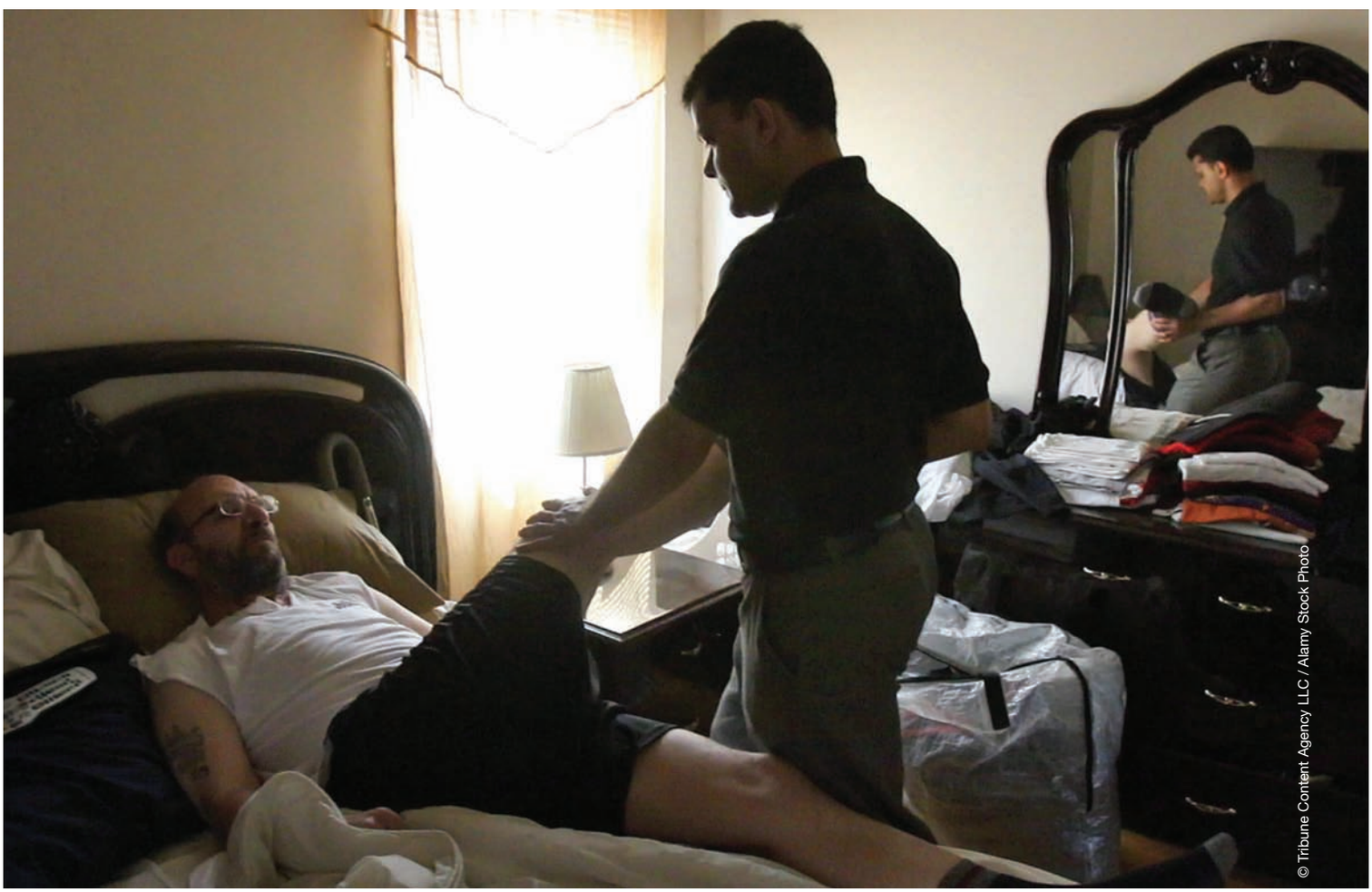
PARKINSON DISEASE



Parkinson disease (PD) is a progressive neurodegenerative disease that affects one million people in the United States. This article reviews the etiology and pathophysiology of PD, risk factors, clinical manifestations, diagnostic criteria, and treatment of this common disease. Implications for home care clinicians are included.

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Parkinson Disease (PD) is a progressive, neurodegenerative disease that causes characteristic motor symptoms of tremor, bradykinesia, and postural instability. It affects approximately 1% to 2% of adults over age 65 and 4% of adults over age 80. Approximately 60,000 Americans are diagnosed with PD annually and more than one million persons are currently living with the disease in the United States (Parkinson Disease Foundation, 2015). Due to rising life expectancy, the number of people with PD is expected to increase by more than 50% by 2030 (Dorsey et al., 2007). Males are predominantly affected with a male-to-female ratio of 3:2. Caucasians are more commonly affected than African Americans or Asians (Wright Willis et al., 2010).

Etiology/Pathophysiology

PD is caused by deterioration of the dopaminergic neurons in the extrapyramidal tract of the midbrain. There is also accumulation of α-synuclein proteins, known as Lewy bodies, in the central, autonomic, and peripheral nervous system. It is unknown what triggers the initiation of PD; however, most investigators point to a combination of genetic and environmental factors (Olanow & Brundin, 2013). The extrapyramidal nerve tract modulates voluntary movements, controls maintenance of posture and coordination of gait. The tract also influences autonomic activity, sequencing of movements, and habitual activities. Degeneration of the neurons that release dopamine causes an imbalance of excitatory (acetylcholine) and inhibitory (dopamine) neurotransmitters in the region. This imbalance causes excessive uncontrollable movements, termed dyskinesias, at times, and lack of movement, known as freezing of gait, at other times (Olanow et al., 2009).

Clinical Manifestations

There is a set of characteristic motor symptoms in PD that include bradykinesia, muscular rigidity, resting tremor, and postural and gait impairment. Bradykinesia is slowed initiation of voluntary movements. The patient has a characteristic stooped posture with a slow, shuffling gait without arm swing. At times, the person may appear stiff without any facial expression. Dyskinesias, which are involuntary choreiform-like movements, are common and typically rhythmic movements of the lower limbs (Gazewood et al., 2013; Olanow et al., 2009). Often, a characteristic resting "pill-rolling" tremor is evident that becomes less prominent with intentional, voluntary movement (Crawford & Zimmerman, 2011). Nonmotor features of PD include olfactory dysfunction, staring appearance, flat affect, cognitive impairment, psychotic symptoms, sleep disorders, autonomic dysfunction, unexplained pain, depression, apathy, and fatigue (Kalia & Lang, 2015). See Table 1 for common signs and symptoms of PD.

PD is a progressive disease that occurs over the course of 10 years or more. In late-stage PD, medication resistance is a major problem. After approximately 17 years of disease, up to 80% of patients with PD have freezing of gait with risk of falls and up to 50% of patients report choking (Hely et al., 2005). Dementia is a late sign, occurring in 60% of patients with 10 years of disease duration and 83% in those with 20-year history (Hely et al., 2008). Late-stage symptoms, such as dementia and falls, are commonly the reason for admission to long-term care and high mortality (Coelho & Ferreira, 2012).

Table 1. Common Signs and Symptoms of Parkinson Disease

Motor Sign/Symptoms	Nonmotor Sign/Symptoms
Tremor	Staring appearance
Bradykinesia	Flat affect
Postural instability	Excessive salivation
Falls	Anosmia
Shuffling gait	Depression/anxiety
Stooped posture	Psychotic symptoms
Dyskinesia	Sleep disruption
Muscle rigidity	Fatigue
"Freezing" episodes	Autonomic dysfunction
Micrographia	Cognitive impairment
	Constipation
	Dysphagia
	Urinary incontinence
	Dysarthria; difficult pronunciation
	Diminished speech volume
	Unexplained pain

Adapted from Kalia, L. V., & Lang, A. E. (2015). Parkinson's disease. *Lancet*, 386(9996), 896-912 and Gazewood, J. D., Richards, D. R., & Clebak, K. (2013). Parkinson disease: An update. *American Family Physician, 87*(4), 267-273.

Diagnosis

PD is a diagnosis commonly based on signs and symptoms. Observation of a sustained response to a trial of dopamine medication (dopamine agonists or levodopa) is also commonly used in diagnosis. There are no remarkable findings on magnetic resonance imaging or computed tomography imaging studies (Kalia & Lang, 2015). Genetic markers for the diagnosis of PD are under investigation. A number of studies are focusing on levels of beta-amyloid, tau, and alpha-synuclein proteins in the cerebrospinal fluid (Pan et al., 2014).

Treatment of PD

Presently, there is no cure for PD; the goal of treatment is to provide symptomatic relief and minimize dyskinesia. There are a wide number of pharmacologic agents used for disorder (Table 2). When symptoms are under control through medication, clinicians and patients commonly call this an "on" state. Conversely, when symptoms are not being adequately controlled by medication, the term used is "off." Patients with PD undergo "on" and "off" fluctuations (Kalia & Lang, 2015).

Pharmacologic Treatment

Pharmacotherapy is begun when symptoms cause disability. Medication treatment regimens depend on age and the symptoms the patient is seeking to control (Connolly & Lang, 2014). Early in PD, some patients seek care for tremor, which can be relieved by a beta-blocker, mainly Propranolol. Alternatively, an anticholinergic, such as benztropine or trihexyphenidyl, or the antipsychotic medication, clozapine has shown good results for minimizing tremor (Connolly & Lang, 2014).

Major motor symptoms occur due to a deficit of dopamine; therefore, replacing dopamine,

Table 2. Commonly Used Medications in Parkinson Disease

Drug	Indicated Use	Potential Side Effects
Levodopa-carbidopa Levodopa-benserazide	Most effective at controlling disability; prolongs ability to perform instrumental activities of daily living	Nausea, dyskinesia, dystonia, confusion, sedation, orthostatic hypotension, psychotic symptoms
Dopamine agonists Bromocriptine Pramipexole Ropinirole Rotigotine Apomorphine (injectable)	Motor symptoms	Nausea, hypotension, leg edema, vivid dreams, impulse control disorder, hallucinations (especially in the elderly), somnolence, and sudden sleep attack
Monoamine oxidase B inhibitors Selegiline Rasagiline	Early mild symptoms, all motor symptoms Commonly an adjunct medication	Selegiline: Stimulant effect, dizziness, headache, confusion, and exacerbation of levodopa adverse effects Rasagiline: Headache, arthralgia, dyspepsia, depression, flulike syndrome, exacerbation of levodopa adverse effects, and constipation Serotonin syndrome
Catechol-O-methyl transferase inhibitors (COMTIs) Entacapone Tolcapone	Early mild symptoms, all motor symptoms Commonly an adjunct medication	Dark-colored urine, exacerbation of levodopa adverse effects, diarrhea, and hepatotoxicity
Anticholinergic Benztropine Trihexyphenidyl	Tremor	Hallucinations, nausea, dry mouth, blurred vision, urinary retention, and constipation
Beta-Blocker Propranolol	Tremor	Fatigue, dizziness, and depression
Antipsychotic Clozapine	Tremor, dyskinesia	Agranulocytosis, myocarditis, seizures, sedation, and orthostatic hypotension
Antiviral Amantadine	Gait dysfunction, dyskinesia	Hallucinations, confusion, blurred vision, ankle edema, livedo reticularis, nausea, dry mouth, and constipation

Adapted from Gazewood, J. D., Richards, D. R., & Clebak, K. (2013). Parkinson disease: An update. *American Family Physician, 87*(4), 267-273 and Connolly, B. S., & Lang, A. E. (2014). Pharmacologic treatment in Parkinson disease: A review. *JAMA, 311*(16), 1670-1683.

stimulating the brain to release dopamine via an agonist, or inhibiting dopamine breakdown are the pharmacologic strategies used in PD (Olanow et al., 2009). Levodopa provides the greatest symptomatic benefit for PD. However, clinicians should delay prescription of levodopa as long as possible because effectiveness diminishes with time. Levodopa is prescribed as a combination of levodopa-carbidopa. Carbidopa inhibits peripheral breakdown of levodopa, thereby allowing a greater proportion of levodopa to act at the central nervous system (Connolly & Lang, 2014). When levodopa or dopamine agonists lose effectiveness, there are several strategies used to enhance efficacy. Strategies include increasing the dosage of the dopamine agonist, adding another dopaminergic medication, dividing the levodopa dose into smaller but more frequent doses, or adding a catechol-O-methyltransferase inhibitor or monoamine oxidase inhibitor to inhibit breakdown of levodopa and dopamine and prolong their effects (Connolly & Lang, 2014). See Table 2 for commonly used medications for treatment of PD.

Deep Brain Stimulation

Deep brain stimulation (DBS) is used in patients who have poorly controlled symptoms despite optimal medical therapy. DBS uses a surgically placed, battery-powered medical device called an implantable pulse generator, similar to a cardiac pacemaker, to deliver electrical stimulation to specific areas in the brain that control movement. The mechanism is incompletely understood but in some individuals, DBS can block abnormal nerve signals that cause PD motor symptoms (Olanow et al., 2009).

Implications for Home Healthcare Clinicians

The patient and caregiver require education regarding how to manage different symptoms of the disease, incorporate therapeutic modalities into daily life, administer medication, and cope with side effects of medication. The clinician should aim to foster the patient's independence and enhance quality of life for the patient and caregiver.

Medication Management

Timing of medications is crucial; if medications are not promptly taken, inability to perform activities can occur. Parkinson's patients can go from "on" to "off" states very quickly. To foster the patient's self-medication, pharmacists can supply patients with easy-open caps. Also, electronic pillboxes are available that can sound an alarm when a medication is due and dispense the exact dose prescribed. It is best to supply medications in liquid form if swallowing difficulty is present. Consider mixing crushed pills (if crushable) or open capsules in applesauce or pudding. Special swallowing cups, Oralflo or Ezy dose, are available for those with dysphagia (Cotton & Heisters, 2012).

Medication Side Effects

The medications needed for PD have many potential side effects. Long-term use of levodopa can cause disabling uncontrollable dyskinesias. Some clinicians recommend using a combination of low-dose levodopa with low-dose dopamine agonist, which can reduce the side effects associated with both substances (Ossig & Reichmann, 2015). According to Johnson (2015), reducing the dopaminergic medication may eliminate hallucinations; however, increased motor disability can occur. Alternatively, the atypical antipsychotic agent, quetiapine (Seroquel), is effective with minimal side effects. Clozapine is also effective, but agranulocytosis is a potential adverse effect (Connolly & Fox, 2014). Pimavanserin, a 5-HT2A agonist, is another medication for PD psychosis (Cummings et al., 2014). It is advisable to avoid use of haldol, olanzapine, and risperidone as severe rigidity can be induced (Kalia & Lang, 2015).

Dopamine agonist treatment is associated with 2- to 3.5-fold increased risk of an impulse control disorder (ICD) (Weintraub et al., 2010). Impulsive behaviors are often motivated by pleasure, gratification, or some other reward and are largely controlled by dopamine in the brain, the same neurotransmitter involved in PD. Examples of ICDs in PD include excessive spending, gambling, hypersexuality, or skin picking (Weintraub et al., 2015). According to a study by Tanwani et al. (2015), zonisamide (a sulphonamide anticonvulsant), naltrexone (an opioid receptor antagonist), clozapine (an antipsychotic), and valproate (antiseizure medication) are all effective in treating ICD.

Gait and Balance

Exercises that specifically strengthen a person's balance, address postural rigidity, and improve flexibility are ideal for reducing risk of falls. Swimming and walking are particularly good exercises for PD. Regular exercise is associated with better quality of life and mobility, and less progression of

disease, less caregiver burden, and less cognitive decline (Oguh et al., 2014). Research has shown exercises that include attentiveness, concentration, focus on activity and movement may be beneficial for balance and may also be neuroprotective, meaning they may slow down, stop, or reverse the progression of PD. Tai Chi and Qi Gong are ancient Chinese healing exercises that include slow and well-designed physical movements that engage the whole body. They focus on controlled breathing to reduce stress, slow stretching, and self-massage. They have been shown to improve balance and flexibility through weight shifting and axial mobility. The exercises can be done standing, sitting, or lying down (Ni et al., 2014). A publication called Parkinson's Disease: Fitness Counts is available and describes exercises to improve balance, muscle strength, and aerobic fitness, and offers suggestions for gait freezing and fall prevention (National Parkinson Foundation, 2014).

Patients with PD who have impaired balance, dyskinesias, freezing of gait episodes, and bradykinesia are at high risk for falls. Various studies show that 35% to 90% of patients with PD report at least one fall per year, and in two thirds of patients, falls are recurrent (Allen et al., 2010). There are a number of assessment tools that can be used to predict falls such as the Tinetti Gait & Balance and Berg Balance Scales. The clinician and caregiver should implement fall risk prevention, which includes assessing the home environment for safety modifications. For patients in wheelchairs, necessary home modifications include 32-in wide doorways, rugs that are fastened to the floor, bathroom grab bars, nonskid surfaces, elevated toilet seat, and a shower bench. For recommended home modifications, see the Parkinson's Disease Foundation Web site at: http://www.pdf.org/en/home_safety.

Dysphagia

Dysphagia occurs in up to 80% of all PD patients in early stages of the disease and up to 95% in the advanced stages. Patients often do not seek medical help until aspiration occurs. A patient may need to have video-fluoroscopy studies of their swallowing reflex and evaluation by a speech pathologist or otolaryngologist (Argolo et al., 2015). Depending on the type and severity of the swallowing dysfunction, food can be offered in different consistencies from pureed to soft textures and in small amounts. Thickeners should be added to fluids to make the liquid the consistency of nectar or honey depending on the severity of dysphagia (Rofes et al., 2014).

Sleep Disorders

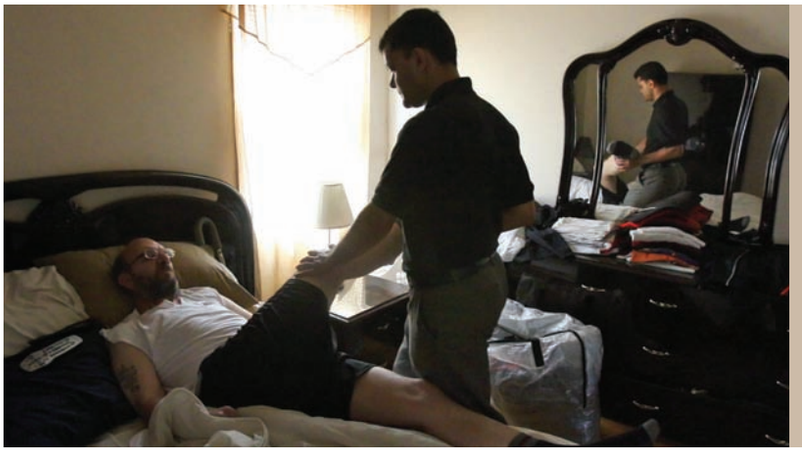
Many people with PD find it difficult to sleep through the night. Rigid muscles, tremors, stiffness, or not being able to roll over in bed can all interfere with sleep, as can the frequent urge to urinate. In addition, many people with PD experience vivid dreams or hallucinations and act out their dreams and nightmares; a problem called rapid eye movement sleep behavior disorder (RBD). In RBD, behaviors include kicking, hitting, punching, jumping, screaming, talking, and crying (Iranzo et al., 2009). Initial management of RBD should focus on safety and protective measures. Sleeping environments should be free of any potentially injurious objects, and furniture arranged to maximize safety. Review of the medication regimen is necessary because tricyclic antidepressants, serotonin, and norepinephrine reuptake inhibitors may induce RBD symptoms. Clonazepam is the recommended pharmacological treatment for RBD (Kalia & Lang, 2015).

Excessive daytime sleepiness affects up to 50% of patients (Knie et al., 2011). Daytime sleepiness is caused by impairment in circadian melatonin secretion. Also, some dopaminergic medications have been associated with episodic sleep attacks (Videnovic et al., 2014).

Interventions include increased daytime activities, exercise, and enforcement of good sleep habits. Modafinil promotes wakefulness via improvement in dopaminergic transmission and does not appear to affect the extrapyramidal motor system (Generali & Cada, 2014). Alternatively, methylphenidate may be used (Johnson, 2015).

Restless Legs Syndrome

Restless Legs Syndrome (RLS) is an irresistible urge to move the legs, usually accompanied by an unpleasant sensation, with worsening in the evening hours and with inactivity, and improvement with movement. Patients suffering from RLS describe unpleasant sensations as a "burning," "itching," "crawling," or "feeling worms under the skin" mainly in lower extremities. Dopamine agonists (ropinirole, pramipexole) should be the first-line therapy for RLS in PD. Levodopa for RLS should be avoided due to the risk of worsening of symptoms (Videnovic & Golombek, 2013). Other pharmacological approaches have included clonazepam and anticonvulsant medications. Gabapentin enacarbil



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(Pregabalin) has been recently approved for the treatment of moderate-to-severe primary RLS in adults. Tricyclic and selective serotonin reuptake inhibitor antidepressants can worsen RLS (Johnson, 2015; Kalia & Lang, 2015).

Autonomic Dysfunction

Common signs of autonomic dysfunction include orthostatic hypotension, erectile dysfunction, constipation, dysphagia, bladder dysfunction, excessive sweating, and sialorrhea (excessive salivation) (Olanow et al., 2009). Orthostatic hypotension may occur spontaneously, commonly after eating, in hot weather, and with physical exertion. Straining, such as during the Valsalva maneuver, can precipitate hypotension (Olanow et al. 2009). Orthostatic vital signs, which include taking the patient's heart rate and blood pressure (BP) when they are lying supine, sitting up, and standing, should be monitored. In the supine position, BP may be higher than in the standing position. Patients should be advised to avoid lying prone, as this can cause drop in BP. If orthostatic hypotension occurs frequently, fludrocortisone (Florinef) can be used and the patient can increase intake of sodium. Desmopressin can also be used at bedtime (Olanow et al., 2009).

Constipation can occur secondary to the disease or as a result of medications taken. Treatment includes stopping anticholinergic medication or treating the constipation with increased water intake, exercise, fiber, stool softeners, or laxatives. Osmotic laxatives such as polyethylene glycol (Miralax) and chloride channel activators (lubiprostone) can be used (Johnson, 2015).

Hyperhidrosis or excessive sweating may occur due to the changes in the hypothalamus in PD. Some patients endure drenching sweats and DBS has been effective in ameliorating this disorder. Sialorrhea (drooling) can cause embarrassment, social isolation, and worsening depression. Medications that can be given for sialorrhea include atropine drops, glycopyrrolate, botulinum toxin A, and botulinum toxin B (Olanow et al., 2009).

Bladder dysfunction is another autonomic disorder that occurs in PD because many patients develop detrusor muscle hyperactivity, which will cause nocturia, urinary frequency, and urgency. Tolterodine (Detrol) has been effective. Erectile dysfunction also often occurs and can be treated with sildenafil (Gazewood et al., 2013).

Depression

It may be difficult to detect depression in the PD patient due to flattened affect, fatigue, somnolence, and motor retardation that are effects of the disease. Depression occurs in up to 40% of patients with PD (Slaughter et al., 2001). Daily exercise should be encouraged as it can improve mood. Medications that can be used include dopamine agonists (Pramipexole), serotonin reuptake inhibitors (Citalopram, escitalopram, fluoxetine, paroxetine, and sertraline), serotonin and norepinephrine reuptake inhibitors (Venlafaxine extended release), and tricyclic antidepressants (Desipramine, nortriptyline) (Johnson, 2015; Olanow et al., 2009).

Cognitive Impairment

Cognitive impairment or dementia is a late development in PD. Studies show 30% to 60% of PD patients suffer cognitive impairment (Aarsland et al., 2005). If the clinician suspects dementia, the Parkinson Neuropsychometric Dementia Assessment (PANDA) tool is recommended (Gasser et al., 2015). The Folstein Mini-Mental Status Examination (MMSE) or the Short Test of Mental Status (STMS) can be alternatively used. The patient's medications should be reviewed as some medications can cause confusion and disorientation. Anticholinergic drugs, selegiline, and sedatives can particularly cause diminished cognitive ability (Olanow et al., 2009). Acetylcholinesterase inhibitors, such as rivastigmine and donepezil, have shown small but clinically significant improvements in cognitive status (Gazewood et al., 2013; Johnson, 2015).

Caregivers' Burden

Clinicians should evaluate the caregivers' capacity at every visit. It is crucial that caregivers are educated about resources in the community that can decrease caregivers' burden. The referral to a

psychotherapist or support group may be necessary for both caregivers and care recipients to manage anxiety, depression, and stress. Also, respite care may be beneficial for caregivers' strain (Bhimani, 2014).

Palliative and Hospice Care

Palliative care should be discussed with the patient and family members throughout the course of the illness. Advanced care planning is the cornerstone of palliative and hospice care and can help provide a longitudinal plan of care for the patient. Home care clinicians should foster open communication about choices for end-of-life care. Dementia and falls that lead to hip fracture are major reasons for admission to long-term care facilities for palliative care. Hospice care is appropriate when a patient has a life expectancy of 6 months or less. Symptoms associated with this final stage of PD include motor fluctuations with medication resistance, dysphagia with episodes of aspiration, weight loss to a body mass index less than 18.5, cognitive and autonomic dysfunction, and falls (Goy et al., 2015).

On the Horizon

According to Lindvall (2015), clinical trials of transplantation of human fetal brain tissue have shown that grafted dopaminergic neurons can restore dopamine release and, in some cases, induce major, long-lasting improvement of motor function. Recent studies demonstrate that standardized preparations of dopaminergic neurons can be generated from stem cell-based therapies and will soon be available for patient application (Gonzalez et al., 2015).

Conclusion

PD is a progressive, neurodegenerative disease that causes characteristic motor symptoms of tremor, bradykinesia, and postural instability. It affects approximately 1% to 2% of adults over age 65 and 4% of adults over age 80. PD is a degenerative disorder of dopaminergic neurons in the brain. Presently, there is no cure for PD; the goal of treatment is to provide symptomatic relief for motor and nonmotor symptoms with medication. Home healthcare clinicians are key players in assessment, support, and education of patients and caregivers. ☗

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Registration Deadline: June 30, 2018

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