target-specific sequences or CT scanning with metal artifact suppression on a soft tissue filter in order to assess the anatomical location of each of the individual electrodes on the lead
2. Use impedance verification to ensure that the lead is electrically intact, and macrostimulation using the implanted electrodes to check the response on the symptoms intraoperatively [24]; programming notes, if available, can be of immense assistance in verifying the intraoperative macrostimulation results (effects and the threshold for side effects); imaging can show if the electrode is in the desired anatomical target, but macrostimulation is necessary to ensure an optimal clinical outcome [43, 44]
3. Recording of the local field potential with DBS electrodes can be a further indicator of symptom response to ablation [44]

Postoperative confirmation of the rescue lesion's location can be obtained by acquiring an MRI or CT scan. Additionally, this can be merged with the scans acquired before making the rescue lesion to audit the location of the same.

In the case presented, a small controlled thalamotomy performed using the above workflow provided satisfactory long-term effects for the HT patient. Thus, rescue lesions through the DBS lead (off-label) could be carefully considered for select patients if no other options are available.

Statement of Ethics

The Ethics Committee of the Research Center for Neuromodulation and Pain (Shiraz, Iran) approved this study, and written informed consent from the presented case is available.

Disclosure Statement

A.R. and O.Y. declare that there are no conflicts of interest relevant to this work. J.V. is an employee of Medtronic; the views expressed here are entirely personal and in no manner indicate the organizational standpoint.

Author Contributions

A.R.: conception and organization of the project, and review and critique of the manuscript; O.Y.: organization and execution of the study, design and execution of data collection, and writing of the first draft; J.V.: execution and review of the study, and review and critique of the manuscript.

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