Deep brain stimulation (DBS) of the basal ganglia and thalamus has been used successfully to treat adults with intractable movement disorders. This success has encouraged the use of DBS in other movement disorders such as childhood dystonia, in which electrodes are inserted into the globus pallidus internus (GPi).4,11,21 The procedure of inserting DBS electrodes involves initial brain imaging, placement of cranial bone fiducials or a stereotactic head frame to establish coordinates for target calculations, creation of a bur hole, and insertion of the stimulating electrode into the GPi. The neurophysiologist uses variations in spontaneous firing rates from single-unit microelectrode recordings (MERs) to identify the location of the electrode tip. Finally, macrostimulation permits confirmation of accurate placement of the permanent electrode and observation of any clinical improvements or side effects.11 Unfortunately, many anesthetic drugs can affect neuronal firing frequency, resulting in the inability to use MERs to guide electrode placement.16 Because the brain is free from pain fibers, the electrode insertion part of the procedure is performed with the patient awake while receiving monitored anesthesia care or an asleep-awake-asleep technique.3,10–12,14,21,22 However, young children with severe dystonia, developmental delay, and other associated cardiorespiratory conditions are not good candidates for awake procedures because their restlessness and anxiety can interfere with the operation. The unsedated patient may also have a higher risk of intracerebral bleeding from intraoperative hypertension.7 In addition, sedation may be associated with cardiorespiratory- and airway-related complications. In this paper we report on our experience in 6 pediatric patients in whom a

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**Preservation of microelectrode recordings with non–GABAergic drugs during deep brain stimulator placement in children**

**Technical note**

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Object. Deep brain stimulation (DBS) has become accepted therapy for intractable dystonia and other movement disorders. The accurate placement of DBS electrodes into the globus pallidus internus is assisted by unimpaired microelectrode recordings (MERs). Many anesthetic and sedative drugs interfere with MERs, requiring the patient to be awake for target localization and neurological testing during the procedure. In this study, a novel anesthetic technique was investigated in pediatric DBS to preserve MERs.

Methods. In this paper, the authors describe a sedative/anesthetic technique using ketamine, remifentanil, dexmedetomidine, and nicardipine in 6 pediatric patients, in whom the avoidance of GABAergic stimulating drugs permitted excellent surgical conditions with no detrimental effects on intraoperative MERs. The quality of the MERs, and the frequency of its use in making electrode placement decisions, was reviewed.

Results. All 6 patients had good-quality MERs. The data were of sufficient quality to make a total of 9 trajectory adjustments.

Conclusions. Microelectrode recordings in pediatric DBS can be preserved with a combination of dexmedetomidine and ketamine, remifentanil, and nicardipine. This preservation of MERs is particularly crucial in electrode placement in children.

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**Key Words** • microelectrode recording • deep brain stimulation • anesthesia • sedative • functional neurosurgery

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Abbreviations used in this paper: DBS = deep brain stimulation; GABA = γ-aminobutyric acid; GPi = globus pallidus internus; LMA = laryngeal mask anesthesia; MER = microelectrode recording.
sedative/anesthetic technique with dexmedetomidine, ketamine, remifentanil, and nicardipine and the avoidance of drugs acting on the γ-aminobutyric acid (GABA) system provided excellent surgical conditions, unimpaired neuroelectrophysiological signals, hemodynamic stability, and a smooth, prompt emergence from anesthesia, allowing immediate neurological assessment after long DBS procedures.

Methods

The preoperative conditions of the patients are presented in Appendix 1 and intraoperative anesthetic medications administered during DBS placement are summarized in Appendix 2. Duration of surgery, anesthesia emergence times, and postoperative care is provided in Appendix 3. All patients were monitored with standard American Society of Anesthesiologists monitors, had wide-bore venous access, an arterial line, and a Foley catheter inserted. Attention was paid to careful patient positioning and maintenance of body temperature. Scalp blocks and local infiltration with 0.25% bupivacaine were performed prior to the fiducial placement and incision, and repeated as needed. The Nexframe Frameless Stereotaxy System (Medtronic) was used to implant the Activa 3309 4-contact electrode (Medtronic) into the target nucleus. Pulse generators were then implanted into a subpectoral pocket and connected to the electrodes for long-term stimulation therapy.

Case 1

A 14-year-old girl with pantothenate kinase deficiency-associated neurodegeneration, developmental delay, severe dystonia, and opisthotonos had a medical history of hereditary spherocytosis with rhabdomyolysis, hypertension controlled with amlodipine, and an episode of respiratory failure requiring ventilation. Anesthesia was induced with 3–3.5 mg/kg of intravenous propofol and tracheal intubation was facilitated with atracurium. A loading dose of dexmedetomidine, 1 μg/kg intravenously over 30 minutes, was followed by an infusion at 0.3–0.5 μg/kg/hr, along with remifentanil at 0.1 μg/kg/min and intermittent ketamine in doses of 0.05–0.2 mg/kg intravenously. No volatile anesthetic agents, nitrous oxide, or supplementary doses of propofol were administered during the procedure. Nicardipine was administered for blood pressure control (1.0–2.5 μg/kg/min intravenously).

In preparation for the awake phase of MER and neurophysiological testing of the procedure, general anesthesia was discontinued by stopping the remifentanil and ketamine infusions and reducing the dexmedetomidine infusion rate to 0–0.5 μg/kg/hr. The ideal place for the permanent electrode was determined by optimal MER signatures within the tract (described below) and lack of adverse side effects upon macrostimulation at greater than 4 V. The patient then received general endotracheal anesthesia with sevoflurane, rocuronium, and fentanyl or morphine for the pulse generator implantation and electrode connection.

Cases 2–6

Cases 2–6 underwent an asleepleve-stimulation procedure after intravenous access was obtained. The patients were put to sleep with ketamine, remifentanil, and dexmedetomidine infusions for the opening, placement of bur holes, durotomies, and placement of the Nexframe apparatus. Patency of the airway was initially maintained with a laryngeal mask. Prior to the MER and permanent electrode placement, the remifentanil and ketamine were discontinued while dexmedetomidine was reduced to 0–0.5 μg/kg/hr and the laryngeal mask anesthesia (LMA) was removed. A child life companion assisted in calming or waking the patient as needed. After MER confirmed proper electrode placement and the permanent electrode was in position, the patient underwent awake macrostimulation to look for evidence of electrode malplacement such as tonic contractions of the contralateral limbs, adverse sensory responses, or reported visual scintillations. Nicardipine infusions were adjusted to maintain normotension during the awake phase. When the neurophysiologist satisfied with the position of the electrode, the electrode was secured and the incision was temporarily closed. The patient then received general endotracheal anesthesia with sevoflurane, rocuronium, and fentanyl or morphine for the pulse generator implantation and electrode connection.

Microelectrode Recording

Microelectrode recording was performed using microTargeting electrodes (Fred Hare Co.) on a StimPilot system for Cases 1–5 (Medtronic) and the Leadpoint system (Medtronic) for Case 6. Microelectrode recording was used to place a total of 12 electrodes in the 6 patients. The GPi was targeted 11 times and the subthalamic nucleus was targeted once. There was good signal-to-noise ratio on all recordings, and each final electrode trajectory possessed recordings that were consistent with traversed and targeted nuclei. Low-frequency pausing cells, low-frequency bursting cells, border cells, kinesthetic cells, and high frequency cells were encountered and used to assess electrode tip location. The average electrode impedance was 500 kOhm. The microelectrode recordings were of sufficient quality to enable a total of 9 trajectory adjustments.

Neurophysiological Testing

As the electrode was advanced from 10 mm above the proposed target, physiological testing was performed. This testing included rapid flexion-extension of the contralateral elbow, wrist, knee, and ankle to detect kinesthetic cell activity changes. Near and beyond the target, optic tract potentials were investigated by placing a strobe light in the patients visual field. When the microelectrode was in the optimal position based upon the MER, the microelectrode tip was retracted and the electrode cannula was advanced 10 mm and microstimulation was performed. The patient was then examined for adverse reactions to the stimulation such as arm and leg contractions that would reflect internal capsule placement or visual phosphenes or scotoma that would reflect optic tract placement. If no adverse reactions were detected, the microelectrode was removed and the 4-contact permanent electrode was placed in that...
position and macrostimulation was performed up to 5 V. If no adverse responses were detected, the electrode was anchored in this position. Case 1 required a general anesthesia technique to control patient movements due to severe dystonia and to allow the placement of an endotracheal tube for airway protection and ventilatory support required due to marginal airway patency and inadequate spontaneous respiratory effort. Although this patient was not able to report subjective adverse responses to microor macrostimulation, the patient did otherwise undergo the same neurophysiological testing.

Results

There were no anesthesia-related complications or cardiovascular or metabolic instability throughout the entire 12-hour procedure. Each patient was awake, stable, and without any signs of new neurological symptoms, and the trachea was extubated 10 minutes after completion of the operation. The neurophysiologists reported excellent quality and pattern of neuroelectrical signals from the globus pallidus (Fig. 1 upper and center) and subthalamic nucleus (Fig. 1 lower) throughout the MER procedure.

Discussion

The anesthesiologist is faced with a number of patient- and procedure-related challenges in managing patients undergoing DBS placement.21 Preservation of neurophysiological electrical activity generated from the globus pallidus is crucial for an optimal placement of the stimulating electrodes and subsequent improvement of the dystonia. Many anesthetic drugs have effects on these signals, but sedation may be required if the patient’s age, emotional instability, or violent uncontrolled dystonia make them unable to tolerate the procedures while awake. Sedation may also be necessary to avoid complications from intraoperative movement and hypertension resulting in intracerebral hemorrhage. The availability of MER is essential in movement disorder surgery in pediatric patients due to the abnormal basal ganglia anatomy frequently encountered, in contrast to the adult movement disorder population. A common approach in children is to employ the asleep-awake-asleep technique. None of the chosen agents alone provides a level of anesthesia necessary for the initial phase of the surgery. A combination of 3 non–GABAergic agents was used to achieve adequate analgesia and sedation without MER compromise. During the awake phase a dexmedetomidine infusion between 0.1 and 0.5 μg/kg/hr was maintained while ketamine and remifentanil were discontinued approximately 30 minutes prior to MER.

Gamma-aminobutyric acid is an important inhibitory neurotransmitter in the basal ganglia.16 Anesthetics such as benzodiazepines, barbiturates, propofol, etomidate, and volatile agents potentiate the postsynaptic GABA effects by several actions that create an increased “open probability” for the GABA receptor channels.17 Recent animal work on normal and MPTP-treated mice has shown abundant GABAergic innervations and GABA receptors in globus pallidus and that the endogenous GABA receptors

Fig. 1. Microelectrode recording screenshots. Upper: Screenshot of a partial tract of an MER performed on the Stimpilot demonstrating recordings consistent with a subcortical tract to the GPi. Center: A fusion of screenshots of an MER tract demonstrating clear frequency changes used to guide the placement of the stimulating electrode into the GPi. Lower: A complete record of an MER tract demonstrating clear frequency changes used to guide the placement of the stimulating electrode into the subthalamic nucleus.
modulate the spontaneous firing of globus pallidus neurons in normal mice. Active spontaneous neuroelectrical firing from the globus pallidus is exceptionally sensitive to even small doses of anesthetic agents due to a large GABAergic input from the striatum. Propofol has been shown to greatly depress global discharge and findings in animal studies support that this effect is at least, in part, GABA receptor mediated. Ketamine and remifentanil have fewer effects on spontaneous neuronal discharge during DBS placement. The effect of μ opioid agonists on the GABAergic system is less clear as there are animal studies that suggest these drugs could modulate the activity of GABA neurons via receptors located mainly at extrasynaptic sites on dendritic plasma membranes. We therefore avoided the use of benzodiazepines, propofol, and volatile anesthetics and discontinued remifentanil during the MER phase.

Dexmedetomidine is a highly selective, short-acting α2 adrenoceptor agonist with sedative anxiolytic, analgesic, and probably neuroprotective effects without significant respiratory depression. Alpha-2 adrenoceptors are a subgroup of noradrenergic receptors distributed within and outside the CNS. The central presynaptic α2 adrenergic receptor is a negative feedback receptor resulting in a decreased catecholamine release from the nerve terminals. Dexmedetomidine appears to exert its sedative and anxiolytic effects through α2-adrenoceptor activation of the pontine nucleus locus coeruleus, a major site of noradrenergic innervation in the CNS. Decreased noradrenergic output from the locus coeruleus allows for increased firing of inhibitory neurons, most importantly the GABA system. This reaction is similar to normal sleep and appears to be the anesthetic mechanism of dexmedetomidine. The central sedative effect of dexmedetomidine differs from other anesthetic agents by its presynaptic action on a subcortical level in the pons and medulla and not by action through the GABAergic system or cerebral cortex. This drug has become increasingly used as an anesthetic agent during functional neurosurgical procedures because it does not interfere with neurophysiological electrical signal monitoring and spontaneous basal ganglia neuron activity, and allows brain mapping during awake craniotomy and MER. It has been used in both adult and pediatric patients who require intraoperative electrocorticography, cortical mapping, or neurocognitive testing.

Ketamine, a cyclohexanone derivate, interacts with N-methyl-D-aspartate subtype glutamate receptors, opioid receptors, monoaminergic receptors, muscarinic receptors, and calcium ion channels. The anesthetic mechanism of ketamine is not well understood, but it does not interact with GABA receptors. Previous reports of anesthetic techniques for DBS have not focused on ketamine for sedation and analgesia during MER. We speculate that a combination of ketamine with dexmedetomidine is advantageous in obtaining an adequate depth of anesthesia without affecting neuronal discharge during the pre-electrode insertion phase. Ketamine may also be beneficial in countering the hemodynamic effects of dexmedetomidine on heart rate.

Our findings suggest that the advantage of this anesthetic technique lies in the selection of non-GABAergic anesthetics. Further studies will be needed to rule out a dose effect. Elias et al. described loss of subthalamic nucleus MERs with increasing doses of dexmedetomidine (above 0.5 μg/kg/hr) that resulted in sedation and decreasing Bispectral Index value.

**Conclusions**

We report 6 pediatric patients, ages 9–18 years, with severe dystonia who successfully underwent bilateral DBS electrode implantation using an anesthetic technique that avoided drugs with GABA receptor action. This anesthetic technique provided excellent surgical conditions, unimpaired neuroelectrophysiological conditions crucial for optimal DBS placement, and hemodynamic stability.

**Disclosure**

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conceptual and design: Curry, Karlberg Hippard, Watcha. Acquisition of data: Curry, Karlberg Hippard. Analysis and interpretation of data: Karlberg Hippard. Stocco. Drafting the article: Curry, Karlberg Hippard, Watcha. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Curry. Administrative/technical/material support: Karlberg Hippard. Study supervision: Karlberg Hippard, Stocco.

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Neurophysiological anesthesia in pediatric DBS


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### Appendix 1: Characteristics of the patients during preoperative evaluation

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Weight (kg)</th>
<th>Diagnosis</th>
<th>Medical History</th>
<th>Medications</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>14, F</td>
<td>26.4</td>
<td>pantothenate kinase deficiency, hereditary spherocytosis, neurogenic hyperthermia, hypertension, retinitis pigmentosa</td>
<td>pneumonia w/ respiratory failure, splenectomy, tendon releases, gastrostomy, baclofen pump insertion</td>
<td>baclofen, dantrolene, clonazepam, lansoprazole, glycopyrrolate, amiodipine, albuterol, azithromycin, acyclovir</td>
<td>severe developmental delay, severe opisthotonus, dysphagia, thick secretions, no interaction w/ parents</td>
</tr>
<tr>
<td>2</td>
<td>14, M</td>
<td>24.7</td>
<td>cerebral palsy (neonatal cerebrovascular accident), hypoxic encephalopathy, severe quadriparetic dystonia (hypokineti form)</td>
<td>tenotomy, baclofen pump insertion, botox injections</td>
<td>baclofen, benzodiazepines, levodopa/carbidopa, trihexyphenidyl</td>
<td>former premature twin 24-wk gestation, sleep disturbance, anxiety, verbally communicative</td>
</tr>
<tr>
<td>3</td>
<td>18, F</td>
<td>62</td>
<td>intrauterine lt middle cerebral artery stroke, severe rt hemidystonia, seizure disorder, depression, migraine</td>
<td>tendon releases, adeno-tonsillectomy &amp; ear tube insertion</td>
<td>baclofen, diazepam, tizanidine, leviracetam, amitriptyline, rizatriptan, escitalopram, botox injections, naproxen</td>
<td>severe pain, anxious, small mouth opening</td>
</tr>
<tr>
<td>4</td>
<td>9, F</td>
<td>44.1</td>
<td>acute myeloid leukemia (in remission), rt basal ganglia infarction, lt-sided dystonia</td>
<td>aspergillosis pneumonia, chemotherapy for leukemia, wedge resection rt upper &amp; middle lobe lung, botox injections</td>
<td>baclofen, clonazepam, carbidopa, levodopa, budesonide, albuterol, ibuprofen</td>
<td>lt-sided dystonia w/ hyperextension, severe pain</td>
</tr>
<tr>
<td>5</td>
<td>9, M</td>
<td>22.5</td>
<td>congenital diaphragmatic hernia, hypotension, stroke &amp; anoxic brain injury, α-1 antitrypsin deficiency</td>
<td>hernia repair, gastrostomy, lysis of peritoneal adhesions</td>
<td>baclofen, benzodiazepines, carbidopa, levodopa, tetrabenzidine</td>
<td>dysarthria, dysphagia, asthma, gastro-esophageal reflux, eczema, awake, alert, ambulatory, converses w/ dysarthric speech</td>
</tr>
<tr>
<td>6</td>
<td>13, M</td>
<td>39</td>
<td>Tourette's syndrome, self-injurious behavior, depression</td>
<td>none</td>
<td>sertraline, melatonin, clonidine, pimozide, guanfacine</td>
<td>obsessive-compulsive behavior disorder, attention-deficit hyperactivity disorder, motor &amp; phonic tics</td>
</tr>
</tbody>
</table>

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### Appendix 2: Details of intraoperative management in each case *

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sensorium</th>
<th>Airway</th>
<th>Drugs</th>
<th>Pre-Electrode Insertion Phase</th>
<th>Electrode Insertion Phase</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>general anesthesia</td>
<td>endotracheal intubation</td>
<td>propofol; atracurium; dexmedetomidine; remifentanil; ketamine; nicardipine; “scalp block”</td>
<td>general anesthesia; endotracheal tube</td>
<td>Dexmedetomidine Infusion (μg/kg/hr): 0.3–0.5, Ketamin Bolus (mg/kg): 0.05–0.2 repeated every 40–45 mins, Remifentanil Infusion (μg/kg/min): 0.1, Nicardipine Infusion (μg/kg/min): 1.0–2.5, Post-Electrode Insertion</td>
</tr>
<tr>
<td>2</td>
<td>general anesthesia</td>
<td>LMA</td>
<td>dexmedetomidine; ketamine; remifentanil; nicardipine; “scalp block”</td>
<td>LMA removed; O₂ by nasal cannula; sedated</td>
<td>Dexmedetomidine Infusion (μg/kg/hr): 0.4–0.8, Ketamin Bolus (mg/kg): none, Remifentanil Infusion (μg/kg/min): stopped 45 mins prior to MER, Nicardipine Infusion (μg/kg/min): 0.5–1.0, Post-Electrode Insertion</td>
</tr>
<tr>
<td>3</td>
<td>general anesthesia</td>
<td>LMA</td>
<td>dexmedetomidine; ketamine; remifentanil; rocuronium; nicardipine; “scalp block”</td>
<td>LMA removed; O₂ nasal cannula; sedated</td>
<td>Dexmedetomidine Infusion (μg/kg/hr): 0.2–0.5, Ketamin Bolus (mg/kg): 0.4 every hr × 2 doses, Remifentanil Infusion (μg/kg/min): stopped 1 hr prior to MER, Nicardipine Infusion (μg/kg/min): 1.5–3.0, Post-Electrode Insertion</td>
</tr>
<tr>
<td>4</td>
<td>general anesthesia</td>
<td>LMA</td>
<td>dexmedetomidine; ketamine; remifentanil; nicardipine; “scalp block”</td>
<td>LMA removed; O₂ nasal cannula; sedated</td>
<td>Dexmedetomidine Infusion (μg/kg/hr): off, Ketamin Bolus (mg/kg): none, Remifentanil Infusion (μg/kg/min): off, Nicardipine Infusion (μg/kg/min): 0.75–1.5, Post-Electrode Insertion</td>
</tr>
<tr>
<td>5</td>
<td>general anesthesia</td>
<td>LMA</td>
<td>dexmedetomidine; ketamine; remifentanil; nicardipine; “scalp block”</td>
<td>LMA removed; O₂ nasal cannula; sedated</td>
<td>Dexmedetomidine Infusion (μg/kg/hr): 0–0.5, Ketamin Bolus (mg/kg): none, Remifentanil Infusion (μg/kg/min): off 30 mins prior to MER, 0.05 at end of MER, Nicardipine Infusion (μg/kg/min): 0.5–1.0, Post-Electrode Insertion</td>
</tr>
<tr>
<td>6</td>
<td>asleep</td>
<td>LMA</td>
<td>dexmedetomidine; ketamine; remifentanil; nicardipine; “scalp block”</td>
<td>LMA removed; O₂ nasal cannula; sedated</td>
<td>Dexmedetomidine Infusion (μg/kg/hr): 0.1–0.5, Ketamin Bolus (mg/kg): 0.5–1.5 every 30 mins, Remifentanil Infusion (μg/kg/min): stopped 45 mins prior to MER, Nicardipine Infusion (μg/kg/min): 0.2–0.6, Post-Electrode Insertion</td>
</tr>
</tbody>
</table>

### Appendix 3: Postoperative management

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Duration of Surgery (hrs)</th>
<th>End of Surgery Until Tracheal Extubation (mins)</th>
<th>Pulse Generator Placement</th>
<th>Post-Anesthesia Care Unit</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>12</td>
<td>10</td>
<td>2 days after DBS insertion</td>
<td>no complications; placement of deep brain electrodes in region of basal ganglia bilaterally (CT scan)</td>
<td>respiratory insufficiency w/ difficulties clearing secretions, tracheostomy 1 mo after DBS surgery</td>
</tr>
<tr>
<td>2</td>
<td>10.3</td>
<td>5</td>
<td>immediately after DBS placement</td>
<td>no complications; MRI &amp; CT confirmed good position of DBS stimulator traversing the bilateral GPi to terminate in the region of substantia innominata; no evidence of intracranial abnormality or complication</td>
<td>postoperative urinary tract infection, initially markedly reduced dystonia of rt leg, little or no improvement in other limbs</td>
</tr>
<tr>
<td>3</td>
<td>10</td>
<td>12</td>
<td>immediately after DBS placement</td>
<td>no complications; DBS in the expected vicinity of the subthalamic nucleus region (CT scan), small extraaxial pneumocephaly</td>
<td>dystonia in rt arm markedly improved at initial &amp; second programming, medication decreased</td>
</tr>
<tr>
<td>4</td>
<td>12.5</td>
<td>10</td>
<td>immediately after DBS placement</td>
<td>immediate postop exacerbation of dystonia &amp; possible seizure activity; tip of the stimulator mildly anterior &amp; lateral to the subthalamic nucleus, rt side basal ganglia atrophy (CT scan); no acute changes</td>
<td>improved baseline tone but has periodic events of painful worsening tone</td>
</tr>
<tr>
<td>5</td>
<td>13.5</td>
<td>10</td>
<td>immediately after DBS placement</td>
<td>DBS electrode tip in bilateral subthalamic nucleus (CT scan); no acute changes</td>
<td>increased dysphagia, dysarthria, &amp; baseline hyperkinesia after discharge home, lt basal ganglia infarct &amp; lt lead breakage &amp; rt basal ganglia fluid collection</td>
</tr>
<tr>
<td>6</td>
<td>12.6</td>
<td>10</td>
<td>immediately after DBS placement</td>
<td>electrodes placed in the location of the inferior basal ganglia (CT scan)</td>
<td>initially reduced tics &amp; calmer, but recurrent nearly constant motor &amp; phonic tics after discharge</td>
</tr>
</tbody>
</table>