Lesson 258: PreAnesthetic Assessment of the Patient With Parkinson’s Disease

PREANESTHETIC ASSESSMENT

Dr. Elizabeth A.M. Frost, who is the editor of this continuing medical education series, is Clinical Professor of Anesthesiology at The Mount Sinai School of Medicine in New York City. She is the author of Clinical Anesthesia in Neurosurgery (Butterworth-Heinemann, Boston) and numerous articles. Dr. Frost is past president of the Anesthesia History Association and former editor of the journal of Anesthesiology History Association and former editor of the journal of Anesthesiology History Association and former editor of the journal of

The administration of levodopa (added 3% in the United States and Canada. PD is defined as a neurodegenerative condition caused by neuronal loss in the dopaminergic substantia nigra pars compacta (SNc). Projections from the SNc to the striatum normally allow natural movement. However, populations of somata are increasingly lost in the SNc, increasing activity of vesicular amine transporters in the basal ganglia leads to an inhibition of thalamic and brain stem nuclei. Thalamic inhibition suppresses the motor system in the cortex and clinical symptoms appear. Dysfunction initially occurs unilaterally in the form of hand tremor (resting to pill rolling), micrographia, limited arm swing, and foot dragging. Eventually, symptoms develop bilaterally as bradykinesia, resting tremor, and postural instability. Intellec- tual function is usually preserved in the early stages of dis- ease but may deteriorate markedly with time. The extent of dopamine loss correlates with the severity of bradykinesia. The administration of levodopa (L-dopa) results in relief of symptoms.1

The treatment of PD is directed at controlling symptoms by enhancing the effects of dopamine and reducing the effects of acetylcholine (ACh). Agents are used that have combined effects on the dopaminergic and cholinergic components of the extrapyramidal system. Pharmacologic therapy common- ly consists of L-dopa together with α-methylhydrazine (added to prevent decarboxylation of L-dopa and thus retard the peripheral breakdown of the drug carbidopa-levodopa), the

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DATE OF RELEASE: NOVEMBER 2006

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November 2006

Anesthesiology News

Elizabeth A.M. Frost, MD, at ElzFrost@aol.com.

Learning Objectives

At the end of this activity, the participant should be able to:

1. Define the epidemiology of Parkinson’s disease (PD).
2. Explain the pathogenesis of PD.
3. Outline a mechanism for the presence of diagnostic Lewy bodies.
4. Discuss the typical clinical scenario involving patients with PD, including both primary and sec- ondary features of the disease.
5. Identify diagnostic clinical features of PD that can be used to differentiate the disease from other neurologic disorders.
6. Identify and discuss the role of pharmacologic therapy in treating patients with PD.
7. Outline surgical approaches to treatment.
8. Discuss anesthetic considerations.
9. Recognize potential perioperative complications.
10. Present a perioperative plan.

Case History

A 58-year-old woman with Parkinson’s disease was scheduled for implantation of a deep brain stimula- tor. A schoolteacher, she had become increasingly debilitated by the disease, requiring increased med- ication that had resulted in major side effects. Her daily medications included levodopa, bromocriptine, selegiline, pramipexole, and amantadine. Her hypertensive condition (blood pressure, 168/92 mm Hg) was treated with hydrochlorothiazide with moderate effect. Physical examination revealed a well-devel- oped woman who appeared older than her stated age, with right-hand tremor, muscle rigidity, and pos- tural instability. At laboratory, chest X-ray, and electrocardiography results were within normal limits.

The patient noted that she was claustrophobic and had difficulty undergoing magnetic resonance imaging studies.
irreversible monoamine oxidase inhibitors selegline and rasagiline, the antiviral drug amantadine, and anticholinergics (eg, benztropine, trihexyphenidyl, and ethopropazine).1–7 Catechol-O-methyltransferase (COMT) inhibitors that prevent the decarboxylation of L-dopa include entacapone-carbidopa levodopa and tolcapone. Dopamine receptor agonists include ergot derivatives (bromocriptine, cabergoline, lisuride, and apomorphine) and nonergot derivatives (pramipexole and ropinirole). Apomorphine is injectable and can be given for rapid rescue therapy during periods of "off effect." These combinations are designed to increase dopamine levels in the brain and blunt peripheral effects.4 Treatment is palliative; drug therapy does not halt neuronal degeneration.2 In addition, the duration of action of sustained-release formulations is only 3 to 6 hours.

Stereotactic therapy often becomes ineffective, surgical strategies have been developed. Stereotactic surgery employs precise lesioning to dampen pathologic neuronal discharge. Procedures such as pallidotomy and thalamotomy that were performed previously are used less now. The lesions are permanent, with a risk for severe side effects such as stroke and neurologic injury. The procedure of choice today is deep brain stimulation, which involves the implantation of stimulatory electrodes and (at a later date) a generator that allows the patient to control the electrodes. Devices are implanted in 2 stages. The first stage is performed with the patient awake or minimal sedation; the second stage is performed with the patient under general anesthesia. The anesthetic management of surgical patients with PD is based on an understanding of risk factors, drug therapies, varying anesthetic requirements, and potential perioperative complications.

Epidemiology

PD is a progressive disorder that typically manifests clinically in people past the age of 65—although it may appear at a much younger age. In the United States alone, PD has been diagnosed in approximately 750,000 to 1 million individuals, making the disorder one of the leading neurologic diseases in the elderly.1 Although several causes of and risk factors for PD have been proposed, its etiology is unclear. However, familial forms—both autosomal-dominant and autosomal-recessive—have been documented. In addition, some studies have found significant environmental factors to be associated with a higher prevalence of disease. Probably both genetic and environmental factors play a limited role in increasing susceptibility.1

Several studies have assessed the risk factors for the eventual development of PD. In a population-based case-control study, Gorell et al examined 10 independent variables. Occupational exposure to both copper and lead for more than 20 years had a population attributable risk of 3.9%. Occupational exposure to insecticides for more than 20 years had a population attributable risk of 8.1%. A family history of PD in first- and second-degree relatives had a population attributable risk of 12.4%. Less than 30 pack-years of smoking or no smoking at all had the highest population attributable risk of 44.1%. Thus, family and occupational history and lifestyle behaviors may be to some extent associated with PD.2 Other studies have confirmed that lifestyle behaviors have some effect. In a case-control study, Nuti et al noted that cigarette smoking had a protective effect. While the study produced insignificant data regarding insecticide exposure, control patients who did not have the disease were much more likely to be chronic cigarette smokers.2 De Reuck et al advanced this association in a retrospective study that found a history of smoking to be inversely associated with onset of the disease. The study concluded that the onset of symptoms is delayed in chronic smokers, possibly indicating a modulating effect of smoke products on the dopaminergic system.3

Many studies have attempted to link PD with family history; only marginal, conflicting data have been produced. Rocca et al performed a historical cohort study of 162 patients with PD and more than 1,000 first-degree relatives. In patients with an early onset of PD, it was noted that first-degree relatives had a small relative risk for development of the disorder. The risk for relatives of patients who had late-onset PD was not identified.4

Compared with the general public, patients with PD have consistently been shown to have a decreased life expectancy. Patients with PD have a median survival of 10.3 years, compared with 13.4 years for age-matched control patients.5 Moreover, a younger age at onset of the disease correlates positively with relative risk for mortality. For patients younger than 67 years at onset of the disease, the relative risk value is 2.04; for those with onset at 67 to 76 years, it is 1.76; and for patients with onset after the age of 76, the relative risk value for mortality is lowest, at 1.48.
Pathogenesis and Molecular Genetics
PD is recognized clinically, and the diagnosis is confirmed in postmortem examination by the presence of Lewy bodies and the loss of dopaminergic neurons in the SNc.1,2 Figure 1. The degeneration of SNc dopaminergic neurons, which project to the striatum as the nigrostriatal pathway, reduces the striatal dopamine content. The genetics of PD suggest a mechanism for the presence of diagnostic Lewy bodies—oxidative stress, mitochondrial dysfunction, decreased production of adenosine triphosphate, diminished degradative activity of the highly energy-dependent ubiquitin-proteasome system, protein aggregation, and eventual disease.13 McNaught et al demonstrated that the administration of synthetic and endogenous proopiomelanocortin inhibitors disrupts the ubiquitin-proteasome system in rats and results in the development of clinical symptoms of PD (bradykinesia, rigidity, and tremor) and pathologic findings (neuronal degeneration and Lewy body-like inclusions in the SNc and other similarly affected areas) within 2 weeks.

Environmental factors may be involved in the pathogenesis of PD. An acute form of PD can be caused by exposure to 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP), a neurotoxic by-product of the illicit synthesis of meperidine analogs. After parental administration, MPTP readily enters the brain and is metabolized by astrocytic monoamine oxidase B to 1-methyl-4-phenylpyridinium (MPP+). MPTP concentrates in dopaminergic SNc neurons, where it inhibits the mitochondrial electron transport chain to result in a depletion of adenosine triphosphate and selective cell death.14 Exposure to pesticides or other environmental toxins may increase the risk for PD.15

Clinical Features
PD primarily affects muscle movement.3 Several symptoms of PD overlap with those of other neurologic disorders. A defining clinical feature of PD is response to treatment with L-dopa; in other words, if a patient presents with ambiguous neuromuscular symptoms does not respond to L-dopa therapy, then a diagnosis of PD is probably inaccurate.16 Typically, the patient presents with unilateral symptoms—micrographia, hand tremor, decreased arm swing, foot dragging—and related to the primary symptoms (bradykinesia, muscular rigidity, tremor, and postural instability) (Table 1). Primary symptoms arise from a loss of dopamine in the basal ganglia, which directly affects the ACh-dopamine balance. Projections of the basal ganglia function to dampen motor neurons in the reticular formation, making possible smooth, coordinated movements. Normally, a balance between the excitatory effects of dopamine and the inhibitory effects of ACh on dampening allows the coordination of motor movements.

In the patient with PD, ACh is the primary neurotransmitter in the basal ganglia, effectively inhibiting the dampening effect. The result is muscular rigidity and a lack of smooth, coordinated movement (Figure 2). The unilateral onset of symptoms is diagnostic of PD; many other neurokinetic disorders present initially with bilateral symptoms. As PD gradually progresses and more dopaminergic neurons are lost, symptoms appear bilaterally and become disabling. Patients with advanced PD usually succumb to postural instability and are eventually confined to a wheelchair. In approximately 10% to 15% of patients with PD, mostly elderly, dementia develops that is similar to the dementia of Alzheimer’s disease. Because these patients may also have pathologic evidence of Alzheimer’s disease, it is important to distinguish between the 2 conditions based on other clinical features. The development of dementia in patients with PD has been found on autopsy to correlate with the widespread dissemination of Lewy bodies, which can be demonstrated by immunohistochemistry.17 Recent studies have shown that depression is associated with PD in nearly 40% of patients. However, this seemingly widespread comorbidity usually goes undiagnosed and untreated. Patients must be monitored for signs of depression and treated accordingly.18 Autonomic symptoms, such as orthostatic hypotension, constipation, and urinary retention, are common in PD but not diagnostic. The imbalance between ACh and dopamine in the striatum leads to the hypercholinergic symptoms mentioned.

Pharmacologic Therapy
Although there is no cure for PD, several medication regimens can be used to manage the symptoms. However, it should be noted that no medication has been shown to retard the progression of the disease.19 The nonsurgical treatment of PD is centered on the replacement of lost dopaminergic neurotransmission and antagonism of unchecked cholinergic neurons in the striatum. The primary goal of the drugs designed for treating PD is to restore the balance between dopamine and ACh in the striatum.20 The increased level of synaptic dopamine resulting from pharmacologic therapy has side effects, mainly nausea, orthostatic hypotension, hallucinations, psychosis, and dyskinesia.21 Perhaps the most serious problem physicians face when administering medication for PD is a pattern of motor fluctuations that typically develops after 4 to 6 years of drug therapy; dyskinesia, a common side effect of L-dopa therapy, is discussed later. Progression to the motor complications (tremors and gait disturbances) of these side effects occurs in nearly half of all patients, and the complications are often debilitating. Current research is focused on finding regimens that minimize drug-induced effects.22

Levodopa-Carbidopa
L-Dopa, a dopaminergic precursor of dopamine, has been the gold standard treatment for PD. Taken orally, L-dopa crosses the blood-brain barrier—unlike dopamine. Remaining dopaminergic neurons take up L-dopa in the SNc and metabolize it to produce the neurotransmitter dopamine. L-Dopa, when administered alone, is rapidly decarboxylated to dopamine outside the central nervous system (CNS) by the enzyme dopamine decarboxylase. An enhanced concentration of dopamine in the periphery causes many adverse side effects, including nausea, vomiting, and hypotension. Because much of the original dose of L-dopa is metabolized before it reaches the CNS, large quantities must be administered to achieve the desired effects. To avoid the effects of L-dopa monotherapy, dual therapy with L-dopa and carbidopa is recommended. Carbidopa blocks the peripheral conversion of L-dopa to dopamine and thus increases the bioavailability of L-dopa while eliminating some of the side effects associated with excessive dopamine in the periphery.23 Problems associated with L-dopa therapy usually center on a “wearing off” of the effect of the drug over time. L-Dopa treatment depends on the presence of viable neurons in the SNc; so a progressive loss of these neurons reduces the efficacy of the drug. Also, an “on/off” motor effect has been described, primarily caused by the extremely short half-life (1-2 hours) of L-dopa. Thus, concentrations in the CNS can vary greatly and lead to fluctuations in symptomatic relief.24 Some studies have shown that L-dopa therapy can eventually lead to dyskinesia due to fluctuating dopamine concentrations, prompting some physicians to save L-dopa therapy for more advanced cases.25 L-Dopa tends to react with various other drugs and vitamins. Pyridoxine (vitamin B6) increases the peripheral breakdown of L-dopa, effectively reducing its bioavailability. L-Dopa should not be administered simultaneously with monoamine oxidase inhibitors because the enhanced production of catecholamines can result in a hypertensive crisis. Monoamine oxidase inhibitors not only increase the levels of peripheral dopamine and other catecholamines but also inhibit the breakdown of L-dopa to further increase peripheral dopamine levels and raise blood pressure. Physicians should be wary of prescribing L-dopa to patients receiving phenothiazine, butyrophenone, or thioxanthen compounds because L-dopa can exacerbate psychiatric symptoms by increasing dopamine concentrations in the CNS. Antipsychotic medications are contraindicated in general because these drugs block dopamine receptors and can by themselves cause PD-like symptoms. In addition, cardiac patients are advised to take L-dopa cautiously because of an increased susceptibility to arrhythmia.26
Lesson 258 continued from page 37

Dopamine Agonists

Dopamine agonists, mainly bromocriptine, pergolide, pramipexole, and ropinirole, are frequently coadministered with L-dopa to reduce the amount needed. These drugs, which rapidly cross the blood–brain barrier, require no metabolism or conversion within the brain. Dopamine agonists function by directly binding to dopamine receptors in the striatum. Similar to L-dopa, dopamine agonists cause side effects of nausea, hypotension, and vomiting associated with increased peripheral levels of dopamine. However, some studies have shown that the side effects are more intense than those of L-dopa therapy alone. Dopamine agonists do not cause dyskinesia.

Dopamine agonists are administered primarily in the early stages of PD because many physicians prefer to “save” L-dopa therapy for the more severe symptoms associated with advanced disease. These agents are also favored for patients who cannot tolerate the dyskinesia associated with L-dopa therapy. In his research on guidelines for the pharmacologic therapy of PD, Kondo noted that dopamine agonists may be preferable for younger patients because they are less likely to be functional when faced with the frequent motor side effects of L-dopa treatment.

Dopamine agonists can be dangerous in patients with a history of myocardial infarction or peripheral vascular disease because peripheral dopamine increases arterial spasm. Much like L-dopa, dopamine agonists have been shown to increase symptoms in patients with psychiatric disorders.

Selegiline

Selegiline, like the dopamine agonists, is typically used for the early treatment of PD to delay the administration of L-dopa. Selegiline acts by selectively inhibiting monoamine oxidase B, the enzyme that breaks down dopamine in the brain. Some forms of PD have been linked to an accelerated metabolism of dopamine, which directly increases the concentration of toxic free radicals that destroy dopamine neurons. Selegiline, by blocking the breakdown of dopamine and subsequent production of free radicals, has been shown to have a neuroprotective effect. Recent studies have indicated that the early administration of selegiline can delay the time to onset of severe symptoms by as much as 50%. The side effects of the drug are generally mild, although high doses can cause hypertensive crisis.

Catechol-O-Methyltransferase Inhibitors

Catechol-O-methyltransferase (COMT) inhibitors, mainly tolcapone and entacapone, are relatively new drugs designed to combat PD. The mechanism of action of these drugs is similar to that of carbidopa in inhibiting the breakdown of L-dopa. The enzyme COMT selectively metabolizes L-dopa in the central and peripheral nervous system, decreasing its bioavailability. The COMT inhibitors, by blocking the enzyme, effectively increase the central uptake of L-dopa. The use of COMT inhibitors simultaneously with L-dopa has been shown to reduce some of the negative motor-related side effects of L-dopa therapy.

In his research on the “on/off” motor effect of L-dopa, Borges showed that COMT inhibitors increase the duration of “on” time and decrease the duration of “off” time. Because COMT inhibitors increase the effective concentrations of L-dopa, their side effects are similar to those of L-dopa—mainly orthostatic hypotension, nausea, and vomiting. Tolcapone has been shown to cause frequent diarrhea and has also been linked to liver failure resulting from fulminating hepatitis. Liver function should be closely monitored. Tolcapone is now used only as an adjunct to the combination of L-dopa and carbidopa in patients with severe fluctuations of symptoms.

Amantadine

The antiviral drug amantadine has anti-Parkinson effects. Although the mechanism is not entirely understood, it is thought that amantadine somehow enhances the synthesis, release, or reuptake of dopamine in remaining neurons of the SNc. Side effects of amantadine—orthostatic hypotension, urinary retention, peripheral edema, dry mouth, restlessness, agitation—are considered to be less frequent and less severe than those of L-dopa therapy. Amantadine has not been shown to have any effect on tremor, although it does decrease symptoms of rigidity and bradykinesia.

Anticholinergic Agents

The anticholinergic agents trihexyphenidyl, benztropine, and biperiden have been used mainly in an adjuvant role in treating PD. These drugs block ACh-dominated cholinergic transmission and restore the balance of ACh and dopamine in the striatum. As with any anticholinergic agent, systemic side effects result from the blocking of parasympathetic stimulation of various organs and include mood changes, dry mouth, dilated pupils, constipation, urinary retention, and visual problems. Anticholinergic drugs are contraindicated in patients with glaucoma, prostatic hypertrophy, or pyloric stenosis.

Surgical Approaches

A diminished response to L-dopa is often observed after several years. Although bradykinesia, rigidity, and tremor can be controlled by L-dopa, axial symptoms such as dysarthria, gait disorders, and postural instability begin to appear and respond poorly to drug therapy. In some patients, intractable tremor, motor fluctuations, and severe dyskinesia can arise, and surgical intervention may be indicated. Stereotactic lesions can be used, or stimulators can be placed at specific points in the basal ganglia to disrupt the pathologic pathways. Pallidotomy has been used in patients who have had little or no response to drug therapy or in those with severe on/off swings. Bradykinesia, rigidity, and drug-related dyskinesia may significantly decrease. The procedure involves the stereotactic placement of a lesion in the ventral posterior portion of the globus pallidus. The disruption of ansa lenticularis inhibitory fibers projecting from the medial globus pallidus to the ventral lateral nucleus of the thalamus resolves bradykinesia and rigidity. The benefits of pallidotomy are most apparent on the side contralateral to the lesion, although some temporary ipsilateral effects have been observed.

More recent surgical strategies involve the implantation of stimulatory electrodes into the thalamus. Subthalamic nucleus, or globus pallidus with a generator that can be powered by the patient. These devices function by transiently inhibiting nearby dysfunctional neurons, thus relieving motor symptoms. Deep brain stimulation of the subthalamic nucleus has become a treatment option. The subthalamic nucleus is a structure below the thalamus that modulates the functioning of the basal ganglia and the thalamus. A surgical team composed of a neurosurgeon, an electrophysiologist, and a neurologist adjusts measurements such as the frequency and pulse width of the stimulus to achieve optimal suppression of symptoms and improvement of function.

Photo reprinted courtesy of Irene Osborn, MD, Mount Sinai School of Medicine, New York, NY.

Figure 3. Operating room during deep brain stimulation. Note the difficulty that may be encountered in attempting to emergently secure the airway.
as pulse and amplitude and the electric port to optimally inhibit output of the subthalamic nucleus to the thalamus.\textsuperscript{1} The procedure, which can be performed bilaterally, relieves symptoms of bradykinesia, rigidity, and tremor, but gait abnormalities and postural instability are known complications to treatment. Postoperative dyskinesia often resolves with subsequent reductions of doses of pharmacologic agents. Eventually, a net improvement of dyskinesia occurs in most patients. Patients with PD who are young and free of dementia and who respond well to drug therapy are ideal candidates for deep brain stimulation; patients who fail to respond favorably to L-dopa are less likely to benefit.\textsuperscript{2}

Neural transplantation has also been shown to relieve the clinical symptoms of PD or MPTP-induced PD. The techni-que involves the stereotactic implantation of fetal mesencephalic neurons or adrenal chromaffin cells into the striatum.\textsuperscript{2,3} However, some researchers assert that any benefit derived from neural transplantation is a result of the associated acute and reactive host brain injury, whereas others maintain that stem cells are more suitable as cellular replacements.\textsuperscript{4,5} For these reasons, neural transplantation has been abandoned, and it does not currently appear to be as promising as stem cell transplant-ation. At present, these techniques are controversial and, experimentally, and not generally available.\textsuperscript{6}

### Anesthetic Considerations and Management

Anesthesiarelated concerns for patients with PD include the following: an increased sensitivity to anesthetic agents; increased risk for laryngospasm, diaphragmatic spasm, and aspiration; hallucinations (visual and tactile); decreased vital capacity that may lead to pulmonary complications; violent tremors; postoperative delirium; muscle tremors that cause electrocardiographic changes mimicking those of ventricular fibrillation; and extrapyramidal symptoms.\textsuperscript{6} Possible side effects of L-dopa consist of a depletion of myocardial stores of norepinephrine, peripheral vasoconstriction, hypovolemia, and orthostatic hypotension.\textsuperscript{4}

The patient who undergoes deep brain stimulation must understand the sequence involved in the prolonged proce-dure—which commonly takes 6 to 7 hours. Cessation of anti-Parkinson therapy begins the evening before surgery to allow observation of symptoms during the procedure and accurate placement of stimulating electrodes. Unfortunately, skeletal muscle rigidity, cramping, and other side effects ensue. Preoperative medication should consist of antiemetics and antacids.

Vigilant clinical monitoring of patients undergoing deep brain stimulation should include blood pressure measurements, electrocardiography, pulse oximetry, and side stream capnography (end-tidal carbon dioxide) via nasal cannula. The stereotactic frame is placed (perhaps in the ward room) and computed tomography or magnetic resonance imaging confirms the placement and rules out hematoma formation. PD medications are given as soon as possible, which usually relieves patient discomfort. Patients are carefully observed overnight for any neurologic changes and are discharged the following day (Table 4).

Patients who present for the insertion of generators (usual-ly 2 weeks later) are often relieved to undergo general anes-thesia for the procedure. In general, patients with PD are sensitive to hypnotic agents unless they frequently take ben-zodiazepines for anxiety. The induction of anesthesia may cause fluctuations in the blood pressure and heart rate sec-ondary to a depletion of intravascular volume and an inade-quate response to hypotension (because of the decreased release of renin and depletion of noradrenenergic stores sec-ondary to L-dopa therapy). Ketamine has been known to cause an exaggerated sympathetic response; there is also a potential hyperkalemic response to succinylcholine.\textsuperscript{3}

During the maintenance of anesthesia, patients respond normally to nondepolarizing muscle relaxants. The use of agents that allow a rapid recovery is suggested.\textsuperscript{3} As previ-ously mentioned, patients with PD do exhibit an increased sensitivity to anesthetic agents. Intraoperative monitoring should include the standard measurements, including temper-ature. The electrocardiogram should be closely moni-tored when anhydromic anti-Parkinson agents (eg, L-dopa) are used. The patient’s airway is of concern because of possible muscle rigidity that may impede airway manipulation and impair ventilation.\textsuperscript{3}

Before extubation, a full recovery from neuromuscular blockade should be ensured. The anesthesia provider should be prepared to treat laryngospasm and impaired ventilation secondary to muscle rigidity. Also, the patient emerging from anesthesia may experience tremors and hallucinations.\textsuperscript{6}

Postoperatively, transient mental confusion may develop in these patients.\textsuperscript{3} Their respiratory status must be observed closely, and the use of incentive spirometry may be indi-cated.\textsuperscript{4} The state of the CNS should be monitored, as there is a potential for delirium and other psychic disturbances. It is imperative to begin anti-Parkinson therapy immediately postoperatively.\textsuperscript{9}

### Complications of Deep Brain Stimulation

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<td>Dislocation of hardware</td>
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<td>Paralysis</td>
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<td>Seizures</td>
<td>Infections</td>
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<td>Venous air embolism</td>
<td>Median cubital vein</td>
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### Anesthetic Considerations For Deep Brain Stimulation

- Head frame and airway access
- Minimal or no sedation
- Multiple locations for administration of anesthesia
- Patient positioning (patient comfort without PD medications)
- Reduction of auditory and electric noise
- Facilitates future adjustments

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<tr>
<th>Table 2. Advantages of Deep Brain Stimulation Over Neuroablative Procedures</th>
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<td>Reversible: Adverse neurologic events are reduced</td>
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<td>Customizable: Tailoring makes therapeutic efficacy more likely</td>
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<td>Accessible: Electrode and generator remain available for future adjustments</td>
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<th>Table 4. Anesthetic Considerations</th>
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See Lesson 258 page 40
Lesson 258 continued from page 39

aspiration. Also, orthostatic hypotension may result in ex-agerated decreases in blood pressure in response to violent anesthetics. Finally, indirectly acting sympathomimetic amines should be avoided if the patient is taking selegiline.

Management of the Case Presented

The anesthesiologist saw the patient at the preanesthetic assessment clinic 2 days before surgery. She was very ner-vous and asked to not be alone during the radiologic studies. She arrived at the hospital at 6:30 am on the day of surgery. After the patient had been sedated with 1.0 mg of midazolam given intravenously in divided doses, the scalp was infiltrated with bupivacaine 0.5% and the head frame was placed. The patient was monitored during the preliminary studies, and 75 mcg of fentanyl was administered in divided doses.

In the operating room, the patient was given propofol in 10-mg increments. However, she was not responsive to the surgeon’s questions, and exsanguination was substitut-ed. She tolerated the 5-hour procedure well and was returned to a monitored unit for overnight observation. Two weeks later, while under general anesthesia with sevoflu-rane, she underwent placement of the stimulator. There were no complications.

Conclusions

PD is a common, progressive, neurodegenerative disorder that primarily affects motor functions. The loss of dopaminergic neurons in the substantia nigra pars compacta leads to decreased levels of dopamine in the striatum and results in the primary motor symptoms of bradykinesia, muscular rigidity, resting tremor, and postural instability. Pathology testing characteristically reveals the diffuse presence of Lewy bodies and global loss of dopaminergic neurons in the substantia nigra pars compacta. Although there is no actual cure for PD, several surgical and nonsurgical therapies are capable of controlling symp-toms. Pharmacologic therapies typically seek to replace dopamine in the striatum and usually consist of L-dopa-car-bidopa and/or a dopamine agonist. Surgical methods include pallidotomy and the neural implantation of devices that block the pathological pathways causing motor symptoms. Anesthesia providers must remain cognizant of the manifestations of motor dysfunction in PD, such as laryngospasm, that can affect ventilation. Moreover, drug interactions and side effects can be complicating factors.

References