Deep brain stimulation was approved by public health insurance in Japan for the treatment of tremor more than 10 years ago, and approximately 4000 Japanese patients have since undergone implantation of DBS units for the treatment of chronic movement disorders refractory to pharmacotherapy. Most neural circuit disorders that can be treated with DBS are chronic or slowly progressive movement disorders. Therefore, patients need to be provided with optimal stimulation parameters suitable for relief from neurological symptoms along the course of disease progression.

Open and/or short circuits are frequent causes of DBS unit malfunction. However, the details of short circuits, particularly their incidence, causes, and clinical effects on the DBS therapy, are not fully understood. Lead fracture is one of the most frequent hardware-related complications of DBS, and it can result in open or short circuits.1,3,6,7,13,14,16,18,20,25–28,30 The term “lead fracture” in most previous studies includes the breakage of leads, extension cables, their inner wires, and/or insulators.1,3,4,6,9–11,13,15,16,18,20,25–28,30 The electrical condition of a DBS system can be checked by routinely reviewing circuit impedances while implanting hardware. However, DBS systems are often not investigated unless unfavorable clinical symptoms are observed. Short circuits can also occur when tissue fluid invades connections between the lead and extension wire due to a laceration or loose connector boot ligature.7 Most previous studies refer to open circuits

Object. The authors undertook this study to investigate the incidence, cause, and clinical influence of short circuits in patients treated with deep brain stimulation (DBS).

Methods. After the incidental identification of a short circuit during routine follow-up, the authors initiated a policy at their institution of routinely evaluating both therapeutic impedance and system impedance at every outpatient DBS follow-up visit, irrespective of the presence of symptoms suggesting possible system malfunction. This study represents a report of their findings after 1 year of this policy.

Results. Implanted DBS leads exhibiting short circuits were identified in 7 patients (8.9% of the patients seen for outpatient follow-up examinations during the 12-month study period). The mean duration from DBS lead implantation to the discovery of the short circuit was 64.7 months. The symptoms revealing short circuits included the wearing off of therapeutic effect, apraxia of eyelid opening, or dysarthria in 6 patients with Parkinson disease (PD), and dystonia deterioration in 1 patient with generalized dystonia. All DBS leads with short circuits had been anchored to the cranium using titanium miniplates. Altering electrode settings resulted in clinical improvement in the 2 PD cases in which patients had specific symptoms of short circuits (2.5%) but not in the other 4 cases. The patient with dystonia underwent repositioning and replacement of a lead because the previous lead was located too anteriorly, but did not experience symptom improvement.

Conclusions. In contrast to the sudden loss of clinical efficacy of DBS caused by an open circuit, short circuits may arise due to a gradual decrease in impedance, causing the insidious development of neurological symptoms via limited or extended potential fields as well as shortened battery longevity. The incidence of short circuits in DBS may be higher than previously thought, especially in cases in which DBS leads are anchored with miniplates. The circuit impedance of DBS should be routinely checked, even after a long history of DBS therapy, especially in cases of miniplate anchoring.

Abbreviations used in this paper: DBS = deep brain stimulation; GPi = globus pallidus internus; MER = microelectrode recording; PD = Parkinson disease; STN = subthalamic nucleus.
as lead fractures. Few have examined short circuits specifically.\textsuperscript{1,25,27} In the current study, we report on 7 patients with short circuits in implanted DBS units (without open circuits) identified within a single year.

**Methods**

This study was approved by the ethics committee of Kaizuka Hospital. Since the first case in which thalamic DBS was used for the treatment of essential tremor in July 1996 until March 2011, a total of 1090 leads have been implanted in Kaizuka Hospital in 721 patients; in 369 of these patients, leads were placed bilaterally (Fig. 1). Between April 1999 and May 2010, implantation surgeries for DBS were performed using single-track MER with a 4-mm craniotomy made with a twist drill. A total of 816 DBS leads were anchored with titanium miniplates, as previously described.\textsuperscript{17,22} In this method, connections between leads and extension cables are placed in the parietal region, dorsal to the superior temporal line (Fig. 2). Since June 2010, the single-track MER method has been replaced with a multi-track MER method\textsuperscript{12,23} using a bur hole, because of superior clinical results. In this new method, dual-floor bur holes are used,\textsuperscript{29} and the leads are anchored with standard bur hole caps included with DBS kits. A routine examination of system impedance among DBS circuits is performed just after connecting the DBS lead (model 3387 or 3389, Medtronic), the extension cable, and the pulse generator (Itrel II or Soletra, model 7424 or 7426, Medtronic) during surgery. Medtronic technical manuals for the Soletra system indicate that short circuits are suggested by an impedance measurement of less than 50 $\Omega$ with a current greater than 250 $\mu$A in a given pair of bipolar configurations among 4 contacts and that open circuits are suggested by an impedance measurement greater than 2000 $\Omega$ with a current less than 7 $\mu$A in a unipolar configuration (that is, with the pulse generator case as an anode and 1 intracerebral contact as a cathode) under conditions of 1.0 V, 210 $\mu$sec pulse width, and 30 Hz frequency.

The first postoperative programming is performed using unipolar stimulation in each patient. The stimulation parameters are reevaluated and adjusted as required at ambulatory visits. When symptoms such as dystartria or tetanic muscle contraction are suspected to be caused by current diffusion into neighboring fibers or nuclei, bipolar stimulation is introduced to focus the therapeutic potential field.

Since the incidental discovery of the first short-circuit case (Case 1 in the present study), therapeutic impedance and system impedances have been examined at every outpatient visit. This study reports on the details of the cases that were identified between June 2010 and May 2011.

**Results**

Of 79 patients who visited Kaizuka Hospital for postoperative follow-up during a single year (June 2010...
Short circuit in DBS

through May 2011), 7 were found to have a short circuit in their DBS system (6 with PD and 1 with generalized dystonia; mean age 64.0 years). These patients were included in the present study. The incidence of short circuits was 8.9% of DBS patients per year and 0.85% of all implanted leads. Patient information for cases of short circuits is summarized in Table 1.

At the time of implantation surgery, the system impedances in the 7 patients were within the normal range, and no open or short circuits were listed in their medical records. These patients exhibited favorable surgical outcomes at the time of discharge and had been followed up as outpatients. Examination of system impedance during follow-up revealed delayed development of short circuits (very low impedance at a certain bipolar pair of contacts). Even in these cases, however, no open circuits were found with any unipolar electrode configuration among 4 contacts. Radiographic examination did not detect any gap or interruption in either the extension cables or DBS lead. However, miniplate anchoring of the DBS lead to the cranium was detected in all leads with short circuits. The mean duration from implantation of DBS to the identification of a short circuit was 64.7 months (range 9–144 months). The symptoms suggesting the presence of short circuits included wearing off of levodopa effect, apraxia of eyelid opening, gait freezing, dysarthria, and sensory shock in the patients with PD and shortened battery longevity in the patient with dystonia. According to their medical records, all 6 PD patients underwent DBS surgery with single-track MER with a craniotomy made with a twist drill, and all DBS leads were anchored with titanium miniplates. Alteration of the contact polarity resulted in alleviation of symptoms in 2 cases (Cases 2 and 4 in Table 1). In the other 4 cases (Cases 1, 3, 5, and 6), the symptoms remained unchanged. In Case 7, MRI revealed that the lead was located in too anterolateral a position, suggesting stimulation of the globus pallidus externus. The lead was replaced and repositioned in the GPi with moderate alleviation of the patient’s dystonia. Thus, the incidence of symptomatic cases with short circuits was 2.5% of DBS patients per year. The clinical courses of Cases 2 and 4 (symptomatic cases) and 5 (asymptomatic case) are detailed in the following section.

Illustrative Cases

Case 2

This 65-year-old man had developed parkinsonism with a limp in his right leg at the age of 45. At the age of 60, he was treated with bilateral STN DBS, and motor fluctuation resulting from long-term levodopa replacement therapy was markedly reduced. During the 5 years before the short circuit was identified, the patient experienced wearing off of therapeutic effect and freezing and propulsive gait during the off phase of medication, causing him to visit our hospital. The patient also exhibited marked dysarthria and drooling. The patient’s left DBS implant delivered bipolar stimulation (3.1 V, pulse width

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Diagnosis &amp; DBS Target</th>
<th>FU (mos)</th>
<th>Cue Symptom†</th>
<th>Side &amp; Short Circuit</th>
<th>Impedance (drain)‡</th>
<th>Contact Polarity Setting</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>49, M</td>
<td>PD (Yahr 3); STN, bilat</td>
<td>9</td>
<td>wearing off, dyskinesia</td>
<td>rt, 2-3</td>
<td>&lt;50 Ω (294 μA)</td>
<td>1-2+ 1-case+ (no change)</td>
</tr>
<tr>
<td>2</td>
<td>65, M</td>
<td>PD (Yahr 3); STN, bilat</td>
<td>66</td>
<td>wearing off, gait freezing, drooling</td>
<td>lt, 0-3</td>
<td>&lt;50 Ω (284 μA)</td>
<td>1-3+ 1-2-case+ (freezing alleviated)</td>
</tr>
<tr>
<td>3</td>
<td>75, F</td>
<td>PD (Yahr 5); GPi, bilat</td>
<td>49</td>
<td>eyelid opening apraxia, neck dyskinesia</td>
<td>lt, 0-2</td>
<td>&lt;50 Ω (271 μA)</td>
<td>1-2+ 1-3+ (no change)</td>
</tr>
<tr>
<td>4</td>
<td>68, F</td>
<td>PD (Yahr 3); STN, bilat</td>
<td>49</td>
<td>dysarthria, shock at turning on DBS</td>
<td>lt, 0-1</td>
<td>&lt;50 Ω (180 μA)</td>
<td>1-3+ 2-3+ (both alleviated)</td>
</tr>
<tr>
<td>5</td>
<td>72, F</td>
<td>PD (Yahr 3); STN, bilat</td>
<td>112</td>
<td>wearing off</td>
<td>lt, 0-3; lt, 1-2</td>
<td>&lt;50 Ω (341 μA); &lt;50 Ω (341 μA)</td>
<td>1-3+ 1-3+ (no change)</td>
</tr>
<tr>
<td>6</td>
<td>62, F</td>
<td>PD (Yahr 4); STN, bilat</td>
<td>144</td>
<td>dysarthria, akinesia</td>
<td>rt, 0-3</td>
<td>&lt;50 Ω (126 μA)</td>
<td>0-2+ 1-2-3+ (no change)</td>
</tr>
<tr>
<td>7</td>
<td>57, M</td>
<td>dystonia; GPi, bilat</td>
<td>24</td>
<td>dystonia deterioration</td>
<td>lt, 0-2</td>
<td>&lt;50 Ω (232 μA)</td>
<td>1-3+ 1-3+ (no change, replaced later)</td>
</tr>
</tbody>
</table>

† Refers to symptoms that were present and might have made one suspect a short circuit. Only 2 patients (Cases 2 and 4) had specific symptoms of short circuit.
‡ Impedances and drains of every bipolar pair of contacts were examined with 210-μsec pulse width, 30-Hz frequency, and 1.0-V amplitude.
90 μsec, 160 Hz) using Contact 1 as a cathode and Contact 3 as an anode. The therapy impedance was 794 Ω with 61 μA. Examination of system impedance revealed a short circuit between Contacts 3 and 0 (< 50 Ω and 284 μA). Contact 0 was 6.5 mm under the midcommissural plane, suggesting a location in the ventral area of the STN. Altering the electrode setting to a unipolar stimulation using Contacts 1 and 2 as cathodes (3.0 V, pulse width 60 μsec, 130 Hz), led to the subjective alleviation of freezing and propulsion, but the sense of wearing off was not subjectively altered. These findings suggested that a short circuit between Contacts 3 (anode) and 0 unexpectedly had limited the spread of the therapeutic potential field around Contact 1 (cathode).

Case 4

This 68-year-old woman had exhibited parkinsonism from the age of 52. At the age of 58, she gradually developed marked anterolateral spinal flexion (camptocormia), which did not respond to levodopa replacement therapy. The patient underwent left GPi DBS and right posteroventral pallidotomy, but her camptocormia was only transiently alleviated. At the age of 64, the left GPi DBS was replaced with bilateral STN DBS. Since then, she had been treated by another physician with bipolar stimulation of Contact 1 as a cathode and Contact 3 as an anode, bilaterally. When the patient visited our hospital after a 4-year interval, she complained of gradual deterioration of dysarthria and sensory shock of the left hemibody in both activating and inactivating the right DBS (3.5 V, pulse width 90 μsec, 130 Hz). The therapy impedance was 1345 Ω with 36 μA. Examination of system impedance revealed a short circuit between Contacts 0 and 1 (< 50 Ω and 180 μA). Switching the cathode from Contact 1 to Contact 2 markedly diminished her dysarthria, with an amplitude of 3.0 V. Sensory shock when turning DBS on and off totally disappeared. These findings suggested that when Contact 1 was used, the right lowermost contact (0) unexpectedly functioned as a cathode as a result of the short circuit, extending the therapeutic potential field posteriorly and inferiorly to the lemniscus medialis and substantia nigra, causing uncomfortable sensory shock and dysarthria, respectively.

Case 5

This 72-year-old woman had developed parkinsonism with tremor in her left hand at the age of 54. At the age of 63, she underwent 2-staged bilateral STN DBS unit implantation, and her motor fluctuation resulting from long-term levodopa replacement therapy was markedly reduced. During the last 3 years before the short circuit was identified, she experienced wearing off of the therapeutic effect of DBS, and her axial symptoms, including frozen gait, small-step gait, and postural instability, gradually recurred. She also experienced visual hallucinations and persecutory delusions. During this period, she had undergone frequent adjustment of the DBS parameters using the unipolar configuration of Contact 1 or 2, or the bipolar configuration of Contact 1 (cathode) and Contact 2 (anode) with no additional therapeutic benefit. Examination of the system impedance revealed double short circuits between Contacts 0 and 3 (< 50 Ω and 341 μA) and between Contacts 1 and 2 (< 50 Ω and 341 μA). The center of Contact 1 was located 6.5 mm under the midcommissural plane, suggesting that the location was in the ventral area of the STN. Further trial stimulation, including unipolar or bipolar configuration with Contact 0 or 3 as the cathode, did not produce a therapeutic effect. In this case, the double short circuits of Contacts 1–2 and 0–3 limited the therapeutic options. The patient refused replacement of the DBS lead; therefore, the electrode configuration could not be changed.

Discussion

Malfunction of DBS hardware immediately after surgery typically suggests a failure in the surgical procedure, such as a loose setscrew in the connection between the lead and extension cables or mechanical damage to the lead. On the other hand, delayed hardware malfunction suggests material fatigue, such as breakage of a lead or cable and/or laceration of the connector boot or lead insulation. The incidence of lead fracture has been reported to be 0%–13.3%.4,7,13,16,18 Most previous reports describe circuit malfunction solely as “electrode or lead fracture” in a broad sense, and the specific incidence of short circuits or open circuits has not been described in detail. Lead fracture includes wire breakage or insulation breakage or both. The wire breakage causes an open circuit in a unipolar configuration, while the insulation breakage causes a short circuit in a bipolar configuration. A wire breakage can sometimes be detected as a gap on radiographic images.4,7,24 Open circuits due to the breakage of a lead or cable result in abrupt cessation of stimulation when it involves the affected contacts. Sudden changes in symptoms and/or onset of new side effects in the absence of change in medication or stimulation settings are indicative of malfunction of DBS circuits, as is a shock-like sensation caused by palpating the hardware.9,26 However, in our Case 2 and Case 4, short circuits were not suggested in radiographic examinations and were associated with no open circuits in a unipolar configuration. In addition, patients with short circuits presented with various types of onset, including subacute, gradual, and insidious patterns, without acute deterioration of clinical symptoms. Thus some of the neurological deterioration mimicked the natural advancement of PD per se. It should be noted that short circuits in DBS hardware could manifest clinically as a gradual decrease in impedance among DBS contacts.

An accurate incidence of short circuits in DBS has not been reported. In the current study, the incidence of short circuits was 0.85% of all implanted leads, including symptomatic (0.24%) and asymptomatic (0.61%) cases. However, this rate is likely to underestimate the incidence or clinical relevance of short circuits, especially in cases in which leads are anchored with miniplates, because impedance examination was not a routine procedure in our institution until our initial discovery of a short-circuit case without an open circuit (Case 1). When system impedance was routinely reviewed at follow-up in every

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In a selected case, irrespective of symptoms, 8.9% of patients evaluated during a single year (June 2010–May 2011) were found to have short circuits (symptomatic 2.5%, asymptomatic 6.3%). Because we examined the system impedance in the patients who visited us in a single year, but not in all the implanted patients, the rate of symptomatic short circuits of “2.5% per year” cannot be compared directly with incidence rates of lead fracture reported in the literature, but should be regarded as reflective of the specific conditions of this study. A prospective systematic investigation by routine impedance monitoring in all cases should be performed to determine the true incidence of short circuits in patients treated with DBS. There may be cases of lowered impedance without associated neurological changes when system impedance is routinely reviewed in all cases, irrespective of whether there are neurological changes. If taking into account asymptomatic short-circuit cases with good DBS control, the incidence of short circuits with our former surgical technique (miniplate anchoring method) may be much higher than expected. Additionally, it is suggested that impedance change alone, without accompanying neurological problems, is not a reason to alter electrode configuration.

Short circuits may occur anywhere in the lead, connector, or extension cable and even inside the pulse generator. Since the first report of the miniplate anchoring method, many neurosurgeons have used miniplates in DBS surgery. We also used this technique in combination with twist-drill surgery with minimal incision to reduce the subgaleal bulk and the risk of skin erosion, although the manufacturer strongly recommended the use of their bur hole cap and ring. The DBS manual describes that the use of alternative anchoring methods (such as glues, cements, and surgical plates) may damage the lead, which would require surgical replacement. The causes of the short circuits were not identified for each case in this study. Lead anchoring with the miniplate was considered as one of the most probable causes of short circuits but the large number of cases using bur hole caps and long-term follow-up are needed to elucidate the contribution of miniplate anchoring for a statistical analysis. Most of the lead fractures were reported to be located proximal to the connection between the leads and the extension cables or in regions where the leads were over-tightened under the miniplates. Since June 2010, the single-track MER method has been replaced with multi-track MER with bur holes, because of superior clinical results. To reduce the bulk of a genuine bur hole cap, we use a dual-floor bur hole method and have abandoned the miniplate anchoring method.

In the presence of a short circuit, bipolar stimulation may produce unexpectedly limited effects or may induce adverse effects on the target structure and neighboring structures. The postulated stimulation fields generated by bipolar stimulation with various patterns of short circuits are shown in Fig. 3, in light of the simulation study reported by Arle et al. According to the predictions of a finite-element computer model, the clinically effective stimulation parameters would be expected to result in the activation of large-diameter myelinated axons over a volume that spreads outside the borders of the STN. In Case 4, observations suggested that dysarthria was generated...
ated by current diffusion to corticobulbar fibers anterior to the STN,\textsuperscript{3} unintentionally causing the ventral contacts to function as cathodes because of the short circuit (Table 1 and Fig. 3C), since ventral contacts are generally closer to the corticobulbar fibers than dorsal contacts.\textsuperscript{19} If a short circuit of a cathode exists beyond an anode (Fig. 3B), an actual potential field may spread in the counter direction. In Case 2, gait freezing may have resulted from a limited potential field due to the 2 anodes (on either side of the cathode) being involved in the short circuit (Table 1 and Fig. 3D). Correcting the contact polarity (addition of the dorsal contact as a cathode as well as changing to unipolar stimulation) alleviated gait freezing in Case 2. Actually, the positive and negative components of the second potential difference around DBS contacts can generate membrane depolarization and hyperpolarization in neurons. Therefore, the therapeutic effect may exhibit more complicated spatial patterns in bipolar stimulation.\textsuperscript{21}

Short circuits are not rare, particularly in outpatient examinations, as demonstrated in this study. If they depend on a mechanical stress and material fatigue in the long term, the incidence of short circuit may presumably be higher than that of open circuit. Further study is required to clarify the accurate incidence of short and open circuits separately, the relationships with anchoring methods, and their impacts on the surgical outcomes.

Conclusions

All short circuits were identified in patients whose DBS leads were anchored using a miniplate rather than the manufacturer’s standard bur hole cap. Based on the results, it was suggested that some short circuits induced unfavorable or unexpected neurological manifestations due to limited or extended potential fields. The incidence of symptomatic short circuits was 2.5% of outpatients followed up in the given study year. More meticulous and frequent examination of circuit impedance may reveal a higher incidence of short circuits, including symptomatic and asymptomatic cases. The development of short circuits is a delayed hardware complication and its manifestation may be mistaken for natural progression of disease. These examinations should be undertaken even after a long duration of DBS therapy, especially in cases with miniplate anchoring of the DBS lead.

Disclosure

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