

Asleep, Awake, Repeat: The Art of Dexmedetomidine-Based Consciousness Cycling in Deep Brain Stimulation: A Case Series

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Abstract

Introduction: Deep Brain Stimulation (DBS) is an established treatment for patients with Parkinson's disease refractory to medical therapy. Anesthetic management during DBS is particularly challenging, as it requires adequate sedation and patient comfort while preserving neurophysiological integrity for Microelectrode Recording (MER). Dexmedetomidine has emerged as a favorable sedative agent due to its minimal respiratory depression and limited interference with basal ganglia activity.

Case: We report a case series of three patients with advanced Parkinson's disease who underwent bilateral DBS using a dexmedetomidine-based asleep-awake-asleep (AAA) anesthetic strategy. All patients received standardized sedation with dexmedetomidine during the asleep phases, followed by discontinuation prior to MER to facilitate intraoperative neurological assessment. Sedation depth was monitored using the Ramsay Sedation Scale (RSS), while Bispectral Index (BIS) was used to monitor the depth of anesthesia. Clinical adequacy of MER, hemodynamic stability, respiratory events, and early postoperative outcomes were evaluated.

Discussion: All patients achieved smooth transitions between sedation states and were fully cooperative during the awake phase (RSS 2). No episodes of apnea, significant hemodynamic instability, or adverse events were observed. MER was successfully performed in all cases, allowing accurate electrode placement in the subthalamic nucleus. Early postoperative evaluation demonstrated favorable clinical improvement without complications.

Conclusion: A dexmedetomidine-based asleep-awake-asleep anesthetic protocol is a safe and effective approach for DBS surgery. Careful titration and appropriate timing of dexmedetomidine discontinuation are essential to optimize MER quality while maintaining patient comfort and physiological stability.

Keywords: Asleep-awake-asleep, deep brain stimulation, dexmedetomidine, microelectrode recording Parkinson's disease, neuroanesthesia

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Introduction

Parkinson's disease (PD) is one of the fastest-growing neurodegenerative disorders worldwide, with a rising prevalence that has been described as a non-infectious pandemic.¹ As the disease advances, long-term pharmacological therapy is frequently complicated by motor fluctuations and dyskinesia, and deep brain stimulation (DBS) has become an established and effective treatment for medication-refractory PD.²

Accurate electrode placement during DBS commonly relies on intraoperative microelectrode recording (MER) to identify characteristic neuronal firing patterns within targets such as the subthalamic nucleus.³ Consequently, anesthetic management during MER-guided DBS presents a unique challenge: sedation must ensure patient comfort and hemodynamic stability while preserving spontaneous ventilation and minimizing interference with electrophysiological signals. Several anesthetic strategies have been

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described for DBS surgery, including fully awake techniques, monitored anesthesia care with sedation, and general anesthesia using either total intravenous anesthesia (TIVA) or volatile-based balanced anesthesia.³ However, anesthetic agents acting on γ -aminobutyric acid (GABA) receptors, including propofol and volatile anesthetics, may suppress basal ganglia neuronal activity and alter MER signal quality.^{3,4} Opioid-based sedation techniques using remifentanyl may improve analgesia but carry the risk of respiratory depression, hypercapnia, and the potential need for airway intervention during awake phases.³ Dexmedetomidine, a selective α_2 -adrenergic agonist, provides cooperative sedation with minimal respiratory depression while preserving spontaneous ventilation, making it attractive for awake neurosurgical procedures. In elderly patients with PD, these characteristics may additionally reduce exposure to anesthetic techniques associated with postoperative cognitive dysfunction and facilitate continuous intraoperative neurological assessment.^{5,6}

Nevertheless, emerging evidence suggests that dexmedetomidine may exert dose-dependent modulation of subthalamic neuronal activity, potentially affecting MER interpretation.^{4,7,8} Therefore, careful titration and appropriate timing of discontinuation before MER acquisition remain important considerations. At our institution, DBS implantation is routinely performed using a structured dexmedetomidine-based asleep–awake–asleep (AAA) protocol to maintain patient comfort while preserving intraoperative electrophysiological evaluation. In this case series, we describe our institutional experience with this protocol, focusing on sedation depth control, timing of dexmedetomidine discontinuation relative to MER acquisition, and perioperative respiratory and hemodynamic stability.

Case Series and Ethical Considerations

This case series describes three patients with Parkinson's disease (PD) refractory to conventional levodopa therapy who underwent primary deep brain stimulation (DBS) using

a structured dexmedetomidine-based asleep–awake–asleep (AAA) anesthetic protocol at our institution. All procedures were performed by a single neurosurgeon. Written informed consent for publication was obtained from all patients. Institutional ethical approval was waived because of the descriptive nature of this case series.

Patient Presentation

The first patient was a 66-year-old man diagnosed with Parkinson's disease in 2019 who presented with progressive body stiffness, bradykinesia, impaired balance during walking and standing, and tremor predominantly affecting the left side. He also complained of impaired chewing and swallowing coordination, occasionally resulting in choking or food spillage during meals. Prior treatments included pharmacological therapy with Madopar, acupuncture, and physiotherapy, with only partial symptomatic improvement before DBS implantation. His medical history included a transient ischemic attack in 2018, prior LASIK surgery, and dental surgery. There was no history of hypertension, diabetes mellitus, or drug allergies. The second patient was a 72-year-old woman who presented with progressively worsening gait disturbance beginning three years before admission.

Over the preceding year, she experienced severe bradykinesia and difficulty moving both lower extremities, accompanied by bilateral knee pain, resulting in substantial limitation of daily activities and near-complete dependence at home. Tremor was observed predominantly in both hands. She also reported constipation, urinary incontinence, and intermittent short-term memory impairment. Despite treatment with Madopar, her symptoms progressively worsened. Her medical history was significant for hypertension without prior surgical history, diabetes mellitus, or known drug allergies. The third patient was a 58-year-old woman who initially presented with body stiffness and right-sided weakness in 2023. Stroke was initially suspected, and she underwent hospitalization with digital subtraction angiography demonstrating normal findings. During hospitalization, she developed progressive tremor and bradykinesia without clinical improvement. Subsequent neurological evaluation established the diagnosis

Table 1. Patient Characteristics

Variable	Patient 1	Patient 2	Patient 3
Sex	Male	Female	Female
Age (year)	66	72	58
BMI (kg/m2)	27.2	29.9	20.3
Duration of PD (year)	6	3	2
Main Symptoms	Tremor, rigidity	Bradykinesia	Rigidity, tremor
Refractory of Levodopa	Yes	Yes	Yes
Comorbidities	TIA/stroke	Hypertension	Atrial fibrillation
ASA Physical Status	III	III	III
MRI Swallow Tail Sign	Absent bilaterally	Absent bilaterally	Absent bilaterally

BMI: Body Mass Index; PD: Parkinson’s Disease; ASA: American Society of Anesthesiologists; MRI: Magnetic Resonance Imaging

of Parkinson’s disease. Initial therapy with Stalevo resulted in temporary improvement; however, symptom control gradually deteriorated, prompting transition to Madopar therapy. Three months before admission, she experienced worsening tremor, rigidity, and bradykinesia with shortened duration of symptomatic relief after medication administration. Her medical history included cardiac arrhythmia treated with catheter ablation in 2016. She reported allergies to ciprofloxacin, levofloxacin, gentamicin, and sulfamethoxazole, without history of hypertension or diabetes mellitus.

Physical examination in all three patients demonstrated muscle rigidity, bradykinesia, postural instability, and minimal tremor, without other significant systemic abnormalities. Brain magnetic resonance imaging with susceptibility-weighted imaging demonstrated bilateral absence of the swallow tail sign in the substantia nigra, supporting the diagnosis of Parkinson’s disease. Preoperative chest radiography showed no pulmonary abnormalities; however, all patients demonstrated cardiothoracic ratio greater than 50% with elongation and calcification of the aorta. Echocardiographic evaluation revealed preserved biventricular systolic function without significant structural abnormalities. Laboratory investigations, including hematological profile, coagulation studies, liver and renal function tests, blood glucose, and electrolyte analysis, showed no clinically significant abnormalities in all patients. Detailed patient characteristics data are

presented in Table 1.

Anesthetic and Surgical Procedure

All patients underwent comprehensive preoperative evaluation before surgery to assess suitability for both the surgical and anesthetic procedures. Preoperative magnetic resonance imaging (MRI) for brain mapping was performed under light sedation to estimate the target coordinates for microelectrode recording (MER) insertion.

The DBS procedure was performed in two stages, consisting of intracranial electrode implantation followed by implantation of the pulse generator. This staged approach was selected to reduce the risk of postoperative infection. The first stage was conducted using an asleep–awake–asleep (AAA) anesthetic technique, whereas the second stage was performed under general anesthesia using total intravenous anesthesia (TIVA). The first stage began with stereotactic frame placement. Patients were positioned supine, and standard monitoring was applied, including electrocardiography, non-invasive blood pressure monitoring, and pulse oximetry. Initial sedation during the first asleep phase was achieved using intermittent intravenous propofol (1 mg·kg⁻¹) combined with dexmedetomidine administered as a loading dose of 1 µg·kg⁻¹ over 10 minutes, followed by continuous infusion at 0.2 – 0.3 µg·kg⁻¹·h⁻¹. Sedation depth was titrated to achieve a Ramsay Sedation Scale (RSS) score of 4–5.

Table 2. Intraoperative Anesthetic and Surgical Data

Variable	Patient 1	Patient 2	Patient 3
DBS target	STN Bilateral	STN Bilateral	STN Bilateral
Asleep Phase 1			
Duration (min)	125	85	80
Sedation Protocol	Intermittent iv propofol (1 mg.kg ⁻¹) + dexmedetomidine loading dose of 1 µg.kg ⁻¹ over 10 minutes, followed by continuous infusion at 0.2 – 0.3 µg.kg ⁻¹ .h ⁻¹		
SBP mean ± SD (mmHg)	124.5 ± 8.6		
	72.4 ± 2.8		
DBP mean ± SD (mmHg)	71.2 ± 3.5		
	4 – 5	4 – 5	4 – 5
HR mean ± SD (beats/min)	140	125	110
RSS	45	30	30
Awake MER Phase	127.3 ± 4.1	133.4 ± 4.8	100.9 ± 10.0
Duration (min)	78.1 ± 3.0	83.3 ± 5.7	70.3 ± 4.5
Duration	71.0 ± 4.8	66.9 ± 2.8	77.0 ± 2.4
Dexmedetomidine Discontinued (min)	2	2	2
SBP mean ± SD (mmHg)	200	175	190
DBP mean ± SD (mmHg)			
HR mean ± SD (beats/min)			
RSS			
Asleep Phase 2			
Duration (min)			
General Anesthesia Protocol	Propofol TCI Schnider model at an effect-site concentration of 3 µg·mL ⁻¹ + dexmedetomidine i nfusion at 0.2 – 0.3 µg·kg ⁻¹ ·h ⁻¹		
SBP mean ± SD (mmHg)	111.1 ± 11.7	127.0 ± 8.3	109.9 ± 12.2
	71.1 ± 7.5	79.4 ± 8.1	71.1 ± 7.1
DBP mean ± SD (mmHg)	72.6 ± 4.3	67.5 ± 2.0	73.6 ± 7.4
	51.1 ± 12.6	50.5 ± 15.8	49.9 ± 15.3
HR mean ± SD	400	340	315
(beats/min)	None	None	None
BIS mean ± SD	None	None	None
Surgical duration (min)	None	None	None
	None	None	None
Apneic episodes	Yes	Yes	Yes
Airway intervention	None	None	None
Conversion to GA	1	1	1
Hemodynamic instability			
Adequate MER acquisition			
Postoperative nausea/vomiting			
VAS score			

DBS: Deep Brain Stimulation; STN: Subthalamic Nucleus; MER: Microelectrode Recording; RSS: Ramsay Sedation Scale; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; HR: Heart Rate; BIS: Bispectral Index; GA: General Anesthesia; VAS: Visual Analog Scale

A scalp block was performed before stereotactic frame placement using 0.375% ropivacaine at four sites, consisting of bilateral supraorbital and greater occipital nerve blocks. Each injection contained 18.75 mg ropivacaine in a total volume of 5 mL. Following frame placement, computed tomography was performed to confirm stereotactic positioning before transfer to the operating room for definitive electrode implantation. After standard antiseptic preparation using povidone-iodine and alcohol, local infiltration was performed using 2% lidocaine with epinephrine 1:80,000. A C-shaped scalp incision was subsequently made, followed by creation of bilateral burr holes and dural opening. Transition to the awake phase was achieved by discontinuing dexmedetomidine after durotomy.

Patients regained consciousness within approximately 15–20 minutes and maintained an RSS score of 2, allowing them to follow commands comfortably without significant pain, anxiety, or agitation. During this phase, MER setup, calibration, and intraoperative stimulation testing were performed while preserving patient cooperation and spontaneous ventilation. A dexmedetomidine-free interval of approximately 30–45 minutes was maintained before initiation of MER targeting the subthalamic nucleus (STN). Corticotomy and insertion of the right-sided MER electrode were performed first, followed by identification of characteristic STN neuronal activity and macrostimulation testing. Intraoperative neurological assessment demonstrated improvement in rigidity, preserved motor strength, satisfactory ocular movement, and improved speech fluency. Definitive DBS electrode placement was confirmed using C-arm fluoroscopy. The same procedure was subsequently repeated on the contralateral side, with similar clinical improvement observed. During the second asleep phase, general anesthesia was induced using propofol target-controlled infusion (TCI) Schnider model at an effect-site concentration of $3 \mu\text{g}\cdot\text{mL}^{-1}$ combined with dexmedetomidine infusion at $0.2\text{--}0.3 \mu\text{g}\cdot\text{kg}^{-1}\cdot\text{h}^{-1}$.

Depth of anesthesia was maintained using bispectral index (BIS) monitoring with a target

range of 40–60. Endotracheal intubation was performed following administration of atracurium $0.5 \text{ mg}\cdot\text{kg}^{-1}$ to facilitate neuromuscular blockade. Subcutaneous tunneling of the extension cable and implantation of the pulse generator were subsequently completed, and impedance testing confirmed satisfactory system function. Surgical wounds were closed in layers. All procedures were completed uneventfully without respiratory compromise, failed MER acquisition, or conversion during the awake phase. Postoperative evaluation demonstrated satisfactory electrode function and significant clinical improvement in all patients. Detailed intraoperative anesthetic and surgical data is presented in Table 2.

Sedation Monitoring

Sedation depth was assessed using the Ramsay Sedation Scale (RSS), ranging from 1 (anxious and agitated) to 6 (unarousable) (Table 3). All patients achieved an RSS of 2 during the awake phase, indicating that they were cooperative, oriented, and calm. Intraoperative blood pressure remained stable, and no episodes

Table 3. Ramsay Sedation Scale

Score	Description
1	Anxious and agitated or restless, or both
2	Cooperative, oriented, and tranquil
3	Drowsy, but responds to commands
4	Asleep, brisk response to light glabellar tap or loud auditory stimulus
5	Asleep, sluggish response to light glabellar tap or loud auditory stimulus
6	Asleep and unarousable

of apnea or adverse events were observed. Bispectral Index (BIS) monitoring was used to assess the depth of anesthesia through electroencephalographic (EEG) signal analysis. The BIS scale ranges from 0 to 100, where 0 represents the absence of cortical activity and 100 indicates full wakefulness (Table 4). Values between 40 and 60 are generally considered to represent an adequate depth of general anesthesia.

Post Operative Care

All three patients underwent awake extubation

Table 4. Bispectral Index Value

BIS Value	Level of Consciousness / Sedation	Clinical Interpretation
100	Fully awake	Normal alert consciousness
80-90	Light sedation	Patient may be relaxed but easily responsive
70-80	Moderate sedation	Reduced awareness; responds to verbal stimulation
60-70	Deep sedation	Low probability of explicit recall
40-60	General anesthesia	Adequate depth for surgical anesthesia
20-40	Deep hypnotic state	Very low brain activity; excessive anesthesia possible
0	No detectable EEG activity	Electrical silence of the brain

Table 5. Aldrete Score

Criteria	Point Value
SpO ₂ (or color)	
Pink (SpO ₂ > 92% on room air)	2
Pale or dusky (SpO ₂ > 90% on oxygen)	1
Cyanotic (SpO ₂ < 90% on oxygen)	0
Respiration	
Can breathe deeply and cough	2
Shallow but adequate exchange	1
Apnea or obstruction	0
Circulation	
Blood pressure within 20% of normal	2
Blood pressure within 20% to 50% of normal	1
Blood pressure deviating > 50% from normal	0
Consciousness	
Awake, alert, and oriented	2
Arousable but readily drifts back to sleep	1
No response	0
Activity	
Moves all extremities	2
Moves two extremities	1
No movement	0

following completion of the surgical procedure and were subsequently monitored in the Post Anesthesia Care Unit (PACU). Neuromuscular blockade was reversed with neostigmine 1 mg IV and atropine 0.5 mg IV in all patients. In the PACU, patients were monitored for level of consciousness, hemodynamic stability, and

pain for 60 minutes. Discharge criteria from the PACU were based on the Aldrete Score (Table 5) and the Visual Analog Scale (VAS). Patients were considered eligible for transfer when the Aldrete Score was ≥ 9 and pain was tolerable (VAS ≤ 2). During monitoring, no hemodynamic changes greater than 20% were observed. All patients were fully conscious and well oriented, had pink skin color, were able to breathe deeply and cough adequately, and could move all four extremities. Postoperative pain was well tolerated in all three patients, with a VAS score of 1. The pain was primarily localized at the surgical wound on the scalp, described as throbbing and stinging in nature. For analgesia, all patients received ketorolac 30 mg IV every 8 hours and paracetamol 1 g IV every 8 hours. No postoperative nausea or vomiting was reported in any of the patients.

Discussion

Parkinson's disease (PD) is one of the fastest-growing neurodegenerative disorders worldwide, and Deep Brain Stimulation (DBS) has become an established therapeutic option for patients with advanced disease refractory to pharmacological therapy.^{1,2} Successful DBS implantation depends on accurate electrode placement, which commonly relies on intraoperative Microelectrode Recording (MER) to identify characteristic neuronal firing patterns within targets such as the subthalamic nucleus (STN).^{3,9,10} Consequently, anesthetic management during MER-guided DBS presents a unique challenge because sedation must maintain patient comfort and hemodynamic stability while preserving spontaneous ventilation and

minimizing interference with electrophysiological recordings.^{3,11} Several anesthetic strategies have been described for DBS surgery, including fully awake techniques, monitored anesthesia care with sedation, and general anesthesia using either total intravenous anesthesia (TIVA) or volatile-based balanced anesthesia.^{3,11} General anesthesia offers advantages in terms of patient immobility, airway protection, and procedural comfort; however, anesthetic agents acting through γ -aminobutyric acid (GABA)-mediated pathways, including propofol and volatile anesthetics, may suppress neuronal activity within the basal ganglia and alter MER signal quality.^{3,4} Propofol has been shown to reduce subthalamic neuronal firing, while volatile anesthetics may similarly impair electrophysiological recordings because of their inhibitory effects on cortical and subcortical neuronal transmission.^{3,4} As a result, DBS performed entirely under conventional general anesthesia may compromise intraoperative target identification and reduce electrode placement accuracy.

Opioid-based sedation techniques, particularly remifentanyl, may provide effective analgesia and favorable hemodynamic control; however, respiratory depression, hypercapnia, upper airway obstruction, and the potential need for airway intervention remain important concerns during awake MER acquisition.^{3,11} Recent evidence also suggests that remifentanyl may modulate subthalamic beta oscillatory activity, potentially affecting electrophysiological recordings despite lacking direct GABAergic effects.¹² Preservation of spontaneous ventilation and reliable patient cooperation is therefore essential during awake phases of DBS surgery. Dexmedetomidine has emerged as an attractive anesthetic option for awake neurosurgical procedures, including DBS implantation. As a selective α_2 -adrenergic agonist, dexmedetomidine produces cooperative sedation resembling physiological sleep while preserving spontaneous ventilation and causing minimal respiratory depression.^{3,5} Compared with propofol- or opioid-based sedation, dexmedetomidine facilitates smoother transition between asleep and awake phases, reduces

agitation, and allows continuous neurological assessment during MER and intraoperative stimulation testing.^{3,5,6} In elderly patients with PD, these characteristics may additionally reduce exposure to anesthetic techniques associated with postoperative cognitive dysfunction (POCD), an important consideration in a population already vulnerable because of advanced age and neurodegenerative disease.⁶

Nevertheless, dexmedetomidine is not entirely free from electrophysiological effects. Dexmedetomidine has been shown to suppress basal ganglia neuronal activity in a dose-dependent manner, with subthalamic neuronal firing remaining suppressed even after patients regain apparent clinical wakefulness.^{4,7,8} Importantly, normalized root mean square neuronal activity remained below baseline despite Ramsay Sedation Scale (RSS) scores consistent with cooperative awakening, suggesting that behavioral sedation scales alone may not accurately reflect complete electrophysiological recovery.⁸ Anesthetic agents are recommended to be discontinued at least 15 minutes before MER acquisition to improve signal reliability; however, subsequent studies suggest that longer washout periods may further optimize neuronal recovery.^{2,8,11}

In the present case series, DBS implantation was performed using a structured dexmedetomidine-based asleep–awake–asleep (AAA) protocol. Dexmedetomidine infusion was discontinued approximately 30–45 minutes before MER acquisition, allowing patients to regain cooperative wakefulness while minimizing potential interference with electrophysiological recordings. Throughout the awake phase, all patients maintained spontaneous ventilation without apnea, oxygen desaturation, or need for airway intervention. Adequate MER signals and satisfactory intraoperative neurological responses were successfully obtained in all cases, while hemodynamic parameters remained stable throughout the procedure. These findings support the feasibility of a dexmedetomidine-based AAA strategy for MER-guided DBS surgery in patients with Parkinson's disease.

Practical Implications and Future Directions

This case series highlights several practical considerations for anesthetic management during DBS surgery. First, a structured dexmedetomidine-based AAA protocol may provide a favorable balance between patient comfort, respiratory safety, and preservation of MER quality. Predictable transition between asleep and awake phases facilitated communication between anesthesiologists, neurosurgeons, and neurophysiology teams while allowing reliable intraoperative neurological assessment.^{6,11} Standardized anesthetic protocols may therefore improve procedural consistency and reduce variability during MER-guided DBS implantation.

Second, the timing of dexmedetomidine discontinuation before MER acquisition appears clinically important. Although patients may regain clinical wakefulness within a relatively short period, electrophysiological recovery may require a longer washout interval.^{4,5,8} The 30–45-minute dexmedetomidine-free interval used in our institutional protocol allowed adequate MER acquisition while maintaining patient comfort and respiratory stability, suggesting that longer washout periods may optimize signal quality during DBS surgery.

Third, the preservation of spontaneous ventilation throughout the awake phase may reduce complications associated with opioid-based sedation or deep general anesthesia, including respiratory depression, hypercapnia, airway manipulation, and potential conversion to general anesthesia.^{3,11,12} These considerations are particularly relevant in elderly patients with Parkinson's disease, who frequently present with multiple comorbidities and increased vulnerability to perioperative neurological complications.⁶ Future research should focus on prospective comparative studies evaluating dexmedetomidine-based protocols against alternative anesthetic strategies, including propofol-based sedation, remifentanil-assisted techniques, and DBS performed entirely under general anesthesia. Objective neurophysiological parameters such as MER amplitude, neuronal

firing rate, beta oscillatory activity, and signal-to-noise ratio should be incorporated to better characterize anesthetic effects on subthalamic neuronal activity.^{5,8} Larger multicenter cohort studies and randomized controlled trials would also be valuable in establishing standardized evidence-based recommendations for anesthetic management during DBS surgery.^{6,11}

Study Limitation

Several limitations of this study should be acknowledged. First, the small sample size inherent to a case series limits the generalizability of the findings, and the results should therefore be interpreted as descriptive and hypothesis-generating rather than definitive.^{6,11} Second, the absence of a control group using alternative anesthetic techniques precludes direct comparison between dexmedetomidine-based sedation and other commonly used approaches, including propofol–remifentanil sedation or volatile-based general anesthesia.^{3,11} Third, electrophysiological outcomes were evaluated primarily through clinical adequacy of MER rather than quantitative neurophysiological analysis. Objective parameters such as normalized root mean square activity, neuronal firing frequency, or beta oscillatory power were not systematically assessed despite prior evidence demonstrating persistent electrophysiological suppression after apparent clinical recovery.^{5,8} Fourth, postoperative outcomes were evaluated only during the early postoperative period, and long-term neurological outcomes, stimulation efficacy, and functional improvement were not assessed.^{2,9} Finally, all procedures were performed at a single institution by the same surgical and anesthetic team, which may introduce institutional and operator bias and limit external validity. Multicenter studies are therefore required to confirm reproducibility and broader applicability of this protocol.

Conclusion

Anesthetic management during Deep Brain Stimulation (DBS) requires a carefully tailored strategy to preserve microelectrode recording

(MER) quality, facilitate accurate electrode placement, maintain patient comfort, and ensure physiological stability. In this case series, a dexmedetomidine-based asleep–awake–asleep (AAA) protocol provided controllable sedation, preserved spontaneous ventilation during the awake phase, and allowed satisfactory intraoperative MER acquisition without respiratory complications or significant hemodynamic instability.

A dexmedetomidine-free interval before MER appeared to facilitate reliable electrophysiological assessment while maintaining patient cooperation and comfort. These findings support the feasibility of dexmedetomidine-based AAA anesthesia as a practical approach for MER-guided DBS procedures in patients with Parkinson's disease. Further prospective studies with larger sample sizes and quantitative neurophysiological evaluation are required to better define the optimal anesthetic strategy and timing of dexmedetomidine discontinuation during DBS surgery.

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