Elevated Mood States in Patients With Parkinson's Disease Treated With Deep Brain Stimulation: Diagnosis and Management Strategies

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Objective: Deep brain stimulation (DBS) is an effective surgical treatment for patients with Parkinson's disease (PD). DBS therapy, particularly with the subthalamic nucleus (STN) target, has been linked to rare psychiatric complications, including depression, impulsivity, irritability, and suicidality. Stimulation-induced elevated mood states can also occur. These episodes rarely meet DSM-5 criteria for mania or hypomania.

Methods: The investigators conducted a chart review of 82 patients with PD treated with DBS.

Results: Nine (11%) patients developed stimulation-induced elevated mood. Five illustrative cases are described (all males with STN DBS; mean age=62.2 years [SD=10.5], mean PD duration=8.6 years [SD=1.6]). Elevated mood states occurred during or shortly after programming changes, when more ventral contacts were used (typically in monopolar mode) and lasted minutes to months. Four patients experienced elevated mood at low amplitudes (1.0 V/1.0 mA); all had psychiatric risk factors (history of impulse-control disorder, dopamine dysregulation syndrome, substance use disorder, and/or bipolar diathesis) that likely contributed to mood destabilization.

Conclusions: Preoperative DBS evaluations should include a thorough assessment of psychiatric risk factors. The term "stimulation-induced elevated mood states" is proposed to describe episodes of elevated, expansive, or irritable mood and psychomotor agitation that occur during or shortly after DBS programming changes and may be associated with increased goal-directed activity, impulsivity, grandiosity, pressured speech, flight of ideas, or decreased need for sleep and may persist beyond stimulation adjustments. This clinical phenomenon should be considered for inclusion in the bipolar disorder category in future DSM revisions, allowing for increased recognition and appropriate management.

J Neuropsychiatry Clin Neurosci 2021; 33:314–320;
doi: 10.1176/appi.neuropsych.20080205

Deep brain stimulation (DBS) is an effective surgical treatment strategy for movement disorders including dystonia, essential tremor, and Parkinson's disease (PD). The cardinal motor symptoms of PD (resting tremor, rigidity, and bradykinesia) respond well to DBS, whereas other motor and nonmotor symptoms have variable improvement (1, 2). Rarely, DBS therapy can worsen nonmotor symptoms of PD or produce psychiatric complications, including mood dysregulation. Acute mood changes can occur during DBS programming sessions, ranging from uncontrollable crying to mirthful laughter (3–5). Longer lasting anxiety, anger, or depressive episodes can also manifest postoperatively, with both subthalamic nucleus (STN) and globus pallidus interna (GPi) targets (2, 6–15). The STN has been more frequently implicated than the GPi. Among patients treated with STN DBS, 4%−15% were reported to develop hypomania or mania (using the authors' original terms), often in the first 3 months after surgery (6). These mood states are typically transient and resolve with DBS setting adjustments; infrequently, they can persist, requiring pharmacological intervention (11–13).

There is no uniformly agreed upon term in the current psychiatric nomenclature to describe elevated mood states associated with DBS therapy. We propose the term "stimulation-induced elevated mood states" to describe behavioral changes consisting of elevated, expansive, or irritable mood and psychomotor agitation that occur during or shortly after DBS programming changes and may be associated with increased goal-directed activity, impulsivity,

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grandiosity, hypersexuality, pressured speech, flight of ideas, or decreased need for sleep. These episodes can also be associated with distractibility, inattention, and poor judgment, as the following case vignettes illustrate. Here we describe five patients who experienced stimulation-induced elevated mood states.

METHODS

This study was approved by the institutional review board at the University of California at San Francisco (UCSF) and consisted of a chart review of all patients with PD who were treated with DBS and followed at the UCSF Movement Disorders and Neuromodulation Center and who were evaluated by the center's psychiatrist between 2015 and 2019 and provided signed research consent. The diagnosis of PD was made by experienced neurologists, on the basis of established clinical criteria. No red flags for atypical parkinsonian syndromes were present. Clinical and demographic factors collected included patients' age, sex, PD duration, and DBS settings; time after surgery when the elevated mood occurred; preoperative and postoperative L-dopa equivalent daily dose; psychiatric personal and family history; and length of follow-up (time from the first elevated mood episode to the most recent neurological visit). Patients' preoperative and postoperative medications are listed in Table S1 in the online supplement.

DBS stimulation parameters include amplitude, measured either as current (mA) or voltage (V); pulse width, indicating duration of each stimulus (measured in μs); and frequency (number of pulses per second, Hz). Monopolar stimulation (where the battery case is positively charged and one contact is negatively charged) produces a higher volume of tissue activated around the contact in a roughly spherical shape, whereas bipolar stimulation (one positive and one negative contact on the same lead) creates a more ellipsoid field, with less electrical current spread (16).

RESULTS

Eighty-two patients were included in the chart review. Nine patients (11%) developed stimulation-induced elevated mood states. Of these, five illustrative cases are included in this study (all males with STN DBS; mean age=62.2 years [SD=10.5]; mean PD duration=8.6 years [SD=1.6]; follow-up period=40 months [SD=25.4]). Four patients were implanted with Medtronic DBS lead model 3389, and one patient (case 2) received a Boston Scientific Intrepid system. DBS leads were implanted under microelectrode recording guidance; postoperative imaging studies confirmed adequate placement in all cases. Elevated mood symptoms lasted 30 minutes to 6 months; in four of the patients, they recurred each time the same contacts were used. Table 1 summarizes the five patients' clinical and demographic characteristics and DBS settings associated with elevated mood.

Case 1

"Mr. A" was a 73-year-old man with no previous psychiatric history who underwent staged bilateral STN DBS for left-predominant tremor and rigidity with motor fluctuations. His right STN was implanted with excellent motoric response, followed by left STN 5 months later.

During a programming session 16 months after right STN surgery, the voltage on contact 1 was raised from 2.7 to 3.5 V. Prior to this, Mr. A had described his mood as calm and serene and had minimal spontaneous speech. Approximately 15 minutes after voltage was raised, Mr. A exhibited psychomotor agitation and became very talkative. Stimulation was lowered back to 2.7 V, and symptoms resolved. Two weeks later, when the same settings were trialed in clinic, elevated mood recurred: Mr. A reported feeling euphoric, paced around the office, crouched in a corner, and grabbed a female examiner's leg. He walked backward and waved his arms as if conducting an orchestra during the neurological examination. Symptoms again resolved with DBS amplitude reduction.

Case 2

"Mr. B" was a 47-year-old man who underwent bilateral STN DBS for left-side predominant symptoms, motor fluctuations, freezing of gait, and dyskinesia. He had a history of dopamine dysregulation syndrome (manifested by compulsive use of dopaminergic medications), which had resolved 1 year previously, and a family history of bipolar disorder.

On postoperative day 2, Mr. B exhibited transient disinhibited behavior and lack of empathy. Stimulation was initiated after 3 weeks. Twelve weeks later, the patient reported he achieved best motor control in a monopolar configuration, although he was also more impulsive, irritable, and anxious, and talked faster with this setting. Medications were reduced and programming changes were made. At the 1-year follow-up, the patient's family reported that he appeared to be "revved up" at times, particularly with one specific DBS configuration. He described episodes lasting 10–15 minutes when he became impulsive and irritable, talked faster than usual, and had trouble focusing. Two of these episodes were associated with panic attacks. These symptoms occurred when he temporarily increased DBS amplitude (usually before physical exercise) and resolved when he lowered it. Mr. B also shared he was taking a lot more medication than prescribed. Of note, the revved-up episodes only occurred when he increased stimulation.

Mr. B's treatment included close psychiatric follow-up, with a behavioral intervention targeting dopamine dysregulation syndrome (keeping daily logs of the exact medication doses and times when he was taking them, taking medication no more than eight times daily, using a pill box, and involving his wife to ensure accountability) and supportive therapy. Dopaminergic medications were reduced. Over time, both the elevated mood and dopamine dysregulation syndrome symptoms resolved. Five years after surgery, the patient's mood remained stable, and his motor symptoms were overall well controlled.

TABLE 1. Clinical and demographic characteristics and deep brain stimulation (DBS) settings associated with elevated mood episodes among patients with Parkinson's disease (PD) (N=5)[a]

Case number	Age (years)	PD duration (years)	Preoperative psychiatric history	Family psychiatric history	LEDD reduction (mg)	DBS settings[b]		Time after surgery[c]	Elevated mood episode duration	Follow-up period[d]
						Left	Right			
1	73	7	Mild impulse-control disorder symptoms, mild NCD	None	818	C+,1−,2−; 2.5 V; 60 μs; 185 Hz	**Interleaved STN1: C+,1−; 3.5 V; 60 μs; 125 Hz** STN2: C+,3−; 2.9 V; 60 μs; 125 Hz	16 months post-right STN implantation; recurred 2 weeks later (same settings)	30 minutes	2 weeks
2[e]	47	11	Dopamine dysregulation syndrome	Bipolar disorder	493–544	**Monopolar C+,2−; 1.5 mA; 60 μs; 130 Hz** Bipolar **2−(15%),3−(85%),4+; 4.0 mA; 40 μs; 130 Hz**	**Monopolar C+,11−; 1.5 mA; 60 μs; 130Hz** Bipolar **10−(15%),11−(85%),12+; 3.8 mA; 40 μs; 130 Hz**	4 months Recurred 7 months later	Brief (intermittent) 10–15 minutes (intermittent)	56 months
3	53	7	Mild NCD, depressive symptoms	Depression, bipolar disorder, substance use disorder	0	**Monopolar C+,1−; 2.0 V; 60 μs; 140 Hz** and **Bipolar 2+,1−; 2.0 V; 60 μs; 130 Hz**	C+,0−; 2.6 V; 60 μs; 155 Hz	4 months	3 months	60 months
4	65	10	Impulse-control disorder, mild NCD, depressive symptoms	Anxiety disorder	627	C+,1−,2−; 3.0 V; 60 μs; 200 Hz	**Monopolar C+,1−; 1.0 V; 60 μs; 150 Hz**	1 month	2 hours	64 months
5	73	8	Major depressive disorder, substance use, in full sustained remission	Depression (bipolar disorder?), alcohol use disorder	150	**Monopolar C+,1−; 2.0 mA; 60 μs; 130 Hz** Same settings >3.1 mA	NA	1 month Recurred 2 months later	1 week Several hours	20 months

[a] C=neurostimulator battery case; LEDD=L-dopa equivalent daily dose; NA=not applicable (unilateral DBS); NCD=neurocognitive disorder; STN=subthalamic nucleus.

[b] Settings associated with elevated mood episodes are indicated in bold. DBS electrode configurations include monopolar (where the battery case is positively charged and one contact is negatively charged), bipolar (one positive and one negative contact on the same lead), double monopolar (case is positive, with two negative contacts), and interleaved (one lead alternates between two different settings with unique contact activation). Contacts on Medtronic lead model 3389 (cases 1, 3, 4, and 5) are numbered 0–4, with 0 being the most ventral (lower) and 4 being the most dorsal (higher) on each lead. Contacts on Boston Scientific Intrepid leads (case 2) are numbered 1–8 (left lead) and 9–16 (right lead), with 1 and 9 being the most ventral and 8 and 16 being the most dorsal.

[c] When the first elevated mood episode occurred.

[d] Time from first elevated mood episode to most recent neurological visit.

[e] The patient also had a brief episode of disinhibited behavior on postoperative day 2, before stimulation was initiated.

Case 3

"Mr. C" was a 53-year-old man with no past psychiatric history who underwent staged bilateral STN DBS surgery for left-side predominant motor symptoms. His right STN was implanted initially, followed by the left STN 1 year later. One month after left STN implantation, stimulation was initiated in monopolar mode; however, Mr. C was unable to tolerate higher voltages due to dyskinesia. A new bipolar group was created, with instructions to slowly increase voltage at home. Four months postoperatively, the patient reported significant stimulation-induced mood changes starting shortly after surgery: he felt "easily agitated, frustrated, snappy, short-tempered, and irritable." For example, when his computer malfunctioned, he punched and broke the screen. These symptoms occurred in both monopolar and bipolar settings but were more severe in monopolar mode and with voltage >2.0 V; they resolved if the left DBS was turned off. Symptoms were addressed by using a double monopolar configuration and avoiding contact 1. Irritability persisted but ultimately improved with the addition of escitalopram 10 mg daily.

Case 4

"Mr. D" was a 65-year-old man who underwent staged bilateral STN DBS for primarily right-sided motor symptoms. He had a history of depression and dopamine agonist-induced impulse-control disorder (consisting of gambling, risky investments, and compulsive behavior surrounding household projects). Impulse-control disorder symptoms resolved after weaning off the dopamine agonist medication. The patient initially underwent left STN DBS surgery with excellent motoric response. His right STN was implanted 2 years later, after he developed left-sided symptoms.

Right STN stimulation was initiated 1 month later. Mr. D was given two monopolar groups (active contact 1− and contact 3−, respectively) to try at home. He reported best tremor control with contact 1; however, within a few hours, he developed dyskinesia and mood changes, described as "goofiness, restlessness, and acting giddy." These symptoms did not occur when using contact 3; however, his tremor was insufficiently controlled. With slow upward amplitude titration, Mr. D was able to tolerate gradually higher voltages (up to 2.9 V) using contact 1 without recurrence of elevated mood.

Case 5

"Mr. E" was a 73-year-old man who underwent left STN DBS for right-sided motor symptoms and motor fluctuations. He had a history significant for major depressive disorder with onset in his youth, with a recent recurrence after receiving the PD diagnosis. He also had history of hallucinogen use, in full sustained remission, and a family history significant for mood and alcohol use disorders; four relatives had died by suicide.

Following initial programming in a monopolar configuration, Mr. E was instructed to slowly increase the amplitude (by 0.1 mA/day) at home. Ten days later, the patient reached 2.0 mA with good motor control. However, family members reported he became "hyper," with reduced sleep and pressured speech. He was researching PD-related information online for 10–12 hours daily and believed he was going to "solve the problem of PD." He also reported flight of ideas. The DBS device was turned off for 1 week and medications were adjusted.

Over the next 8 weeks, Mr. E slowly increased stimulation to 2.8 mA but became more disorganized, impulsive, and irritable. He was reprogrammed to a bipolar configuration and DBS amplitude was reduced to 2.5 mA. Elevated mood symptoms resolved, although motor control was insufficient. On his own, Mr. E increased the amplitude and again developed pressured speech, talkativeness, and increased goal-directed activity. These symptoms occurred at amplitudes >3.1 mA and improved when the DBS was turned off. Reducing amplitude to 2.5 mA led to lasting symptom resolution.

Table 2 highlights management strategies for stimulation-induced elevated mood, specifying those used in the five patients presented here (12–15, 17, 18).

DISCUSSION

We described five men with PD treated with STN DBS who experienced stimulation-induced elevated mood states during or shortly after DBS programming changes, lasting minutes to weeks. Elevated or irritable mood was distinct from baseline and associated with psychomotor agitation, as well as a variable combination of additional symptoms. These episodes occurred after crossing a stimulation threshold, typically when using ventral contacts. In contrast to prior studies, where amplitudes >3.0 V have been noted, four of our patients experienced elevated mood symptoms at amplitudes as low as 1.0 V or 1.0 mA. Occurrence of elevated mood episodes at lower amplitudes in patients with adequately placed DBS leads suggests the presence of underlying risk factors that need to be better understood in order to anticipate and manage this potentially harmful complication of DBS therapy. Screening for risk factors for stimulation-induced elevated mood should be part of every DBS candidacy evaluation.

All four patients who developed elevated mood at lower amplitudes had psychiatric comorbidities that likely increased their sensitivity to the physiological effects of DBS, making them more prone to mood destabilization when stimulation was initiated or increased. One patient had a history of impulse-control disorder, one had concurrent dopamine dysregulation syndrome, and a third had a history of substance use; two (possibly three) also had a family history of bipolar disorder. Addictive disorders such as impulse-control disorder, dopamine dysregulation syndrome, and substance use disorders involve activation of dopaminergic reward pathways and may increase the risk of stimulation-induced elevated mood (19, 20). Although DBS can be an effective treatment for impulse-control disorder, mainly by

TABLE 2. Management strategies for stimulation-induced elevated mood states in patients with Parkinson's disease[a]

Strategy	Case number [b]
Deep brain stimulation modifications	
Stimulation adjustments	
Amplitude reduction	1, 5
Temporary discontinuation	3, 5
Slower amplitude titration	4
Reprogramming in bipolar mode	2, 5
Reprogramming in double monopolar mode	3
Reprogramming in interleaved mode	
Pulse width reduction	
Frequency reduction	
Use of more dorsal (higher) contacts	3, 4
Surgical lead repositioning (if necessary)	
Use of directional leads to steer current	
Use of adaptive approaches (in the future)	
Parkinson's disease medication management	
Reduction or discontinuation of dopamine agonist	1, 2
Discontinuation of "booster" or "rescue" medications: subcutaneous apomorphine or rapid-acting levodopa (if comorbid dopamine dysregulation syndrome)	2
Reduction of other dopaminergic medications	1, 2, 4, 5
Psychiatric management	
Exploration of use of alcohol, drugs, over-the-counter medications, or other agents (and discontinuation of any offending agent)	
Psychotherapy	
Behavioral intervention	2, 5
Motivational interviewing (for comorbid dopamine dysregulation syndrome or substance use disorder)	2
Pharmacotherapy	
Antidepressant/anxiolytic (for anxiety, depression, and impulse-control disorder symptoms)	3
Mood stabilizer (if elevated mood symptoms persist)	
Antipsychotic (if psychotic symptoms are present)	
Electroconvulsive treatment (for severe, treatment-resistant mood and/or psychotic symptoms)	

[a] For further details, see references 12–15, 17, 18.
[b] The strategy was used for the patient in the specified case number.

TABLE 3. Risk factors associated with stimulation-induced elevated mood states in patients with Parkinson's disease (PD)[a]

Risk factor	Case number[b]
Patient-related factors	
Male	1, 2, 3, 4, 5
Young onset PD	2, 3
Dopamine agonist use	1, 2, 3
Substance use (past or current)	5
Impulse-control disorder	1 (symptoms only), 4
Dopamine dysregulation syndrome	2
High burden of preoperative motor symptoms	2
High burden of preoperative psychiatric symptoms	2, 4, 5
Family history of bipolar disorder	2, 3, 5 (possible)
Stimulation-related factors	
Electrodes placed ventral to the subthalamic nucleus	None
Ventromedial contacts	1, 2, 3, 4, 5
Voltage >3 V	1
Monopolar stimulation mode	2, 3, 4, 5

[a] For further details, see references 7–11, 14, 15, 17, 25.
[b] The risk factor is reported for the patient in the specified case number.

dopaminergic agent dose reduction, STN stimulation can impair decision making and increase impulsivity (21). Three of our patients had mild cognitive deficits, which may additionally reflect dysfunction of the frontal subcortical networks. Recent studies have shown that both structural (prefrontal cortex atrophy) and functional (preoperative frontal function scores in patients undergoing STN DBS) indicators are associated with severity of neuropsychiatric symptoms in patients with PD (22, 23).

Individuals with a family history of bipolar disorder may have a bipolar diathesis—that is, a genetic vulnerability that predisposes them to mood destabilization with antidepressant treatment (medications, electroconvulsive therapy, or phototherapy). Akiskal et al. (24) coined the term "bipolar III" to describe antidepressant-induced hypomania in previously depressed individuals. In a study of approximately 500 patients, those classified as bipolar III were more likely to have a family history of bipolar disorder and completed suicide, a personal history of psychosis and suicidality, and greater chronicity and severity of depressive symptoms, compared with patients with bipolar II disorder (24). Herzog et al. described a 65-year-old woman with PD and no past psychiatric history but a family history of bipolar disorder who underwent thalamotomy for tremor, followed by bilateral STN DBS (13). This woman developed a sustained stimulation-induced episode of mania with psychosis, requiring treatment with clozapine and carbamazepine.

Risk factors for stimulation-induced elevated mood are summarized in Table 3, based on literature review and the present case series (7–11, 14, 15, 17, 25).

The STN has been classically divided into three functionally distinct territories: the dorsolateral motor territory, the ventromedial associative part, and the medial limbic territory (26). Although the precise pathophysiologic mechanism of STN stimulation-induced elevated mood is not fully elucidated, it has been postulated to result from the spread of current into neighboring areas such as the medial limbic territory or ventromedial associative region (particularly with monopolar configurations). This can lead to dysregulation of the mesolimbic dopamine circuits involved in emotional regulation, addiction, and learning (14, 15, 20, 27). Using diffusion tensor imaging, Coenen et al. (28) demonstrated recruitment of median forebrain bundle (MFB) fibers. The MFB is a crucial component of the mesolimbic dopamine system and therefore the reward circuitry; it has been

implicated in mood disorders, addictive behavior, and learning (15). Coenen et al. hypothesized that elevated mood states are caused by inadvertent MFB activation (28).

In another original investigation, Schilbach et al. (11) studied regional cerebral flow (rCBF) using positron emission tomography scan in one patient with STN DBS while activating a ventral contact (which had caused an elevated mood state) compared with using a more dorsal contact (mood-neutral). They found a differential increase of rCBF in the right dorsolateral prefrontal cortex, right middle temporal gyrus, and dorsal anterior cingulate cortex, suggesting that patients with underlying psychiatric vulnerabilities may develop a pattern of activation similar to that seen in bipolar disorder (11).

Several case reports have also shown that leads placed ventral to the STN (in the substantia nigra) may result in acute depressive or elevated mood states (3, 9). Correct lead location was confirmed in all our patients. It is important to note that the contacts that caused elevated mood were associated with optimal motor benefit in several cases. As such, multiple strategies may be necessary to address elevated mood while maintaining motor benefit (Table 2). Newer directional leads, DBS systems with multiple independent current sources, and future adaptive DBS paradigms may also be useful in optimizing motor benefit without negatively influencing mood.

One of the patients we described (Mr. B) had severe co-occurring dopamine dysregulation syndrome. He experienced elevated mood symptoms on postoperative day 2, before his DBS device was activated. The high dopaminergic load in a patient with bipolar diathesis, perhaps combined with the lesional effect, were most likely contributors. Dopaminergic agents can independently destabilize mood or have additive effects to DBS, which is why it is paramount to address both comorbidities (15).

Elevated mood symptoms recurred in four of our five patients when the same contacts were used. One patient (Mr. D) was able to tolerate gradually higher voltages with the same contact. This may indicate a habituation process, as suggested by Tommasi et al. (5), allowing patients to safely use configurations that initially caused adverse effects if amplitude is slowly increased. Slow stimulation titration can be helpful with patients at higher risk of developing postoperative elevated mood.

None of the patients described here met DSM criteria for bipolar disorder (29). The use of the terms hypomania or mania in previous reports was technically inaccurate, since duration criteria for hypomanic or manic episodes were rarely met and adjusting DBS settings or turning off the device usually led to symptom resolution (7–10). In DSM-5, criteria for hypomanic or manic episodes are met when symptoms occur in response to antidepressant treatment (e.g., medications, electroconvulsive treatment) but persist at a fully syndromal level beyond the physiological effect of that treatment (29). As DBS therapy becomes more frequently used for the treatment of both movement disorders and psychiatric conditions, the inclusion of stimulation-induced elevated mood in the DSM warrants consideration. Proposed criteria for this new clinical entity are presented in Table 4. This phenomenon has also been described in patients treated with DBS for treatment-refractory major depressive disorder or obsessive-compulsive disorder involving stimulation of the anterior limb of the internal capsule or the ventral internal capsule/ventral striatum (VC/VS) (30–32). Interestingly, in a series of 20 patients with VC/VS DBS, a personal or family history of bipolar disorder did not predict occurrence of stimulation-induced hypomania (32).

TABLE 4. Proposed diagnostic criteria for stimulation-induced elevated mood states

Criteria
Core features[a]
Elevated, expansive, or irritable mood
Psychomotor agitation
Behavioral changes occurred during or shortly after deep brain stimulation programming changes
Supportive features[b]
Increase in goal-directed activity
Impulsivity
Pressured speech
Decreased need for sleep
Grandiosity
Flight of ideas
Hypersexuality
Duration: Symptoms typically resolve with deep brain stimulation setting adjustments but may persist beyond adjustments.

[a] All core features must be present.
[b] One or more supportive features may be present.

Limitations of this study include selection bias; that is, only patients with severe behavioral symptoms are referred for psychiatric evaluations, and thus not all patients who had DBS during the study period were included. All patients in this case series had STN DBS, which has been linked to mood dysregulation more than the GPi target. Future studies should include systematic multisite reviews of all patients treated with DBS during a certain period, followed prospectively. Strengths of our study include the detailed documentation of programming settings, availability of psychiatric evaluations, and length of postoperative follow-up (up to 5 years), allowing the longitudinal tracking of symptoms and evaluation of the effectiveness of management strategies.

CONCLUSIONS

Stimulation-induced elevated mood states are underdiagnosed clinical phenomena. Psychiatric factors (history of impulse-control disorder, dopamine dysregulation syndrome, substance use disorder, and/or bipolar diathesis) can increase risk and should be identified early during the preoperative DBS candidacy evaluation. Inclusion in future DSM revisions would facilitate the recognition and management of this rare but nevertheless potentially severe DBS-related complication.

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Dr. Spiegel has received funding from Medtronic. Dr. Brown has received research grant support from Biogen, the Gateway Institute for Brain Research, and the Michael J. Fox Foundation. Dr. Starr has received research grant support from Boston Scientific and Medtronic. Dr. Ostrem has received research grant support from Boston Scientific and consulting fees from Abbott. The other authors report no financial relationships with commercial interests.

The authors thank the study participants and their families. The authors also thank Nicholas Galifianakis, M.D., M.P.H., for assistance with data collection.

The authors have confirmed that details of the cases have been disguised to protect patients' privacy.

Previously presented in part as a poster at the annual meeting of the American Psychiatric Association, San Francisco, May 20–22, 2019, and the International Congress of Parkinson's Disease and Movement Disorders, Nice, France, September 22–26, 2019.

Received August 1, 2020; revision received December 23, 2020; accepted January 11, 2021; published online July 2, 2021.

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