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An Experimental Study of Deep Brain Stimulation Lead Fracture: Possible Fatigue Mechanisms and Prevention Approach

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Objectives: Lead fracture is a common and troublesome hardware-related complication in deep brain stimulation therapy. Frequent cervical movements are suspected as the main cause, but the underlying mechanisms are still unclear. We propose the integrity of the helical structure of the lead wires is important and conduct systematic experiments to demonstrate this. We aim to provide a new view on how lead fracture takes place.

Materials and Methods: Flexural fatigue tests were conducted on intact and stretched lead wires with a custom-made testing machine. Number of cycles until failure was recorded as the fatigue life, and the fracture morphology was observed under optical and scanning electron microscopes.

Results: The fatigue life of the lead wires showed dramatic decline with the severity of deformation, from 434,112 \pm 10,277 cycles for an intact specimen down to 19,435 \pm 2,622 cycles for a specimen elongated by approximately 20%. The morphology of the fractures revealed characteristic beach marks and striations indicating a fatigue failure.

Conclusion: We demonstrate that integrity of the helical structure of the wires is crucial to the fatigue performance of the lead. Although the results cannot be directly extrapolated to human subjects, they suggest a possible lead fracture mechanism. The implanted lead may undergo deformation due to large-amplitude motions (e.g., falls) and develop fracture due to the deterioration in fatigue resistance, especially when it is placed at or migrates to the neck. It may be possible to effectively protect the lead by using certain surgical techniques during implantation, such as placing the connector on the calvaria or in a drilled trough at the retroauricular region with reliable fixation.

Keywords: Deep brain stimulation, fatigue, hardware failure, lead fracture, surgical technique

Conflict of Interest: The authors reported no conflict of interest.

INTRODUCTION

Deep brain stimulation (DBS) has become a widely accepted surgical therapy in treating a variety of movement disorders, such as Parkinson's disease (PD) (1), essential tremor (2), and dystonia (3). Its application further extends to treatment for other refractory diseases, including obsessive-compulsive disorder (4), epilepsy (5), major depression (6), and, potentially, Alzheimer's disease (7). So far, more than 100,000 patients worldwide have received the therapy, and the number continues to grow rapidly.

DBS is generally considered as a permanent and lifelong treatment based on implantation of a set of foreign materials, which is under constant risk of causing or suffering damage. A variety of hardware-related complications during the long-term implantation period have been reported in the literature, including infection, lead migration or misplacement, skin erosion, and lead fracture (8–11). In different studies the incidence in patients was found to vary from 6.7% to 49% (12,13). Lead fracture is recognized as a common complication, affecting about 5% of the patients who undergo DBS surgery (14,15). Patients usually experience abrupt symptom deterioration with abnormally high electrode impedance when examined. Breaches can be visualized by X-ray inspection in some cases (16). The fractured lead needs to be replaced by surgery, which causes suffering and economic loss to the patients.

The incidence of lead fracture may significantly increase when the connector between the lead and extension cable is placed at the cervical level (14). Higher fracture rates have been observed in dystonia and essential tremor patients, as these patients may exhibit more cervical movement (17,18). In such patients, the lead constantly bears more varying stress induced by neck bending, tilting,

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or twisting in daily life. Therefore, it is suspected that cervical movements transmit flexion-extension movements, especially rotation, to the extension cable and then to the lead, causing the weaker lead to break (14). However, the details of the fracture mechanisms still remain unclear.

The DBS lead used in our study incorporates four helical wires, which endow the lead with an elongation rate of more than 150%. Hence, the lead is actually more likely to experience fatigue failure than be snapped directly. Considering that severe deformation of the wire structure has been observed in explanted broken leads (14), we suspect that the distortion of the helix played an important role in the fracture. In this study, we conducted a series of fatigue tests on deformed lead wires by prestretching and observed decline in wire fatigue life. The results demonstrate that the integrity of the helical structure of the wire is crucial to its resistance to fatigue, shedding a new light on the causes of DBS lead fracture.

MATERIALS AND METHODS

Lead Specimen

Fragments of the lead used in our clinic were used for the fatigue test. As shown in Figure 1, the specimen consisted of four helical platinum–iridium alloy (Pt-Ir) wires coated with fluoropolymer inside a polyurethane sheath. The diameter of wire was 0.1 mm and the outer diameter of the helix 0.8 mm. The sheath had an outer diameter of 1.3 mm and a wall thickness of 0.2 mm.

Flexural Fatigue Test

The specimens were tested using a custom-made cable flexural fatigue test system, as described in Figure 2. It consisted primarily of a fixer to hold the specimen, a stepping motor with a driver to provide reciprocating rotary motion, a circuit to detect wire integrity, a controller module including a microprocessor, a display panel to display experiment information and cycle counts, and an input panel to input the experiment parameters and control command.

The testing scheme was adapted from the ISO standard for the pacing lead (19). The test specimen was mounted on the fixer. Then it was driven to repeatedly bend 90° bilaterally at a rate of 2 Hz around a radius of 6 mm. A weight of 30 g was attached to the bottom of the specimen to make it bend faithfully. Intact as well as variously stretched specimens were tested, and the bending count until failure of each wire was monitored. The test stopped when all the four helical wires broke.

Fracture Morphology

After fatigue failure, the specimen was first observed under an optical microscope, and microscopic morphology of the fracture





surface was then examined using scanning electron microscopes (SEMs): a JEOL JSM-6301F model (JEOL Ltd., Tokyo, Japan) operated at 15 kV and a Hitachi S-5500 model (Hitachi High-Technologies Corporation, Tokyo, Japan) operated at 5 kV for low- and high-magnification observation respectively.

RESULTS

Fatigue Resistance Performance

As shown in Figure 3, the resistance to flexural fatigue of the helical wires declined significantly with the elongation rate of the specimen. On average, the intact wires could bear 434,113 cycles of bending without failure. When the length was stretched approximately 20%, however, the wires were much more prone to fatigue failure. The fatigue life dropped to only 19,435 cycles—more than 20-fold deterioration. From the experimental data, the relationship between the fatigue life *N* (on a logarithmic scale) and elongation rate δ could be roughly described by

$$\delta = -0.062 \times \log(N) + 0.794 \tag{1}$$

Morphology of the Fracture

Figure 4 shows the typical appearance of lead wires after fatigue fracture. Usually, the breaking points of the four wires formed a cluster. They might align roughly axially at one side of the helix (Fig. 4a) or at both sides (Fig. 4b). Because the test was not stopped until all wires broke, multisite fractures sometimes occurred on individual wires, as shown in Figure 4b. In clinical practice, usually only one or two of the four wires would be used to transmit therapeutic current, corresponding to unipolar or bipolar modes of stimulation. When lead fracture occurred, the wires would not break at the same time. Therefore, there is a chance that the inactive wires could break first, which would have no influence on the therapeutic effect of DBS. The patient would continue his daily life as always until abnormality appeared in the active wire, along with deterioration of disease symptoms. Hence, multisite fracture is also possible in reality.

The typical microscopic morphology of the fracture surfaces was observed by SEM, as shown in Figure 5. Usually, a fatigue surface can be divided into three regions (20). The crack starts at the origin and then grows slowly across the fatigue zone. As the crack grows, the remaining material bears more stress due to the reduction of area. Eventually, a point is reached where the remaining material is overstressed and develops fast rupture, resulting in the appearance of an overload zone or instantaneous zone. In Figure 5a, several radial ratchet marks that developed into axially oriented secondary cracks along the axial direction could be seen, and there were smooth areas at the edge between these ratchet marks, which indicate the existence of multiple fatigue crack origins. Characteristic beach marks were clearly observable and are more evident in the close-up in Figure 5b. Such marks result from variations in the crack growth rate induced by substantial load variations across the piece. At even higher magnification, fatigue striations could be observed, as shown in Figure 5c. Such striations are formed by successive blunting and resharpening of the crack tip and show the impact of each stress cycle experienced by the local material. The beach marks and striations provided clear evidence of fatigue crack growth. Fast rupture of the central instantaneous zone resulted in a rugged surface, as shown in Figure 5d. It demonstrated ductile fracture of the Pt-Ir alloy when overloaded. The alloy had a face-centered cubic crystal structure and possessed relatively more slip systems and



Figure 2. (a) Schematic and (b) photo of the cable flexural fatigue test system.



Figure 3. Flexural fatigue test results.

more compact alignment, making it more prone to ductile fracture (21).

The ratchet marks were formed when cracks nucleated at different points joined together, while the multiple origins of cracks indicated high stress concentration at the periphery of the wire. There were more ratchet marks at the intrados of the helix, reflecting higher concentration of stress therein. The radial orientation of the ratchet marks and the shape of the instantaneous zone demonstrated that there were combined loads on the wire. A combination of torsion and bending or tension was probably resulted from the flexural deformation of the lead due to the helical structure.

DISCUSSION

DBS is a long-term therapy. The traditional stimulator, powered by a nonrechargeable battery, can usually last for 2–5 years. Surgical replacement is needed when the battery is depleted. Meanwhile, a newly introduced rechargeable stimulator has a service life anticipated at nearly 10 years. For both kinds of battery, however, the lead is always intended for lifelong usage. It will not be removed or replaced unless the therapy is terminated or adverse events happen, such as lead fracture.

Lead fracture is recognized as a common hardware-related complication in clinical practice, and the incidence reported varies between different studies, ranging between 0% and 17.9% in implanted leads (8,9,12,14,15,18,22–33). The majority of diagnoses occur between 6 and 24 months after implantation (8,12,33). Nevertheless, lead fracture is, on average, detected after a longer period at about 36 months, with a range between 7 and 84 months or even longer (14,18). Generally, it is reported to affect 1.1% of patients (34), with the rate increasing to about 5% in the long term (12,15,29).

If the patient encounters abrupt change in symptoms and/or new onset of side effects in the absence of change in medication or stimulation settings, then there is a possibility of lead fracture. An assessment protocol has been proposed by Farris et al., including inquiry regarding relevant events, electrode impedance and current measurement, X-ray inspection, palpation, and so on (16). Rupture in the lead wires usually leads to measurements of abnormally high electrode impedance and very low current and might be observed in radiological studies. In some cases, impedance and current measurements might vary significantly in different body positions, usually associated with an intermittent pattern of stimulation. This is probably because only a microfracture is developed at the time and the two poles of the open circuit can still connect temporarily when altering neck or head positions (16,18). Shock sensations or electrical paresthesia along the trajectory of the wires might also be felt (16,29).

The lead is reported to be more vulnerable when the connector between the lead and the extension cable is placed at the neck, below the mastoid (12,14). The incidence of lead fracture can reach 16.7% in patients in whom this is the case (12). A majority of the breakage occurs at a distance of approximately 10.7 mm from the connector (14). It is also reported that patients with cervical dystonia (17,30), dyskinesia (9), or essential tremor (12,18) bear a higher risk of lead fracture. In these patients, more frequent and vigorous cervical movements are expected if the symptoms are not completely controlled by the therapy. It is plausible that the cervical movements impose continually varying load on the lead and greatly accelerate the fatigue failure of the wires. However, lead fracture is also possible when the connector is placed on the calvaria. Some leads are crushed by the fixing miniplate (11,17,35), while others are damaged due to vigorous scalp massage treatments (25) or head trauma (31). In some cases, the lead is originally placed on the scalp or in the retroauricular region but later migrates to the neck due to falls or body movements, and lead fracture follows (9,11,28,30).

Although movements can impose a higher level of stress on the lead, however, it is noticeable that only a small proportion of leads encounter breakage in the long run, even if the connector is placed at the neck (14). The lead wires adopt a helical structure that has a higher tolerance of stress. In our flexural tests the wires had a fatigue



Figure 4. Optical images showing the fractures of the samples that were (a) stretched 4% and (b) stretched 19%. Dotted circles mark the breaking points.



Figure 5. Scanning electron micrographs. (a) Typical fracture surface. Image obtained with the JSM-6301F. (b) Magnified view of the fatigue zone showing the beach marks. Image obtained with the JSM-6301F. (c) Higher-magnification image obtained with the S-5500 showing the fatigue striations in the fatigue zone. (d) Magnified view of the instantaneous zone. Image obtained with the JSM-6301F.

life of more than 400,000 cycles, suggesting them to be capable of surviving the normal level of stress in daily life for a rather long time. However, once the helical structure was stretched, the resistance to fatigue of the wires dropped dramatically. As shown in Figure 3, decline of the fatigue life had roughly an exponential relation to the elongation rate. The fatigue life could drop approximately 20-fold after deformation by a rate of slightly less than 20%. Note that due to the complexity of the in vivo environment, these results cannot be directly extrapolated to human subjects. Nevertheless, they suggest a possible mechanism, as severe deformation in the lead wires was observed in the explanted fractured lead (14). The lead might come to rupture due to the deterioration of fatigue

resistance resulted from deformation of the helix. There are a variety of situations that could result in deformation of the helical wires, such as falls or vigorous sports (16).

Daily activities may impose complex movements on the lead passing through the neck because of the diversity of lead path or configuration. Various kinds of distortion of the lead wires were observed in clinic, including stretching, twisting and bending, and could be relevant to the breakage (29). Fernandez et al. proposed that the flexion–extension and particularly the rotational movements induced by cervical movements are transmitted to the lead and finally cause fracture (14). However, no further examinations, such as of the microscopic morphology of the rupture surface, were



Figure 6. Demonstration of a surgical technique to handle the connector during implantation by placing it in a drilled bone trough behind the ear and fixing with titanium plates.

reported. Thus, it was still impossible to verify which cause dominated.

In order to lower the risk of lead fracture, patients may want to avoid participating in activities that involve large-amplitude motion, such as vigorous sports. Nevertheless, proper management of the connector during implantation to prevent the lead from bearing load can be more effective. The extension cable is much more robust than the lead. It possesses not only much higher strength but also a fatigue life more than two orders of magnitude longer. Thus, it readily survives the stress of daily activities and rarely fails (26,27,30). Placing the connector high on the head is proposed (22,36). Clinical retrospective studies have reported that lead fracture is rare with this technique (12,30,37). This procedure might increase the incidence of infection or skin erosion if the connector is too bulky (14); however, this can be avoided by lowering the profile of the connector and/or using specific surgical techniques, such as drilling a trough (9,38) or avoiding putting the connector directly under the suture line (28). Another method is to place the connector in the retroauricular region (11,28). Proper anchoring is necessary to prevent it from sliding downward, for example by suturing the inferior necking-down part of the connector to the fascia (11). A more reliable way is to drill a trough and to further apply a fixation device such as a miniplate, as shown in Figure 6. This method can enhance fixation as well as diminish the height of the connector so as to reduce the incidence of infection or skin erosion. Note that when applying a miniplate, care should be taken to secure but not crush the lead or the extension (8).

As discussed above, surgical technique is important in lowering the incidence of lead fracture. Avoiding placing the connector below the mastoid and using a reliable fixation method to prevent migration are considered the key issues (11,12). Surgical techniques play an important role in reducing other hardware-related complications (e.g., erosion) as well (10). However, a large diversity of these techniques can still be seen in the literature. Also, many studies have disclosed the existence of a learning curve (18,22,23,26,39). Therefore, we propose that there is still a need to further enhance communications in the field and to make a joint effort to work out a consensus on standard procedures.

CONCLUSION

We demonstrated the importance of the helical structure of DBS lead wires in preserving their fatigue resistance. The fatigue life showed severe decline when the helix was deformed. Fatigue failure characteristics were revealed in morphology examination, which could be further referred to in the analysis of the explanted leads. The results might shed a new light on how such leads develop rupture. It is likely that large-amplitude motion damages the integrity of the helix and leads to deterioration in fatigue performance, and then frequent movements cause the fatigue failure of the lead. This gives a theoretical explanation of why more attention should be paid to surgical technique so as to prevent the lead from bearing load and thus avoid lead deformation and fracture.

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Authorship Statement

Dr. Changqing Jiang conducted the study, and prepared the manuscript with direction of Dr. Luming Li. Mr. Xiaolong Mo helped to build the testing system. Dr. Fangang Meng provided important information about the proposed surgical technique. Mr. Yantao Dong, Dr. Hongwei Hao, Dr. Xiqiao Feng, and Dr. Luming Li reviewed the manuscript. All the authors approved the manuscript.

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REFERENCES

- Krack P, Batir A, Van Blercom N et al. Five-year follow-up of bilateral stimulation of the subthalamic nucleus in advanced Parkinson's disease. N Engl J Med 2003;349:1925–1934.
- Schuurman PR, Bosch DA, Bossuyt P et al. A comparison of continuous thalamic stimulation and thalamotomy for suppression of severe tremor. N Engl J Med 2000;342:461–468.
- Vidailhet M, Vercueil L, Houeto JL et al. Bilateral deep-brain stimulation of the globus pallidus in primary generalized dystonia. N Engl J Med 2005;352:459–467.
- Greenberg BD, Malone DA, Friehs GM et al. Three-year outcomes in deep brain stimulation for highly resistant obsessive-compulsive disorder. *Neuropsychopharmacology* 2006;31:2384–2393.
- Hodaie M, Wennberg RA, Dostrovsky JO, Lozano AM. Chronic anterior thalamus stimulation for intractable epilepsy. *Epilepsia* 2002;43:603–608.
- Mayberg HS, Lozano AM, Voon V et al. Deep brain stimulation for treatmentresistant depression. *Neuron* 2005;45:651–660.
- 7. Bible E. Alzheimer disease: enhanced functional connectivity in AD after deep brain stimulation. *Nat Rev Neurol* 2012;8:356.
- Joint C, Nandi D, Parkin S, Gregory R, Aziz T. Hardware-related problems of deep brain stimulation. *Mov Disord* 2002;173:S175–S180.
- Oh MY, Abosch A, Kim SH, Lang AE, Lozano AM. Long-term hardware-related complications of deep brain stimulation. *Neurosurgery* 2002;50:1268–1276.
- Baizabal Carvallo JF, Simpson R, Jankovic J. Diagnosis and treatment of complications related to deep brain stimulation hardware. *Mov Disord* 2011;26:1398–1406.
- 11. Chan D, Zhu XL, Yeung J et al. Complications of deep brain stimulation: a collective review. *Asian J Surg* 2009;32:258–263.
- 12. Blomstedt P, Hariz MI. Hardware-related complications of deep brain stimulation: a ten year experience. *Acta Neurochir* 2005;147:1061–1064.
- Zhang K, Bhatia S, Oh MY, Cohen D, Angle C, Whiting D. Long-term results of thalamic deep brain stimulation for essential tremor. J Neurosurg 2010;112:1271–1276.
- Fernandez FS, Vega M, Ramos AA, Gonzalez FF, Aragoneses BL. Lead fractures in deep brain stimulation during long-term follow-up. *Parkinsons Dis* 2010;2010: 409356.

- Boviatsis EJ, Stavrinou LC, Themistocleous M, Kouyialis AT, Sakas DE. Surgical and hardware complications of deep brain stimulation. A seven-year experience and review of the literature. *Acta Neurochir* 2010;152:2053–2062.
- Farris S, Vitek J, Giroux ML. Deep brain stimulation hardware complications: the role of electrode impedance and current measurements. *Mov Disord* 2008;23:755–760.
- 17. Yianni J, Nandi D, Shad A, Bain P, Gregory R, Aziz T. Increased risk of lead fracture and migration in dystonia compared with other movement disorders following deep brain stimulation. *J Clin Neurosci* 2004;11:243–245.
- Carvallo JFB, Mostile G, Almaguer M, Davidson A, Simpson R, Jankovic J. Deep brain stimulation hardware complications in patients with movement disorders: risk factors and clinical correlations. *Stereotact Funct Neurosurg* 2012;90:300–306.
- International Organization for Standardization. Implants for surgery: active implantable medical devices. Part 2: cardiac pacemakers. 2005. ISO 14708-2:2005.
- 20. Campbell FC. *Fatigue and fracture: understanding the basics*. Materials Park, OH: ASM International, 2012.
- 21. Raykhtsaum G. Platinum alloys: a selective review of the available literature. *Platinum Met Rev* 2013;57:202–213.
- Kondziolka D, Whiting D, Germanwala A, Oh M. Hardware-related complications after placement of thalamic deep brain stimulator systems. *Stereotact Funct Neurosurg* 2002;79:228–233.
- Lyons KE, Wilkinson SB, Overman J, Pahwa R. Surgical and hardware complications of subthalamic stimulation – a series of 160 procedures. *Neurology* 2004;63:612– 616.
- Constantoyannis C, Berk C, Honey CR, Mendez I, Brownstone RM. Reducing hardware-related complications of deep brain stimulation. *Can J Neurol Sci* 2005; 32:194–200.
- 25. Goodman RR, Kim B, McClelland S et al. Operative techniques and morbidity with subthalamic nucleus deep brain stimulation in 100 consecutive patients with advanced Parkinson's disease. J Neurol Neurosurg Psychiatry 2006;77:12–17.
- Voges J, Waerzeggers Y, Maarouf M et al. Deep-brain stimulation: long-term analysis of complications caused by hardware and surgery – experiences from a single centre. J Neurol Neurosurg Psychiatry 2006;77:868–872.

- Vergani F, Landi A, Pirillo D, Cilia R, Antonini A, Sganzerla EP. Surgical, medical, and hardware adverse events in a series of 141 patients undergoing subthalamic deep brain stimulation for Parkinson disease. *World Neurosurg* 2010;73:338–344.
- Falowski S, Ooi YC, Smith A, Metman LV, Bakay RAE. An evaluation of hardware and surgical complications with deep brain stimulation based on diagnosis and lead location. *Stereotact Funct Neurosurg* 2012;90:173–180.
- Guridi J, Rodriguez-Oroz MC, Alegre M, Obeso JA. Hardware complications in deep brain stimulation: electrode impedance and loss of clinical benefit. *Parkinsonism Relat Disord* 2012;18:765–769.
- Panov F, Gologorsky Y, Connors G, Tagliati M, Miravite J, Alterman RL. Deep brain stimulation in DYT1 dystonia: a 10-year experience. *Neurosurgery* 2013;73:86–93.
- Petrossian MT, Paul LR, Multhaupt-Buell TJ et al. Pallidal deep brain stimulation for dystonia: a case series clinical article. J Neurosurg Pediatr 2013;12:582–587.
- Fenoy AJ Jr, Simpson RK. Risks of common complications in deep brain stimulation surgery: management and avoidance. J Neurosurg 2014;120:132–139.
- Paluzzi A, Belli A, Bain P, Liu X, Aziz TM. Operative and hardware complications of deep brain stimulation for movement disorders. Br J Neurosurg 2006;20:290–295.
- Hamani C, Richter E, Schwalb JM, Lozano AM. Bilateral subthalamic nucleus stimulation for Parkinson's disease: a systematic review the clinical literature. *Neurosur*gery 2005;56:1313–1321.
- Samura K, Miyagi Y, Okamoto T et al. Short circuit in deep brain stimulation clinical article. J Neurosurg 2012;117:955–961.
- Schwalb JM, Riina HA, Skolnick B, Jaggi JL, Simuni T, Baltuch GH. Revision of deep brain stimulator for tremor. Technical note. J Neurosurg 2001;94:1010–1012.
- Allert N, Markou M, Miskiewicz AA, Nolden L, Karbe H. Electrode dysfunctions in patients with deep brain stimulation: a clinical retrospective study. *Acta Neurochir* 2011;153:2343–2349.
- Hu X, Jiang X, Zhou X et al. Avoidance and management of surgical and hardwarerelated complications of deep brain stimulation. *Stereotact Funct Neurosurg* 2010;88:296–303.
- Doshi PK. Long-term surgical and hardware-related complications of deep brain stimulation. Stereotact Funct Neurosurg 2011;89:89–95.