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Parkinson disease:

Enhance nursing knowledge

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Abstract: Parkinson disease (PD) is a progressive, incurable disease caused by dopamine deficiency. This article provides an overview of this neurodegenerative disorder and offers information for optimal outcomes.

Keywords: dopamine, levodopa, Lewy bodies, motor functions, Parkinson disease, PD

PARKINSON DISEASE (PD) is a progressive, incurable neurodegenerative disorder caused by a deficiency of the active neurotransmitter dopamine.[1] Affecting approximately 7 million individuals worldwide, PD is the second most common neurodegenerative disorder after Alzheimer disease. Its incidence is expected to double over the next 20 years.[2,3] Although rare in individuals under age 50, PD increases in prevalence with age and affects approximately 4% of those over age 80.[4,5]

There is no cure for PD, but a correct diagnosis is important in determining treatment strategies and eligibility for clinical trials and epidemiologic studies.[6] This article discusses the diagnosis and treatment of PD for optimal patient outcomes.

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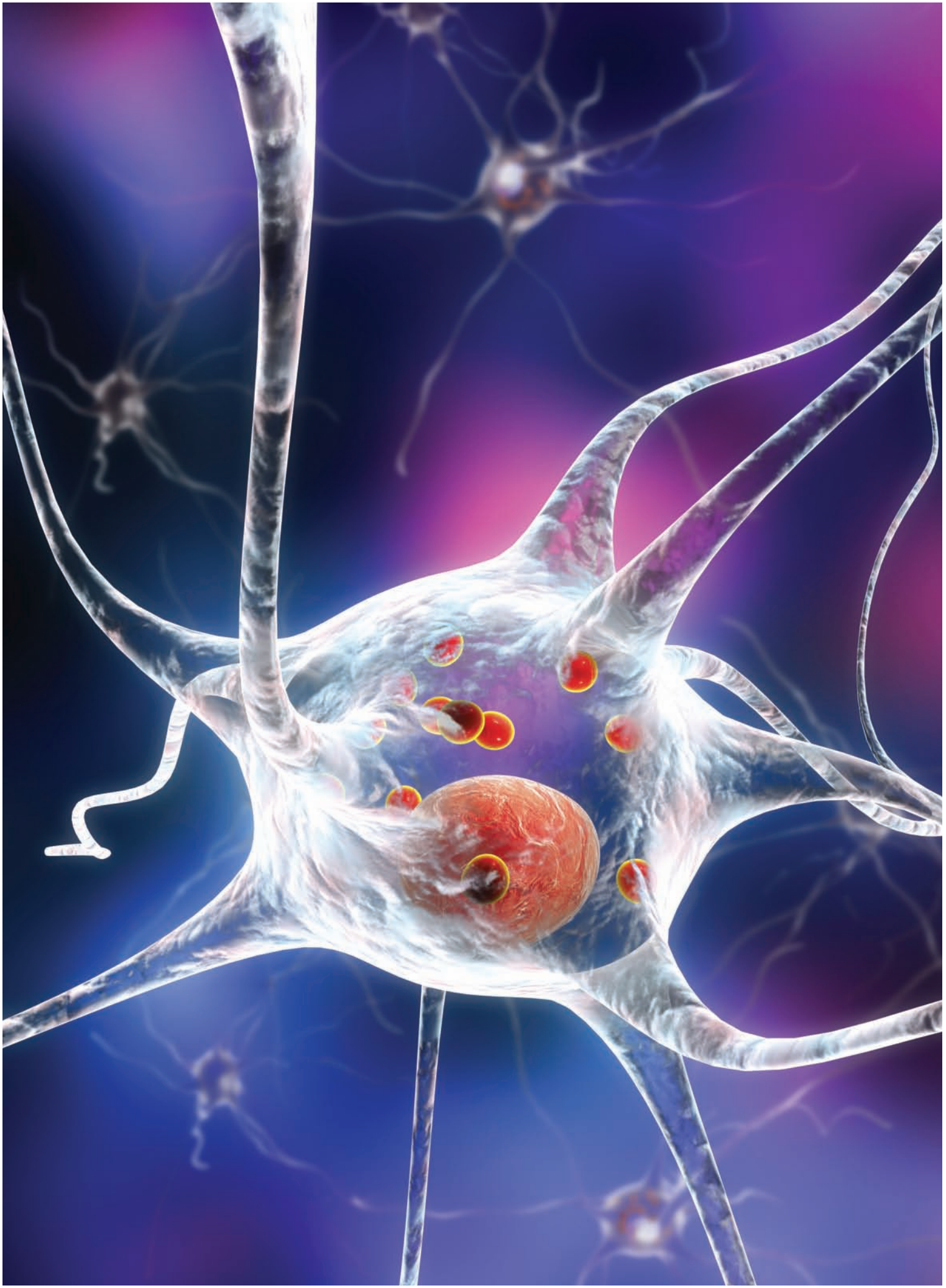


Background

Dopamine is a neurotransmitter produced in the substantia nigra, which is part of the basal ganglia. Present in both the central and peripheral nervous systems, dopamine regulates motor and nonmotor functions.[3,4,7] The principal pathologic characteristic of PD is the progressive degeneration of dopamine-producing neurons, resulting in a significant reduction in available dopamine.[4] This dopamine deficiency causes PD.[1,3]

Pathophysiology

PD is characterized by two pathologic processes:[1,5]

- premature loss of dopamine-producing neurons
- accumulation of Lewy bodies (abnormal aggregates of a misfolded protein).

PD can be classified as familial or idiopathic. Familial PD results from specific genetic mutations, and evidence suggests a genetic correlation when patients under age 50 begin to experience symptoms.[3,8] Between 10% and 16% of early- and late-onset PD cases can be linked to a first- or second-degree relative.[5] Most cases are idiopathic, however, suggesting that nongenetic and environmental factors may be important contributors in the disease's onset and progression. For example, prolonged exposure to certain environmental toxins such as heavy metals can damage dopamine-producing neurons, leading to dopamine deficiency and an increased risk for PD.[1]

Dopamine-producing neurons in the substantia nigra are prominently affected by neurodegeneration, but the cause of degeneration and impairment is not fully understood.[3,9] A key known neuropathology in PD is Lewy body deposition, however, which results from abnormal aggregates of a misfolded protein called alpha-synuclein.[1,10] The presence of Lewy bodies caused by misfolded alpha-synuclein may result in neuron dysfunction and degeneration that involves the substantia nigra, neurotransmitter systems and pathways, and many other areas of the brain.[1,5,10]

Dopamine and acetylcholine exist in a balance to transmit signals throughout the basal ganglia, including the substantia nigra. The degeneration of dopamine- and acetylcholine-producing structures leads to deficiencies in both neurotransmitters. These deficiencies correlate to the motor and non-motor manifestations of PD. Although there are deficiencies in both neurotransmitters, dopamine levels are affected more than acetylcholine, leading to the non-motor manifestations of PD. Pharmacologic therapies are aimed at promoting and maintaining adequate levels of each neurotransmitter.[11] (See *The role of dopamine*.)

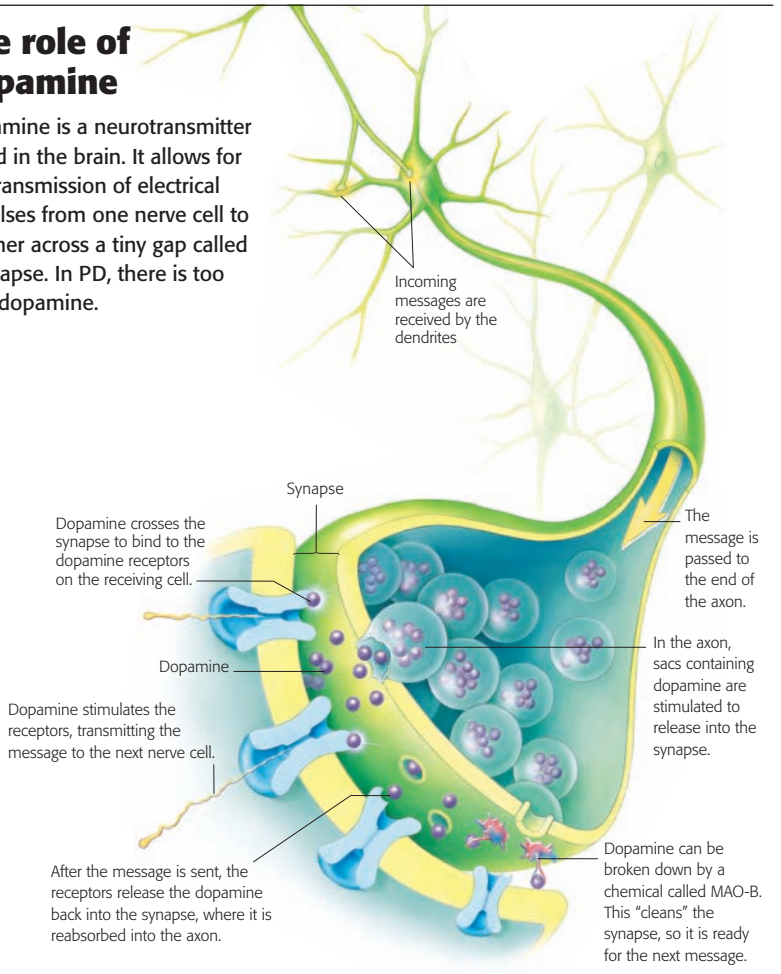
Signs and symptoms

PD is characterized by both motor and nonmotor signs and symptoms. Although there are many important nonmotor symptoms, this article focuses on motor signs, which present early and worsen throughout the course of the disease.[1,9]

Motor manifestations begin asymmetrically. The site of damage in the brain responsible for PD motor signs is contralateral to the side of signs affecting the body.[2,5,6,12,13] These become bilateral as the disease progresses. Signs and symptoms may

The role of dopamine

Dopamine is a neurotransmitter found in the brain. It allows for the transmission of electrical impulses from one nerve cell to another across a tiny gap called a synapse. In PD, there is too little dopamine.



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include various altered and impaired movements, such as bradykinesia or generalized slowed movements, resting tremors, muscle stiffness causing rigidity and slowness, hypomimia or masked facial expression, micrographia or small handwriting, hypophonia or soft voice, and small shuffling steps, as well as impaired balance and coordination, postural instability, and gait difficulty.[7,13,14]

Bradykinesia occurs when approximately 70% to 80% of dopamine-producing neurons have failed. It is considered the strongest clinical indicator of dopamine deficiency in PD.[1,11] Upper extremity resting tremors, known as pill rolling, resolve with movement. Hypomimia initially presents as a reduced blink rate. Dysphagia may lead to drooling or sialorrhea.

Gait and postural disorders seen in PD may lead to falls.[3,4] Patients experience shuffling steps that lead to balance, gait, and postural instability. Other common gait disturbances include diminished arm swing, foot and leg dragging, a fast-moving, forward-leaning movement called festination, and freezing of gait, which patients may describe as being stuck in place or glued to the floor.[9]

Unintended weight loss is another common clinical finding in patients with PD. Although the exact mechanisms are unclear, it is known that body weight depends on the balance between energy expenditure and dietary intake. Whether this unintended weight loss is the consequence of increased energy expenditure from tremors and muscle rigidity, decreased energy intake due to dysphagia or loss of sense of smell (anosmia), or a combination of both remains unclear. However, a recent study suggests that unintended weight loss in patients with PD is likely a result of disease progression rather than involuntary movements or decreased food intake.[15]

Due to dopamine and acetylcholine imbalances in the brain and central nervous system, patients with autonomic nervous system dysfunction may present with urinary incontinence, sexual dysfunction, constipation, orthostatic hypotension, sleep disturbances, and anosmia. They may also experience deteriorating physical function complicated by fatigue, muscle cramps, and pain.[2,5]

Acetylcholine reduction in PD correlates with cognitive decline. Cognitive impairments may include memory loss. Psychological and emotional effects include anxiety, fear, panic, and a sense of powerlessness associated with loss of independence. Patients have reported embarrassment, shame, and anger due to loss of dignity and distress from changing family relationships. Similarly, they have also reported feelings of identity loss, failure, and reduced self-worth, which could lead to social isolation, depression, and suicidal ideation.[16]

Currently, a needs-based approach is recommended to understand the consequences of living with PD and explore means of improving quality of life.[16] As the disease progresses, emotional and psychological counseling from a mental health professional may be recommended, as well as spiritual support according to patient preference.

Stages of PD[1,35]

Stage 1	During the initial stage, patients have mild signs and symptoms that generally do not interfere with daily activities. Tremor and other movement signs occur unilaterally only. Changes in posture, walking, and facial expressions may occur.
Stage 2	Signs and symptoms begin to worsen. Tremor, rigidity, and other movement disorders become bilateral. Gait problems and poor posture may be apparent. Patients are still able to live alone, but daily tasks become more difficult and take more time.
Stage 3	In midstage PD, loss of balance and bradykinesia are hallmarks. Falls are more common. Patients may still be fully independent, but their signs and symptoms significantly impair activities of daily living such as dressing and eating.
Stage 4	Signs and symptoms are now severe and limiting. Patients may be able to stand without assistance, but movement requires a walker. Patients require help with activities of daily living and are unable to live alone.
Stage 5	This is the most advanced and debilitating stage. Stiffness in the legs may make it impossible to stand or walk. Patients require a wheelchair or may be bedridden. Around-the-clock nursing care is required for all activities. Patients may experience hallucinations and delusions.

Diagnosis

Typically, PD does not become symptomatic until 70% to 80% of the body's dopamine-producing neurons have been lost.[1] It is not commonly recognized or diagnosed in the early stages due to a long latency period between the initial neuron damage and symptom onset (see *Stages of PD*).[1]

Diagnosis is based on data collected during the patient's health history and physical assessment; specifically, a minimum of two cardinal motor signs such as resting tremor and bradykinesia.[5]

Neurodiagnostic imaging may also be used to help diagnose PD and rule out other disorders.[1] Using radiotracers and computer techniques to generate 3-D images, both positron emission tomography (PET) and single-photon emission computed tomography (SPECT),

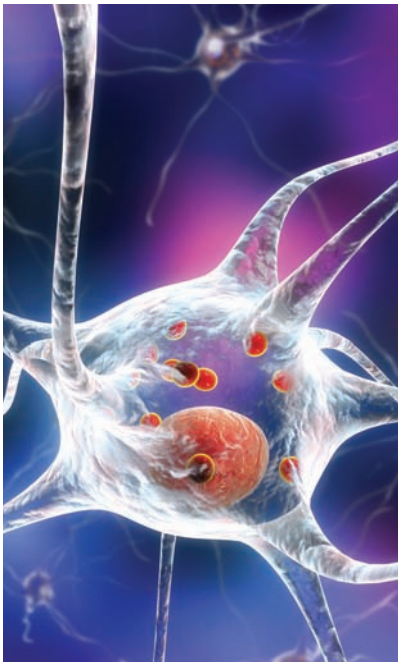
can detect a loss of dopamine-producing neurons. Imaging results alone are not sufficient to diagnose PD, but they can be used to support a diagnosis in patients with classic signs and symptoms.[1,12,17]

Diffusion-weighted imaging is a form of MRI that measures the rate of water diffusion through tissue to determine its structural details. A higher measured diffusivity of water molecules may translate to greater water mobility due to dead cells and reduced tissue volume. This technique is useful for differentiating PD from other disorders with similar presentations.[1,5]

A dopamine transporter (DaT) scan is a diagnostic imaging study used to differentiate or confirm abnormalities in dopamine transmission in the basal ganglia. DaT scans are performed via SPECT using a radioactive pharmaceutical agent that binds to DaT proteins in the presynaptic membrane on the terminals of dopaminergic projections from the substantia nigra and in structures such as the striatum in the basal ganglia. It provides a marker for dopamine terminal innervation. DaT-SPECT imaging can help clinicians monitor the degeneration of presynaptic terminals in dopaminergic neurons by visualizing the quantity of DaT proteins.[1]

Pharmacologic management

PD cannot be cured, but medication can help patients manage signs and symptoms.[1] Because of the complex and fluctuating patterns in both the occurrence and progression of PD signs and symptoms, the disease is typically perceived as unpredictable and difficult to manage.[16] Current therapies target both dopamine and acetylcholine production. For example, rivastigmine inhibits cholinesterase from breaking down acetylcholine. It is indicated to treat cognitive dysfunction in patients with PD.[11]



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Typically, PD is treated with dopaminergic drugs or dopamine agonists to counter the lack of dopamine in the substantia nigra. These medications alleviate PD signs and symptoms temporarily by enhancing dopamine levels, mimicking the action of dopamine, or inhibiting the body's ability to metabolize dopamine.[1,18]

Levodopa, which is the immediate metabolic precursor of dopamine, has been an effective PD medication for more than 40 years. When administered orally, it is converted to dopamine in the liver before crossing the blood-brain barrier.[19] This can lead to adverse reactions such as nausea, vomiting, and hypotension.[4,19] To prevent this, it is used in combination with a peripheral decarboxylase inhibitor, such as carbidopa in the US.[19] Carbidopa inhibits decarboxylation and slows the breakdown of dopamine until it passes through the blood-brain barrier for increased availability of levodopa and a longer duration of its therapeutic effects.[1,4,20] Because approximately 80% of patients with idiopathic PD respond to levodopa, diagnosis should be reevaluated after levodopa has been prescribed on a trial basis.[5]

Patients with PD may experience motor fluctuations such as slow, extended muscle spasms and dyskinesias or abnormal involuntary movements that cause rapid jerking. Also known as the on-off effect, this occurs when the therapeutic effect of levodopa wears off before the next scheduled dose.[14]

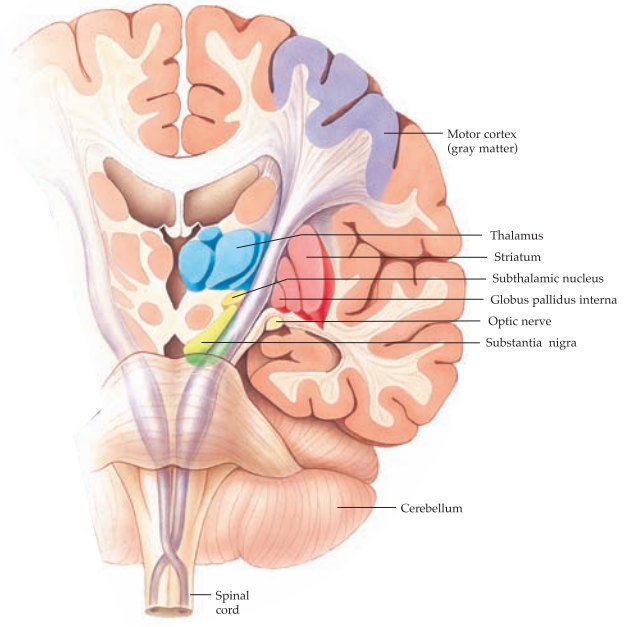
Recently, a carbidopa and levodopa enteral suspension received FDA approval for the management of motor fluctuations in patients with advanced PD, providing an option for patients who cannot take or tolerate oral forms of the drug.[21] Additionally, it eliminates absorption issues sometimes associated with oral levodopa and the first pass effect. Indicated for use over a 16-hour period via infusion, it is administered into the jejunum through a percutaneous endoscopic jejunostomy tube using a portable infusion pump.[21] The daily dose is equivalent to oral levodopa, varying depending on patient response, and includes a morning dose, a continuous dose, and additional doses as necessary.[21]

The carbidopa and levodopa enteral suspension has no current age limitations or neurocognitive exclusions, but it is contraindicated for patients on nonselective monoamine oxidase inhibitors.[5,21]

Studies of carbidopa and levodopa enteral suspension have shown a significant reduction in motor fluctuations and dyskinesias in advanced PD, but the drug does not eliminate signs and symptoms completely. In a 7-year follow-up study of 59 patients with advanced

What is Parkinson disease?

Parkinson disease (PD) is a slowly progressive, degenerative disease of the brain. It affects nerve cells in the basal ganglia and the substantia nigra. Nerve cells in the substantia nigra produce dopamine, an important neurotransmitter that acts as a chemical messenger in brain circuits to plan and control body movement. For reasons not yet understood, the nerve cells in the substantia nigra die. When 70% to 80% of dopamine-producing neurons are lost, signs and symptoms such as tremors, slowness of movement, stiffness, and balance problems occur.[1] PD usually occurs in men and women in their 60s but can occur earlier. The cause is largely unknown.



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PD, 90% of patients treated with the enteral suspension reported improved quality of life, autonomy, and clinical status.[5] Modified-release levodopa, such as controlled-release medications, may be used to reduce motor fluctuations, but these drugs are not typically a provider's first choice.

Monoamine oxidase-B inhibitors (MAO-BIs) used to manage PD include rasagiline, selegiline, and safinamide. They work by inhibiting the metabolism of dopamine in the substantia nigra.[2] MAO-BIs can be taken alone or in combination with other anti-Parkinson drugs.[14] This drug class blocks the MAO-B enzyme system in the central nervous system by inhibiting the breakdown and potentiating the duration of dopamine, serotonin, and norepinephrine.[14,22]

Although beneficial, MAO-BIs may cause a hypertensive crisis when taken with stimulants; for example, in concurrent use with some common over-the-counter (OTC) drugs, such as cough and cold medication. Understanding drug-food interactions with some MAO-BIs, such as rasagiline and selegiline, is important. Because these drugs prevent the metabolism of dopamine, foods and beverages that contain tyramine, which causes the release of norepinephrine, can lead to severe hypertension or intracranial hemorrhage when taken together.[22]

Catechol-O-methyltransferase (COMT) inhibitors represent another class of medications used for PD. When used in combination with levodopa, COMT inhibitors such as tolcapone or entacapone inhibit the metabolism of peripheral dopamine, increase the effect of levodopa, and reduced the off effect.[4,23] Additionally, they increase the uptake of levodopa and the concentration of dopamine in the brain for improved clinical effectiveness.[4]

COMT inhibitors should be taken with food to minimize gastrointestinal upset. As these medications may result in dark-colored urine, inform patients and caregivers that this is expected and harmless. Similarly, instruct patients and caregivers to report any signs and symptoms such as jaundice or right upper quadrant pain due to potential hepatic dysfunction.[2,22] Some evidence suggests that combinations of levodopa and COMT inhibitors are associated with earlier onset and increased frequency of dyskinesias.[14]

Dopamine agonists, which may be prescribed to reduce the off effect of other PD medications, include pramipexole, ropinirole, and bromocriptine.[5] This drug class is slightly less effective than levodopa and tends to have an increased rate of adverse reactions, such as sedation, lower extremity edema, visual hallucinations, and impulse control disorders (compulsive gambling, eating, and shopping).[14]

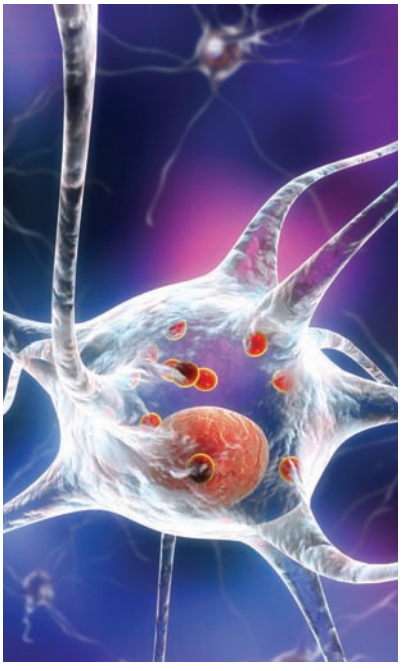
Anticholinergics may be administered to reduce tremors in patients with PD under age 70 who do not have significant bradykinesia, akinesia (failure of willed movement to occur), or difficulty walking. They may be taken alone or with levodopa or dopamine agonists in patients with more advanced PD. There are several anticholinergic medications available, including trihexyphenidyl, benztropine, orphenadrine, procyclidine, and biperiden. They all are considered equally effective.[14] Drugs in this class may cause dry mouth, which can be treated with artificial saliva products, frequent oral care, fluids, sugarless gum, or hard candies.[2,22]

Antivirals such as amantadine may be considered to reduce dyskinesias and are effective in 60% to

70% of patients with PD.[5] When combined with levodopa, amantadine is particularly beneficial in reducing dyskinesias in those with advanced disease.[14]

Because PD is a chronic, progressive neurodegenerative disorder, these medications may become less effective over time and will not relieve other nonmotor signs and symptoms alone. Developing an individualized strategy for effective pharmacotherapy over the course of the disease is important, as is taking both motor and nonmotor manifestations of PD into consideration.[4] Because dyskinesias and motor fluctuations may develop after long-term use or high dosages of medication, patients may require multiple medication adjustments with adjunctive treatments.[5] To avoid acute adverse reactions such as neuroleptic malignant syndrome, antiparkinsonian medications should not be withdrawn abruptly.[5]

If a patient is admitted to the hospital, attempt to maintain the same medication time schedule that the patient uses at home for these medications. Each patient has a highly specific and individualized drug schedule, so medication reconciliation is very important. Providers must be aware of safe practices, such as built-in alerts, neurologic consultations, surgical times, and managing swallowing and NPO status to avoid potential delays, discontinuations, and contraindications.[24] The consequences of a forgotten or late dose may be uncomfortable, distressing, and last hours. Advise patients to contact their healthcare provider for guidance in these instances.[22]



All medications, especially dopamine agonists, must be taken exactly as prescribed with round-the-clock dosing.

Surgical options

Deep brain stimulation (DBS) is a therapeutic option used in certain brain-related disorders. When PD symptoms become severe and medications can no longer moderate them, DBS and other surgical options may be considered. For example, thalamotomy, pallidotomy, and subthalamotomy may be used for treatment in areas with limited resources for DBS.[25]

The DBS system consists of a programmable infraclavicular internal pulse generator (IPG) and electrodes implanted in the targeted deep brain structure such as the subthalamic nucleus or globus pallidus. The IPG is the power source and connects to the intracranial electrodes via an extension cable tunneled subcutaneously from the cranial area to the infraclavicular area, often on the right side.[26,27]

The effect of DBS on neurons may lead to neurogenesis and neuroplasticity and can improve motor problems, such as dyskinesia and tremor, as well as other levodopa-responsive signs and symptoms for 3 to 5 years following the implantation.[1,25] Recent studies have demonstrated that DBS is a viable therapeutic option even in the early stages of PD.[25]

Complications associated with DBS can be broken into three categories:[25,28]

- **Surgical:** Surgical complications are infrequent but may include confusion, seizures, cerebral hemorrhage, cerebral spinal fluid leak, permanent neurological damage, and mortality in rare cases.
- **Hardware:** Complications associated with DBS hardware include lead misplacement, migration, or failure requiring replacement. Additionally, malfunction of the IPG can lead to a loss of symptomatic benefits, requiring replacement.
- **Stimulation:** Usually mild, stimulation-related complications may include paresthesias, dysarthria, dizziness, eyelid-opening apraxia, and facial contractions. These are often resolved by reprogramming the stimulator.

Typically, DBS complications are related to the placement of the intracranial electrodes during surgery. Infection occurs in 1.2% to 15.2% of patients and may require device removal and antibiotics; intracranial hemorrhage occurs in 5% of patients and may cause permanent neurologic deficits or mortality (up to 1.1% of patients).[4,26] Additionally, diathermy, or the therapeutic production of heat by high-frequency electric currents, electrocautery, and MRI,

Resources

American Parkinson Disease Association: www.apdaparkinson.org
Parkinson's Foundation: www.parkinson.org
The Michael J. Fox Foundation for Parkinson's Research: www.michaeljfox.org

should be avoided in patients with DBS systems due to the potential for thermal injury to brain tissue, reprogramming of the device, damage causing device malfunction, and patient harm.[5,29,30] DBS may also interfere with patient monitors and therapeutic devices, such as pacemakers and implantable cardioverter-defibrillators.[30]

Nursing considerations

Nursing care must be holistic and comprehensive. Patients with PD experience motor and nonmotor dysfunction and deficits that worsen with time, putting them at risk for physical impairments and loss of mobility, falls, urinary retention, and constipation related to decreased peristalsis, and malnutrition from a combination of the disease process and adverse reactions from prescribed medications. Additionally, they may experience disturbances in body image related to changes in their appearance due to the disease process or a knowledge deficit due to complex and evolving lifelong treatments.[22]

Nutrition recommendations include adequate dietary fiber and fluids to prevent or reduce constipation associated with the disease process and drug therapies.[22] A Mediterranean diet, characterized by high intake of vegetables, legumes, fruits, and cereals, may help prevent weight loss. This diet includes a high intake of unsaturated fats and a low intake of saturated fats. Increased caloric intake is also recommended for PD patients, as well as adequate calcium and vitamin D to prevent osteoporosis.[31]

Recent PD research has shown the benefits of exercise in improving motor performance and potentially slowing motor and neural degeneration.[5,32,33] For some patients, exercise and physical therapy are options for maintaining and possibly improving motor strength, flexibility, and function. Evidence suggests that physiotherapy may be beneficial in addressing specific motor features, including falls, freezing of gait, and overall physical deconditioning.

For those with early-stage PD, nurses should encourage a variety of exercises such as walking or dance therapy. Speech therapy may also be considered to improve voice volume. Similarly, occupational therapy can be utilized for practical home issues related to daily living and may help with driving assessments.[5]

Aerobic, strengthening, and stretching exercises can have a positive mental and physical effect. Exercise can improve balance, quality of life, and socialization. All exercise programs should be done in collaboration with the patient's primary provider.[22] The Lee Silverman voice treatment (LSVT) BIG study is an exercise-based physical or occupational therapy. It was derived from the LSVT LOUD, a speech therapy utilized in the management of deficits such as hypophonia. The LSVT Programs may have a positive impact on mobility and cognitive ability in patients with PD.[34]

Patient and family education

In early-stage PD, nurses must educate and support patients and families in strategies to maintain a safe and active lifestyle. Over the course of the disease, nurses also provide education about prescribed medications.

Emphasize that all drugs, especially dopamine agonists, must be taken exactly as prescribed with round-the-clock dosing. Inform patients that some medications require titration and take several weeks to achieve the desired therapeutic response. Extended- or sustained-release drugs should never be crushed, chewed, or altered in any way. Patients and caregivers may also require information about extra doses when traveling or attending functions in which motor control is important.[22]

While patients are treated with PD medication, any changes in the patient's clinical status must be reported to the provider immediately. These may include visual blurring, altered mental status, lethargy, irregular pulse, or severe uncontrolled movements. Emergency response teams should be alerted for more severe conditions such as difficulty breathing.[2,22]

Because the disease process and effects of the medication may lead to postural hypotension and syncope, slow position changes and graduated compression stockings may be appropriate. Unless approved by the provider, patients should avoid alcohol and OTC medications and herbal supplements as they may interfere with prescribed medications. As patients progress from early- to advanced-stage PD, education and support regarding palliative care may be necessary.[22]

Living with PD

PD is a chronic progressive neurodegenerative disease resulting from dopamine deficiency and leading to both motor and nonmotor signs and symptoms. It is a lifelong disease, and holistic approaches that address all aspects of patient care are essential for optimal patient outcomes. ■

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