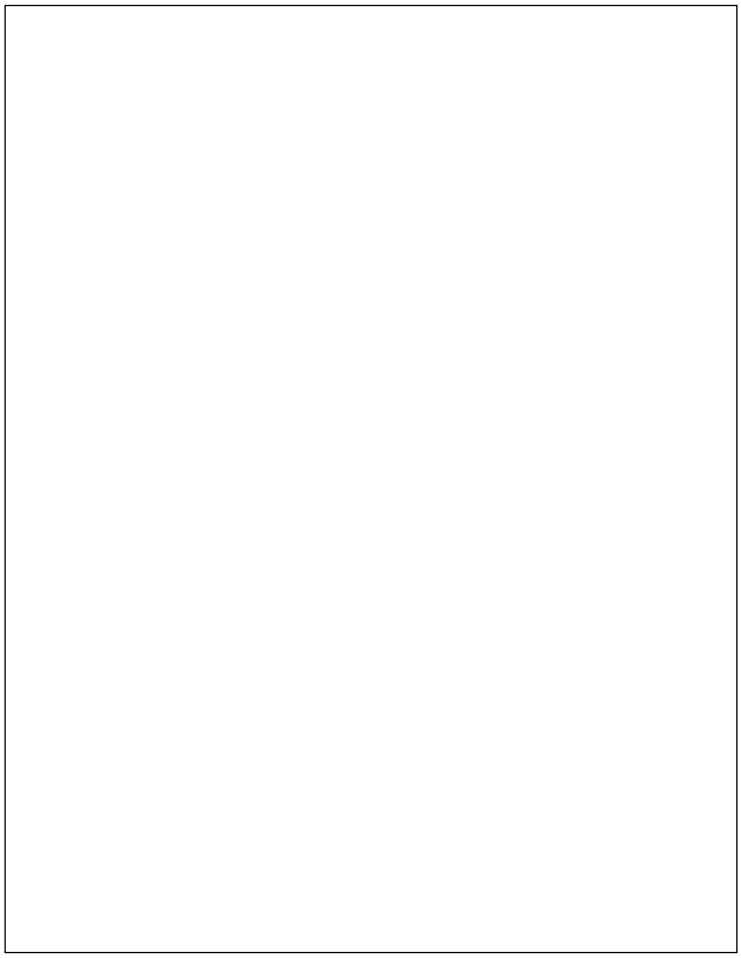
Provided for non-commercial research and education use.
Not for reproduction, distribution or commercial use.



This article appeared in a journal published by Elsevier. The attached copy is furnished to the author for internal non-commercial research and education use, including for instruction at the author's institution and sharing with colleagues.

Other uses, including reproduction and distribution, or selling or licensing copies, or posting to personal, institutional or third party websites are prohibited.

In most cases authors are permitted to post their version of the article (e.g. in Word or Tex form) to their personal website or institutional repository. Authors requiring further information regarding Elsevier's archiving and manuscript policies are encouraged to visit:

http://www.elsevier.com/authorsrights

Author's Personal Copy

Check for updates

# Nucleus Accumbens Deep Brain Stimulation for Obsessive-Compulsive Disorder and Aggression in an Autistic Patient: A Case Report and Hypothesis of the Role of Nucleus Accumbens in Autism and Comorbid Symptoms

***Paresh K. Doshi[1], Anaita Hegde[2], Amit Desai[3]***

**Key words**
- Aggression
- Autism
- Nucleus accumbens deep brain stimulation
- Obsessive-compulsive disorder

**Abbreviations and Acronyms**
**ASD**: Autism spectrum disorder
**DBS**: Deep brain stimulation
**HFS**: High-frequency stimulation
**MD**: Mediodorsal
**MRI**: Magnetic resonance imaging
**NAc**: Nucleus accumbens
**OCD**: Obsessive-compulsive disorder
**OFC**: Orbitofrontal cortex
**VS**: Ventral striatum
**YBOCS**: Yale-Brown Obsessive Compulsive Scale

*From the Departments of [1]Neurosurgery, [2]Pediatrics, and [3]Psychiatry, Jaslok Hospital and Research Centre, Mumbai, India*

*To whom correspondence should be addressed: Paresh K. Doshi, M.Ch.*
*[E-mail: pareshkd@gmail.com]*

*Citation: World Neurosurg. (2019) 125:387-391.*
*https://doi.org/10.1016/j.wneu.2019.02.021*

*Journal homepage: **www.journals.elsevier.com/world-neurosurgery***

*Available online: **www.sciencedirect.com***

*1878-8750/$ - see front matter © 2019 Published by Elsevier Inc.*

BACKGROUND: **Autism spectrum disorder represents a set of developmental disorders characterized by lack of social interaction and verbal and nonverbal communication in the first 3 years of life. It is also associated with several comorbidities, including epilepsy, aggression, self-mutilating behavior, and obsessive-compulsive behavior. In some cases, obsessive-compulsive disorder (OCD) develops. The nucleus accumbens (NAc) plays a key role in reward circuitry and is involved in the control of OCD and aggression.**

CASE DESCRIPTION: **A 42-year-old woman with autism was offered NAc deep brain stimulation for her comorbidities of OCD and aggression. The NAc was targeted using standard stereotactic methods, and postoperative scans confirmed the position of the active electrode to be within the NAc. The patient experienced significant symptom relief. At 1-year follow-up, the Yale-Brown Obsessive Compulsive Scale score for OCD, excluding items 1–5 of the scale, improved from 19 to 5. Hamilton Depression Scale and Hamilton Anxiety Scale scores similarly improved from 20 to 15 and from 30 to 18, respectively. Social Communication Questionnaire Current version for autism score improved from 26 to 16. Subscores for reciprocal social interactionimproved from 13 to 8; for communication improved from 5 to 4; and for restricted, repetitive, and stereotyped patterns of behavior improved from 6 to 3.**

CONCLUSIONS: **This case report illustrated the role of the NAc in OCD and aggression in an autistic patient. We discussed the role of the NAc as a target to explain the outcome of this case.**

## INTRODUCTION

Autism spectrum disorder (ASD) represents a set of developmental disorders characterized by lack of social interaction and verbal and nonverbal communication in the first 3 years of life.[1] The prevalence rate of autism has been reported as 1%–2% in various studies.[2] DSM-5 adopted the term ASD with a dyadic definition of core symptoms as early onset of difficulties in social interaction and communication and repetitive, restricted behavior, interests, or activities.[3] Two disturbing comorbidities of ASD are obsessive-compulsive behavior and self-injurious behavior with a reported incidence of 17% and 32%, respectively.[4,5] Other comorbidities include epilepsy, gastrointestinal disturbances, immune dysregulation, and mood disorders.[6] Medical management includes antipsychotics, mood stabilizers, sedatives, $\alpha_2$-adrenergics, and behavioral therapy. Despite aggressive treatment, some patients remain refractory.[7]

The nucleus accumbens (NAc) and basolateral amygdala are a part of the circuit involved in the expression of autism and related behavioral symptoms.[8] Deep brain stimulation (DBS) of these structures has been tried in 4 cases of ASD—1 case in amygdala, 2 cases in globus pallidus internus, and 1 case in NAc—with limited success.[9–11] NAc DBS has also been used to control obsessive-compulsive disorder (OCD) and aggression. We offered NAc DBS to a patient with autism to control her aggression and OCD and were surprised to find improvement in her autistic symptoms. We present this case as an example to understand the complex role of the NAc in autism.

## CASE DESCRIPTION

This case presentation was approved by the scientific committee and the ethics committee of our institute (project code 857). Informed consent was obtained from the caretaker and legal guardian after explaining the procedure in detail in accordance with the recommendations of the task force on neurosurgery for psychiatric disorders.[12]

We report a 42-year-old woman with autism who underwent bilateral NAc DBS for control of OCD and aggression. The patient had normal developmental

Author's Personal Copy



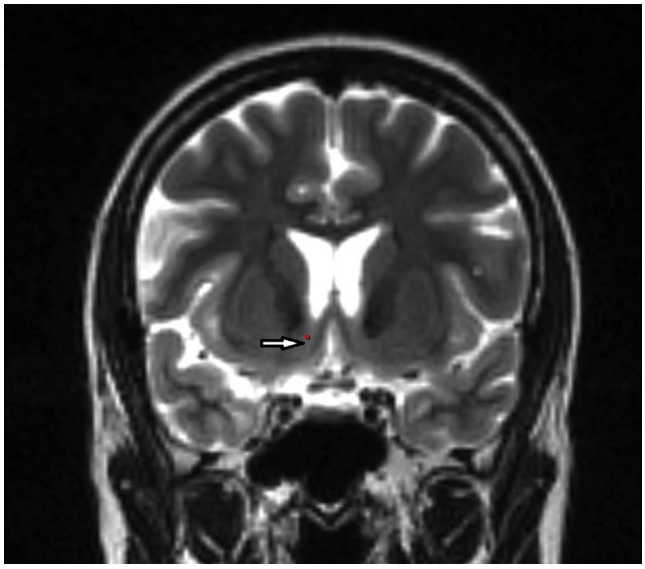


**Figure 1.** Magnetic resonance imaging T2-weighted sequence showing the left nucleus accumbens target (*arrow*).

milestones up to the age of 3 years; thereafter, she stopped communicating by words. Autism was diagnosed at the age of 5 years. Until the age of 21 years, she attended a special school. During this time, she was almost nonverbal, although occasionally she would speak 1 or 2 words. Her relative noted that she understood English, Hindi, and Punjabi languages and had the capability of speaking but did not use it.

At an early age, the patient began showing signs of obsessive-compulsive behavior. This was in the form of organizing her toys, purposefully making a mess of things, and refusing to be toilet trained. In her late 30s and early 40s, she started developing changes in behavior patterns reflecting symptoms of OCD and aggression, including focusing entirely on her defecation; checking out toilets; incessantly switching on and off lights of the house; and having violent outbursts against family members, caregivers, and herself, including hitting and injuring others and herself. She also developed a voracious appetite and stubbornly refused to be toilet trained. At the age of 40 years, she had 3 episodes of generalized seizures at monthly intervals. She also developed rest tremors in both upper limbs. By the time she presented to us, she was on several drugs, including aripiprazole, quetiapine, haloperidol, guanfacine hydrochloride, lamotrigine, alprazolam, propranolol, vitamin $B_6$, and magnesium. Clomipramine made her hyperactive and had to be stopped. The treating psychiatrists had confirmed that she was not responsive to 2 selective serotonin reuptake inhibitors. Because of her autism, she could not manage any cognitive or behavioral therapy; hence she fit the criteria for refractory OCD surgery. The seizure disorder and rest tremors were considered to be side effects of her psychiatric medications.

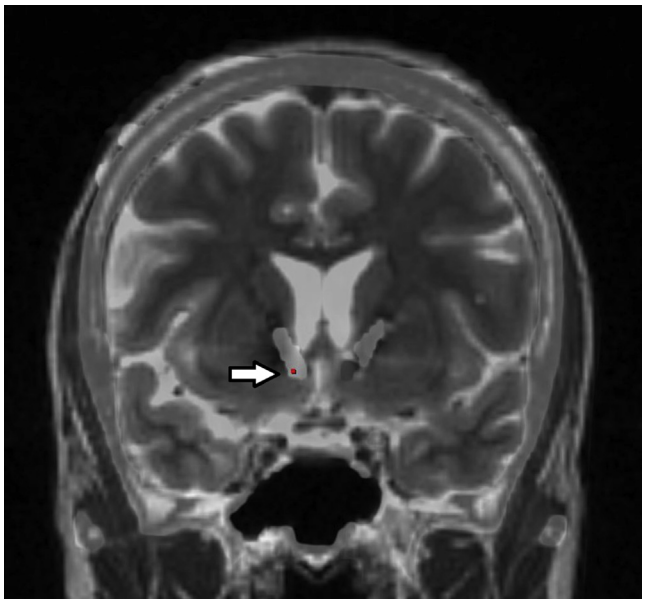


**Figure 2.** Postoperative computed tomography scan fused with preoperative magnetic resonance imaging. The *arrow* pointing to the dot indicates the active contact point.

On admission, the patient seemed to be composed, attentive, and listening to all conversations. She avoided making eye contact. She would put things in order if not kept properly. She was noted to have continuous compulsive acts and if stopped would become irritated. There were no features suggestive of her experiencing hallucinations or paranoid delusion. She had to be restrained or would go out and check the doors of the rooms in the ward. Magnetic resonance imaging (MRI) of the brain was normal. She underwent neuropsychological and psychiatric evaluations 3 days before surgery. After evaluation by a multidisciplinary team comprising a neurologist, psychiatrist, psychologist, neurosurgeon, and specialist nurse, it was decided to offer her NAc DBS. The NAc was targeted based on fusion of T1-weighted and T2-weighted MRI (Figure 1) and stereotactic computed tomography. The coordinates for the right and left NAc were 6.38 and 6.58 lateral to the anterior commissure, 3.51 and 4.13 anterior to the anterior commissure, and 4.71 and

Author's Personal Copy



3.74 below the anterior commissure—posterior commissure plane. Postoperative computed tomography scan confirmed that 1 of the stimulating contact points was in the NAc on both sides (Figure 2). We started the stimulation on the third day postoperatively. The stimulation parameters at the last visit for the right and left leads were 60 μs pulse width, 130 Hz frequency, and 2.6V/2.6V current. The electrode combination was 1&2(−)3(+) and 9&10(−)11(+) for the left and right electrodes, respectively, with bipolar settings on both sides. These were the parameters that we found to be effective in other cases of NAc stimulation for OCD.

In the days immediately after surgery, the patient showed a marked difference in her behavior. She became more manageable and gradually started urinating in the toilet. She started visiting malls and movie theaters with the family. She stopped checking the doors and toilets wherever she visited. On day 12 after surgery, her mother reported that she did "namaste," a form of thank you, to a waiter who brought food to her; the patient had not been seen to do this gesture since she was 3 years old. She started developing eye contact and smiled if happy. She was able to concentrate and meditate for 1 hour at the temple, which she did not do before the DBS surgery. Her mother stated, "My daughter has become very expressive of her opinions. She is becoming her own advocate in life. She has not hit anyone or herself since the surgery."

This behavioral change was reflected in changes in various rating scale scores. Her Yale-Brown Obsessive Compulsive Scale (YBOCS) score could be only partially completed, as she was almost mute. Her YBOCS score, excluding items 1–5 of YBOCS, improved from 19 preoperatively to 5 at 1-year follow-up. The scores for additional YBOCS items of 11–16 was 17. This is more relevant in this patient, as it reflects the patient's behavior. The score for YBOCS items 11–16 improved to 5, and score for item 18, which reflects the response to the treatment, was rated as 5 out of a total score of 6, indicating "much improved." Her Hamilton Depression Scale and Hamilton Anxiety Scale scores similarly improved from 20 to 15 and from 30 to 18, respectively. Her Social Communication Questionnaire Current version for autism score improved from 26 to 16. Subscores for reciprocal social interaction improved from 13 to 8; for communication improved from 5 to 4; and for restricted, repetitive, and stereotyped patterns of behavior improved from 6 to 3.

## DISCUSSION

### Autism and Reward

Most interventions targeting social communicative skill defects in ASD rely on the principles of applied behavior analysis, where desired behaviors are reinforced using a variety of rewards.[13] Stimulus-reward association learning that persists through adulthood has been repeatedly highlighted as an area of difficulty for children with ASD.[14]

Reward comprises of 2 components—one is liking and the other is wanting. Wanting is the anticipatory drive component of the reward, whereas liking is related to the pleasurable effect of the reward. In autistic patients, the wanting component of the reward mechanism is deficient. Separate neurobiologic and neurochemical substrates have been identified to be responsible for these responses. Ventral striatum (VS), including NAc, is the central hub that receives major afferents from the orbitofrontal cortex (OFC), anterior cingulate cortex (ACC), and medial temporal lobe, including amygdala. Dopamine is the neurotransmitter predominately associated with reward processing.[15,16] Functional MRI studies found recruitment of VS and NAc in response to monetary reward cues, suggesting that VS, particularly the NAc, functions as a general, modality-independent mediator of reward wanting.[17] Reward liking, by contrast, has been primarily associated with activations in medial prefrontal cortex, particularly the medial OFC and the ventral anterior cingulate cortex.[18] Four of 5 functional MRI studies reported diminished VS activation in patients with ASD when processing either social or monetary reward versus nonreward.[8] Preliminary evidence indicates that the motivational component of reward (the wanting component) may be particularly compromised in individuals with ASD. This is reflected in blunted VS activity. This indicates the VS and NAc are key structures in the dysfunctional circuit in patients with ASD.[19]

### Obsessive-Compulsive Behavior and OCD

OCD and restricted and repetitive patterns of behavior, interests, and activities inherent to ASD share a series of characteristics that can make their differential diagnosis extremely difficult. Differentiation between the 2 disorders is highly important at the clinical level owing to treatment implications. In ASD, the restricted and repetitive patterns of behavior or activities represent a pleasurable affective experience, intrinsically motivating and reinforcing. In patients with ASD, these patterns of behavior are egosyntonic; that is, individuals feel good about themselves when they perform the behavior, without the behavior generating any conflict or negative judgment about themselves, and the behavior even becomes a source of pleasure and satisfaction, intrinsically motivating and reinforcing.[20,21] In OCD, obsessions and compulsions are egodystonic. That is, they are perceived as intrusive, unwanted, and unpleasant. DSM-IV-TR clarifies that obsessions and compulsions "provoke significant clinical discomfort" in individuals who suffer from them. Therefore, it is important to determine the amount of anxiety associated with these symptoms, as in OCD, they will cause severe anxiety to resist them or put them away.

Our patient had obsessive-compulsive behavior symptoms from childhood in the form of organizing toys and creating a purposefull mess, especially uprooting plants. It was not until the age of 25 years that the symptoms changed to be more severe and disturbing and the patient had to be started on drug therapy. The symptoms initially responded to treatment, but later, despite treatment, the symptoms changed, as described earlier, and were present most of the time. The patient was aware of her symptoms and would express anguish (through her facial expression) over her behavior.

### NAc as a Target

The NAc is considered to be a key structure related to social reward response in ASD.[22-24] Scott-Van Zeeland et al.[19] found that the activity within the VS was decreased in autistic patients, and this

Author's Personal Copy



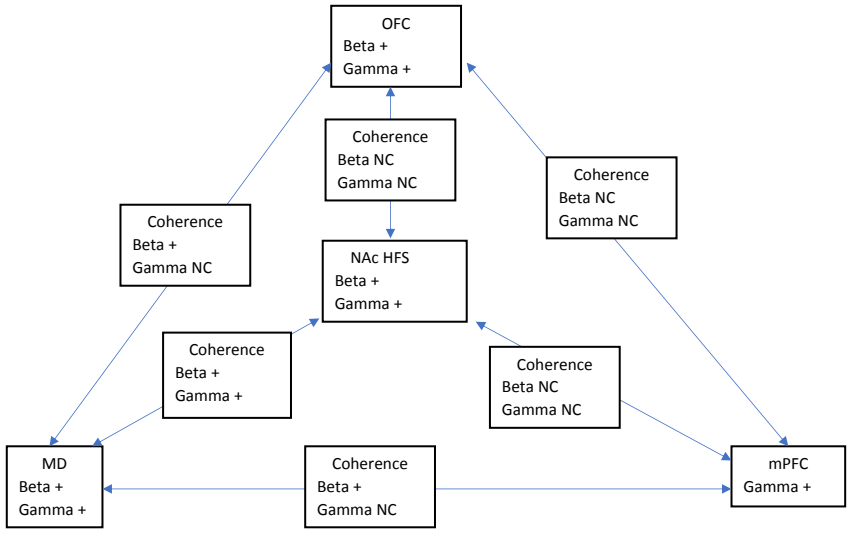



**Figure 3.** Changes in local field potentials and coherence produced by high-frequency stimulation of nucleus accumbens. OFC, orbitofrontal cortex; NC, no change; NAc, nucleus accumbens; HFS, high-frequency stimulation; MD, mediodorsal; mPFC, medial prefrontal cortex; +, increase.

may be the reason for impaired social reciprocity. These observations make NAc a potential target to modulate in ASD.

NAc shell high-frequency stimulation (HFS) also increased concentrations of NAc dopamine and serotonin, which are shown to be responsible for reducing the symptoms of OCD.[25] McCracken and Grace[26] showed that HFS of NAc core increased beta power (13–30 Hz) in OFC and mediodorsal (MD) thalamus and spontaneously increased gamma power (30–70 Hz) in OFC, medial prefrontal cortex, and MD thalamus. HFS additionally increased frequency-dependent (high frequency) beta coherence between MD thalamus and OFC, medial prefrontal cortex, and NAC and gamma coherence between OFC-PFC and OFC-MD thalamus. These fast oscillations are generated by synchronization in inhibitory interneuron networks, leading to a postulation that NAc HFS may generate therapeutic effects via synchronization of inhibition in dysfunctional OCD circuits.[27] The widespread and specific alterations in synchronous local field potential activity produced by HF NAC DBS provide further evidence that the therapeutic actions of DBS depend not only on the activity in stimulated nucleus but also are due to widespread changes along the thalamocortical circuit (Figure 3).[26] The therapeutic proof of the role of NAc is further obtained from a number of case reports that have documented benefit of stimulation of VS, including NAc in patients with OCD. This benefit seems to persist even with blinding to stimulation state and in larger randomized trials.[28-30] Hence, NAc forms one of the important targets for the control of OCD symptoms.

Amygdala has been a target to treat aggressive behavior for >50 years. Narabayashi and Uno[31] performed bilateral amygdalotomy to control epilepsy and aggression. Strum et al. performed DBS in the basolateral amygdala and found improvement in self-injurious behavior and autism symptoms in 1 patient.[10] The NAc is the main input nucleus of basal ganglia, receiving direct inputs from subiculum and amygdala, hippocampus, thalamus, and prelimbic and prefrontal cortex and indirect inputs from ventral tegmental area and substantia nigra.[32,33] It has been used as a target for DBS to control aggression and has been shown to reduce aggression when performed for other indications.[34,35] This makes NAc another target to modulate aggression.

In the present case, the stimulation parameters had a wide field of coverage, as only 1 of the 3 contact points used was in the NAc. Moreover, similar results have been obtained by stimulation of white matter tracts of anterior limb of internal capsule. Data suggest that the common element is activation of the descending fibers that either traverse or end in NAc.[26,36] The aforementioned concepts are further strengthened by the clinical improvements seen in our patient. The change in social behavior was striking and rewarding for the family. The response to OCD and aggression were in line with the expected role of the NAc and its response to similar symptoms in other diseases. However, the response for the autistic symptoms was unexpected, as the only report of NAc DBS in autism had no details about such a response except subjective description.[9]

Park et al.[9] reported NAc DBS in autism and aggression. They reported a 40% (subjective) decrease in self-injurious behavior and improved symptoms of autism in the form of eye contact and expression and comprehension of language skills. They also found that the cortical gray matter volume decreased and the intracranial volume increased in this patient at 2-year follow-up.

### Limitations

ASD is a complex disorder, with several theories explaining the pathogenesis. This is only the second case report of NAc DBS for autism, and hence this experience should be treated as experimental unless confirmed by larger studies. Another limitation is that we could not perform a sham stimulation in this patient, and the possibility of an additional placebo effect should be kept in mind.

## CONCLUSIONS

The present case report reviews the key role that NAc may play in OCD and aggression in patients with autism. The positive response to NAc DBS in improving the symptoms of autism opens up the possibility of further research in this area.

## ACKNOWLEDGMENTS

The authors thank Jaslok Hospital and Research Centre for supporting this work and Ms. Bharati Karkera for helping in the collection of data for the article.

Author's Personal Copy



## REFERENCES


1. Just MA, Cherkassky VL, Buchweitz A, Keller TA, Mitchell TM. Identifying autism from neural representations of social interactions: neurocognitive markers of autism. *PLoS One*. 2014;9:e113879.
2. Mattila ML, Kielinen M, Linna SL, et al. Autism spectrum disorders according to DSM-IV-TR and comparison with DSM-5 draft criteria: an epidemiological study. *J Am Acad Child Adolesc Psychiatry*. 2011;50:583-592.e511.
3. First MB. Diagnostic and Statistical Manual of Mental Disorders, 5th edition, and clinical utility. *J Nerv Ment Dis*. 2013;201:727-729.
4. Adler BA, Wink LK, Early M, et al. Drug-refractory aggression, self-injurious behavior, and severe tantrums in autism spectrum disorders: a chart review study. *Autism*. 2015;19:102-106.
5. van Steensel FJ, Bogels SM, Perrin S. Anxiety disorders in children and adolescents with autistic spectrum disorders: a meta-analysis. *Clin Child Fam Psychol Rev*. 2011;14:302-317.
6. Berney TP. Autism—an evolving concept. *Br J Psychiatry*. 2000;176:20-25.
7. Richards C, Oliver C, Nelson L, Moss J. Self-injurious behaviour in individuals with autism spectrum disorder and intellectual disability. *J Intellect Disabil Res*. 2012;56:476-489.
8. Kohls G, Chevallier C, Troiani V, Schultz RT. Social "wanting" dysfunction in autism: neurobiological underpinnings and treatment implications. *J Neurodev Disord*. 2012;4:10.
9. Park HR, Kim IH, Kang H, et al. Nucleus accumbens deep brain stimulation for a patient with self-injurious behavior and autism spectrum disorder: functional and structural changes of the brain: report of a case and review of literature. *Acta Neurochir*. 2017;159:137-143.
10. Sturm V, Fricke O, Buhrle CP, et al. DBS in the basolateral amygdala improves symptoms of autism and related self-injurious behavior: a case report and hypothesis on the pathogenesis of the disorder. *Front Hum Neurosci*. 2012;6:341.
11. Stocco A, Baizabal-Carvallo JF. Deep brain stimulation for severe secondary stereotypies. *Parkinsonism Relat Disord*. 2014;20:1035-1036.
12. Nuttin B, Wu H, Mayberg H, et al. Consensus on guidelines for stereotactic neurosurgery for psychiatric disorders. *J Neurol Neurosurg Psychiatry*. 2014;85:1003-1008.
13. Virues-Ortega J. Applied behavior analytic intervention for autism in early childhood: meta-analysis, meta-regression and dose-response meta-analysis of multiple outcomes. *Clin Psychol Rev*. 2010;30:387-399.
14. Dawson G, Osterling J, Rinaldi J, Carver L, McPartland J. Brief report: Recognition memory and stimulus-reward associations: indirect support for the role of ventromedial prefrontal dysfunction in autism. *J Autism Dev Disord*. 2001;31:337-341.
15. Schultz W. Multiple dopamine functions at different time courses. *Annu Rev Neurosci*. 2007;30:259-288.
16. Schultz W. Predictive reward signal of dopamine neurons. *J Neurophysiol*. 1998;80:1-27.
17. Knutson B, Taylor J, Kaufman M, Peterson R, Glover G. Distributed neural representation of expected value. *J Neurosci*. 2005;25:4806-4812.
18. Grabenhorst F, Rolls ET. Value, pleasure and choice in the ventral prefrontal cortex. *Trends Cogn Sci*. 2011;15:56-67.
19. Scott-Van Zeeland AA, Dapretto M, Ghahremani DG, Poldrack RA, Bookheimer SY. Reward processing in autism. *Autism Res*. 2010;3:53-67.
20. Paula-Perez I. Differential diagnosis between obsessive compulsive disorder and restrictive and repetitive behavioural patterns, activities and interests in autism spectrum disorders. *Rev Psiquiatr Salud Ment*. 2013;6:178-186.
21. Klin A, Saulnier CA, Sparrow SS, Cicchetti DV, Volkmar FR, Lord C. Social and communication abilities and disabilities in higher functioning individuals with autism spectrum disorders: the Vineland and the ADOS. *J Autism Dev Disord*. 2007;37:748-759.
22. Neto LL, Oliveira E, Correia F, Ferreira AG. The human nucleus accumbens: where is it? A stereotactic, anatomical and magnetic resonance imaging study. *Neuromodulation*. 2008;11:13-22.
23. Gasic GP, Smoller JW, Perlis RH, et al. BDNF, relative preference, and reward circuitry responses to emotional communication. *Am J Med Genet B Neuropsychiatr Genet*. 2009;150B:762-781.
24. Dichter GS, Richey JA, Rittenberg AM, Sabatino A, Bodfish JW. Reward circuitry function in autism during face anticipation and outcomes. *J Autism Dev Disord*. 2012;42:147-160.
25. Sesia T, Bulthuis V, Tan S, et al. Deep brain stimulation of the nucleus accumbens shell increases impulsive behavior and tissue levels of dopamine and serotonin. *Exp Neurol*. 2010;225:302-309.
26. McCracken CB, Grace AA. Nucleus accumbens deep brain stimulation produces region-specific alterations in local field potential oscillations and evoked responses in vivo. *J Neurosci*. 2009;29:5354-5363.
27. Traub RD, Bibbig A, LeBeau FE, Buhl EH, Whittington MA. Cellular mechanisms of neuronal population oscillations in the hippocampus in vitro. *Annu Rev Neurosci*. 2004;27:247-278.
28. Nuttin B, Cosyns P, Demeulemeester H, Gybels J, Meyerson B. Electrical stimulation in anterior limbs of internal capsules in patients with obsessive-compulsive disorder. *Lancet*. 1999;354:1526.
29. Nuttin BJ, Gabriels LA, Cosyns PR, et al. Long-term electrical capsular stimulation in patients with obsessive-compulsive disorder. *Neurosurgery*. 2003;52:1263-1272 [discussion: 1272-1274].
30. Denys D, Mantione M, Figee M, et al. Deep brain stimulation of the nucleus accumbens for treatment-refractory obsessive-compulsive disorder. *Arch Gen Psychiatry*. 2010;67:1061-1068.
31. Narabayashi H, Uno M. Long range results of stereotaxic amygdalotomy for behavior disorders. *Confin Neurol*. 1966;27:168-171.
32. Mogenson GJ, Jones DL, Yim CY. From motivation to action: functional interface between the limbic system and the motor system. *Prog Neurobiol*. 1980;14:69-97.
33. Lopes da Silva FH, Arnolds DE, Neijt HC. A functional link between the limbic cortex and ventral striatum: physiology of the subiculum accumbens pathway. *Exp Brain Res*. 1984;55:205-214.
34. Harat M, Rudas M, Zielinski P, Birska J, Sokal P. Deep brain stimulation in pathological aggression. *Stereotact Funct Neurosurg*. 2015;93:310-315.
35. Tomycz ND, Whiting DM, Oh MY. Deep brain stimulation for obesity—from theoretical foundations to designing the first human pilot study. *Neurosurg Rev*. 2012;35:37-42 [discussion: 42-43].
36. van den Munckhof P, Bosch DA, Mantione MH, Figee M, Denys DA, Schuurman PR. Active stimulation site of nucleus accumbens deep brain stimulation in obsessive-compulsive disorder is localized in the ventral internal capsule. *Acta Neurochir Suppl*. 2013;117:53-59.


*Conflict of interest statement: The authors declare that the article content was composed in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.*

*Received 18 December 2018; accepted 12 February 2019*

*Citation: World Neurosurg. (2019) 125:387-391.*
*https://doi.org/10.1016/j.wneu.2019.02.021*

*Journal homepage:* ***www.journals.elsevier.com/world-neurosurgery***

*Available online:* ***www.sciencedirect.com***

*1878-8750/$ - see front matter © 2019 Published by Elsevier Inc.*