Long-Term Neuropsychological Safety of Subgenual Cingulate Gyrus Deep Brain Stimulation for Treatment-Resistant Depression

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Deep brain stimulation (DBS) of the subgenual cingulate gyrus (SCG) is a promising investigational intervention for treatment-resistant depression (TRD), but long-term outcome data are limited. Serial neuropsychological evaluations, using a comprehensive battery, were conducted on four subjects with TRD prior to surgery, and up to 42 months post-operatively. Reliable change methodology suggested general stability and/or select statistically reliable improvement in cognitive abilities over time. This is the first known set of multi-year neuropsychological follow-up data for SCG DBS for TRD. Observed improvements are likely attributable to reduced depressive symptomatology, recovery of functional capacities, and/or specific practice effects of repeated assessment.

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Within the past decade, scientific and public interest in deep brain stimulation (DBS) as a promising investigational therapy for treatment-resistant depression (TRD) has dramatically increased.[1] DBS is a well-established first-line neurosurgical treatment for movement disorders and involves MRI-guided stereotaxic placement of electrodes within selected brain regions. An implantable pulse generator, typically placed in the chest, delivers focal stimulation to the electrodes according to clinician-programmed parameters. Relative to other types of neurosurgery, the main advantages of DBS are its reversibility and its adjustability in terms of stimulation settings.

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The subgenual cingulate gyrus (SCG), the ventral capsule/ventral striatum, and the nucleus accumbens (NAcc) have been the most studied neurosurgical target areas for DBS in TRD to date, although single-case studies have identified other targets with positive symptomatic outcomes.[2–6] Large-scale controlled trials are already underway for some targets, with follow-up data suggesting that DBS for TRD is generally safe and moderately efficacious over the short term. However, continued long-term monitoring and data collection are needed to determine whether DBS represents a viable option for individuals who have not responded to standard treatments for depression and who continue to experience severe distress and functional impairments.

Concerns regarding the impact of DBS on cognition in those with TRD have been raised, given that the implant procedure and active stimulation may create functional lesions at the selected targets, and given that depression, per se, is already associated with cognitive dysfunction.[7–11] Clinical evaluation using neuropsychological measures appears clearly warranted in light of the following: 1) scarce data on long-term neuropsychological function following DBS for TRD; 2) previous findings that implicate the SCG in cognitive and memory function[12]; 3) inconsistent findings that other treatments for depression (such as ECT or certain classes of antidepressant medications) are associated with cognitive impairment[13,14]; and 4) previous findings suggesting that DBS for Parkinson's disease may result in mild cognitive deficits depending on surgical trajectory and electrode location.[15,16]

Unfortunately, because of the relative novelty of this intervention for TRD, there have been no reliable data that describe long-term cognitive outcomes beyond the typical 12-month endpoint in studies of DBS for depression.[17,18] This article presents the first report of multi-year follow-up neuropsychological data for individuals receiving DBS for TRD.

METHODS

Four consecutive subjects (one female, three male) from the Canadian multi-center DBS pilot study[19] targeting the SCG (Brodmann Area 25, Cg25) provided written informed consent to participate in a long-term follow-up (LTFU) protocol. A fifth subject who participated in the pilot study at this site was explanted due to non-response and was, thus, not enrolled in the LTFU protocol. However, analysis of this individual's neuropsychological test scores from baseline through the 12-month period did not reveal any pattern of statistically reliable decline in performance. The research protocol was approved by the Clinical Research Ethics Board at the University of British Columbia and by the Vancouver Coastal Health Research Institute/Vancouver Coastal Health Authority.

All subjects underwent DBS implant surgeries with the Libra constant-current internal pulse generator device (St. Jude Medical) between January and December of 2006, as previously described,[19] and entered the LTFU portion of the study between September 2007 and August 2008. All subjects met DSM-IV-TR[20] criteria for Major Depressive Disorder (MDD) and had documented histories of resistance to standard treatments for MDD, including adequate trials with a minimum of four different classes of antidepressant medications and psychotherapy. Three of the four subjects were also unresponsive to ECT (the procedure contraindicated in the fourth because of the presence of a cerebral venous angioma). Patients did not have significant psychiatric or medical comorbidities and were deemed suitable neurosurgical candidates.

Subjects had received active DBS for approximately 42 months at the time of long-term neuropsychological follow-up. Following implantation, three of the four subjects had one contact activated per hemisphere, and the fourth had two contacts per hemisphere. DBS treatment parameters were initially programmed using continuous monopolar cathode stimulation, 130 Hertz frequency, 91 microseconds pulse widths, and 2.0–4.5 milliamps amplitude. Over time, settings were adjusted, as dictated by patients' responses and clinical need. All patients continued to take psychiatric medications after receiving DBS implants and into the LTFU period, with infrequent, standard-care medication adjustments as warranted. Clinical reassessments occurred every 6 months in the LTFU period. Demographic information for all four subjects, and as a group, is presented in Table 1.

A comprehensive neuropsychological test battery was administered prior to surgery and again at 3, 6, 12 months, and an average of 42 months (LTFU) post-operatively. The test battery encompassed a number of domains of cognitive functioning including general intellectual ability, language ability, attention and working memory, verbal and visual memory, visuoperceptual and visuoconstructional ability, speed of information processing, and executive functioning. Specifically, the test battery was

TABLE 1. Patient Demographics

	S1	S2	S3	S4	Group
Gender	M	M	F	M	1F/3M
Age at baseline, in years	35	59	46	39	45.2
Age at MDD onset, in years	18	20	18	26	20.5
Lifetime number of MDD episodes	4	2	3	3	3.0
Length of current MDD episode, in years	9	3	12	9	8.3
Past ECT	Yes	No	Yes	Yes	75%
Past psychotherapy	Yes	Yes	Yes	Yes	100%
Family history of MDD	No	Yes	No	Yes	50%
Melancholic features	Yes	Yes	Yes	No	75%
Baseline HRSD-17 score	34	28	34	20	29.0
Baseline IDS score	60	31	58	44	48.3
Baseline MADRS score	47	34	42	36	39.8
NAART estimated premorbid IQ	122	115	115	118	117.5

S, subject; MDD, major depressive disorder; ECT, electroconvulsive therapy; HRSD–17, Hamilton Rating Scale for Depression (17 item); IDS, Inventory of Depressive Symptomatology; MADRS, Montgomery-Åsberg Depression Rating Scale; NAART, North American Adult Reading Test; IQ, intelligence quotient.

TABLE 2. Summary of Clinical Assessments Across Time

Subject and rating scale	Baseline	3 mo	6 mo	12 mo	LTFU
Subject 1— HRSD-17	34	21	25	16	14
Subject 1—IDS	60	39	42	26	29
Subject 1— MADRS	47	30	30	20	23
Subject 2—HRSD-17	28	13	9	15	14
Subject 2—IDS	31	30	26	29	24
Subject 2—MADRS	34	31	26	21	26
Subject 3—HRSD-17	34	25	21	13	22
Subject 3—IDS	58	48	38	25	41
Subject 3—MADRS	42	42	24	21	33
Subject 4—HRSD-17	20	11	9	11	12
Subject 4 – IDS	47	32	25	30	22
Subject 4—MADRS	36	18	14	17	14

composed of the following measures: North American Adult Reading Test (NAART)[21]; Wechsler Adult Intelligence Scale (WAIS–III)[22]; selected subtests (Logical Memory I and II, Family Pictures I and II) from the Wechsler Memory Scale–3rd Edition (WMS–III)[23]; selected subtests (Trail Making, Fluency, Color-Word) from the Delis-Kaplan Executive Function System (DKEFS)[24,25]; California Verbal Learning Test–II (CVLT–II)[26]; Benton Visual Form Discrimination Test (VFDT)[27]; and the Boston Naming Test (BNT).[28] Further description of these standard neuropsychological measures is not included here but can be found in Spreen and Strauss.[29] Where available (DKEFS Verbal Fluency, CVLT–II), alternate versions of these measures were used across testing sessions to mitigate practice effects. Depression was assessed via the Montgomery-Åsberg Depression Scale (MADRS),[30] the Hamilton Rating Scale for Depression (HRSD),[31] and the Inventory of Depressive Symptomatology (IDS).[32,33] Total scores for clinical scales are reported in Table 2. In almost all cases, neuropsychological evaluations occurred within 7 days of the clinical assessments.

Neuropsychological test results were analyzed using the most appropriate normative sample with correction for age, gender, and education, where possible, derived from their respective test manuals. Z-score conversions were used for consistency across all data. Reliable change methodology[34] was used to evaluate for change in cognitive functioning, as this takes into account the test-retest reliability of the neuropsychological instruments in question. (RCI confidence interval=Initial test score +/− (Z*SEdiff) where SEdiff=Sqrt [(SEM1^2) + (SEM2^2)]. SEM1=Standard error of measurement Time1, SEM2=SEM at time 2, and SEM=SD*Sqrt[1-r12]. A 90% confidence interval was used, so 5% at each tail (i.e., Z score of 1.645).

RESULTS

Self-Reported Cognitive Function Following DBS

Although not assessed as part of the formal research protocol, all four subjects reported cognitive difficulties after DBS implantation during regular study visits, starting at least 1 year post-implant. Specifically, subjects reported short-term memory deficits, paraphasic errors of speech, and word-finding difficulties. In most instances, family members corroborated subjects' self-reports and provided anecdotes about observed forgetfulness or speech errors. Some subjects also reported perceived worsening of cognitive deficits over time and concern about the impact of DBS on overall cognitive functioning.

Objectively Measured Cognitive Function at Baseline

No clinically significant cognitive problems were identified in any of the subjects at baseline. Subjects performed within normal limits (i.e., "low average" range or better; $z \geq -1.3$) on all measures with the exception of subject 2, whose performance fell below normal limits on tests of category fluency ($z=-1.7$); color naming speed ($z=-2.7$); visual scanning speed, ($z=-2.3$); and letter sequencing speed ($z=-1.7$). As a group, subject performance fell within the "high average" to "superior" range on intellectual function—a finding consistent with, or exceeding, NAART estimated premorbid IQ scores.[35]

Cognitive Function across Time

The primary approach for evaluating cognitive change was to compare performance at baseline to each of the follow up evaluation points. General cognitive stability and many areas of statistically reliable improvement were noted in subjects across follow-up time points, with only minor variability, as indicated in Table 3. These include statistically reliable improvements in general intellectual ability (WAIS–III Full Scale IQ) in all four participants, including at LTFU (subjects 1, 2, and 4). Similar improvements in overall memory ability (WMS–III Total Memory Composite) were also noted in three of the four participants (subjects 1, 3, and 4), including at LTFU (subjects 1 and 3). Additional areas of statistically reliable change for all the specific neuropsychological abilities are noted in Table 3.

With respect to declines in cognitive performance, subject 1 displayed a statistically reliable decline in category fluency at 3 months and at LTFU, but not at 6 or at 12 months. Subject 2 displayed a statistically reliable decline in free recall for word lists after a short-delay (CVLT–II) at LTFU only, and in terms of long-delay free recall at 3 months but not at other time points. Subject 3 displayed a statistically reliable decline on the WAIS–III Working Memory Index at 3 and 12 months, but not at 6 months or at LTFU. No other statistically reliable declines were apparent and no consistent pattern across time points for any individual subjects, or across subjects, was apparent, suggesting that these isolated findings reflect normal variability over a high number of assessments.

Medication Changes during the Course of the Study

With regard to medication adjustments/changes and their potential influence on observed cognitive performance of subjects, medication changes are included in Table 4 below.

Medication adjustments were made per standard care for all participants and additional medications were initiated for some participants as noted in Table 4. No clear pattern of cognitive change associated with medications was apparent but this could not be evaluated formally using statistical methods due to the small sample size.

DISCUSSION

Despite fairly consistent reports by our subjects of post-implant word-finding difficulties, paraphasic errors of speech, and short-term memory issues, the neuropsychological data collected at various post-surgical time-points, including at 42 months, did not indicate a systematic decline in any area of assessed cognitive functioning compared with pre-surgical performance. The results suggest that DBS is not only effective in reducing depressive symptomatology in the context of treatment-resistant depression, but that it also does not result in systematic detectable cognitive declines. However, it must be acknowledged that both pro-cognitive effects of reduced depressive symptomatology and/or practice effects from repeated test administration (particularly during the first 12 months) could be offsetting or masking some degree of cognitive decline.

With respect to the discrepancy between self-report and objectively measured cognitive function, there are several possible explanations. First, it is possible that having undergone brain surgery resulted in a heightened level of self-monitoring of internal cognitive performance. Another possibility is that self reports of cognitive impairment correlate poorly with objective indices of neuropsychological performance, particularly in patients with affective disorders.[35,36] Alternately, subjects' self-reports may reflect subtle changes in cognitive efficiency that fell below the sensitivity of the cognitive measures. Finally, it is also possible that the surgical procedure/stimulation, itself, altered *perception* of cognitive performance without altering actual performance. However, the fact that cognitive changes were objectively confirmed by relatives suggests that self-perception or self-monitoring was not the only responsible factor.

This report represents the first published long-term follow-up data on DBS in treatment-resistant depression. Further, complete neuropsychological data on all four participants across time points were available, the

TABLE 3. Selected Neuropsychological Results Across Time

	Baseline	3 Months	6 Months	12 Months	LTFU
General Intellectual Ability					
Subject 1	1.9	2.9[a]	3.4[a]	3.0[a]	3.0[a]
Subject 2	0.8	0.7	1.3[a]	0.9	1.4[a]
Subject 3	1.8	1.9	2.5[a]	2.9[a]	2.1
Subject 4	1.1	1.8[a]	1.5[a]	2.1[a]	2.1[a]
Verbal Delayed Recall—List Learning					
Subject 1	0.0	1.5[a]	1.0[a]	1.0[a]	1.5[a]
Subject 2	–1.0	–3.0[b]	–1.0	0.0	–2.0
Subject 3	0.5	1.5	1.5	1.5	1.5
Subject 4	1.0	1.0	1.5	1.0	1.0
Verbal Delayed Recall—Story Recall					
Subject 1	0.7	1.7	2.0[a]	1.7	1.3
Subject 2	–0.3	–0.3	0.7	0.7	0.0
Subject 3	1.7	2.0	2.0	2.3	2.0
Subject 4	1.3	1.3	2.0	2.0	1.7
Visual Delayed Recall					
Subject 1	–0.3	1.0[a]	2.3[a]	2.3[a]	0.7
Subject 2	–1.0	–1.3	–0.7	–0.7	–0.7
Subject 3	0.7	1.7	2.3[a]	2.3[a]	2.3[a]
Subject 4	0.7	1.0	1.3	2.0[a]	2.3[a]
Naming					
Subject 1	0.8	0.8	1.1	1.1	1.1
Subject 2	0.6	0.8	1.0	1.0	1.5[a]
Subject 3	–0.9	0.4[a]	0.4[a]	0.7[a]	0.4[a]
Subject 4	1.1	0.7	1.1	0.7	1.1
Verbal Fluency—Phonemic					
Subject 1	0.7	1.7	2.0[a]	1.7	1.7
Subject 2	–1.0	–0.7	–1.3	–0.7	–2.0
Subject 3	1.0	1.7	2.0	2.3[a]	2.0
Subject 4	0.3	1.0	0.3	0.7	1.3
Verbal Fluency—Category					
Subject 1	2.0	0.7[b]	2.0	2.0	0.7[b]
Subject 2	–1.7	–1.3	–2.0	–1.3	–1.7
Subject 3	1.3	1.7	1.7	1.7	1.0
Subject 4	0.7	1.3	1.7	1.7	2.0[a]
Executive Functioning—Switching					
Subject 1	1.3	0.7	1.7	2.0	2.3
Subject 2	–0.7	–1.0	–1.0	–2.3	–1.3
Subject 3	0.7	0.0	–0.7	0.3	0.0
Subject 4	0.7	0.3	1.3	2.3	1.3
Executive Functioning—Inhibition					
Subject 1	1.0	1.0	1.0	0.7	1.0
Subject 2	–1.3	–0.7	0.3[a]	–0.7	–0.7
Subject 3	0.7	0.7	1.0	0.7	0.3
Subject 4	1.0	0.7	1.3	0.3	0.0
Visual Perception					
Subject 1	0.7	0.7	0.7	0.7	0.7
Subject 2	–0.1	–0.1	0.9	–0.5	–0.1
Subject 3	0.9	0.9	0.9	0.9	0.9
Subject 4	0.7	0.7	0.1	0.7	0.7
Attention/Working Memory					
Subject 1	1.3	1.4	2.2[a]	1.7	1.9
Subject 2	0.6	0.5	1.0	0.6	1.0
Subject 3	2.4	1.6[b]	2.2	1.6[b]	1.7
Subject 4	0.9	1.0	0.7	1.3	1.9[a]
Processing Speed					
Subject 1	0.4	0.5	0.7	1.1	1.5[a]
Subject 2	–1.3	–1.3	–0.8	–1.1	–0.6
Subject 3	1.3	2.3[a]	2.3[a]	2.1	1.7
Subject 4	0.2	0.2	0.5	0.4	0.9

LTFU, long-term follow-up (average of 42 months).

Results presented in z-scores based on published normative data. General intellectual ability: Wechsler Adult Intelligence Scale–III (WAIS–III) Full Scale IQ; verbal delayed recall–list learning: California Verbal Learning Test–2 standard and alternate forms; verbal delayed recall–story recall: Wechsler Memory Scales–III (WMS–III) Logical Memory; visual delayed recall: WMS-III Family Pictures; verbal fluency – phonemic: Delis Kaplan Executive Function System (DKEFS); verbal fluency–category: DKEFS; executive function–switching: DKEFS Fluency Switching; executive function–inhibition: DKEFS Color-word interference test; visual perception: Benton Visual Form Discrimination Test; attention/working memory: WAIS-III Working Memory Index; processing speed: WAIS III Processing Speed Index.

[a]Denotes statistically reliable improvement compared with baseline.

[b]Denotes statistically reliably decline compared with baseline.

Underlined scores represent scores falling within the "Impaired" range defined as 1.4 SD below the mean or greater.[38]

TABLE 4. Summary of Medications Across Study Duration

Patient 1	Patient 2	Patient 3	Patient 4
Prebaseline	**Prebaseline**	**Prebaseline**	**Prebaseline**
trazodone	olanzapine	levothyroxine	escitalopram
zopiclone	dextroamphetamine sulfate	fluoxetine	nortriptyline
lorazepam prn	phenobarbital	sertraline	topiramate
triazolam prn	gabapentin	lorazepam	dexedrine SR
vitamin B12	levothyroxine	dextroamphetamine sulfate	dexedrine IR
Added post-implant	fluvoxamine	loxapine	**Added post-implant**
bupropion	clonazepam	zopiclone	NA
Added before month 12 evaluation	**Added post-implant**	temazepam	**Added before month 12 evaluation**
methylphenidate	doxepin	**Added post-implant**	NA
Added before month 30 evaluation	sertraline	risperidone	**Added before month 30 evaluation**
NA	**Added before month 12 evaluation**	clonazepam	NA
	NA	**Added before month 12 evaluation**	
	Added before month 30 evaluation	trazodone	
	bromocriptine	**Added before month 30 evaluation**	
	cytomel	quetiapine XR	
	mirtazapine	duloxetine	

data collection procedure remained consistent across time points, and reliable change methodology was utilized. Although these data are not yet available, a 6-year post-operative follow-up is planned, and will provide further information with respect to the long-term stability of cognitive function following DBS for TRD.

As is the case with all research, this study had some specific limitations that may have influenced the results, as well as the conclusions that could be drawn. First, this study involved a small sample size of only four participants without a control group. Second, the high baseline intellectual ability of the sample is not characteristic of the general population and may limit the generalizability of the results. Third, despite efforts to control for reliability of neuropsychological instruments using reliable change methodology, the influence of repeated exposure to the test materials over multiple time points, particularly over the first 12 months of the study, was apparent. For example, despite already performing at a high-average to superior IQ level at the pre-surgical baseline, all four subjects displayed statistically reliable improvements in IQ at a number of time points, including at LTFU. Given that IQ is generally thought of as relatively stable, it is likely that the improved scores reflect some element of practice and increasing familiarity with the test materials and testing procedures.[37] (The exception to this is subject 2, who displayed a number of impaired scores at baseline and some statistically improved scores at LTFU [e.g., visual scanning speed, letter sequencing speed] that may reflect reversal of depression-related psychomotor slowing at baseline). It is also possible that these findings reflect pro-cognitive effects of reduced depression symptoms. However, 12-month follow-up data in a similar sample (Subgenual Cingulate Gyrus DBS for refractory depression) did not find an association between changes in cognitive function and improvement in mood.[18] In addition, 12-month follow-up data in a sample that received nucleus accumbens DBS for refractory depression revealed pro-cognitive effects that were independent of the antidepressant effects of DBS or changes in DBS parameters.[17] Further, a recent review of cognition and depression found little empirical support for pervasive depression-related deficits in general cognition, identifying only deficits in control of attention when not well-controlled by task demands.[8] Further, it has been identified that cognitive deficits persist in euthymic chronic unipolar depression suggesting an independent substrate from the depressed state contributing to cognitive deficits in this population.[39,40] Fourth, it is possible that medication or parameter changes influenced cognitive performance. However, no systematic relationship was noted between medication changes or parameter changes and subjective or objective measures of cognitive functioning in the follow-up period.

Although further research is needed in a larger sample to replicate the current LTFU findings and hopefully address some of the limitations of this study, this study provides preliminary reassurance of stability of cognitive functioning up to 42 months

post-surgery and no evidence to suggest systematic observable declines in any area of cognitive ability.

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Erratum

In the Winter 2014 issue, Chien-Han Lai, M.D., should have been listed as co-author for the Letters to the Editor "Long-Term Duloxetine Withdrawal Syndrome and Management in a Depressed Patient" (J Neuropsychiatry Clin Neurosci 2014; 26:E04) and "Improvement in Psychotic Symptoms and Social Functioning After Augmentation of Paliperidone With Clozapine in a Patient With Schizoaffective Disorder" J Neuropsychiatry Clin Neurosci 2014; 26:E26).