Anesth Pain Med. 2023 June; 13(3):e136093. https://doi.org/10.5812/aapm-136093.

Published online 2023 May 6. **Review Article**

Anesthetic Considerations for Cataract Surgery in Patients with Parkinson's Disease: A Narrative Review

Alyssa Chiew [1], David Mathew [1], Chandra M. Kumar [1, *], Edwin Seet [2], Farnad Imani [3] and Seyed-Hossein Khademi [4, **]

[1] Department of Anaesthesia, Khoo Teck Puat Hospital, Yishun, Singapore
[2] Department of Anaesthesia, Yong Loo Lin School of Medicine, National University of Singapore, Singapore
[3] Pain Research Center, Department of Anesthesiology and Pain Medicine, Iran University of Medical Sciences, Tehran, Iran
[4] Department of Anesthesiology, School of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran

[*] *Corresponding author*: Department of Anaesthesia, Khoo Teck Puat Hospital, Yishun, Singapore. Email: chandra.kumar2406@gmail.com
[**] *Corresponding author*: Department of Anesthesiology, School of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran. Email: khademihs@mums.ac.ir

Received 2023 March 04; **Revised** 2023 April 05; **Accepted** 2023 April 10.

Abstract

Parkinson's disease (PD) is a chronic neurological degenerative disease affecting the central nervous system, which is responsible for progressive disorders such as slow movements, tremors, rigidity, and cognitive disorders. There are no specific recommendations and guidelines for anesthetic management of patients with PD undergoing ophthalmic procedures. This narrative review aims to summarise the anesthetic considerations in patients with PD presenting for cataract surgery.

Keywords: Cataract, Parkinson's Disease, General Anaesthesia, Local Anaesthesia, Regional Anaesthesia

1. Context

Parkinson's disease (PD) is the second most common neurodegenerative disease of the elderly in frequency after Alzheimer's disease. It is a chronic condition which affects the central nervous system. It is responsible for the progressive disorder, including the triad of cardinal motor symptoms: Rigidity, bradykinesia, and tremor, and symptoms related to autonomic dysfunction and cognitive disorders. Symptoms of PD are due to the loss of about 50 - 70% of dopaminergic neurons in the basal ganglia, which generally worsens as the disease advances (1).

Since patients with PD now enjoy a greater life expectancy with improvement in therapies, they are more prone to develop age-related cataracts requiring surgery and anesthesia. Ophthalmologists usually request general anesthesia (GA) for cataract surgery because of involuntary movement. However, GA may mask neurological symptoms in the intraoperative period and exacerbate symptoms in the postoperative period, along with possible drug interactions between anesthetic and concurrent drugs used in PD. Patients with PD may benefit from regional anesthesia (RA) because regular medications can be taken effectively and on time, even though the baseline tremor may pose possible technical challenges for the surgeons.

There is no published data on anesthetic management or guidelines specific to the choice and conduct of anesthesia for cataract surgery in patients with PD. We conducted a literature review to highlight new information regarding anesthetic considerations for cataract surgery in patients with PD, with the aim of enhancing patient well-being and ensuring their safety.

2. Search Strategy

In January 2023, a comprehensive search was performed on MEDLINE (Ovid), PubMed, EMBASE, CINAHL, Google Scholar, and Cochrane databases to locate English language articles published between January 1970 and December 2022. The search terms used included various combinations of "anesthesia" and "Parkinson's disease". Additionally, relevant case studies were identified, and relevant articles were sourced from the references of the searched articles (no double-blind controlled studies were found; thus, consort flow diagram was not needed). The quality and applicability of anesthesia techniques for cataract surgery in patients with Parkinson's disease were evaluated by the authors based on these articles. As a result, 51 relevant articles were included in this review.

2.1. Aetiology

The exact cause of PD has yet to be fully understood. However, it is considered a disease of the basal ganglia with clinical features arising from the degeneration of dopaminergic neurons of the substantia nigra (2). This neurodegenerative disease is characterized by the progressive development of bradykinesia, resting tremor, and rigidity (1). As the disease progresses, patients develop impairment in both gross and fine motor skills (3). A recent study by Baille et al. showed that inspiratory muscle strength is compromised, even in the early stages of PD, at a mean disease duration of about two years (4). The pathogenesis of PD is also believed to be attributed to a combination of genetic and environmental factors leading to oxidative stress, dysfunctional mitochondria, cellular calcium imbalance, neuroinflammation, and other neurotransmitter system deficits (5). Some specific causes also identified include mutations in certain genes, exposure to toxins such as pesticides, and head injuries.

2.2. Pathogenesis

The pathophysiology of PD is believed to involve the loss of dopamine-producing neurons in the brain (6). Dopamine is a neurotransmitter crucial in regulating motor control and other vital functions such as cognitive function, attention, motivation, and reproduction (7). Following degeneration and death of dopamine-producing neurons in the substantia nigra, the level of dopamine in the brain drops, which eventually contributes to the motor symptoms of the disease, such as tremors, stiffness, and difficulty with movement.

2.3. Symptomatology

PD is a neurological disorder which causes tremors and disabling motor, neuropsychiatric and autonomic dysfunction. The presentation of PD can be very diverse and varied, and each patient may experience different disabilities during the disease. Symptoms may also vary from person to person and change over time (8).

A tremor in hands, arms, legs, jaw, and face is most pronounced at rest rather than during movement, and it may be worse on one side of the body and may even be absent in some patients. The tremor is typically described as a “pill-rolling” tremor, which refers to the back-and-forth rubbing of the thumb and forefinger (8). Other features of the tremor may include a slight trembling or shaking in the limbs, head, face, or jaw, and it may be accompanied by stiffness or difficulty with coordination. In some cases, the tremor may be so severe that it interferes with daily activities such as writing, eating, or dressing.

In contrast, non-motor symptoms such as autonomic features, neuropsychiatric disturbances, and additional features such as fatigue, insomnia, and hypersalivation may precede the motor syndrome and even the diagnosis of PD (9).

PD may have autonomic dysfunction manifestations, including postural hypotension, gastrointestinal symptoms, and urinary control disturbances. Postural instability, difficulty maintaining balance and coordination with a shuffling gait, or minor, quick steps may be seen. Loss of facial expression (hypomimia) may be present. Phonation and swallowing are affected. The chest wall movement is reduced, and a ventilatory defect may occur. The neuropsychiatric symptoms include depression, anxiety, apathy, agitation, and psychosis, which can significantly impact a patient’s quality of life, and are often under-recognized (10). Mood changes are also observed, which may reflect the deterioration of central nervous system function.

3. Management of PD

There is currently no cure for PD, and the treatment plan varies depending on the individual’s symptoms, stage of the disease, and overall health condition. The symptoms of each patient with PD are unique, and an individualized treatment plan must be established with the help of a neurologist (11).

3.1. Medical

The medical management of PD typically involves a combination of drugs and therapies to help manage tremors and symptoms and improve quality of life. These drugs (Table 1) include levodopa-carbidopa, ergot or non-ergot dopamine agonists, monoamine oxidase inhibitors (MAOI) B inhibitors, Catechol-O-methyl transferase (COMT) inhibitors, anticholinergics and N-methyl-D-aspartate (NMDA) receptor antagonist (12).

3.2. Supportive Measures

Supportive therapies include physical therapy (improves mobility, balance, and flexibility, and can help to prevent falls), occupational therapy (improves fine motor skills, such as writing and buttoning clothes), and speech therapy (improve speech and swallowing difficulties). Measures such as physical therapy (Tai Chi or yoga), occupational therapy (adaptive devices and techniques: such as special utensils or writing aids), and lifestyle

Table 1. Parkinson's Medications

No	Categories of Parkinson's Medications	Examples	Side-effects	Anesthetic Implications
1	Levodopa: The most effective drug to treat motor symptoms is mainly bradykinesia, but it can also be used for tremors and rigidity.	Levodopa; levodopa-carbidopa (to enhance the effectiveness of levodopa and minimize its adverse effects)	Nausea, sleepiness, dizziness, headache; others: confusion, hallucinations, delusions, agitation, psychosis; long-term (5 to 10 years) use can be associated with motor fluctuations and dyskinesia.	Risk of Parkinsonism-hyperpyrexia syndrome (PHS) on acute withdrawal; short half-life: Will require enteral administration in prolonged procedures; avoid halothane as it may sensitize the heart to catecholamines which may cause levodopa-induced arrhythmias
2	Monoamine oxidase B inhibitors (MAOBIs): It works by obstructing the activity of MAOB enzymes, which deactivate dopamine.	Selegiline; rasagiline; safinamide	Selegiline commonly causes nausea, headaches, and insomnia. Additionally, in elderly Parkinson's patients, its use may be restricted due to the potential for confusion.	Risk of serotonin syndrome (agitation, muscle rigidity, and hyperthermia) with opioids such as meperidine (pethidine); risk of exaggerated vasoconstrictor effects in patients taking direct-acting sympathomimetics and MAOBIs
3	Dopamine agonists (ergot or non-ergot): It functions by directly activating the dopamine receptors in the brain to replicate the effects of dopamine.	Pramipexole; ropinirole; rotigotine (trans-dermal); apomorphine (SC infusion)	Nausea, sedation, leg swelling, visual hallucinations, impulse control disorders, orthostatic hypotension; apomorphine is highly emetogenic.	Risk of dopamine agonist withdrawal syndrome (DAWS) on acute withdrawal; to prevent the use of medications such as metoclopramide, butyrophenones (haloperidol, droperidol), and phenothiazines (promethazine, prochlorperazine) that have anti-dopaminergic effects, which may exacerbate Parkinson's disease symptoms
4	Catechol-O-methyl transferase inhibitors (COMTIs): To prolong and enhance the effect of levodopa by preventing the breakdown of dopamine by COMT	Tolcapone; entacapone	Dyskinesia, hallucinations, confusion, nausea, diarrhoea, orange discoloration of the urine, and postural hypotension; tolcapone can cause deranged LFTs and potential hepatotoxicity. Hence entacapone should be used in those at risk of hepatotoxicity.	Risk of exaggerated sympathetic response when used together with drugs metabolized by COMT pathway, e.g., adrenaline, hence the need to reduce the dose when used concurrently
5	Anticholinergics: They are used to reduce symptoms of tremors in PD patients aged 70 and below who do not have significant akinesia or difficulty walking.	Trihexyphenidyl; benztropine; orphenadrine; procyclidine; biperiden	Dry mouth, blurred vision, constipation, nausea, difficulty emptying the bladder, impaired sweating, tachyarrhythmia, delirium, memory issues, hallucinations	Centrally-acting anticholinergic drugs, like atropine, may trigger central anticholinergic syndrome, which can cause confusion, drowsiness, and agitation. As a result, glycopyrrolate, a peripherally acting alternative, is a safer choice.
6	N-methyl-D-aspartate (NMDA) receptor antagonist: The antiviral drug was initially developed to prevent influenza but was found to improve mild symptoms (tremors, akinesia, rigidity) by an indirect dopamine-stimulating effect.	Amantadine	Visual hallucinations, confusion livedo reticularis (blotchy, purple-colored areas of skin found on the wrists and legs), ankle swelling	
7	Treatment for non-motor symptoms: Anti-depressants; medications for PD dementia	TCAs (amitriptyline, nortriptyline); SSRIs (citalopram, paroxetine, sertraline); SNRIs (venlafaxine, duloxetine); acetylcholinesterase inhibitors (rivastigmine, donepezil)	Risk of extrapyramidal side effects with SSRIs and SNRIs	The use of TCA anti-depressants may potentiate levodopa-induced arrhythmias.; risk of serotonin syndrome with concurrent use of MAOB and some anti-depressants; the use of acetylcholinesterase inhibitors, such as rivastigmine, donepezil, and galantamine, in Parkinson's disease dementia patients has been linked to extended succinylcholine effects, which can last up to 50 minutes, as well as heightened resistance to non-depolarizing neuromuscular blocking drugs.

Abbreviations: COMT, Catechol-O-methyl transferase; PD, Parkinson's disease.

changes (avoiding triggers that worsen tremors: stress, and fatigue) may confer help in compensating for tremors and perform daily activities more easily to people with PD (13).

3.3. Surgical

In patients with advanced PD with debilitating symptoms unresponsive to conventional drugs, neurosurgical treatments include deep brain stimulation, lesion surgery, and focused ultrasound thalamic surgery (1). Deep brain stimulation (DBS) is the most commonly used surgical procedure that involves implanting a small device in the brain that sends electrical impulses to specific areas, which can help reduce tremors (14).

4. Cataract Development in Patients with PD

There is some evidence to suggest a possible association between cataracts and PD. Both individuals with PD may be at an increased risk of developing cataracts, and individuals with cataracts may have an increased risk of developing PD. A retrospective cohort study conducted to analyze the database of nearly 50,000 people in Taiwan showed a 26% increased risk of PD in cataract patients (15).

Visual disturbances are a common problem in patients with PD. Most patients present with decreased visual acuity, contrast sensitivity, and color discrimination which may be related to dopamine in the eye (16). Other diseases, such as dry eye syndrome and glaucoma, frequently occur in patients with PD and may contribute to visual disturbances (17). Cataracts are characterized by the gradual clouding of the crystalline lens caused by a variety of risk factors, including exposure to ultraviolet light. Oxidative stress is a significant contributor to the formation of cataracts, as lens DNA, proteins, and lipids are often oxidized (18). Excessive oxidative stress is thought to be one of the mechanisms responsible for Parkinson's disease neurodegeneration, which may explain the increased incidence of cataracts in PD patients (19).

5. Choice of Anaesthetic Modality for Cataract Surgery in Patients with PD

The tremors and muscle rigidity in PD make it difficult for the patient to keep their heads still during the procedure. Also, some patients with PD may have difficulty cooperating with the procedure or may have anxiety or agitation. GA can help to relax the muscles and prevent tremors, allowing the ophthalmologist to perform the procedure unhindered. However, GA is not without risk and may only be necessary for some patients. Regional anesthesia can be an attractive choice for cataract surgery if the patient is cooperative and compliant and the disease is in the early stages where head and body tremors are unlikely to interfere with surgery (20).

6. Pre-operative Anaesthetic Considerations

Pre-operative considerations broadly encompass system evaluation, operative scheduling, and medication scheduling (21). Patients with PD may have other associated comorbidities and poor general health; therefore, a system evaluation during the pre-operative assessment is also essential.

6.1. System Evaluation

6.1.1. Cardiovascular Diseases

Common cardiac problems in patients with PD include bradycardia, orthostatic hypotension, and an increased risk of heart disease due to a combination of factors such as immobility and concurrent drug use in treating PD. Some drugs used in PD, particularly those that affect dopamine levels, may also increase the risk of arrhythmia and sudden cardiac death (22). Some medications used to treat PD can cause QT prolongation, such as domperidone and citalopram, which make patients more susceptible to arrhythmias such as torsade de pointes and ventricular fibrillation (23). Dysautonomia is common and can result in fluctuations in blood pressure. Persistent hypotension can be managed with a vasopressor such as phenylephrine (24), and supine hypertension can be managed with short-acting vasodilators (25). Patients with PD tend to be smokers and hypertensive as well.

6.1.2. Respiratory Diseases

Common respiratory problems in patients with PD include dyspnoea and impaired cough reflex (26), leading to the risk of aspiration and pneumonia. Dyspnoea is caused by muscle weakness and poor lung function (27). Other potential issues include sleep-related breathing disorders like apnoea (27, 28). In addition, it has been demonstrated that the respiratory function of such patients can be subnormal due to restrictive and obstructive elements (27).

6.1.3. Gastrointestinal Diseases

Common gastrointestinal problems in patients with PD include constipation, dysphagia, and gastroparesis (29). Constipation is caused by a combination of factors

such as decreased physical activity and side effects of drugs used in the treatment, in addition to the muscles and nerves that control bowel movement being affected in PD. Patients with PD are at risk of dysphagia, with up to 80% experiencing it during the course of the disease (30). In addition, they may also have sialorrhoea which predisposes them to aspiration (31). Gastroparesis, a condition in which the stomach takes too long to empty its contents, can also occur in PD and cause symptoms such as nausea, vomiting, and abdominal pain. Gastroparesis is mainly due to nerve damage, further compounded by levodopa treatment (32).

6.1.4. Genitourinary Diseases

Genitourinary problems in patients with PD include urinary incontinence, urgency, and frequency due to changes in the muscles and nerves that control the bladder and urinary tract, manifesting as signs of urgency and frequency and predisposing to urinary tract infections (31). Although unclear, some medications used for PD may also cause side effects such as urinary retention or difficulty starting urination (33). In addition, PD can cause sexual dysfunction, commonly erectile dysfunction in men and orgasm dysfunction, and reduced libido in females (34), which is often under-reported.

6.1.5. Neuropsychiatric Diseases

Cognitive deficits can manifest in patients with PD, including the insidious onset of cognitive impairment and dementia, which may predispose them to an increased risk for postoperative cognitive disorders or delayed emergence after GA (35). Moreover, depression, anxiety, apathy, and hallucinations are also often reported (36). Patients with PD may also have underlying sleep disturbances such as rapid eye movement (REM), sleep behavior disorders, sleep fragmentations, and insomnia (37).

7. Pre-operative Optimisation

Optimizing a patient with PD before surgery is essential in ensuring the best possible outcome. A multidisciplinary approach involving the neurologist, surgeon, and anesthetist is required to manage such patients. PD symptoms should be well-controlled before surgery. Medication reconciliation and timely administration are critical facets of managing such patients. Otherwise, they risk being in the "off"-state (when the Parkinson's symptoms return) or have worsening dysphagia or dyskinesias (38).

7.1. Concurrent Medications and Their Implications for Anaesthesia

The decision to continue or discontinue medications for PD during the peri-operative period should be carefully considered, weighing risks versus benefits (Table 1).

7.1.1. Discontinuing PD Medications

Stopping the medication before surgery can cause the following issues:

(1) PD medications control symptoms such as tremors, stiffness, and difficulty with movement. Discontinuing these medications can cause a sudden worsening of symptoms, known as "off" periods, which can be debilitating and make it difficult for the patient to perform daily activities (38);

(2) Some PD medications, can cause abnormal, uncontrollable movements. These movements can occur when medication is discontinued, making it difficult for the patient to move around and perform daily activities (39);

(3) There is an increased risk of falls. PD affects balance and coordination, and discontinuing medication can increase the risk of falls and other accidents (40);

(4) PD causes depression and anxiety, and discontinuing medication can worsen these symptoms, affecting the patient's overall quality of life (41);

(5) Patients with PD may develop motor fluctuations, which means that the patient may experience periods when medication is less effective in controlling symptoms. Discontinuing medication can worsen these fluctuations and make it more difficult to manage symptoms (42).

7.1.2. Continuing PD Medications

The drugs should be continued promptly during the pre-operative period, especially during the nil by mouth. This would allow better control of the condition and avoid unwanted complications. In cases where oral medication is strictly not permitted, consultation with a neurologist would be prudent to determine alternative formulations of equivalent medications that can be administered. This will aim to maintain an optimal level of dopaminergic stimulation throughout the peri-operative period as close to the baseline as possible. If RA is performed for cataract surgery, it is prudent to continue Parkinson's medications.

7.2. Patients fitted with Deep Brain Stimulation

Some patients with advanced PD may also have a deep brain stimulator (DBS) implanted. Patients with a DBS implanted will need to have their implantable pulse generator device turned off prior to surgery and turned on at the

end of surgery to avoid any recurrence of any Parkinson's symptoms (31). Diathermy should be avoided, and bipolar should be used when necessary.

8. Consent, Medication Timing, and Scheduling for Surgery

8.1. Consent

Patients with PD may be cognitively impaired, so it would be essential to assess them carefully if they can consent. Depending on institutional practices, a legal guardian might be required to participate in the consent-taking process. Communication difficulties: PD can affect a patient's communication ability, making it difficult to obtain informed consent. Limited understanding of the procedure: Patients with PD may have a limited understanding of the surgical procedure and the risks and benefits of the surgery. In cases where the patient cannot provide informed consent, a legal guardian or a healthcare proxy may be able to consent on the patient's behalf. It is essential to have detailed documentation of the consent process and to have it signed by the patient, or their legal representative, to ensure that the patient's autonomy and rights are respected.

8.2. Miedication Timing

Patients in advanced stages of PD are vulnerable to complications during the perioperative period. Careful attention must be paid to the timing of their medication doses, as sudden cessation of these drugs can cause a rapid resurgence or deterioration of symptoms. In severe cases, it may trigger a condition known as a neuroleptic malignant syndrome, which can have serious consequences. Hence, scheduling surgery for such patients would be advisable, resulting in the least disruption to their medication schedule. This might include being scheduled as the first case of the day to minimize long fasting periods and potentially missed medications.

8.3. Scheduling of Surgery

Each patient of PD is unique, and the specific scheduling of surgery should be tailored to meet the patient's individual needs. Factors such as the patient's overall health, type of surgery, medication timing, and availability of healthcare team members may dictate surgery scheduling.

9. General Anaesthesia

Patients with PD have tremors and rigidity, which may impair their ability to undergo cataract surgery under RA due to positioning and the inability to lay still for surgery. Hence, GA is the preferred choice of ophthalmologists. However, GA does have some specific considerations for patients with PD. There are pros and cons of using GA in patients with PD. GA carries a risk of complications such as respiratory depression, which can be more pronounced in patients with PD (20). GA can adversely affect PD symptoms, such as worsening tremors, stiffness, and difficulty with movement (43). PD medications can interact with medications used during GA (20). GA may cause postoperative neurocognitive dysfunction in some patients, which can be more pronounced in patients with PD (44).

If GA is undertaken, it would be advisable to undertake a rapid sequence induction because of the aspiration risk that might be present. The use of propofol in patients with PD has been reported to cause chorea and dystonic dyskinesia in two patients undergoing stereotactic pallidotomy (20). Thiopentone can exacerbate Parkinsonism, and ketamine can cause a sympathetic surge and cardiovascular complications (1). In modern-day anesthesia, propofol would be the drug of choice for induction of anesthesia. For anesthesia maintenance, halothane is arrhythmogenic and should be avoided (45). Other inhalational agents, such as enflurane and sevoflurane, have been deemed safe for patients with PD.

Opioids can potentially cause muscle rigidity, and patients with PD can be more sensitive to this (45). In addition, opioids such as meperidine (pethidine) and tramadol should be avoided as they predispose patients to serotonin syndrome. Non-depolarising muscle relaxants can be safely used in patients with PD.

The patients fitted with a DBS implanted should have their implantable pulse generator device turned off prior to surgery and turned on at the end of surgery to avoid any recurrence of any Parkinson's symptoms (31). Diathermy should be avoided, and bipolar should be used when necessary.

9.1. Postoperative Considerations After GA

Parkinson's medications should be reinstated as soon as possible according to the schedule post-operatively, and this would avoid the worsening of PD symptoms post-operatively. Patients undergoing cataract surgery should be allowed to take it orally post-operatively; hence, there should not be a delay in administering their medications.

Nausea and vomiting are common side effects of GA, especially with the administration of opioids. However, caution should be exercised with some commonly used drugs for nausea or vomiting. Metoclopramide and prochlorperazine should be avoided, given their dopaminergic-blocking activity (46). Ondansetron and domperidone would be preferred in patients with PD.

Pain is usually uncommon after cataract surgery, and simple administration of topical anesthesia is sufficient. However, if analgesia needs to be administered postoperatively, non-opioids drugs should be considered. Opioids such as tramadol and methadone should be avoided in patients taking MAOB inhibitors as they can increase the risk of serotonin syndrome.

10. Regional Anaesthesia for Cataract Surgery in Patients with PD

Cataract surgery is mainly performed under local and RA. The decision to use local and RA in patients with PD should be made on a case-by-case basis, in collaboration with neurologists, ophthalmologists, and anesthetists, taking into account the general health of the patient, the type of surgery and the potential impact of PD on the surgical outcome. However, careful patient selection is paramount to the success of cataract surgery if scheduled under RA. The patient's anxiety should be addressed, and the surgeons should assess motor manifestations of PD to determine if they will interfere with the surgical process. Patients should receive an explanation about the process and expected duration of surgery and have an appreciation of what to expect. This will aid in the cooperativity central to a successful regional technique.

If the surgical procedure is expected to be complex, the ophthalmic block can be considered more effective than topical anesthesia (21). A sub-Tenon's block or topical anesthesia can be an excellent alternative to a GA. However, adrenaline-containing local anesthetic solution for ophthalmic RA can exacerbate cardiovascular complications in patients with dysautonomia. Nevertheless, adrenaline is typically not used in ophthalmic blocks to avoid the risk of inducing ocular ischemia, making this issue less significant (47).

The advantages of a local and regional technique include reduced drug interactions, less hemodynamic instability, no need for airway manipulation, and reduced postoperative confusion (21). A regional technique for cataract surgery brings about a host of benefits if suitable to be carried out. Local and regional anesthesia allows the patient to remain awake and alert during the procedure, which can help to preserve cognitive function and minimize the risk of postoperative confusion (48). Local anesthesia does not affect PD as the patient remains awake and alert during the procedure, which can help to preserve the patient's symptom control. Local and regional anesthesia provides pain control after cataract surgery.

10.1. Sedation with Local/Regional Anesthesia

Cataract surgery can be performed under monitored anesthetic care. The Royal College of Anaesthetists and the Royal College of Ophthalmologists in the United Kingdom advise that an anesthetist should supervise this procedure (49). Sedation scores such as the Ramsay sedation scores or Richmond Agitation Sedation Scale (RASS) can be utilized to monitor the depth of sedation.

Most commnly used drugs include propofol, fentanyl, midazolam, and dexmedetomidine. Dexmedetomidine is an alpha-2 agonist that can be used as an alternative for sedation during cataract surgery. A lower incidence of postoperative delirium (around 3%) has been observed with its use, as compared to propofol (50%) or midazolam (50%) (50). With a regional ophthalmic technique, these adjuvant medications can provide optimal surgical conditions and patient comfort without much drug interaction (51). The pros and cons of general and regional anesthesia are addressed in Table 2.

11. Conclusions

PD is an increasingly encountered disease of the elderly which presents considerable anesthetic challenges. There are advantages and disadvantages to either a regional or a general anesthetic technique for cataract surgery. The pathophysiology of the disease, including the medication regimes and potential complications, must be considered carefully with a multidisciplinary approach before deciding the best anesthetic modality for the patient presenting for elective cataract surgery. Hence, it is essential for every anesthesiologist to be knowledgeable about the unique requirements and necessities of patients with Parkinson's disease to improve peri-operative anesthetic for the best surgical outcome after cataract surgery.

Footnotes

Authors' Contribution: W. L., A. C., D. M., C. M. K., E. S., F. I., and S. H. K. have contributed to the concept, design, conducting research, drafting, and finalising the manuscript.

Table 2. Pros and Cons of General and Regional Anesthesia

	General Anaesthesia	Regional Anaesthesia
Advantages	(1) Abolishes unwanted movements and tremors, which can subsequently allow for smooth conduct of surgery. (2) The concern about patient anxiety and cooperation can be abolished as the patient will remain immobile for the period of surgery and allow the surgeons to perform the surgery in a timely manner. (3) There is no need to administer an ophthalmic block for anesthesia and the associated risks involved. (4) The airway will be secured, as compared to a partial or failed regional technique which might require sedation and an unsecured airway with potential complications such as hypoxia and hypercarbia in the event of over-sedation.	(1) There are reduced drug-drug interactions (due to fewer drugs being involved), less hemodynamic instability, no need for airway manipulation, and reduced postoperative confusion. (2) Pain control is better post-operatively as the anesthetic agent can continue to provide analgesic effects post-surgery. Therefore, fewer systemic analgesic medications must be given, with fewer medication interactions. (3) There is usually no delay in the resumption of oral intake post-operatively, and Parkinson's medications can be resumed without anticipated delay.
Disadvantages	(1) Can worsen tremors and stiffness and result in movement difficulties post-op. (2) Potential exacerbation of respiratory complications as well as neurocognitive dysfunction post-operatively. In addition, such patients are at higher risk of aspiration, which may predispose them to aspiration events if a GA is undertaken. (3) Medication interactions and cessation must be considered (further details under the medication interaction section). (4) Nausea and vomiting are common GA side effects. However, there may be concerns with using medications such as metoclopramide for its treatment because of its dopaminergic blocking capability. In addition, with nausea/committing, patients may not be able to resume diet promptly, which can impair their regular PD medication intake.	(1) There is a need for an anesthetic agent to be administered, either in the form of a topical block or an ophthalmic block. The regional techniques can be associated with risks such as infection and bleeding. (2) Adrenaline-containing blocks can exacerbate cardiovascular complications in patients with dysautonomia. (3) There might be a need for sedation in the event of a failed or partial regional technique, which can potentially bring about issues with sedatives and their potential complications.

Conflict of Interests: The authors have no conflicts of interest to disclose regarding funding and research support, employment, personal financial interest, patents, and consultation fees within the last five years. One of the authors (F. I.) is the reviewer of this journal. Based on journal policy, this author was completely excluded from any review of this article.

Funding/Support: No funding/support was received for this manuscript.

References

1. Nicholson G, Pereira AC, Hall GM. Parkinson's disease and anaesthesia. *Br J Anaesth*. 2002;**89**(6):904–16. [PubMed ID: 12453936]. https://doi.org/10.1093/bja/aef268.
2. DeLong MR, Juncos JL. Parkinson's Disease and Other Movement Disorders. In: Kasper D, Fauci A, Hauser S, Longo D, Jameson J, Loscalzo J, editors. *Harrison's principles of internal medicine*. 17th ed. New York, USA: Mcgraw-hill; 2008. p. 2406–17.
3. Malling AS, Jensen BR. Motor intensive anti-gravity training improves performance in dynamic balance related tasks in persons with Parkinson's disease. *Gait Posture*. 2016;**43**:141–7. [PubMed ID: 26444077]. https://doi.org/10.1016/j.gaitpost.2015.09.013.
4. Baille G, Perez T, Devos D, Deken V, Defebvre L, Moreau C. Early occurrence of inspiratory muscle weakness in Parkinson's disease. *PLoS One*. 2018;**13**(1). e0190400. [PubMed ID: 29329328]. [PubMed Central ID: PMC5766081]. https://doi.org/10.1371/journal.pone.0190400.
5. Zaman V, Shields DC, Shams R, Drasites KP, Matzelle D, Haque A, et al. Cellular and molecular pathophysiology in the progression of Parkinson's disease. *Metab Brain Dis*. 2021;**36**(5):815–27. [PubMed ID: 33599945]. [PubMed Central ID: PMC8170715]. https://doi.org/10.1007/s11011-021-00689-5.
6. Dauer W, Przedborski S. Parkinson's disease: mechanisms and models. *Neuron*. 2003;**39**(6):889–909. [PubMed ID: 12971891]. https://doi.org/10.1016/s0896-6273(03)00568-3.
7. Klein MO, Battagello DS, Cardoso AR, Hauser DN, Bittencourt JC, Correa RG. Dopamine: Functions, Signaling, and Association with Neurological Diseases. *Cell Mol Neurobiol*. 2019;**39**(1):31–59. [PubMed ID: 30446950]. https://doi.org/10.1007/s10571-018-0632-3.
8. Sveinbjornsdottir S. The clinical symptoms of Parkinson's disease. *J Neurochem*. 2016;**139 Suppl 1**:318–24. [PubMed ID: 27401947]. https://doi.org/10.1111/jnc.13691.
9. Schrag A, Horsfall L, Walters K, Noyce A, Petersen I. Prediagnostic presentations of Parkinson's disease in primary care: a case-control study. *Lancet Neurol*. 2015;**14**(1):57–64. [PubMed ID: 25435387]. https://doi.org/10.1016/S1474-4422(14)70287-X.
10. Ray S, Agarwal P. Depression and Anxiety in Parkinson Disease. *Clin Geriatr Med*. 2020;**36**(1):93–104. [PubMed ID: 31733705]. https://doi.org/10.1016/j.cger.2019.09.012.
11. Poewe W. The natural history of Parkinson's disease. *J Neurol*. 2006;**253 Suppl 7**:VII2–6. [PubMed ID: 17131223]. https://doi.org/10.1007/s00415-006-7002-7.
12. Tripathi KD. Chapter-31 Antiparkinsonian Drugs. In: Tripathi KD, editor. *Essentials of Medical Pharmacology*. New Delhi, India: Jaypee Publishers; 2013. p. 425–34. https://doi.org/10.5005/jp/books/12021_33.
13. Armstrong MJ, Okun MS. Diagnosis and Treatment of Parkinson Disease: A Review. *JAMA*. 2020;**323**(6):548–60. [PubMed ID: 32044947]. https://doi.org/10.1001/jama.2019.22360.
14. Kogan M, McGuire M, Riley J. Deep Brain Stimulation for Parkinson Disease. *Neurosurg Clin N Am*. 2019;**30**(2):137–46. [PubMed ID: 30898266]. https://doi.org/10.1016/j.nec.2019.01.001.
15. Lai SW, Lin CL, Liao KF, Chang-Ou KC. Increased risk of Parkinson's disease in cataract patients: a population-based cohort study. *Parkinsonism Relat Disord*. 2015;**21**(1):68–71. [PubMed ID: 25466927]. https://doi.org/10.1016/j.parkreldis.2014.11.005.
16. Weil RS, Schrag AE, Warren JD, Crutch SJ, Lees AJ, Morris HR. Visual dysfunction in Parkinson's disease. *Brain*. 2016;**139**(11):2827–43. [PubMed ID: 27412389]. [PubMed Central ID: PMC5091042]. https://doi.org/10.1093/brain/aww175.
17. Nowacka B, Lubinski W, Honczarenko K, Potemkowski A, Safranow K. Ophthalmological features of Parkinson disease. *Med Sci Monit*. 2014;**20**:2243–9. [PubMed ID: 25387009]. [PubMed Central ID: PMC4238794]. https://doi.org/10.12659/MSM.890861.
18. Sacca SC, Bolognesi C, Battistella A, Bagnis A, Izzotti A. Gene-environment interactions in ocular diseases. *Mu-*

tat Res. 2009;**667**(1-2):98–117. [PubMed ID: 19046976]. https://doi.org/10.1016/j.mrfmmm.2008.11.002.
19. Kaur J, Kukreja S, Kaur A, Malhotra N, Kaur R. The oxidative stress in cataract patients. *J Clin Diagn Res*. 2012;**6**(10):1629–32. [PubMed ID: 23373015]. [PubMed Central ID: PMC3552191]. https://doi.org/10.7860/JCDR/2012/4856.2626.
20. Shaikh SI, Verma H. Parkinson's disease and anaesthesia. *Indian J Anaesth*. 2011;**55**(3):228–34. [PubMed ID: 21808393]. [PubMed Central ID: PMC3141145]. https://doi.org/10.4103/0019-5049.82658.
21. Yim RLH, Leung KMM, Poon CCM, Irwin MG. Peri-operative management of patients with Parkinson's disease. *Anaesthesia*. 2022;**77 Suppl 1**:123–33. [PubMed ID: 35001381]. https://doi.org/10.1111/anae.15617.
22. Renoux C, Dell'Aniello S, Khairy P, Marras C, Bugden S, Turin TC, et al. Ventricular tachyarrhythmia and sudden cardiac death with domperidone use in Parkinson's disease. *Br J Clin Pharmacol*. 2016;**82**(2):461–72. [PubMed ID: 27062307]. [PubMed Central ID: PMC4972162]. https://doi.org/10.1111/bcp.12964.
23. Oka H, Mochio S, Sato H, Katayama K. Prolongation of QTc interval in patients with Parkinson's disease. *Eur Neurol*. 1997;**37**(3):186–9. [PubMed ID: 9137930]. https://doi.org/10.1159/000117432.
24. Stirt JA, Frantz RA, Gunz EF, Conolly ME. Anesthesia, Catecholamines, and Hemodynamics in Autonomic Dysfunction. *Anesth Analg*. 1982;**61**(8). https://doi.org/10.1213/00000539-198208000-00016.
25. Mustafa HI, Fessel JP, Barwise J, Shannon JR, Raj SR, Diedrich A, et al. Dysautonomia: perioperative implications. *Anesthesiology*. 2012;**116**(1):205–15. [PubMed ID: 22143168]. [PubMed Central ID: PMC3296831]. https://doi.org/10.1097/ALN.0b013e31823db712.
26. Ebihara S, Saito H, Kanda A, Nakajoh M, Takahashi H, Arai H, et al. Impaired efficacy of cough in patients with Parkinson disease. *Chest*. 2003;**124**(3):1009–15. [PubMed ID: 12970031]. https://doi.org/10.1378/chest.124.3.1009.
27. Docu Axelerad A, Stroe AZ, Arghir OC, Docu Axelerad D, Gogu AE. Respiratory Dysfunctions in Parkinson's Disease Patients. *Brain Sci*. 2021;**11**(5). [PubMed ID: 34064360]. [PubMed Central ID: PMC8147845]. https://doi.org/10.3390/brainsci11050595.
28. Crosta F, Desideri G, Marini C. Obstructive sleep apnea syndrome in Parkinson's disease and other parkinsonisms. *Funct Neurol*. 2017;**32**(3):137–41. [PubMed ID: 29042002]. [PubMed Central ID: PMC5726349]. https://doi.org/10.11138/fneur/2017.32.3.137.
29. Kim JS, Sung HY. Gastrointestinal Autonomic Dysfunction in Patients with Parkinson's Disease. *J Mov Disord*. 2015;**8**(2):76–82. [PubMed ID: 26090079]. [PubMed Central ID: PMC4460543]. https://doi.org/10.14802/jmd.15008.
30. Suttrup I, Warnecke T. Dysphagia in Parkinson's Disease. *Dysphagia*. 2016;**31**(1):24–32. [PubMed ID: 26590572]. https://doi.org/10.1007/s00455-015-9671-9.
31. Lenka A, Mittal SO, Lamotte G, Pagan FL. A Pragmatic Approach to the Perioperative Management of Parkinson's Disease. *Can J Neurol Sci*. 2021;**48**(3):299–307. [PubMed ID: 32959743]. https://doi.org/10.1017/cjn.2020.211.
32. Heetun ZS, Quigley EM. Gastroparesis and Parkinson's disease: a systematic review. *Parkinsonism Relat Disord*. 2012;**18**(5):433–40. [PubMed ID: 22209346]. https://doi.org/10.1016/j.parkreldis.2011.12.004.
33. Connolly BS, Lang AE. Pharmacological treatment of Parkinson disease: a review. *JAMA*. 2014;**311**(16):1670–83. [PubMed ID: 24756517]. https://doi.org/10.1001/jama.2014.3654.
34. Kinateder T, Marinho D, Gruber D, Hatzler L, Ebersbach G, Gandor F. Sexual Dysfunctions in Parkinson's Disease and Their Influence on Partnership-Data of the PRISM Study. *Brain Sci*. 2022;**12**(2). [PubMed ID: 35203923]. [PubMed Central ID: PMC8869894]. https://doi.org/10.3390/brainsci12020159.
35. Fang C, Lv L, Mao S, Dong H, Liu B. Cognition Deficits in Parkinson's Disease: Mechanisms and Treatment. *Parkinsons Dis*. 2020;**2020**:2076942. [PubMed ID: 32269747]. [PubMed Central ID: PMC7128056]. https://doi.org/10.1155/2020/2076942.
36. Aarsland D, Bronnick K, Ehrt U, De Deyn PP, Tekin S, Emre M, et al. Neuropsychiatric symptoms in patients with Parkinson's disease and dementia: frequency, profile and associated care giver stress. *J Neurol Neurosurg Psychiatry*. 2007;**78**(1):36–42. [PubMed ID: 16820421]. [PubMed Central ID: PMC2117797]. https://doi.org/10.1136/jnnp.2005.083113.
37. Suzuki K, Miyamoto M, Miyamoto T, Iwanami M, Hirata K. Sleep disturbances associated with Parkinson's disease. *Parkinsons Dis*. 2011;**2011**:219056. [PubMed ID: 21876839]. [PubMed Central ID: PMC3159123]. https://doi.org/10.4061/2011/219056.
38. Derry CP, Shah KJ, Caie L, Counsell CE. Medication management in people with Parkinson's disease during surgical admissions. *Postgrad Med J*. 2010;**86**(1016):334–7. [PubMed ID: 20547599]. https://doi.org/10.1136/pgmj.2009.080432.
39. Zesiewicz TA, Sullivan KL, Hauser RA. Levodopa-induced dyskinesia in Parkinson's disease: epidemiology, etiology, and treatment. *Curr Neurol Neurosci Rep*. 2007;**7**(4):302–10. [PubMed ID: 17618536]. https://doi.org/10.1007/s11910-007-0046-y.
40. Grimbergen YA, Munneke M, Bloem BR. Falls in Parkinson's disease. *Curr Opin Neurol*. 2004;**17**(4):405–15. [PubMed ID: 15247535]. https://doi.org/10.1097/01.wco.0000137530.68867.93.
41. Schrag A, Taddei RN. Depression and Anxiety in Parkinson's Disease. *Int Rev Neurobiol*. 2017;**133**:623–55. [PubMed ID: 28802935]. https://doi.org/10.1016/bs.irn.2017.05.024.
42. Jankovic J. Motor fluctuations and dyskinesias in Parkinson's disease: clinical manifestations. *Mov Disord*. 2005;**20 Suppl 11**:S11–6. [PubMed ID: 15822109]. https://doi.org/10.1002/mds.20458.
43. Krauss JK, Akeyson EW, Giam P, Jankovic J. Propofol-Induced Dyskinesias in Parkinson's Disease. *Anesth Analg*. 1996;**83**(2):420–2. https://doi.org/10.1213/00000539-199608000-00037.
44. Price CC, Levy SA, Tanner J, Garvan C, Ward J, Akbar F, et al. Orthopedic Surgery and Post-Operative Cognitive Decline in Idiopathic Parkinson's Disease: Considerations from a Pilot Study. *J Parkinsons Dis*. 2015;**5**(4):893–905. [PubMed ID: 26683785]. [PubMed Central ID: PMC4810448]. https://doi.org/10.3233/JPD-150632.
45. Buxton JA, Gauthier T, Kinshella MW, Godwin J. A 52-year-old man with fentanyl-induced muscle rigidity. *CMAJ*. 2018;**190**(17):E539–41. [PubMed ID: 29712673]. [PubMed Central ID: PMC5929893]. https://doi.org/10.1503/cmaj.171468.
46. Kim S, Cheon SM, Suh HS. Association Between Drug Exposure and Occurrence of Parkinsonism in Korea: A Population-Based Case-Control Study. *Ann Pharmacother*. 2019;**53**(11):1102–10. [PubMed ID: 31216861]. https://doi.org/10.1177/1060028019859543.
47. Kumar CM, Eid H, Dodds C. Sub-Tenon's anaesthesia: complications and their prevention. *Eye (Lond)*. 2011;**25**(6):694–703. [PubMed ID: 21455245]. [PubMed Central ID: PMC3178142]. https://doi.org/10.1038/eye.2011.69.
48. Kumar CM, Palte HD, Chua AWY, Sinha R, Shah SB, Imani F, et al. Anesthesia Considerations for Cataract Surgery in Patients with Schizophrenia: A Narrative Review. *Anesth Pain Med*. 2021;**11**(2). e113750. [PubMed ID: 34336627]. [PubMed Central ID: PMC8314087]. https://doi.org/10.5812/aapm.113750.
49. Kumar CM, Eke T, Dodds C, Deane JS, El-Hindy N, Johnston RL, et al. Local anaesthesia for ophthalmic surgery–new guidelines from the Royal College of Anaesthetists and the Royal College of Ophthalmologists. *Eye (Lond)*. 2012;**26**(6):897–8. [PubMed ID: 22538216]. [PubMed Central ID: PMC3376304]. https://doi.org/10.1038/eye.2012.82.
50. Imani F, Zaman B, De Negri P. Postoperative Pain Management: Role of Dexmedetomidine as an Adjuvant. *Anesth Pain Med*. 2020;**10**(6). e112176. [PubMed ID: 34150582]. [PubMed Central ID: PMC8207883].

https://doi.org/10.5812/aapm.112176.
51. Kumar CM, Chua AWY, Imani F, Sehat-Kashani S. Practical Considerations for Dexmedetomidine Sedation in Adult Cataract Surgery Under Local/Regional Anesthesia: A Narrative Review. *Anesth Pain Med*. 2021;**11**(4). e118271. [PubMed ID: 34692445]. [PubMed Central ID: PMC8520679]. https://doi.org/10.5812/aapm.118271.