

ORIGINAL ARTICLE

ANT-DBS in epilepsy shows no effect on selected neuropsychiatric tests

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Objectives: Deep brain stimulation of the anterior thalamic nucleus (ANT-DBS) is an established option in treatment-resistant epilepsy and obtained FDA approval in 2018. Increased psychiatric comorbidity is well known in epilepsy. The main objective of this study was to investigate possible neuropsychiatric treatment-related changes in patients receiving ANT-DBS.

Materials and Methods: Bilateral ANT electrodes were implanted in 18 adult patients with refractory epilepsy in a randomized, double-blinded study. Immediately after implantation, patients were randomized to stimulation ON ($n=8$) or OFF ($n=10$) for the first 6 months (blinded phase). During the next six months (open phase), both groups received active stimulation. Neuropsychiatric assessment was conducted before implantation (T1), at the end of the blinded period (T2), and 1 year after implantation (T3).

Results: Comparing preoperative status (T1) and 12 months (T3), postoperative outcome in all patients did not show significant differences between the two groups for any of the applied tests. Groupwise comparisons across the two first time points (the blinded period, representing the randomized controlled trial) showed no significant differences between the two groups in any of the neuropsychiatric parameters studied. Comparing test results after 6 months of stimulation in both groups (sum of ON group T1 to T2 and OFF group T2 to T3) did not show significant changes for any of the psychiatric assessments.

Conclusions: Our results indicate that ANT-DBS has limited effect concerning psychiatric issues. Subjective side effects were, however, reported in individual patients.

KEYWORDS

anterior thalamic nuclei deep brain stimulation, drug-resistant epilepsy, neuropsychiatric assessments

1 | INTRODUCTION

It is well known that there is a close connection between epilepsy and psychiatric comorbidities.¹ According to literature, up to 30% of newly diagnosed people with epilepsy and as much as 50% of people with refractory epilepsy experience psychiatric, psychosocial,

or cognitive challenges.²⁻⁴ Refractory epilepsy is a condition that affects all sides of life, and it is associated with increased risk of mortality, morbidities, injuries, and sudden unexpected death in epilepsy (SUDEP), as well as unacceptable side effects of antiepileptic medication. In this context, deep brain stimulation (DBS) has become an important tool shown to be one of the most promising

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neuromodulatory techniques today.^{3,4} The SANTE study,⁵ the only double-blinded randomized study for a long time, showed a sustained effect for 2 years of bilateral stimulation to the ANT, and in the open follow-up continuation after 5 years a 69% reduction of seizure frequency from baseline.⁶

To the best of our knowledge, the Oslo study is the only other ANT-DBS study with a prospective, double-blinded, randomized design. Our study included adult patients suffering from pharmacoresistant, focal epilepsy. We have so far published three articles regarding different aspects of this stimulation-based treatment: reporting the effect of stimulation on seizure frequency and the possible side effects,⁷ discussing the use of direct versus preset coordinates for the implantation of electrodes,⁸ and investigating cognitive changes in patients receiving ANT-DBS.⁹ Our findings were not as promising as the SANTE results, but when considering all our patients and comparing 6 months of stimulation with baseline, there was a significant, 22% reduction in frequency of all seizures.⁷

An increased psychiatric burden in epilepsy is known. Furthermore, the anatomical connections between the ANT and the anterior cingulate and orbitomedial prefrontal cortex may influence emotional and executive functions.¹⁰ Little is known about how stimulation to the ANT may influence neuropsychiatric outcome. This paper's main objective is to analyze the effect of stimulation to the ANT on neuropsychiatric parameters.

2 | MATERIAL AND METHODS

2.1 | Patients and study design

A detailed description of the study design has already been given in the main paper,⁷ including inclusion and exclusion criteria, surgical procedures, stimulation parameters, side effects, and seizure types. The study design is shown in [Figure 1](#).

In short, bilateral ANT electrodes were implanted into 18 patients, 11 women and 7 men suffering from focal, pharmacoresistant epilepsy. Antiepileptic treatment was kept unchanged from 3 months prior to operation. Immediately after implantation, patients were randomized to stimulation ON ($n = 8$) or OFF ($n = 10$) for the first 6 months (blinded phase). During the following 6-months (open phase), both groups received stimulation.

Our study was originally planned to include 40 patients. However, the halfway interim analysis revealed that a number of patients had an increased seizure frequency under active stimulation, and there was no difference between patients with and without stimulation after the blinded period. We therefore chose to stop further inclusion.⁷

Neuropsychiatric assessments were performed before implantation (T1), at the end of the blinded period (T2), and at the end of the open period, 1 year after implantation (T3).

At inclusion, information about the study was given, and informed consent was signed by the patient and the investigator. The study was conducted according to the Declaration of Helsinki and approved for by the Regional Committee for Medical and Health Research Ethics, REC South-East, Norway.

2.2 | Neuropsychiatric assessment

Neuropsychiatric assessments were obtained by patients filling out psychometric inventories preoperatively and at six and 12 months postoperatively. The psychometric inventory contained the following: HADS, MADRS, IDS. The patients also filled out Qualie-89. In addition, the patients went through a thorough interview by an experienced psychiatrist regarding subjective experiences with the treatment, including side-effects.

The Hospital Anxiety and Depression Scale (HADS) is a 14-item self-report measure, widely used as screening instrument for symptoms

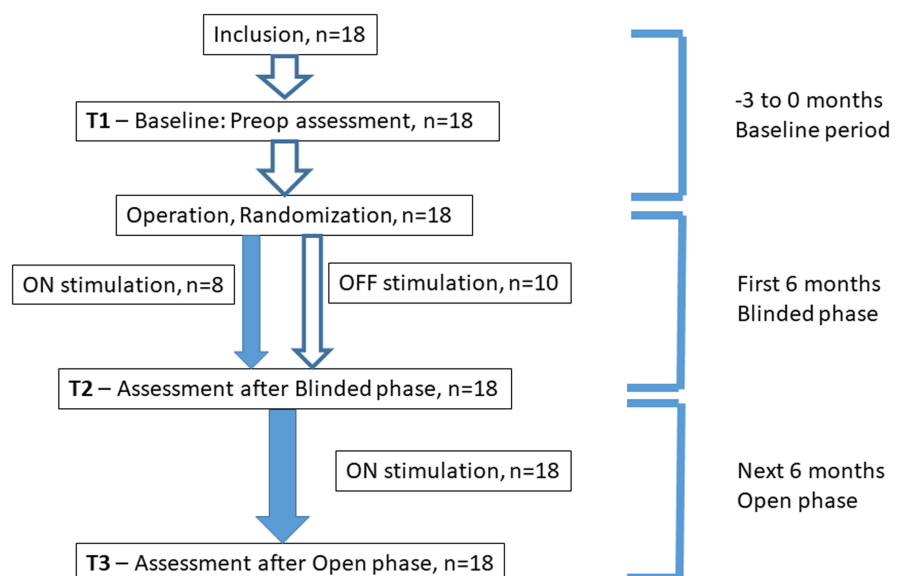


FIGURE 1 Study design

of psychological distress among patients with somatic disorders. HADS consists of 14 items separated into two subscales, one for anxiety (HADA, 7 items) and one for depression (HADD, 7 items). Each of the 14 items are scored from 0–3, giving a score between 0 and 21 for each of the subscales. Higher scores indicate more severe symptoms.¹¹

The *Inventory of Depressive Symptomatology (IDS)* is a 30-item clinician-rated questionnaire used to assess depression severity in clinical trials and practice. Each of the 30 items are scored from 0–3, giving a score between 0 and 90. Higher scores indicate more severe symptoms.¹²

Montgomery and Åsberg Depression Rating Scale (MADRS) is a 10-item clinician-rated questionnaire widely used to assess depression severity in clinical trials and practice. Each of the 10 items are scored from 0–6, giving a score range of 0–60. Higher scores indicate more severe symptoms.¹³

Qualie-89 is an inventory that contains 17 multi-item measures of overall quality of life, emotional well-being, role limitations due to emotional problems, social support, social isolation, energy/fatigue, worry about seizure, medication effects, health discouragement, work/driving/social function, attention/concentration, language, memory, physical function, pain, role limitations due to physical problems, and health perceptions.¹⁴

In addition to structured neuropsychiatric assessment through these inventories, all patients were every third month personally interviewed concerning symptoms and experiences.

2.3 | Statistics

Statistical analyses were performed using IBM SPSS version 25. We used independent sample *T*-test to discover possible differences between the two groups using the psychiatric tests MADRS, IDS, and HAD and the *Qualie-89* after the 6 months blinded period (at T2). We also performed paired sample *T*-test analyses for all patients comparing preoperative data (T1) and 12 months postoperative data (T3) to examine whether there were any differences, and before and after 6 months of stimulation for all patients (comparing T1 with T2 in patients randomized to ON stimulation during the first 6 months blinded period, and T2 to T3 for patients randomized to OFF stimulation during the first 6 months).

3 | RESULTS

The patients' characteristics are given in [Table 1](#). There were no significant differences between the ON and OFF groups regarding seizure frequency, seizure types (FBTC, FAI, FA), age, sex, and number of antiseizure medications.

Comparing preoperative outcomes (T1) and 12 months postoperative outcomes (T3) in all patients did not show significant differences between the two groups for any of the applied tests; MADRS,

HADA, HADD, IDS, and *Qualie-89* ([Table 2](#)). There were no significant differences between ON and OFF groups at baseline.

Groupwise comparisons across the two first time points (the blinded period representing the randomized controlled trial) showed no significant differences between the two groups in any of the neuropsychiatric parameters studied ([Table 3](#)).

Comparing test results in *Qualie-89*, HADA, HADD, IDS, and MADRS after 6 months of stimulation in both groups (sum of ON group T1 to T2 and OFF group T2 to T3) did not show significant changes for any of the psychiatric assessments ([Table 4](#)).

Of our 18 patients, one patient reported an intense sense of joy and happiness each time the stimulator was activated, that is every sixth minute. This sense lasted for 1 min and then subsided. Over time, he grew customized to these symptoms and they tapered off.

Another patient reported strange thoughts and a feeling of being outside herself after the stimulator was turned on at 6 months postoperatively. The voltage was reduced which minimized and finally stopped her psychic reaction to the stimulation.

Other symptoms reported in individual patients during the study period included memory deficit, temporary depression, and difficulties finding words. Other patients reported better sleep, more energy, and better cognitive functioning.

4 | DISCUSSION

In the present ANT-DBS study, we did not find significant differences in neuropsychiatric parameters between the stimulated group and the control group at the end of the blinded phase at 6 months. Neither did we find differences between the two groups after 6 months of active stimulation. Looking at all patients, there were no significant neuropsychiatric differences when baseline data were compared with results at the end of the total 12-month study period. All these findings indicate that stimulation of the anterior thalamus does not alter psychiatric measures.

Our findings from the current neuropsychiatric and a previous neuropsychological study⁹ correspond with the results from the SANTE study⁵ where no significant differences in cognition and mood were found between the stimulated group and the control group at the end of their 3 months blinded phase.⁵ In addition, they found that six out of eight subjects with depression in the blinded phase had a history of diagnosed depression prior to treatment.

Trøster et al¹⁵ published an article analyzing the neurobehavioral adverse events, neuropsychological data and long-term neurobehavioral outcome from the SANTE study. They found that depression and memory-related adverse events were not associated with reliable changes on objective measures or 7-year neurobehavioral outcome. Monitoring and neuropsychological assessment of depression and memory were nevertheless recommended from a theoretical standpoint and because more memory and depression adverse events occurred in the active stimulation group than in the control group.

TABLE 1 Patient characteristics

No	Etiology	Co-morbidity	Seizure semiology	ASMs	EEG	MR cerebrum	Prior surgery
1	Idiopathic	No	FA, FIA, FBTC, ATYPICAL FIA and FBTC	VPA, LTG, CZP	Epileptic activity in several foci, multifocal	Venous angioma, right temporo-parietal	Callosotomy, VNS
2	Idiopathic	Asthma	FA, FIA, FBTC	LAC, LEV, CLB	Theta, right temporal	Postoperative changes	Temporal resection right
3	Idiopathic	No	FA, FIA, FBTC	CBZ, CLB	Low amplitude theta	negative	NO
4	Meningitis, head trauma	No	FIA	LTG, OXC, CLB	Rhythmic theta in central sagittal region, left hemisphere	negative	NO
5	Cortical dysplasia left	No	FA, FIA, FBTC	PB, PHT, CLB	Focal dysrhythmia left temporal	Cortical dysplasia in relation to the Sylvian fissure, left	NO
6	Idiopathic	Encephalopathy	FA, FIA, FBTC	LAC, PB, OXC	Epileptic activity from both hemispheres, general dysrhythmia	Negative	Temporal resection right, VNS
7	Post-traumatic?	No	FA, FIA	LTG, PHT	Possible epileptic activity left temporal	Negative	NO
8	Idiopathic	D. M encephalopathy	FA, FIA, FBTC	VPA, LTG	Suspect epileptic activity in both temporal regions	Negative	NO
9	Idiopathic	No	FA, FIA, FBTC	VPA, LTG	Left temporal activity, possible ictal start in both temporal lobes	Bilateral hippocampal sclerosis	Left temporal resection
10	Posttraumatic/idiopathic	Encephalopathy	FA, FIA, FBTC	VPA, LTG, TPM	Possible ictal start in the left hemisphere, temporolateral	Negative	NO
11	Idiopathic	Hyperlipidemia hypertension	FIA	VPA, LTG	Epileptic activity in both temporal region	Negative	NO
12	Idiopathic	No	FA, FIA, FBTC	CBZ, VPA, LTG	Theta- and epileptic activity in multiple foci	Negative	VNS
13	Postoperative, resection cavity, oligodendroglioma right side	No	FA, FIA, FBTC	OXC, CZP, TPM	Epileptic activity frontocentrally right	Oligodendro-glioma frontally right, gliosis	Resection in 2003 and 2009
14	Idiopathic	No	FA, FIA, FBTC	TPM, LTG	Theta on both sides parietooccipitally	Negative	NO
15	Cerebral paresis, multiple infarctions	Hypertension, cerebral palsy	FIA	OXC, VPA, CLB	Epileptic activity in motor cortex left side	Infarcts, left side behind the central region	Subpial transections centrally left hemisphere
16	Idiopathic, born 10 weeks premature	Thyroiditis	FA, FIA, FBTC	OXC, CZP, TPM	Theta left side, incl epileptic activity on both sides temporally	Negative	VNS
17	Idiopathic	No	FA, FIA, FBTC, atypical FBTC with tonic seizures	FBM, OXC	Seizure starts in both temporal regions	Negative	VNS
18	Idiopathic, encephalopathy	Hypertension	FA, FIA, FBTC, atypical FBTC with tonic seizures	VPA, OXC	Interictal epileptic activity temp right, no focus	Encephalomalacia/gliosis	VNS

Abbreviations: CBZ, carbamazepine; CLB, clobazam; CZP, clonazepam; FA, focal aware seizures; FBTC, focal to bilateral tonic-clonic seizures; FIA, focal impaired awareness seizures; KD, ketogenic diet; LCM, lacosamide; LEV, levetiracetam; LTG, lamotrigine; OXC, oxcarbazepine; PB, phenobarbital; PHT, phenytoin; VNS, vagal nerve stimulation; VPA, valproate.

	<i>n</i>	T1; mean (SD)	T3; mean (SD)	<i>p</i> -Value, paired sample <i>t</i> -test from T1 to T3score
Qualie-89	13	68.3 (10.6)	70.9 (14.8)	<i>p</i> = .46
HADA	13	5.2 (5.1)	3.7 (3.5)	<i>p</i> = .28
HADD	13	2.9 (2.8)	3.0 (2.8)	<i>p</i> = .89
IDS	15	6.3 (6.9)	6.5 (5.8)	<i>p</i> = .91
MADRS	16	5.4 (5.7)	5.3 (5.5)	<i>p</i> = .94

TABLE 2 Neuropsychiatric parameters before (T1) and after (T3) study end at 12 months

Abbreviations: HAD, The Hospital Anxiety and Depression Scale divided into two subscales; one for anxiety - HADA, and one for depression - HADD; IDS, The Inventory of Depressive Symptomatology; MADRS, Montgomery and Åsberg Depression Rating Scale; Qualie-89, An inventory that contains 17 multi-item measures of overall quality of life.

TABLE 3 Effect of DBS during the first 6 months, the randomized controlled trial

	<i>n</i>	T1; mean (SD)	T2; mean (SD)	<i>p</i> -Value, independent sample <i>T</i> -test of the change score
Qualie-89	Off: <i>n</i> = 8	70.6 (9.6)	69.8 (12.3)	<i>p</i> = .15
	On: <i>n</i> = 6	64.8 (10.9)	70.2 (15.8)	
HADA	Off: <i>n</i> = 8	4.1 (4.2)	2.6 (2.8)	<i>p</i> = .55
	On: <i>n</i> = 4	6.8 (7.3)	3.0 (1.4)	
HADD	Off: <i>n</i> = 8	3.1 (3.4)	2.8 (4.0)	<i>p</i> = .62
	On: <i>n</i> = 4	2.3 (1.7)	2.8 (1.3)	
IDS	Off: <i>n</i> = 9	6.4 (8.5)	7.8 (7.6)	<i>p</i> = .40
	On: <i>n</i> = 7	6.1 (3.8)	5.9 (3.8)	
MADRS	Off: <i>n</i> = 10	6.9 (6.9)	5.1 (3.8)	<i>p</i> = .79
	On: <i>n</i> = 7	4.0 (3.2)	2.7 (2.3)	

Abbreviations: HAD, The Hospital Anxiety and Depression Scale divided into two subscales; one for anxiety - HADA and one for depression - HADD; IDS; The Inventory of Depressive Symptomatology; MADRS; Montgomery and Åsberg Depression Rating Scale; Qualie-89; An inventory that contains 17 multi-item measures of overall quality of life.

	<i>n</i>	Before stimulation; mean (SD)	6 months after stimulation; <i>n</i> (SD)	<i>p</i> -Value, paired sample <i>T</i> -test
Qualie-89	14	67.7 (11.6)	69.6 (14.6)	.43
HADA	12	4.0 (4.8)	3.9 (3.4)	.96
HADD	12	2.6 (3.3)	3.3 (2.6)	.28
IDS	15	6.9 (6.3)	6.8 (5.7)	.94
MADRS	16	4.4 (3.5)	4.9 (5.6)	.65

TABLE 4 All patients after 6 months of stimulation

Note: Sum of ON group from T1 to T2 and OFF group from T2 to T3.

Abbreviations: HAD, The Hospital Anxiety and Depression Scale divided into two subscales; one for anxiety - HADA and one for depression - HADD; IDS, The Inventory of Depressive Symptomatology; MADRS, Montgomery and Åsberg Depression Rating Scale; Qualie-89, An inventory that contains 17 multi-item measures of overall quality of life.

In a Finnish study conducted in 2018 by Järvenpää et al.,¹⁶ 22 patients were implanted with ANT-DBS for refractory epilepsy. At group level, no changes in mood were observed during ANT-DBS treatment. Two patients with former histories of depression experienced sudden depressive symptoms related to DBS programming settings. In addition, two patients with no previous histories of psychosis gradually developed paranoid and anxiety symptoms that were relieved after changing the programming settings.

DBS has been a well-established treatment in movement disorders for many years. In a neuropsychiatric study from Oslo Pham

et al.¹⁷ found increased impulsivity after 3 months of subthalamic nucleus stimulation in patients with Parkinson's disease. No other neuropsychiatric changes were found, but they reported better executive function in everyday life.¹⁸ This is in line with our own findings that ANT-DBS may have a positive influence on executive functioning.⁹

As concerns the patient experiencing joy and happiness, every time, the stimulator was activated; this might be explained by stimulation of the dorsal part of the cingulate gyrus through the thalamo-cingulate tract. These structures are part of the circle of

Papez, which is again believed to be crucial for the anatomical and functional basis of ANT-DBS against epilepsy. In a paper from Bijanki et al. from 2018,¹⁹ they performed an awake craniotomy procedure to confirm safe resection margins in the treatment of a patient's epilepsy, at the same time giving direct electrical stimulation to the left dorsal anterior cingulum bundle. Stimulating the dorsal part of anterior cingulum induced acute outward signs of high spirits, subjective reports of happiness and relaxation, and persistent objective behavioral features of positive affect. The cingulum bundle has recently also been suggested to mediate part of the therapeutic effects of DBS for depression.²⁰

Data from the SANTE study showed a gradual improvement in neuropsychological outcome and quality of life from baseline based on the Liverpool seizure severity scale and Qualie-31 scores.²¹ Neuropsychological test composite scores showed statistically significant gains from baseline to 5 years including attention, executive function, depression, tension anxiety, total mood disturbance, and subjective cognitive function. Improvement in neuropsychological parameters paralleled improvement in seizure control.

We found a significant reduction in frequency for all seizures after 6 months of stimulation, but there were no significant neuropsychiatric changes over the same period. Although some of our patients thus did respond positively with reduced seizure frequency, there were only minor differences between patients on neuropsychiatric assessments, indicating no clear relation between effect of the stimulation and neuropsychiatric side effects. However, the number of patients were too small for further statistical evaluation. For the same reason, a possible relation between stimulation site and neuropsychiatric effects cannot be entirely ruled out. Still, in accordance with other studies the results are encouraging and suggest that DBS on a group level is safe with few side effects.²² The mechanisms behind DBS are still enigmatic.²⁰ However, the gradual improvement over years seen in patients treated with ANT-DBS^{22,23} could imply a neuromodulatory effect, an aspect that should be further investigated.

Our patients constitute complex medical challenges including an increased psychic burden which is not obviously recognized. Studies have suggested that the relationship between seizures and depression or suicidal behavior may be bidirectional. People with epilepsy may have undiagnosed and untreated psychiatric illnesses and depression.²⁴ A thorough neuropsychiatric examination is mandatory in epilepsy surgery and should be included also prior to brain stimulation procedures.

AUTHOR CONTRIBUTIONS

HH, ET, ED, KO, and JR-P was involved in study design. HH examined the patients and drafted the manuscript. KO was responsible for the neuropsychiatric interviews and (together with HH) for the statistical analyzes. JR-P, AK, and AE performed the implantations. All authors have been involved in interpretation of data and revision of the manuscript.

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CONFLICT OF INTEREST

No conflicts of interest reported.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

PEER REVIEW

The peer review history for this article is available at <https://publons.com/publon/10.1111/ane.13658>.

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