HHS Public Access

Author manuscript

Curr Opin Anaesthesiol. Author manuscript; available in PMC 2016 October 01.

Published in final edited form as:

Curr Opin Anaesthesiol. 2015 October ; 28(5): 505–510. doi:10.1097/ACO.0000000000000230.

Anaesthesia for deep brain stimulation: a review

Ryan Grant[a], **Shaun E. Gruenbaum**[b], and **Jason Gerrard**[a]

[a]Department of Neurosurgery, Yale School of Medicine, Yale-New Haven Medical Center, New Haven, Connecticut, USA

[b]Department of Anesthesia, Yale School of Medicine, Yale-New Haven Medical Center, New Haven, Connecticut, USA

Abstract

Purpose of review—Deep brain stimulation (DBS) is a well tolerated and efficacious surgical treatment for movement disorders, chronic pain, psychiatric disorder, and a growing number of neurological disorders. Given that the brain targets are deep and small, accurate electrode placement is commonly accomplished by utilizing frame-based systems. DBS electrode placement is confirmed by microlectrode recordings and macrostimulation to optimize and verify target placement. With a reliance on electrophysiology, proper anaesthetic management is paramount to balance patient comfort without interfering with neurophysiology.

Recent findings—To achieve optimal pain control, generous amounts of local anaesthesia are instilled into the planned incision. During the opening and closing states, conscious sedation is the prevailing method of anaesthesia. The preferred agents are dexmedetomidine, propofol, and remifentanil, as they affect neurocognitive testing the least, and shorter acting. All the agents are turned off 15–30 min prior to microelectrode recording. Dexmedetomidine has gained popularity in DBS procedures, but has some considerations at higher doses. The addition of ketamine is helpful for pediatric cases.

Summary—DBS is a robust surgical treatment for a variety of neurological disorders. Appropriate anaesthetic agents that achieve patient comfort without interfering with electrophysiology are paramount.

Keywords

anaesthesia; DBS; deep brain stimulation; macrostimulation; microelectrode recording

Correspondence to Jason Gerrard, MD, PhD, Chief of Functional and Stereotactic Neurosurgery, Yale-New Haven Hospital, Yale University School of Medicine, Tompkins Memorial Pavilion, 789 Howard Avenue, 4th Floor New Haven, CT 06519, USA. Tel: +1 203 785 4891; jason.gerrard@yale.edu.

Financial support and sponsorship

None.

Conflicts of interest

J.L.G. has received honoraria as a consultant from Medtronic. For the remaining authors no conflicts of interest were declared.

INTRODUCTION

Deep brain stimulation (DBS) is an efficacious surgical treatment for a variety of conditions including movement disorders [1], chronic pain [2■], and psychiatric disorders [3]. The brain targets are deep and small in size, usually requiring a frame to increase accuracy via a coordinate system [4], as well as intraoperative electrophysiological guidance using microelectrode recording (MER) and macrostimulation testing to verify that symptoms improve with minimal side-effects [5]. Given that most centers perform DBS awake, proper anaesthetic management is paramount to optimize microelectrode recordings and stimulation testing. However, the effects of anaesthetic agents on the neuronal activity of various target nuclei are unknown. In addition, these can be challenging anaesthesia cases because these patients frequently have multiple medical problems, which compound the necessity of maintaining patient comfort and optimizing neurophysiological recordings. Here we review the latest DBS anaesthetic management, as well as preferred agents and an update on the expanding experience with dexmedetomidine in DBS.

THERAPEUTIC TARGETS FOR DEEP BRAIN STIMULATION

The most common targets for movement disorders are the subthalamic nucleus (STN) [6], globus pallidus interna (GPi) [6], and the ventralis intermedius nucleus of the thalamus [7]. For psychiatric disorders, such as obsessive-compulsive disorder, targets include the subcallosal cingulate gyrus [8], the anterior limb of the internal capsule [9], and the nucleus accumbens [10■]. There are several other targets that are Food and Drug Administration-approved or off-label/experimental for a variety of conditions including epilepsy, depression, Tourette syndrome, headache, obesity, Alzheimer's disease, and the minimally conscious state, which are beyond the scope of this short review. Obviously, the intended target is chosen to achieve the best therapeutic effect and anaesthetic agents must be chosen that give the least interference with microelectrode recordings, while at the same time promoting patient comfort.

BRIEF SURGICAL TECHNIQUE

DBS implantation is performed in a single or two-stage operation. In the latter, the electrode is implanted in the brain separately from the pulse generator given concerns for increased infection risk. After the stereotactic frame is placed, an MRI or conputed tomography is obtained for localization and the appropriate target coordinates are chosen based on the anatomical imaging using computer software. The patient's frame is then attached to the operating room table and a geometric arc is placed to allow the target to be achieved from any angle given a stable radius. From here, a skin incision is planned, local anaesthetic placed, a burr hole drilled, and finally the dura excised. The intraoperative electrode(s) are inserted into the brain and brought to a location 10–25 mm above the target site and advanced in 0.5–1 mm increments along the planned trajectory, with the recordings used to confirm accurate localization of the therapeutic target via microelectrode recording and macroelectrode stimulation. More specifically, the variations in spontaneous background firing, spike discharges, and movement-related changes in fire rates confirm electrode path and optimal target implantation [5].

ANAESTHETIC MANAGEMENT AND IMPLICATIONS

A diversity of techniques have been reported to provide good operative conditions in the awake state while maintaining the airway. Anaesthesia has ranged from conscious sedation utilizing propofol and/or dexmedetomidine, along with small amounts of remifentanil, to general anaesthesia with endotracheal intubation, utilizing IV or inhalation agents [5]. The two major conscious sedation techniques are asleep-awake-asleep (AAA) and monitored anaesthetic care (MAC) with sedation [11]. The former is usually not used with adult DBS as it requires general anaesthesia, but is commonplace in the pediatric population [12■■] given a more challenging age, inability to cooperate, as well as emotional instability. Furthermore, the most common indication for DBS in the pediatric population is poorly controlled dystonia, which makes it difficult to tolerate portions of an awake procedure [12■■]. Regardless, awake techniques provide the best conditions for intraoperative neurophysiology and stimulation testing. Nevertheless, we should note that some providers perform the entire DBS implantation under general anaesthesia, while reporting appropriate microelectrode recordings; however, no stimulation testing can be conducted [11]. In our experience, conscious sedation techniques, with all medications withheld for at least 15 min prior to MER, provides the most reliable and robust data to assist accurate DBS electrode placement.

Additionally, blood pressure control is paramount during DBS surgery and it must be noted that many diseases that require DBS can present with autonomic dysfunction, such as orthostatic hypotension, excessive sweating, constipation, incontinence, and autonomic instability that can lead to sudden, exaggerated, or uncertain responses to central nervous system blockade. This potential autonomic instability needs to be kept in the forefront of the clinicians' minds so as to not be caught off guard. Similarly, respiratory dysfunction for uncoordinated involuntary muscle movement is also possible, as well as gastrointetstinal symptoms that can result in nausea and vomiting [13]. Given this possibility, some practitioners recommend preoperative acid aspiration prophylaxis [13]. In the end, all of these potential symptoms from the variety of disease states can complicate anaesthesia and appropriate preparations should be made.

Premedication

Given that DBS patients have unique neurodegeneration or aberrant pathways, anaesthetic agents must be chosen appropriately to achieve anxiolysis without oversedation (Table 1) [11,12■■,13–17,18■■, 19–22]. For blood pressure control, beta-blockers are avoided as they can mask or complicate intraoperative tremor testing. Clonidine can be helpful, as it is less likely to induce cognitive impairment [11]. Midazolam can help prevent nausea, but it can easily cause oversedation in this population [11]. Furthermore, benzodiazinepines can produce a paradoxical agitation and this has led to some anaesthesiologists not giving any sedative premedication [11]. Droperidol must be avoided in patients with Parkinson's disease and dystonia, given the potential for exacerbation of symptomatology [11]. For nausea and vomiting, metoclopramide blocks dopamine receptors and can cause extrapyramidal side-effects and is not the best first-line agent in patients with movement disorders. Ondansetron is a better first-line choice, as well as dexamethasone [14].

Local

For awake DBS implantation, local anaesthesia with or without a scalp block is an absolute must to aid in patient comfort and critical to minimize opioid and sedative requirements. Long-acting local anaesthetics are helpful if available, such as bupivacaine (0.5%), levobupivacaine (0.5%), and ropivacaine (0.75%), which all should be supplemented with epinephrine to reduce systemic absorption and to elongate duration [23]. Furthermore, adequate scalp blocks are known to improve recovery profiles and postoperative pain [24]. We prefer the combination of rapid onset and long-acting local anaesthesia by combining bupivacaine (0.5%) and lidocaine (1%). In addition, bicarbonate can be added to the local anaesthetic to decrease the initial burning discomfort as the injection is initiated. The local anaesthetic is placed in the pin sites prior to frame placement at the beginning of the procedure. After imaging is obtained and the patient is positioned on the operating table, the scalp block is placed with a wide margin around the planned incision. If needed, the dura can be anaesthestized as well. Reinfiltration at closure can be utilized if there is discomfort reported during the late stages of the surgery. These local anaesthetic techniques are supplemented with sedation to facilitate the scalp opening and burr hole(s).

Monitored anaesthesia care

MAC with sedation is the most common technique employed during DBS surgery with sedation during the opening, mainly for drilling the burr hole and the closing portions. The airway is not manipulated and oxygen by nasal cannula or facemask is employed. The preferred agents are those that affect neurocognitive testing the least, provide pain relief, and are short-acting, such as propofol, remifentanil, and dexmedetomidine (Table 1). Boluses and infusions of propofol and fentanyl or remifentanil have generally been used in the past, with a shift toward using dexmedetomidine. Patients are induced with propofol and a short-acting opioid, such as fentanyl, remifentanil, sufentanil, or alfentanil, with propofol doses ranging from 30 to 180 μg/kg/min [11]. It is important to note that in Parkinson's patients, lower-than-average propofol doses are needed, given the neurodegenerative disease [16]. For pain control, remifentanil dosing of 0.03–0.09 μg/kg/ min is often employed during opening and closing [11]. All infusions are turned off after the burr hole is made, at least 15 min before MER begins. Although some practitioners have reported that they can keep running low-dose propofol or dexmedetomidine without MER interference, it is our practice to turn off all infusions. If necessary, a low dose of remifentanil or dexmedetomidine throughout the awake portion of the case can be continued; doses range from 0.005 to 0.01 μg/kg/min for remifentanil and 0.02to 0.5 μg/kg/hr for dexmedetomidine [11,21]. Some groups have reported ongoing infusions of dexmedetomidine of 0.2–0.8 μg/kg/h with preservation of movement disorder symptoms and electrode recordings during the entire case [11,20]. Furthermore, meticulous blood pressure control is required for DBS surgery. Maintaining systolic blood pressure below 140 or less than 20% over the patient's baseline has been shown to reduce the odds of a clinically significant hemorrhage [25]. As patients awaken from conscious sedation, the blood pressure should be expected to rise. Calcium channel blockers, such as nicardipine, are recommended. Beta-blockers can be utilized if tremor testing is not required. Once the electrode(s) are secured in position and testing completed, sedation is again initiated.

Pediatric anaesthesia care

The AAA technique is the common method for DBS surgery in children. Anaesthesia is often induced with 3–3.5 mg/kg of propofol, in combination with a loading dose of 1 μg/kg of dexmedetomidine over 30 min with a maintenance infusion of 0.3–0.5 mg/kg/hr[12■■]. This is supplemented with remifentanil at 0.1 μg/kg/min and intermittent doses of ketamine of 0.05–0.02 mg/kg IV, all with the avoidance of volatile anaesthetic agents or nitrous oxide [12■■]. If blood pressure management is required, nicardipine is employed [12■■]. In preparation for the awake portion of the case, general anaesthesia is stopped by discontinuing the remifentanil and ketamine and reducing the dexmedetomidine to 0–0.5 μg/kg/h [12■■]. For the closing, in which no further recordings or stimulation is required, employing general anaesthesia with volatile gases is reasonable.

ANAESTHETIC EFFECTS ON MICROELECTRODE RECORDING AND STIMULATION

The main purpose of awake DBS implantation is to obtain microelectrode recordings and to monitor patient symptomatology during test stimulation, ensuring optimal target placement. Thus, avoidance of anaesthetic agents that interfere with these two purposes is required (Table 1). Overall, anaesthetic agents affect the background spontaneous firing and the neuronal spike activity patterns of basal ganglia nuclei [17], mainly through activation of gamma-aminobutyric acid (GABA) receptors. GABA is the major inhibitory neurotransmitter within the basal ganglia [26]. Anaesthetics such as benzodiazepines, barbiturates, propofol, etomidate, and volatile agents all potentiate the inhibitory actions of GABA within the basal ganglia and can worsen or abolish MER [12■■]. This is particularly true in the globus pallidus, where abundant GABAergic innervations are present. Not surprisingly, all of the above listed anaesthestics have the most profound effect on neuronal activity within GPi.

Benzodiazepines are direct GABA agonists that can abolish MER [11] and induce dyskinesias [27]; thus, they are a poor choice for DBS in movement disorder patients. If midazolam is needed, we recommend a single small dose prior to starting the procedure. That is, the dose is administered hours before MER and most often provides anxiolysis during frame placement with the added benefit of amnesia. However, propofol is the most commonly used agent in DBS, and has differential effects on each target [5]. Propofol has been shown to cause a global depression in neuronal discharge [28]. Animal studies have shown that this effect is at least partially mediated by GABA receptor activation [28]. The mechanism(s) by which short-acting mu opioid receptor agonists, such as fentanyl and remifentanil, affect MER are less well understood. There are some data, however, suggesting that they may modulate the activity of GABAergic neurons [29,30]. Therefore, although there are reports of successful MER under continuous infusions of propofol or remifentanil, it is recommended that all anaesthetic agents be turned off at least 15 min prior to MER and left off until electrode stimulation testing is completed. Although not routinely used in adults, ketamine has few effects on MER and can be useful in pediatric patients undergoing DBS [12■■,31].

The trend in DBS surgery involves an increasing popularity of dexmedetomidine in awake neuro-surgical procedures. Dexmedetomidine is a short-acting selective alpha-2 adrenoreceptor agonist and does not have any established effect on GABA receptors. Dexmedetomidine has sedative, analgesic, and anxiolytic effects without significantly depressing the respiratory system. It has become widely utilized in neurological ICU settings and several recent reports suggest that it is the ideal anaesthetic for DBS [11,20,21]. Some of these reports suggest that dexmedetomidine at low doses (<0.5 μg/kg/h) does not significantly impact the quality of MER in either the GPi or STN [11,20]. It has also been used successfully in the pediatric population for AAA technique as well [12■■]. In general, the goal to minimize anaesthesia affects on MER requires avoidance of GABA-potentiating medications. Thus, the combination of dexmedetomidine with a short-acting narcotic such as remifentanil will work well for conscious sedation in adult DBS cases the majority of the time. There are, however, contrary reports suggesting that dexmedetomidine does impact MER and it has been the experience of the authors that even at low dosing, continuous infusions of dexmedetomidine can lower overall neuronal firing. Thus, neurophysiologists and neurosurgeons must be consulted during DBS surgery and made aware of the decision to continue dexmedetomidine during MER. Regardless of the selected anaesthetic, the authors promote discontinuation of all infusions 10–15 min prior to MER whenever possible.

There are a few additional important considerations for the use of dexmedetomidine during DBS. First, dexmedetomidine is well known to cause hypotension and this response can be magnified in patients with Parkinson's disease [21]. Anaesthesiologists must be prepared for this potential response and furthermore, prepared for the inevitable rise in blood pressure once the infusions are discontinued. In addition, postapproval data and a controlled clinical trial demonstrated an increase in the incidence of some adverse advents such as paradoxical agitation (14% incidence at infusion rates >1.1 μg/kg/h) [21,32] at higher doses. This particular adverse event can be potentially dangerous in a patient fixated to the operating room table in a head frame. Therefore, awareness and recognition of this potential adverse reaction are necessary as well as an alternative plan for conscious sedation, especially in patients requiring higher dose infusions.

CONCLUSION

DBS is an effective surgical treatment for movement disorders, psychiatric disorders, chronic pain, and newer emerging conditions. The targets for DBS are small and deep, most centers perform awake microelectrode recordings and stimulation testing to confirm accurate target placement, demonstrate appropriate symptom relief and test for side-effects. We advocate for awake recordings and stimulation testing in the operating room, with all infusions turned off for at least 15 min. Anaesthetic agents have a wide range of effects on the electrophysiological recordings; both surgeons and anaesthesiologists need to be aware of how each agent can affect the intended target's electrophysiology. Dexmedetomidine is gaining popularity, often works well during awake neurosurgical procedures such as DBS, and has fewer effects on MER. Some studies of dexmedetomidine, however, have shown an increase in side-effects, including paradoxical agitation at high doses and the team should be

aware of this rare issue. If absolutely required, general anaesthesia with select agents can still permit successful DBS surgery.

Acknowledgements

We would like to thank our families who allow us the time to treat of our patients and we would like to thank our patients who continuously challenge us daily for perfection.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:

■ of special interest

■■ of outstanding interest

1. Larson PS. Deep brain stimulation for movement disorders. Neurotherapeutics. 2014; 11:465–474. [PubMed: 24833244]

2■. Kovanlikaya I, Heier L, Kaplitt M. Treatment of chronic pain: diffusion tensor imaging identification of the ventroposterolateral nucleus confirmed with successful deep brain stimulation. Stereotact Funct Neurosurg. 2014; 92:365–371. [PubMed: 25359091] [This study used diffusion tensor imaging to successfully identify the thalamic nuclei prior to DBS surgery.]

3. Kocabicak E, Temel Y, Hollig A, et al. Current perspectives on deep brain stimulation for severe neurological and psychiatric disorders. Neuropsychiatr Dis Treat. 2015; 11:1051–1066. [PubMed: 25914538]

4. Akakin A, Yilmaz B, Kilic T, Rhoton AL Jr. Anatomy of the subthalamic nucleus, with correlation of deep brain stimulation. J Neurosurg. 2015; 11:1–5.

5. Venkatraghavan L, Manninen P. Anesthesia for deep brain stimulation. Curr Opin Anaesthesiol. 2011; 24:495–499. [PubMed: 21772140]

6. Williams NR, Foote KD, Okun MS. Stn vs. Gpi deep brain stimulation: translating the rematch into clinical practice. Mov Disord Clin Pract. 2014; 1:24–35. [PubMed: 24779023]

7. Benabid AL, Torres N. New targets for dbs. Parkinsonism Relat Disord. 2012; 18(Suppl 1):S21–S23. [PubMed: 22166437]

8. Luigjes J, de Kwaasteniet BP, de Koning PP, et al. Surgery for psychiatric disorders. World Neurosurg. 2013; 80:S31, e17–28. [PubMed: 22465369]

9. Tierney TS, Abd-El-Barr MM, Stanford AD, et al. Deep brain stimulation and ablation for obsessive compulsive disorder: evolution of contemporary indications, targets and techniques. Int J Neurosci. 2014; 124:394–402. [PubMed: 24099662]

10■. Islam L, Franzini A, Messina G, et al. Deep brain stimulation of the nucleus accumbens and bed nucleus of stria terminalis for obsessive-compulsive disorder: a case series. World Neurosurg. 2015; 83:657–663. [PubMed: 25527882] [This case series described the successful use of DBS of the nucleus accumbens and bed nucleus of the stria terminalis to treat obsessive compulsive disorder.]

11. Erickson KM, Cole DJ. Anesthetic considerations for awake craniotomy for epilepsy and functional neurosurgery. Anesthesiol Clin. 2012; 30:241–268. [PubMed: 22901609]

12■■. Hippard HK, Watcha M, Stocco AJ, Curry D. Preservation of microelectrode recordings with nongabaergic drugs during deep brain stimulator placement in children. J Neurosurg Pediatr. 2014; 14:279–286. [PubMed: 24995822] [This study demonstrated that microelectrode recordings can be preserved in pediatric DBS implantation when children were administered a sedative/anaesthetic technique that included ketamine, remifentanil, dexmedetomidine, and nicardipine.]

13. Shaikh SI, Verma H. Parkinson's disease and anaesthesia. Indian J Anaesth. 2011; 55:228–234. [PubMed: 21808393]

14. Chen MS, Hong CL, Chung HS, et al. Dexamethasone effectively reduces postoperative nausea and vomiting in a general surgical adult patient population. Chang Gung Med J. 2006; 29:175–181. [PubMed: 16767966]

15. Venkatraghavan L, Luciano M, Manninen P. Review article: anesthetic management of patients undergoing deep brain stimulator insertion. Anesth Analg. 2010; 110:1138–1145. [PubMed: 20142347]

16. Fabregas N, Rapado J, Gambus PL, et al. Modeling of the sedative and airway obstruction effects of propofol in patients with parkinson disease undergoing stereotactic surgery. Anesthesiology. 2002; 97:1378–1386. [PubMed: 12459662]

17. Raz A, Eimerl D, Zaidel A, et al. Propofol decreases neuronal population spiking activity in the subthalamic nucleus of parkinsonian patients. Anesth Analg. 2010; 111:1285–1289. [PubMed: 20841416]

18■■. Kim W, Song IH, Lim YH, et al. Influence of propofol and fentanyl on deep brain stimulation of the subthalamic nucleus. J Korean Med Sci. 2014; 29:1278–1286. [PubMed: 25246748] [This study suggests that microelectrode recording signals during subthalamic nucleus DBS surgery in patients with Parkinson's disease can be successful with propofol and/or fentanyl infusions.]

19. Zesiewicz TA, Hauser RA, Freeman A, et al. Fentanyl-induced bradykinesia and rigidity after deep brain stimulation in a patient with parkinson disease. Clin Neuropharmacol. 2009; 32:48–50. [PubMed: 19471184]

20. Rozet I, Muangman S, Vavilala MS, et al. Clinical experience with dexmedetomidine for implantation of deep brain stimulators in parkinson's disease. Anesth Analg. 2006; 103:1224–1228. [PubMed: 17056959]

21. Rozet I. Anesthesia for functional neurosurgery: the role of dexmedetomidine. Curr Opin Anaesthesiol. 2008; 21:537–543. [PubMed: 18784476]

22. Elias WJ, Durieux ME, Huss D, Frysinger RC. Dexmedetomidine and arousal affect subthalamic neurons. Movement Disord. 2008; 23:1317–1320. [PubMed: 18442130]

23. Kerscher C, Zimmermann M, Graf BM, Hansen E. Scalp blocks. A useful technique for neurosurgery, dermatology, plastic surgery and pain therapy. Der Anaesthesist. 2009; 58:949–958. quiz 959–960. [PubMed: 19779756]

24. Hwang JY, Bang JS, Oh CW, et al. Effect of scalp blocks with levobupivacaine on recovery profiles after craniotomy for aneurysm clipping: a randomized, double-blind, and controlled study. World Neurosurg. 2015; 83:108–113. [PubMed: 23743219]

25. Chakrabarti R, Ghazanwy M, Tewari A. Anesthetic challenges for deep brain stimulation: a systematic approach. North Am J Med Sci. 2014; 6:359–369.

26. Galvan A, Wichmann T. Gabaergic circuits in the basal ganglia and movement disorders. Prog Brain Res. 2007; 160:287–312. [PubMed: 17499121]

27. Davies A. Midazolam-induced dyskinesia. Palliative Med. 2000; 14:435–436.

28. Fan W, Zhu X, Wu L, et al. Propofol: An anesthetic possessing neuroprotective effects. Eur Rev Med Pharmacol Sci. 2015; 19:1520–1529. [PubMed: 25967729]

29. Kouvaras E, Asprodini EK, Asouchidou I, et al. Fentanyl treatment reduces gabaergic inhibition in the ca1 area of the hippocampus 24 h after acute exposure to the drug. Neuropharmacology. 2008; 55:1172–1182. [PubMed: 18706433]

30. Griffioen KJ, Venkatesan P, Huang ZG, et al. Fentanyl inhibits gabaergic neurotransmission to cardiac vagal neurons in the nucleus ambiguus. Brain Res. 2004; 1007(1–2):109–115. [PubMed: 15064141]

31. Furmaga H, Park HJ, Cooperrider J, et al. Effects of ketamine and propofol on motor evoked potentials elicited by intracranial microstimulation during deep brain stimulation. Front Syst Neurosci. 2014; 8:89. [PubMed: 24904312]

32. Zhu M, Wang H, Zhu A, et al. Meta-analysis of dexmedetomidine on emergence agitation and recovery profiles in children after sevoflurane anesthesia: different administration and different dosage. PloS One. 2015; 10:e0123728. [PubMed: 25874562]

KEY POINTS

- DBS is an effective surgical treatment for movement disorders, psychiatric disorders, chronic pain, and newer emerging conditions.
- Conscious sedation for the opening and closing parts of the procedure are preferred, with an awake state during the microlectrode recordings and macrostimulation to optimize target placement and alleviate patient symptomatology.
- Generous amounts of long-acting local anaesthetic (potentially a full scalp block) should be infused throughout the incision with bupivacaine (0.5%), levobupivacaine (0.5%), or ropivacaine (0.75%), which all should be supplemented with epinephrine.
- Preferred sedative agents are dexmedetomidine, propofol, and remifentanil, as they affect neurocognitive testing the least, are shorter acting, and provide therapeutic comfort levels.
- For the pediatric population, the addition of ketamine during the opening and closing can be useful.

Table 1

Advantages and disadvantages of drugs used in deep brain stimulation

Agents	Dose	Advantages	Disadvantages
Premedications			
Anxiolytics		Anxiolysis, antinausea	Oversedation, paradoxical agitation [11]
Benzodiazepines			
Antiemetics			
Droperidol			
Metoclopramide			Avoid in Parkinson/dystonia as can exacerbate symptoms [11]
Ondansetron		Better first-line antiemetic [14]	Can causes extrapyramidal side effects
Dexamethasone		Better first-line antiemetic [14]	
GABA receptor agonists			
Benzodiazepines		Anxiolysis [11]	Can induce dyskinesia [15]
Propofol	30–180 μg/kg/min (induction)	Short-acting, less neurocognitive effects than other agents [11]	Abolishes MER and alters threshold stimulation [15]
			Parkinson patients may require lower doses [13,16,17]
			Can induce dyskinesia [15]
			MER attenuation [15]
Opioids			
Fentanyl	0.03–0.1 μg/kg/min (induction)	Short-acting	Rigidity and tremor suppression [15,18■■]
Remifentanil			Bradykinesia [19]
Sufentanil			
Alfentanil			
Alpha-2 agonist			
Dexmedetomidine	1 μg/kg loading [12■■]	Non-GABAergic	High dose abolishes MER [15]
	0.02–0.8 μg/kg/h [11,18■■]	Less effect on MER [11,20]	Hypotension [20,21]
	0.3–0.5 μg/kg/h [12■■] induction in kids	Some authors advocate as ideal agent [11,20,21]	Bradycardia [20,21]
		Good analgesic [11,20,21]	Increased risk of agitation at higher doses [20,21]
		Potential to use during case with preservation of movement disorder symptoms [11,20]	
		Respiratory drive preserved	
		Easily arousable [22]	