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impairment, including dropping out of school and an inability to leave the home. He had undergone adequate trials of most selective serotonin reuptake inhibitors (SSRIs) (paroxetine, fluoxetine, fluvoxamine, escitalopram, and sertraline), both as monotherapy and in combination with several pharmacologic augmentation strategies (diazepam, topiramate, buspirone, olanzapine, aripiprazole, risperidone, memantine). In addition, he underwent cognitive-behavioral therapy using exposure response prevention on a weekly basis for 20 weeks. At the time of surgery, his Yale-Brown Obsessive Compulsive Scale<sup>2</sup> (Y-BOCS) score was 32, consistent with extreme illness severity. After ethical review board approval and providing informed consent, he underwent bilateral implantation of electrodes targeting the nucleus accumbens.

We performed cognitive assessments at baseline (prestimulation), 8 months after DBS began, and then yearly for 3 years. Cognitive tasks were taken from the Cambridge Neuropsychological Test Automated Battery (www.camcog.com) and included the Stop-Signal Test (assessing ability to suppress prepotent motor responses) and the Intradimensional/Extradimensional Set Shift Task (examining rule learning and behavioral flexibility).

Prior research<sup>3</sup> in a sample of OCD patients, their first-degree relatives, and unrelated healthy volunteers demonstrated that variation in motor inhibitory control (Stop-Signal Test) was correlated with gray matter density changes in an extensive system comprising orbitofrontal, cingulate, and parietal cortical areas, as well as striatal and other subcortical regions, and that these inhibition-related brain systems might be considered a neurocognitive endophenotype for OCD. Such cognitive assessments conducted over time have the potential to help researchers and clinicians better understand whether symptom improvement coincides with cognitive processing improvement several years after surgical intervention. To control for potential practice effects, cognitive tasks were also performed at the same yearly intervals in age- and gender-matched controls with no current or lifetime psychiatric disorders based on a structured clinical interview by a board-certified psychiatrist.<sup>3</sup>

At the time of surgery, our patient was taking the following medications (stable doses for at least 3 years): clomipramine 250 mg/d; ziprasidone 120 mg/d; and clonazepam 1 mg 3 times daily. These medications continued unchanged throughout the study. DBS was not interrupted for battery change over the course of the study, and the DBS settings were held constant after an initial period of adjustment. He experienced symptom improvement 8 months after surgery (Y-BOCS score of 10), and over the course of the 3 years, his Y-BOCS scores were consistently 8–10. In addition to OCD symptom improvement, the patient exhibited a dramatic improvement in social and occupational health. He obtained and maintained a job and has re-developed meaningful social relationships (including dating). His motor inhibitory performance

**Long-Term Deep-Brain Stimulation Treatment for Obsessive-Compulsive Disorder**

**To the Editor:** Three years ago, we reported<sup>1</sup> on a successful case of deep-brain stimulation (DBS), targeted at the nucleus accumbens, in a 30-year-old man with obsessive-compulsive disorder (OCD). Here we provide the follow-up to that case.

**Case report.** At presentation, our patient had had a 5-year history of OCD (*DSM-IV*) with contamination obsessions and washing compulsions. The OCD had resulted in severe functional

**Table 1. Neurocognitive Performance (mean z score<sup>a</sup>) Over Time at Baseline and During Deep-Brain Stimulation Treatment Compared to Longitudinal Normative Data**

Cognitive Measure	Baseline		1-Year		2-Year		3-Year	
	Rating	Mean z Score	Rating	Mean z Score	Rating	Mean z Score	Rating	Mean z Score
Stop-Signal Test reaction time (ms)	211.6	1.0	204.7	0.8	205.0	0.2	151.8	-0.3
Stop-Signal Test median go reaction time (ms)	449	0.1	379	-0.4	346	-0.5	345	-0.5
Intradimensional/Extradimensional Set Shift Task, pre-Extradimensional errors	6	0.3	7	1.0	8	1.6	5	0.0
Intradimensional/Extradimensional Set Shift Task, Extradimensional errors	1	-0.8	2	-0.5	0	-0.7	2	-0.5

<sup>a</sup>In all cases, positive z scores indicate worse performance versus healthy controls, and negative scores indicate better performance versus healthy controls.

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(ie, the candidate cognitive endophenotype for OCD) was impaired at baseline and did not improve to the levels of controls until the third year after surgery (Table 1).

Despite OCD-symptom improvement almost immediately after surgery, our results suggest that response inhibition did not improve until several years later. Previous research<sup>4</sup> suggests that stop-signal impairment in OCD persists despite first-line intervention with SSRIs. Acute dopamine manipulation of the nucleus accumbens core had no effect on response inhibition in rodents,<sup>5</sup> while the accumbens shell does appear to be involved in aspects of inhibitory control.<sup>6</sup> We speculate that chronic DBS of the accumbens core led to indirect upstream effects on other neural regions (eg, accumbens shell or subthalamic nucleus), which in turn were responsible for the cognitive improvement reported.

These findings highlight that certain cognitive deficits in OCD, refractory to usual interventions, may resolve with prolonged DBS treatment in OCD and be temporally dissociable from symptom improvement. Further research is needed to confirm these findings, rule out nonspecific contributing factors (eg, improvement in social functioning itself resulting in cognitive change), and characterize the neural mechanisms involved. The results also lead to an important question: once cognitive deficits normalize, could stimulation for DBS in OCD be turned off without deficits and symptoms recurring?

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**Jon E. Grant, JD, MD, MPH<sup>a</sup>**

jongrant@uchicago.edu

**Brian L. Odlaug, MPH<sup>b</sup>**

**Samuel R. Chamberlain, MD, PhD<sup>c</sup>**

<sup>a</sup>Department of Psychiatry and Behavioral Neuroscience, University of Chicago, Illinois

<sup>b</sup>Department of Public Health, Faculty of Health and Medical Sciences, University of Copenhagen, Denmark

<sup>c</sup>Department of Psychiatry, University of Cambridge; and Cambridge and Peterborough NHS Foundation Trust (CPFT), Cambridge, United Kingdom

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