# Bipolar deep brain stimulation permits routine EKG, EEG, and polysomnography

**Abstract**—As the population of patients treated with deep brain stimulation (DBS) grows and the patients age, more will require routine or emergent electrophysiologic tests. DBS artifact may render these uninterpretable, whereas stopping DBS may release symptoms that confound evaluation. The authors find that monopolar, but not bipolar, stimulation produces significant artifact during EKG, EEG, and polysomnography.

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Since the Food and Drug Administration approval of deep brain stimulation (DBS) in the United States for Parkinson disease (PD), essential tremor, and dystonia, estimates for PD implantations alone stood at 30,000 by 2004. DBS will become even more common as other indications are tested, such as Tourette syndrome,<sup>2</sup> myoclonic dystonia,<sup>3,4</sup> epilepsy,<sup>5</sup> and neuropsychiatric diseases. Because of the growing prevalence of DBS and the aging of patients with DBS, more will require electrodiagnostic procedures (such as EKG, EEG, and polysomnography) for evaluation of acute or chronic disease. DBS generates electrical artifact, a confounder that may obscure important diagnostic information. It was recently shown that bipolar DBS produces far less electrical artifact than monopolar DBS in EKG.7 The purpose of our investigation is to confirm these earlier findings and to compare artifact produced from bipolar and monopolar DBS in routine EKG, EEG, and PSG studies.

**Methods.** Subjects. Subject A. A 67-year-old man with bilateral subthalamic nucleus (STN) DBS for tremor-dominant PD underwent voluntary EKG and EEG. The DBS electrodes (Medtronic model 3389) were connected to subclavicular pulse generators (Medtronic, Soletra). He was initially treated with monopolar configurations, but programming sessions over the next 2 years yielded optimal clinical effects with bipolar settings: 4.3 V, 160 Hz, contacts 3+, 0-, pulse width 90 microseconds (right) and 120 microseconds (left).

Following informed consent, standard EKG and EEG were obtained initially at baseline bipolar DBS settings (figures 1A and 2A), with DBS off (see figures 1B and 2B), followed by a monopolar configuration (0-, case+, 3.0 V) (see figures 1C and 2C).

<u>Subject B.</u> A 66-year-old man was treated effectively with a right thalamotomy and a left ventralis intermedius thalamic DBS for tremor. He developed postoperative hypervigilance that evolved into excessive daytime sleepiness and fractionated sleep.

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He was prepared for overnight polysomnography by setting DBS to a bipolar configuration (3+, 0-, 120 microseconds, 160 Hz, 5.3 V).

Results. Figure 1A shows the V2 lead trace clipped from the standard 12-lead EKG recorded during bipolar stimulation (see figure 1B during temporary discontinuation of DBS and figure 1C during monopolar stimulation). The bipolar configuration produced clinically insignificant artifact (see figure 1A) that was similar to the "DBS off" condition (see figure 1B). In contrast, monopolar settings produced a large-amplitude artifact on EKG that obscured the P and T waves and QRS complex on all leads, rendering the study uninterpretable (see figure 1C).

Figure 2 shows samples of optimal, occipital wakeful EEG rhythm. The montage included channels with both long and short interelectrode distances. The EEG demonstrated interpretable signal during bipolar stimulation (see figure 2A) with minimal differences between bipolar mode and DBS off (see figure 2, A and B). Resting tremor was visible in the submental EMG during DBS off (see figure 2B) and persisted through monopolar DBS settings (see figure 2C), demonstrating that deterioration in clinical status may develop quickly during attempts to record with DBS off. During bilateral monopolar DBS, the high-frequency artifact obscured most channels of the EEG and was most marked on long interelectrode distances (see figure 2C).

Subject B underwent an overnight polysomnography using standard lead configurations (see figure E-1 on the *Neurology* Web site at www.neurology.org). The polysomnography was free of artifact, and sleep scoring via standard electroocular channels, EEG, and chin muscle activity was not impeded by continuous bipolar DBS.

**Discussion.** These findings demonstrate that routine electrodiagnostic tests such as EKG, EEG, and polysomnography are possible in patients during continuous DBS. Adequate electrodiagnostic results are only possible, however, when the DBS is programmed with a bipolar configuration.

The problem of DBS interference with electrodiagnostic tests will increase with the rising use of DBS and the aging of existing DBS patients. Ideally, practitioners, especially emergency personnel, would become familiar with DBS and its effects. Typically, however, time and DBS-specific equipment and expertise are lacking outside of movement disorder clinics. DBS interference may cause potentially dangerous problems, or at least troublesome delays, in

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Figure 1. V2 traces clipped from a standard 12-lead EKG from Subject A with bilateral subthalamic nucleus deep brain stimulation (DBS). Trace A = bilateral DBS in bipolar configuration (4.3 V, 3+, 0-, 160 Hz, pulse width left side 120 microseconds, right side 90

microseconds). Trace B = DBS Off. Trace C = left DBS on monopolar (3.0 V, case +, 0-, 160 Hz, pulse width 90 microseconds). All traces: gain 10 mm/mV, bandpass 0.15 to 150 Hz, 60-Hz notch filter on.

cases of unconsciousness or severe trauma when emergent testing is required.

Discontinuing DBS may reduce interference but result in other technical problems during electrodiagnostic tests. For example, patients with tremor uncontrolled by DBS may exhibit electromyography or movement artifact (see tremor artifact in EMG channel of figure 2B).

Acute alteration of DBS settings may also result in patient discomfort or inconvenience. As medications are reduced on average by about 40% after effective subthalamic DBS treatment for PD,<sup>8</sup> the likelihood of acute discomfort is high in PD patients who abruptly have DBS discontinued.<sup>9</sup> In our experience, a PD patient may require up to an hour to regain the ability to walk safely after 30 minutes off DBS. Longer-term studies such as overnight polysomnography may become both extremely uncomfortable for the patient and of questionable validity due to the dramatic deterioration of the motor status of the patient.

Any of the four intracranial contacts on the DBS electrode can be programmed to be anode (positive) or cathode (negative). In a (pseudo)-monopolar configuration, the case of the pulse generator serves as the anode and intracranial contact(s) as cathode, resulting in a long-dipole electrical field. In a bipolar configuration, intracranial electrodes serve as anode and cathode, thereby forming a short electrical dipole that affects a relatively smaller volume of tissue and generates far less artifact.

Patients with DBS are most commonly programmed in monopolar mode for time efficiency.

Finding an optimal setting among the dozen possible two-contact bipolar configurations is time intensive. Bipolar settings are thus typically used where side effects limit voltage to suboptimal levels in monopolar mode.<sup>10</sup>

There is considerable debate and few data concerning optimal DBS parameters, in particular monopolar vs bipolar configuration. It is also likely that optimal parameters are different across different DBS targets to treat different diseases.

In our experience, bipolar configurations have proven effective in thalamic DBS to control tremor. The rapid response of tremor to thalamic DBS is easy to observe. It is more difficult to evaluate different DBS configurations in PD, because motor symptoms may respond hours after changes in settings. Some PD patients exhibiting side effects from monopolar stimulation can achieve good clinical results with bipolar stimulation, suggesting that this may be an effective option in a PD patient with indications for EKG or polysomnography.

Although the dystonia response to DBS evolves over months, we have observed excellent results in our clinic with either long dipole (e.g., 3+, 0-) or monopolar settings. Pending a study with the power to clearly define the relative merits, there is good reason to attempt a bipolar configuration in any DBS patient who may need electrodiagnostic testing. A practical starting point may be to use the best monopolar contact as cathode in a bipolar setting. Polarity of the dipole (anode above or anode below) plays an important role in both efficacy and side effects.

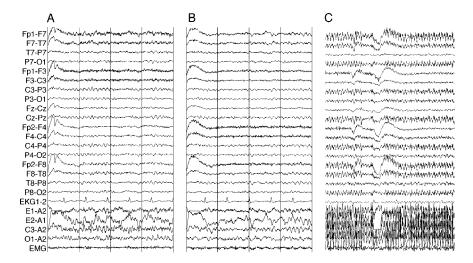


Figure 2. Four-second excerpts of waking EEG from Subject A with bilateral subthalamic nuclear deep brain stimulation (DBS) for Parkinson disease. (A) Bilateral DBS on in bipolar mode (see legend for figure 1). (B) Both DBS implants off. (C) Bilateral monopolar DBS (3.0 V, case +, 0-, 160 Hz, 90 microseconds. Bar = 100  $\mu$ V; time marks = 1 second.

#### References

- 1. Lozano AM, Hamani C. The future of deep brain stimulation. J Clin Neurophysiol 2004;21:68-69.
- Visser-Vandewalle V, Temel Y, Boon P, et al. Chronic bilateral thalamic stimulation: a new therapeutic approach in intractable Tourette syndrome. Report of three cases. J Neurosurg 2003;99:1094-1100.
- 3. Krauss JK. Deep brain stimulation for dystonia in adults. Overview and developments. Stereotact Funct Neurosurg 2002;78:168-182.
- 4. Krauss JK, Loher TJ, Weigel R, Capelle HH, Weber S, Burgunder J-M. Chronic stimulation of the globus pallidus internus for treatment of non-dYT1 generalized dystonia and choreoathetosis: 2-year follow up. J Neurosurg 2003;98:785-792.
- 5. Kerrigan JF, Litt B, Fisher RS, et al. Electrical stimulation of the anterior nucleus of the thalamus for the treatment of intractable epilepsy. Epilepsia 2004;45:346-354.

- 6. Greenberg BD, Rezai AR. Mechanisms and the current state of deep brain stimulation in neuropsychiatry. CNS Spectr 2003;8:522-526.
- 7. Constantoyannis C, Heilbron B, Honey CR. Electrocardiogram artifacts caused by deep brain stimulation. Can J Neurol Sci 2004;31:343-346.
- 8. Kleiner-Fisman G, Fisman DN, Sime E, Saint-Cyr JA, Lozano AM, Lang AE. Long-term follow up of bilateral deep brain stimulation of the subthalamic nucleus in patients with advanced Parkinson disease. J Neurosurg 2003;99:489-495.
- 9. Chou KL, Siderowf AD, Jaggi JL, Liang GS, Baltuch GH. Unilateral battery depletion in Parkinson's disease patients treated with bilateral subthalamic nucleus deep brain stimulation may require urgent surgical replacement. Stereotact Funct Neurosurg 2004;82:153-155.
- 10. Volkmann J, Herzog J, Kopper F, Deuschl G. Introduction to the programming of deep brain stimulators. Mov Disord 2002;17(suppl 3): S181-S187.

## **Neuro** *Images*

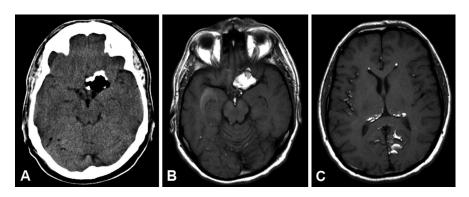


Figure. Non-enhanced CT shows a lowdensity mass with mural calcifications in the juxtasellar region (A). T1weighted MRI without contrast reveals high signal of the lesion representing its fatty content (B) and hyperintense droplets in the interpeduncular cistern (B), the frontal horns and sulci (C) after subarachnoid rupture. The anterior misregistration of the lipid droplets which appear to lie anterior to the ventricles (C) is a chemical shift artifact along read-out gradient direction.

### An MRI view of a ruptured dermoid cyst

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Intracranial dermoid cysts are developmental masses which arise from inclusions of ectodermoidal elements in the neural

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groove and typically contain fatty components.1 Rupture into the subarachnoid space is rare and may produce chemical meningitis. We report a 44-year-old patient presenting with a single complex focal seizure. MRI (figure) showed diffuse subarachnoid fatty deposits, but CSF revealed only a slight pleocytosis without signs of chemical meningitis. Seizures have been identified as typical signs after dermoid rupture especially in elderly patients.2 However, the monosymptomatic presentation in our patient was unusual in view of the extensive subarachnoid spread of fatty material.

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- 1. Smirniotopoulos JG, Chiechi MV. Teratomas, dermoids, and epidermoids of the head and neck. Radiographics 1995;15:1437-1455.
- 2. Stendel R, Pietila TA, Lehmann K, Kurth R, Suess O, Brock M. Ruptured intracranial dermoid cysts. Surg Neurol 2002;57:391-398.



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