Parkinson’s Disease

HANDBOOK
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Revised by Dr. Rebecca Gilbert, Vice President and Chief Scientific Officer, APDA, 2021.

Thank you to Ling Pan, MD, Assistant Professor of Neurology, NYU Grossman School of Medicine, for reviewing the section on Deep Brain Stimulation.

The information contained in this booklet is solely for the information of the reader. It should not be used for treatment purposes, but rather for discussion with the patient’s own physician.
A LETTER TO THE READER

The American Parkinson Disease Association (APDA) is here to provide you with the necessary information and resources to better manage Parkinson’s disease (PD). If you or someone close to you has been diagnosed with PD, you may feel overwhelmed. This handbook has been designed to help by providing valuable information about PD, including common symptoms, available treatments for disease management, practical tips on caring for someone with PD, and research currently underway in PD. In addition, resources that may be of help to you are provided at the end of this handbook.

As you read through this handbook, keep in mind that PD affects each person differently, so don’t worry if you come across information that does not apply to you or your loved one’s current situation. This handbook is intended to provide information for all of those affected by PD.

In dealing with PD, your partnership with your neurologist is extremely valuable. It is important to consult with a qualified physician you trust about the course of PD management that is best for you. The information in this handbook should better prepare you to talk to your physician and the additional healthcare providers with whom you will work. Managing any challenge is easier when working with a team, and PD is no exception. Ideally, you will be collaborating with a group of healthcare professionals with expertise in PD to better manage your care. This handbook will introduce you to many of the possible members of your healthcare team.

Your personal support team is also extremely valuable when living with PD. Maintaining your current networks of support (family, friends, groups you belong to) is crucial but also consider branching out to find others who can support your care. This handbook will provide information about resources and support services that are available to help you remain active and maintain important social connections as well as build new ones.

At APDA, every day, we provide the support, education, and research that will help everyone impacted by PD live life to the fullest. To accomplish this, APDA funds important research and provides education and support to individuals living with PD, their family members, and the community at large. In addition to reading this handbook, visit APDA’s website at apdaparkinson.org for more information and also consider contacting an APDA Information & Referral Coordinator who provides services in your community.

APDA is here to help you take a more active role in the successful management of PD. As President and CEO of APDA, I want to welcome you to the Association and its many resources. The American Parkinson Disease Association wants you to look to the future with hope and optimism.

Sincerely,

Leslie A. Chambers
President and CEO
American Parkinson Disease Association
WHAT IS PARKINSON’S DISEASE?

So what exactly is Parkinson’s disease (PD)? PD is a type of movement disorder* that can affect the ability to perform common, daily activities. Although PD is associated with a wide range of symptoms, there are features of PD that most people with the condition will experience. These symptoms are typically divided into those that affect movement (motor symptoms) and those that do not (non-motor symptoms).

The most common motor symptoms of PD are tremor (a form of rhythmic shaking), stiffness or rigidity of the muscles, and slowness of movement (called bradykinesia). A person with PD may also have trouble with posture, balance, coordination, and walking. Common non-motor symptoms of PD include sleep problems, constipation, anxiety, depression, and fatigue, among others. You will learn about the motor and non-motor symptoms of PD in greater detail in the next chapter.

It is important to note that, although there are common symptoms of PD, they can vary greatly from person to person. Moreover, how these symptoms change over time and whether other symptoms of PD emerge differ from person to person. Most people who develop the symptoms of PD do so sometime after the age of 50, but PD can affect younger persons as well. There are an estimated 1 million Americans living with PD and more than 10 million people worldwide.

*Note: Bolded terms are defined in the glossary at the back of this booklet.
How PD Affects the Brain

PD is a neurodegenerative disease. There is a loss of neurons (nerve cells) in certain areas of the brain, including a region called the substantia nigra (sub-STAN-she-uh NYE-gruh), Latin for “black substance.” The neurons in this region (which appear dark under a microscope) produce a neurotransmitter (a chemical messenger that allows neurons to communicate) called dopamine. Dopamine helps to regulate movement. As the number of cells in the substantia nigra decreases, there is less dopamine available in the brain. Dopamine is important to maintain normal movement patterns. This loss of dopamine is the reason that many treatments for PD are intended to increase dopamine levels in the brain. Treatment for PD will be explained in more detail in Chapter 4.

Loss of neurons in other parts of the brain also occurs in PD, and accounts for some of the non-motor symptoms of the disease.

In addition to decreases in dopamine and the cells that make dopamine, you might also read or hear about a protein called alpha-synuclein (AL-fa-sin-NUKE-lee-un). Studies suggest that alpha-synuclein normally helps neurons communicate with each other. In PD, the protein clumps up in microscopic aggregates called Lewy (LOO-ee) bodies. Researchers believe that alpha-synuclein build-up contributes to the development of PD, and that it may be possible to develop new treatments that reverse this build-up. Future research will hopefully tell us more about this protein.

Theories About Cause

The cause of most cases of PD is still unknown, although scientists believe that both genetics and environment interact to cause PD in most people who have it. Currently, there is an enormous amount of research directed at producing more answers about what causes PD and how it might be prevented or cured. When physicians diagnose PD, they often describe it as idiopathic (ID-ee-oh-PATH-ik). This simply means that the cause of PD is not known.

Genetic Factors

There are several genes that, when mutated, can increase the risk of PD. One of these, called LRRK2, is particularly frequent in families of North African or Ashkenazi Jewish descent. Mutations in the alpha-synuclein gene have also been found to trigger PD, but these are quite rare. Other genes that contribute to PD include the GBA gene, the parkin gene and the DJ-1 gene. However, in the large majority of cases, no primary genetic cause can be found. As studies of PD continue, it is likely that more genetic risk factors will be discovered. In addition, as medications are developed that target specific gene mutations, it will become increasingly important for individuals with PD to understand their particular genetic profile.

Environmental Factors

Certain environmental factors, such as significant exposure to pesticides or solvents and repeated head injuries, can increase the risk of PD. Most people do not have a clear environmental cause for their PD, and because many years can pass between exposure to an environmental factor and the appearance of PD symptoms, the connection is often difficult to establish. However, environmental factors do influence the development of PD, perhaps particularly in people who also have a genetic susceptibility.

Some environmental factors are associated with a lower risk of PD, such as exposure to caffeine and exercise (more on exercise later).
Other Risk Factors
There are other things that put an individual at higher risk for developing PD. The main risk factor is age, with the incidence of PD increasing with age. Men also have a higher risk of PD than women. The actual links between these factors and PD are not completely understood.

Now that you know a bit more about what PD actually is, the following chapter will provide greater detail about what to expect in terms of symptoms.

TIPS FOR UNDERSTANDING MORE ABOUT PD
Every day we learn more about the causes of and treatments for PD as results from new research emerge. APDA currently funds a diverse research portfolio and supports Centers for Advanced Research across the country.
To learn more about APDA’s research programs, visit apdaparkinson.org/research.

Chapter 2

SYMPTOMS
This chapter describes the most common symptoms of PD. Remember once again that, although there are typical symptoms of PD, these can vary greatly from individual to individual—both in terms of their intensity and how they progress. Motor symptoms involve movement, while non-motor symptoms do not.

Motor and Related Symptoms of PD
There are six primary motor symptoms of PD: tremor, rigidity, bradykinesia (slow movement), postural instability (balance problems), walking/gait problems, and vocal symptoms. Observing these symptoms during the neurologic exam is the main way that physicians diagnose PD.

Tremor
The characteristic tremor in PD is a slow, rhythmic tremor that typically starts in one hand, foot, or leg and can eventually affect both sides of the body. Tremor can also occur in the jaw, chin, mouth, or tongue. The classic tremor of PD is a resting tremor, which is strongest when the affected limb is at rest, and may become less apparent or even disappear during a purposeful movement. An action tremor (a tremor that occurs with intentional movement) may also be a feature of PD. In addition, some people with PD can experience a feeling of internal tremor, which is not necessarily noticeable to others.

Essential tremor is a neurologic movement disorder in which tremor is the major symptom, where the tremor is typically an action tremor, rather than the resting tremor of PD. Essential tremor is a different disorder than PD, but is sometimes mistaken for it, especially early in the disease.
Because resting tremor is a hallmark of PD, its presence (at least in some form) is a strong clue for the diagnosis of idiopathic PD. However, there are other types of tremors that can be mistaken for the tremor of PD and conversely, about 30% of people with PD never develop a tremor. It is also important to note, that if a person experiences symptoms of PD, other diagnoses may be possible, including other movement disorders, especially if he/she does not exhibit the characteristic tremor of PD. Disorders that have some of the motor signs and symptoms of idiopathic PD are called parkinsonian syndromes or parkinsonism. Therefore, it is helpful to consult with a physician who has specialized training in movement disorders to assess the quality of any recurrent or persistent tremor.

**Rigidity**

Rigidity refers to a tightness or stiffness of the limbs or torso. Rigidity, especially in the early stages of PD, may be wrongly attributed to arthritis or orthopedic problems, such as a rotator cuff injury.

**Bradykinesia**

Meaning “slow movement,” bradykinesia is a frequent symptom of PD and related movement disorders. In addition to a general slowness of movement, the bradykinesia of PD is typically demonstrated by a reduced or mask-like expression of the face (hypomimia), a decreased blink rate of the eyes, and problems with fine motor coordination (for example, difficulties buttoning a shirt). Having trouble turning over in bed and slow, small handwriting (micrographia) are other signs of bradykinesia.

**Postural Instability**

More pronounced in the later stages of PD, postural instability includes the inability to maintain a steady, upright posture or to prevent a fall when thrown off balance. Such balance problems in PD are associated with a tendency to list or fall backward (retropulsion); in fact, a light push can cause the individual with PD to continue stepping backward or to even fall down. Prominent postural instability early in the disease may indicate that the correct diagnosis is one of the other parkinsonian syndromes rather than PD.

**Walking or Gait Difficulties**

Bradykinesia and postural instability both contribute to walking, or gait, difficulties in PD, particularly as the disease progresses. A common, early symptom of PD is a decrease in the natural swing of one or both arms when walking. Later, steps may become slow and small, and a shuffling gait may appear. Gait problems in PD can also include a tendency to propel forward with rapid, short steps (festination). People with advanced PD may experience episodes of freezing, in which the feet feel as though they are glued to the floor.

**Vocal Symptoms**

In addition to the core motor symptoms of PD, changes in the voice are common. Generally, these are believed to be at least partly due to bradykinesia. In PD, the voice may become softer, or it may start off strong and then fade away. There may be a loss of the normal variation in volume and emotion in the voice, so that the individual may speak in a monotone. In more advanced PD, speaking may become rapid, with the words crowded together, or stuttering may occur. It is beneficial to see a speech language pathologist (SLP) in order to address any emerging communication difficulties as early as possible. You can find an SLP by visiting this website www.asha.org/slp and/or by speaking to your neurologist.

LSVT LOUD is an effective voice treatment program that involves retraining of vocal use and increasing the voice volume. For more information visit www.lsvtglobal.com.
Non-Motor Symptoms of PD

Because PD is a type of movement disorder, the associated non-motor symptoms can be overlooked. However, there are several common symptoms of PD that do not primarily involve movement. Some of these non-motor symptoms such as decreased smell, depression, sleep disorders, and constipation can precede motor symptoms by years or even decades.

Disturbances in the Sense of Smell

A reduced sensitivity to odors (hyposmia) or a loss of smell (anosmia) is often an early symptom of PD.

Sleep Problems

Sleep problems are commonly experienced by people with PD. The inability to fall asleep, or primary insomnia, is less common than the inability to stay asleep, or secondary insomnia. Some people with PD disrupt the normal sleep-wake cycle by taking catnaps throughout the day; doing this may lead to an inability to sleep at night. Other individuals with PD have vivid dreams, although these are more typically due to side effects of medications for PD. People with PD may also talk or thrash in their sleep, particularly during the rapid eye movement (REM) sleep stage (REM sleep behavior disorder).

Depression and Anxiety

Depression is a fairly common non-motor symptom of PD. It can range in severity and may improve with PD treatment, anti-depressant medications, or psychotherapy, such as cognitive behavioral therapy (CBT). Group or family therapy may also help alleviate depression.

Fatigue

Fatigue is a complex symptom of PD that is not fully understood. It is known, however, that fatigue is significantly associated with depression and sleep disorders.

Cognitive Decline

Particularly in more advanced PD or in older people with PD, problems with thinking, word finding, and judgment are common. Many individuals report difficulties in multitasking and organizing daily activities. Confusion may also be a side effect of some PD medications.

Weight Loss

Loss of weight is a common symptom of PD, particularly in the later stages of the illness. If weight loss is significant and unintended, your physician should perform an examination to exclude other medical causes of weight loss. While there can be a great deal of weight loss with PD, it will typically level off. There are different causes of weight loss in patients with PD, including decreased appetite (anorexia), swallowing difficulties, gastrointestinal problems such as chronic constipation, or depression. The constant motion of an advanced resting tremor or involuntary movements may burn many calories and can also be the cause of weight loss.

Gastrointestinal Issues

Disturbances of the gastrointestinal system are common in PD. Constipation, in particular, occurs frequently because PD may slow the automatic movement of the digestive system; however, side effects of medications may also contribute to constipation. Reduced swallowing and associated drooling or collection of saliva are often seen in PD. Nausea and vomiting occur occasionally in untreated PD, but more often these symptoms are related to medication side effects. Nausea and vomiting are most frequent when treatment for PD first begins.
Lightheadedness
Separate from the balance problems of postural instability but contributing to gait problems, lightheadedness or a faint feeling occurs often in PD. This symptom is related to the body’s inability to quickly regulate blood pressure, particularly when sitting up from a lying position or standing from a sitting position. This phenomenon is known as orthostatic or postural hypotension. Feelings of lightheadedness may also be increased by certain medications for PD. When severe, lightheadedness may cause black-outs or fainting.

Urinary Issues
Urinary frequency (the need to urinate often) and urinary urgency (the feeling that one must urinate right away, even if the bladder is not full) are other possible symptoms of PD. These symptoms occur because the normal reflex mechanisms that control the bladder are disrupted. Urinary problems may be worse at night, when a person is lying flat. There may also be problems with initiating a urine stream (urinary hesitancy), slowness of urination, and overfill of the bladder. It should be noted that urinary symptoms in older men specifically may be caused by an age-related enlargement of the prostate gland and not PD.

Sexual Concerns
Changes in sexual desire, or libido, is another non-motor symptom of PD that is often under-recognized. Sexual desire may be reduced in some cases because of complex psychological issues. In other cases, a reduced libido can be a direct effect of PD. Treatment with PD drugs frequently improves sexual desire and, in some cases, even increases it to a troublesome level. In men, the inability to achieve or maintain an erection (impotence) can occur; however, impotence may also be related to other age-related changes in the body or other conditions.

Sweating
Excessive sweating is a relatively common sign of PD, particularly if the disease is untreated. It happens most often in the upper body.

Melanoma
Individuals with PD may have an increased risk of melanoma, a serious type of skin cancer. As a result, people with PD should undergo annual skin examinations with a dermatologist. If you notice any troubling skin lesions, be sure to talk to your physician about them.

Most people with PD will experience both motor and non-motor symptoms. As the disease progresses, most people will begin to experience more symptoms, though exactly which ones, and how severe they are, will vary from person to person. Talk with your physician about new symptoms as they arise to determine the best treatment.

The next chapter will provide more information on how PD is diagnosed.

**PRIMARY MOTOR SYMPTOMS OF PD**
- Tremor
- Rigidity
- Bradykinesia (slowness of movement)
- Postural instability (balance problems)*
- Walking or gait difficulties*

*May be more apparent in the later stages of illness*
DIAGNOSIS

PD is usually diagnosed clinically, meaning that a physician looks for the presence or absence of the possible symptoms of PD by interviewing the patient and performing a detailed neurologic examination.

PD can often be identified by a general neurologist, who is trained to diagnose and treat neurologic disorders. To avoid misdiagnosis, consultation with a movement disorder specialist is recommended. A movement disorder specialist is a physician who has undergone additional, subspecialty training in the diagnosis and treatment of movement disorders, such as PD, after training in general neurology.

What to Expect at the First Physician Visit

When you or someone you know first visits a physician for the evaluation of possible PD symptoms, it is helpful to know what to expect. During the first visit, the physician should:

- Take a complete and careful medical history
- Take your blood pressure while you sit and stand
- Assess your thinking (or cognitive) skills
- Examine your facial expression
- Look for tremor in your face, hands, arms, and legs
- Examine whether there is stiffness in your arms, legs, torso, or shoulders
- Examine your ability to perform rapid movements with your hands and feet
- Determine whether you can get up easily from a chair, especially without using your arms
- Examine your walking pattern
- Assess your balance as you stand

Typically, a trained physician will only consider the diagnosis of PD if the person being examined has at least two of the three core motor symptoms of PD, namely tremor, bradykinesia, and rigidity. At the end of your visit, the physician should discuss with you why you may or may not have PD and the level of certainty about the diagnosis. This determination is based on your medical history and examination at this visit.

Tools to Aid Diagnosis

In addition to taking a history and performing a detailed neurologic examination, physicians can sometimes use brain imaging to help support a diagnosis of PD in particular situations. However, imaging studies have their limitations in the diagnosis of PD and are typically used only in select patients. Brain imaging is not routinely performed by neurologists or movement disorder specialists when they are considering the diagnosis of PD, especially if the person’s symptoms strongly suggest to the physician that idiopathic PD is the correct diagnosis.

Rather, use of imaging is most helpful when the diagnosis is uncertain, or when physicians are looking for changes in the brain that are more typical of one of several parkinsonian syndromes (and not idiopathic PD) and other conditions that can mimic PD. Imaging studies that may be used include magnetic resonance imaging (MRI), which examines the structure of the brain, and DaTscan, an imaging test that measures dopamine function in the brain. Although there are no specific features on MRI that confirm a PD diagnosis, certain features on MRI can suggest that the diagnosis is a parkinsonian syndrome other than PD. DaTscan is approved by the Food and Drug Administration (FDA) to help differentiate idiopathic PD from other disorders with similar symptoms, such as essential tremor. Essential tremor is occasionally difficult to distinguish from PD in its earliest stages. Because other parkinsonian syndromes may also have abnormal dopamine function, DaTscan cannot reliably distinguish between PD and other parkinsonian syndromes. Most physicians’ offices will have
access to MRI; however, DaTscan imaging may only be available at larger hospitals or medical centers.

A DaTscan may be particularly useful for the refinement of a diagnosis if a person with PD symptoms does not respond to the usual PD medications.

Other imaging studies that can be done, but that are not used routinely in the clinic, include positron emission tomography (PET), which can measure certain brain functions. Although PET can be used as a tool to assist with the diagnosis of PD and parkinsonian syndromes, it is largely reserved for research purposes at this point.

In the case of idiopathic PD, there is typically a positive, predictable response to PD medication; in the case of other parkinsonian syndromes, the response to medication may not be particularly robust, or it may be absent entirely (the next chapter will talk more about PD treatments).

Unfortunately, there are no standard biological tests for PD, such as a blood test. However, researchers are actively trying to find a PD biomarker or measurable characteristic in the body which indicates that disease is present. A biomarker could be any test that could help confirm the diagnosis of PD and follow disease progression.

The Healthcare Provider Team
In addition to a general neurologist or movement disorder specialist, you or someone you know with PD symptoms or a PD diagnosis may encounter several other healthcare providers. In fact, a team approach to the management of PD usually provides the best outcomes in the long term.

Key members of the PD healthcare team may include nurses, physician assistants, physical therapists, dieticians, social workers, occupational therapists, neuropsychologists, and speech therapists, among others. All of these individuals can play important roles in the successful management of PD, although their input may not be immediate or necessary throughout the entire course of PD. Also, seeing your primary care physician (a family physician or general internist) is important to regularly assess, maintain, and monitor your general physical and mental health.

HOW TO PREPARE FOR YOUR FIRST PHYSICIAN VISIT
- Bring your complete medical and surgical history. If you have medical records from other physicians, particularly if these records are extensive, have copies of them forwarded to the neurologist or movement disorder specialist before your visit. Be sure to include results from any brain imaging studies that you may have undergone.
- Bring a complete list of medications (prescription and over-the-counter) you take. Also include any nutritional or vitamin supplements that you take regularly. Because some medications can produce or exacerbate the symptoms of PD, it is crucial to provide a complete list of medications and supplements that you are currently taking or have taken within the last year or so.
- Know your family medical history, particularly with respect to any first-degree relatives with tremor or other symptoms resembling those of PD.
- Be prepared to share any history related to alcohol use, tobacco use, or the use of illicit drugs.
- Be prepared to discuss your living situation, social support system, and employment (if relevant), and how you are coping with your symptoms, both physically and mentally.
- Because a first-time office visit can feel overwhelming, particularly one at a larger medical center, bring along a trusted family member or friend for support and assistance. Among other benefits, this person is extremely useful for helping you gather and remember information. Ask this person to take notes during your visit. Sometimes the physician may allow you to record the conversation.
It’s Not Idiopathic PD, What Could It Be?

There are several other conditions that might produce symptoms that can be mistaken for PD. Here are some possibilities:

- Medication side effects: Certain drugs can produce or exacerbate the symptoms of PD.
- Essential tremor: This is a relatively common cause of tremor. A general neurologist or movement disorder specialist is the best physician to help differentiate between essential tremor and PD.
- A parkinsonian syndrome: The symptoms of several neurologic conditions are similar to those of idiopathic PD, but they are often managed differently and often do not respond to the typical medications for PD. Parkinsonian syndromes include multiple system atrophy, progressive supranuclear palsy, and corticobasal degeneration.
- Toxicity from exposure to certain metals or carbon monoxide

Remember: Only a general neurologist or movement disorder specialist can tell you with reasonable certainty if you have idiopathic PD. If for some reason you are not comfortable with the results of your first physician visit, getting a second opinion from another general neurologist or movement disorder specialist is always an option. It is important that you feel comfortable with your physician to ensure the best possible outcome for you.

Once you receive a diagnosis of PD, it is time to discuss treatment options with your physician. The next chapter will discuss current treatments for PD.

TREATMENTS

Once you are diagnosed with PD, your focus should be on improving your symptoms and maintaining an active and positive lifestyle. Although there is currently no cure for PD, it is possible to successfully manage symptoms through healthy choices, medications, and, in some cases, surgery.

Treatment needs change over the course of the disease. At every stage, it is important to maintain physical activity, eat a healthy diet, and pay attention to your mental health. Early treatment with medication can help most people with PD maintain an active lifestyle and continue working. Medications are adjusted throughout the course of the disease, in order to maintain the best control of symptoms and avoid major side effects. Adjusting medications can be complex, and is one of the best reasons to be seen by a movement disorders specialist.

LIVING WITH PD

Living with PD involves addressing symptoms through:

- Lifestyle, including regular exercise and a healthy diet
- Medications and other treatments
- A supportive social network
- A strong partnership with your healthcare team
**Daily Living**

**Exercise and Daily Activity**

In the management of PD, your lifestyle is one of the first things on which you will want to focus. Starting or continuing a schedule of regular exercise can make a big difference in your mobility, both in the short and long term. In fact, several research studies have shown that regular exercise routines of aerobic activity, strength training, or Tai Chi can help to maintain, or even improve, mobility, balance, and coordination in people with PD. People with PD also report the physical (and mental) benefits of swimming, cycling, dancing, and even non-contact boxing. Whatever you enjoy to stay mobile is the best activity for you, as you will be more likely to stay committed to it. Generally speaking, in the case of PD, the more active you are, the more active you’ll stay.

If you did not exercise regularly before your diagnosis, or if you are unsure about your level of fitness or stamina, talk to your primary care physician first. It’s important to have your overall health, and specifically your cardiac status, evaluated before starting any new exercise regimen. Also, a physical therapist is a great resource for finding out what your body can tolerate and what you can do safely on a regular basis. Your primary care physician or neurologist can provide you with a referral to a physical therapist. Regardless of your level of fitness, an early evaluation by a physical therapist can be very valuable. Among other benefits, a physical therapist can help you individualize your exercise regimen to suit your needs and capabilities.

The APDA Rehabilitation Resource Center at Boston University was established to help people with PD access information on exercise recommendations. This center provides callers an opportunity to speak with a licensed physical therapist who can answer questions about exercise and resources in the caller’s area. Find out more at [www.bu.edu/neurorehab/resource-center](http://www.bu.edu/neurorehab/resource-center).

In addition to physical therapists, occupational therapists can help people with PD better manage their daily activities, particularly as the disease progresses. Occupational therapists can help you make the most of your mobility with any number of daily activities—whether it’s writing, typing, cooking, driving, bathing, dressing, or grooming. Modifications for work and to the workplace environment also fall under the expertise of the occupational therapist.

A speech and language pathologist will evaluate and treat changes in voice volume and speech patterns. The Lee Silverman Voice Treatment (LSVT) program, an evidence-based therapy to increase loudness, is provided by many practitioners. A symptom that may develop as PD advances is dysphagia (dis-FAY-jiuh), or difficulty swallowing. This requires careful assessment and treatment to avoid complications due to swallowing problems. Speech-language pathologists can help treat swallowing difficulties.

**Diet**

There is no one diet that is recommended for PD, but healthy eating in general is always a good choice. For example, eating several servings of fruits and vegetables a day increases fiber intake and can help alleviate constipation, in addition to promoting general health. Also, drinking plenty of water or other non-alcoholic and caffeine-free beverages ensures adequate hydration and may reduce the likelihood of low blood pressure and constipation.

There is evidence that the Mediterranean diet and the MIND (Mediterranean-DASH Intervention for Neurodegenerative Delay) diet are brain-healthy. These diets are characterized by vegetables, fruits, whole grains, legumes and nuts, moderate amounts of low-fat proteins such as chicken and fish and fats centered around olive oil.

Registered dieticians are great resources for reviewing your diet and making recommendations about healthy foods and daily calorie counts. A balanced diet should ensure that you get the
recommended daily supply of vitamins to maintain your overall health. There is currently no evidence resulting from a well-designed trial in PD patients that a specific vitamin or nutritional supplement is useful in the management of PD, but research is actively ongoing in this area. Depending on your bone health, however, your primary care physician may recommend vitamin D and/or calcium supplements.

There has been much attention given to the possibility that antioxidants prevent or slow the progression of PD. Antioxidants are substances that remove toxic free radicals, which are produced by cells in the body during injury or stress. In cells, these free radicals promote something called oxidative stress, a condition associated with cell loss and aging. The overproduction of free radicals and oxidative stress may also contribute to the development of PD. Antioxidants, such as vitamin E and coenzyme Q10, remove free radicals to reduce the effects of oxidative stress. However, a large study published in the early 1990s showed that supplemental vitamin E did not slow the progression of PD; in fact, people with PD who took supplemental vitamin E fared worse than those who did not. As a result, supplemental vitamin E is not recommended for people with PD. In addition, another study showed that coenzyme Q10 did not provide any clinical benefit to people with PD over a placebo (sugar pill). Nevertheless, antioxidants obtained through your diet may still be beneficial, and research in this area continues.

AN EYE ON ANTIOXIDANTS
The following “super foods” contain high levels of antioxidants and other important vitamins:

- Grapes
- Blue and red berries
- Nuts
- Dark green vegetables, such as spinach, broccoli, and kale
- Sweet potatoes and carrots
- Tea, especially green tea
- Whole grains
- Beans, such as soybeans, lentils, and black-eyed peas
- Fish, such as tuna, salmon, and sardines

Depending on your prescribed medications, you may need to adjust your diet further. Your physician and pharmacist will tell you if your medications need to be taken at certain times of day or with or without certain foods or beverages. In some people with PD, dietary protein (contained in chicken, beef, fish, etc.) may affect the absorption of levodopa (LEE-voe-DOPE-ah), a common treatment for PD. In addition, if you are taking medications that include levodopa, you may need to adjust the time that you take iron supplements (if you take them), because these supplements can affect the absorption of levodopa from the gastrointestinal tract.
Medications for the Motor Symptoms of PD

Although there is no cure for PD, there are several classes of medications available for the successful treatment of motor symptoms throughout the course of the disease. Be sure to talk with your general neurologist or movement disorder specialist about your most troubling symptoms and your goals for medical therapy. Some medications work better than others for specific symptoms of PD. Make sure you provide your physicians with a complete list of medications (both prescription and over-the-counter) and any vitamin or nutritional supplements that you may be taking.

The benefits of medications can only be obtained if you have access to them and take them as directed. Some medications for PD are available in generic forms or through special programs, so that they are more affordable. Some medications for PD are available in extended release or other forms, which allows for less frequent or easier dosing. Talk to your general neurologist or movement disorder specialist about your situation as well as any preferences for obtaining and taking your medications. Remember that it is important to review with your prescribing physician or pharmacist the side effects of all of your medications, both prescription and over-the-counter, and how they may interact with your other medications or alcohol. Nurses, physician assistants, and pharmacists are also extremely valuable sources of information regarding all of your medications, including how they should be taken, their side effects, and how they may interact with other medications.

A Word About Motor Complications

Motor complications refer to disease-related and treatment-related difficulties that typically develop after several years of uncomplicated treatment of PD. As the disease progresses, a person with PD may begin to develop “off” time, when their medications are not sufficient to maintain good symptom control throughout the day. Doses and timing can usually be adjusted, and new medications added, to reduce “off” time.

In advanced disease, “off” periods may become unpredictable and less responsive to medication changes.

Levodopa treatment increases the risk for development of dyskinesias [dis-keh- NEE-zhee-ahs]), which are uncontrolled movements, especially at the time of peak brain dopamine levels or when brain dopamine levels are in flux. Dyskinesias may not be troublesome, and many people with PD report they prefer some dyskinesia with good motor symptom control to no dyskinesia with less complete symptom control. Sometimes, however, dyskinesias can be socially distressing and/or disabling. Development of significant dyskinesias is often the point at which surgery is considered, which will be discussed later.

Wearable technology refers to devices worn by a patient that capture movement information. These devices can help monitor motor fluctuations and help with medication management.

Carbidopa-levodopa (Sinemet®, Rytary®, Parcopa®, Inbrija®, generics)
The most effective treatment for PD is the combination medication of carbidopa-levodopa which is intended to increase brain levels of dopamine, that are deficient in people with PD. Levodopa, which is converted to dopamine in the brain, increases brain levels of dopamine. Levodopa reduces tremor, stiffness, and slow movement in people with idiopathic PD. Carbidopa prevents levodopa from being broken down in the bloodstream before it reaches the brain. Therefore, the addition of carbidopa allows levodopa to get into the brain more efficiently. Available in various strengths and delivery systems, carbidopa-levodopa is typically started at a low dosage to avoid nausea and vomiting, which can occur when the dose is increased too rapidly. The dosage is then increased over time as tolerated and until optimal therapeutic benefits are experienced. Carbidopa-levodopa is available in the United States as an immediate-release tablet (Sinemet® and generic), two types of extended-release formulations (carbidopa/
levodopa ER available only in the generic version and Rytary®), and an orally disintegrating tablet (Parcopa®). Levodopa alone is also available in an inhaled version (Inbrija®), to be used as needed if medication effects wear off between oral doses of carbidopa-levodopa.

Side effects of carbidopa-levodopa treatment include nausea, orthostatic hypotension, sleepiness (which can be sudden, called sleep attacks), impulse control behaviors, hallucinations, and confusion. Levodopa also contributes to the development of dyskinesias.

**Carbidopa-levodopa Enteral Suspension (Duopa™)**

An alternative form of carbidopa-levodopa (Duopa™) was approved by the FDA in 2015. It is intended for people with more advanced disease, whose symptoms are no longer responding well to oral carbidopa-levodopa. Instead of taking a pill, people with PD can receive carbidopa-levodopa in a gel form through an infusion pump. The pump delivers the medication directly into the small intestine through a surgically placed tube. The advantage of a continuous infusion of the carbidopa-levodopa is less immobility or “off” time from levodopa.

The side effects of the carbidopa-levodopa infusion are similar to those of oral carbidopa-levodopa, but may be associated with a higher incidence of peripheral neuropathy (numbness or loss of sensation in the fingers or feet).

**Dopamine Agonists: Pramipexole (Mirapex®, Mirapex ER®), Ropinirole (Requip®, Requip XL®), Rotigotine (Neupro®), Apomorphine (Apokyn®, Kynmobi®)**

Dopamine agonists are a little different from carbidopa-levodopa in that, instead of increasing dopamine levels in the brain, they mimic the activity of dopamine. They can be given alone in the early stages of PD, or as an adjunct to carbidopa-levodopa or other PD medications later on in the disease course. Dopamine agonists are available in immediate-release as pramipexole (Mirapex®) and ropinirole (Requip®) or extended-release pramipexole (Mirapex ER®) and extended-release ropinirole (Requip XL®) formulations. One of the dopamine agonists, rotigotine (Neupro®), is available as a skin patch. As with carbidopa-levodopa, dopamine agonists are typically started at a low dosage and titrated upward as tolerated and until optimal therapeutic benefits are experienced.

Apomorphine is a dopamine agonist whose effects have a rapid onset. It can be used as a rescue medication for someone whose dose of Levodopa wears off unexpectedly before the next dose is due. Apomorphine comes in two forms, Kynmobi®, a sublingual film, and Apokyn®, an under-the-skin injection.

The side effects of dopamine agonists are similar to those of carbidopa-levodopa, although impulse control disorders and sudden onset of sleepiness can be more pronounced. Apokyn® in particular can cause severe nausea, so initially, it must be given with a medication that reduces or prevents nausea.

**COMT Inhibitors: Entacapone (Comtan®), Tolcapone (Tasmar®), Opicapone (Ongentys®)**

COMT inhibitors are sometimes used with carbidopa-levodopa. Like carbidopa, they prevent the breakdown of levodopa before it reaches the brain. The result is that a more reliable supply of levodopa enters the brain, where it can be converted to dopamine. COMT (catechol-O-methyltransferase) inhibitors are typically prescribed to treat frequent “off” times with levodopa therapy. The COMT inhibitor entacapone (Comtan®) is available as a combination pill with carbidopa-levodopa (Stalevo®). Whereas a dose of entacapone is taken with every dose of levodopa, opicapone (Ongentys®) is taken once daily.

Sometimes COMT inhibitors can increase the side effects associated with levodopa therapy. Diarrhea and discolored bodily fluids such as urine can occur with entacapone. Regular blood tests for liver function are required with the use of tolcapone (Tasmar®).
Selective MAO-B Inhibitors: Rasagiline (Azilect®), Selegiline (Eldepryl®, Zelapar®), Safinamide (Xadago®)

Selective MAO-B inhibitors block the MAO-B enzyme in the brain, which breaks down dopamine. This is another way to increase dopamine levels in the brain. MAO-B inhibitors can be used alone or with other PD medications. Selective MAO-B inhibitors may be prescribed to complement carbidopa-levodopa therapy, particularly if individuals experience “wearing-off” symptoms while taking levodopa. The selective MAO-B inhibitors for PD are available in swallowed pill form, as rasagiline (Azilect®) and selegiline (Eldepryl®), or as an orally disintegrating tablet of selegiline (Zelapar®). Safinamide (Xadago®) is approved as an add-on therapy to carbidopa/levodopa for the treatment of “off” time. It is an MAOB inhibitor, but has other mechanisms of action such as inhibition of glutamate release.

Side effects of selective MAO-B inhibitors include mild nausea, dry mouth, lightheadedness, constipation, and, occasionally, hallucinations and confusion. Previous restrictions on the intake of foods containing tyramine (for example, aged cheeses, red wine, and draft beers) with selective MAO-B inhibitors have been relaxed by the Food and Drug Administration (FDA). However, MAO-B inhibitors can interact with other medications, such as certain antidepressants, nasal decongestants, and narcotic pain medications. If you are having a procedure in the hospital, you may be advised to stop your MAO-B inhibitor 2 weeks prior to the procedure and restart it 1-2 weeks after the procedure to avoid any unintentional medication interactions. Your physician or pharmacist can help you to understand these potential interactions.

Anticholinergics: Benztropine (Cogentin®), Trihexyphenidyl (Artane®)

Anticholinergics are often used for the management of PD as adjunct medications to other PD therapies. Anticholinergics are frequently prescribed to reduce the characteristic tremor of PD or to ease the problems associated with the wearing off of levodopa therapy. Common side effects of anticholinergics include confusion, hallucinations, constipation, dry mouth, and urinary problems. As a result, the use of anticholinergics is typically limited to younger people with PD (under the age of 70). These anticholinergics should also be avoided in combination with antihistamines, certain psychiatric drugs, and alcohol.

Amantadine Formulations

Also used to prevent or treat influenza, amantadine (Symmetrel®) has been observed to ease the tremor of PD as well as muscle rigidity. It is typically used as an adjunct medication to other therapies for PD. In addition, it is used to decrease dyskinesias or involuntary movements caused by levodopa. Common side effects include lightheadedness, dry mouth, constipation, vivid dreams, lacy rash, typically on the legs, and swelling of the ankles. Amantadine dosing needs to be decreased in those with kidney disease. It may also interact with or enhance the side effects of anticholinergics and levodopa therapy. Amantadine is available in pill and syrup forms.

Two amantadine extended-release formulations are available. Gocovri® is taken once daily at night and is indicated for both the treatment of levodopa-induced dyskinesias and for reduction of “off” time. Osmolex® ER is taken once daily in the morning and is indicated for the treatment of PD motor symptoms.

Adenosine inhibitors: Istradefylline (Nourianz™)

Adenosine inhibitors block the effects of the adenosine receptor. Like dopamine, adenosine is a neurotransmitter that
works in the basal ganglia, the deep structures of the brain that are affected in PD. However, to some degree, adenosine and dopamine have opposite effects, so that inhibiting the adenosine receptor improves motor function. Istradefylline is indicated for use as an add-on treatment to carbidopa/levodopa for those experiencing “off” episodes.

**Surgery**

**Deep Brain Stimulation**

Deep brain stimulation (DBS) is a neurosurgical procedure for people with PD who retain a good response to levodopa, but who have developed significant motor fluctuations including dyskinesias. DBS may also be used to treat medication-resistant tremor. By stimulating specific points in the motor control circuits in the brain, DBS “rebalances” the circuits, restoring normal movement control to some degree. In most cases, this allows the person with PD to reduce their dosage of levodopa, and thus reduce their dyskinesias, while maintaining good symptom control.

DBS involves the implantation of permanent, thin electrodes into selected deep parts of the brain. There are contacts along the electrodes that can deliver electricity to a very targeted area in the brain. A battery-operated pulse generator, much like a cardiac pacemaker, is implanted under the skin of the chest or abdomen. The pulse generator is connected to the stimulator electrodes via wires, which are tunneled underneath the skin of the scalp and neck (see image on next page). The DBS procedure is associated with a small chance of infection, stroke, bleeding, or complications associated with anesthesia. The equipment is not visible underneath the clothes, and causes no discomfort in daily use.

Many different stimulation variables can be programmed wirelessly for maximum symptom control. These include which contacts along the electrode should deliver the stimulation, and the size, frequency, and duration of the pulses of electricity delivered. Patients also have a limited ability to change parameters on their DBS systems wirelessly. The batteries can be replaced by an outpatient procedure when necessary, typically after several years.

DBS technology is constantly evolving with many new developments in recent years. Certain systems offer rechargeable batteries which do not need to be replaced as frequently as non-rechargeable batteries. The newer DBS systems are MRI-conditional and can be placed in MRI-mode or turned off for the duration of an MRI, making the process of getting an MRI with a DBS system in place much simpler than with older systems.

In the past, the surgery was only performed when a patient was awake (albeit under mild sedation and with appropriate pain control). This allowed the surgeon to monitor the patient’s symptom as the electrode was placed to ensure accurate targeting of the electrode. Certain surgeons will now perform the surgery in the bore of a CT scanner or MRI machine, allowing for visualization of the advancing electrode and optimization of placement. When performed this way, the patient can be completely asleep during the procedure.

Although DBS is typically done at more advanced stages of disease, the FDA has also approved DBS for earlier stage PD. Talk with your doctor to see if DBS is an appropriate treatment option for your clinical situation.
The available DBS systems for PD are all slightly different. Each offers its own technology in programming the stimulation variables, with the goal of maximizing symptom control and minimizing the side effects of stimulation.

**Abbott**
The Infinity™ system uses directional leads. This means that the contacts on the electrodes are sub-divided into sections around the circumference of the electrode and stimulation can be applied to different sections. This allows for the field of stimulation to be steered in a particular direction, thereby reducing side effects of stimulation. Abbott also offers Neurosphere™, a remote programming option that allows clinicians to connect with patients through a wireless network and perform DBS programming through a telemedicine appointment.

**Boston Scientific**
The Vercise™ system offers two different electrodes, one with eight contacts, more than other leads, and one which offers directional stimulation. The electrodes use MICC or multiple independent current control, which means that each contact has its own energy source and allows for a more customizable field of stimulation.

**Medtronic**
The SenSight™ system uses directional leads allowing for a customized field of stimulation. The SenSight™ system is compatible with the Percept™ PC Neurostimulator with BrainSense™ technology, which captures local field potentials (LFPs) from the neurons that are in contact with the DBS electrode. DBS adjustments can be made based on LFP patterns of an individual patient as they correlate with reported symptoms and medication ingestion.

**Focused Ultrasound**
Focused ultrasound (FUS) is a procedure that uses targeted ultrasound waves to heat up and lesion a precise location in the brain. By destroying a very specific area of the PD brain, the normal circuitry in the brain can be restored. Focused ultrasound is currently approved for treatment of tremor in PD.

**Treatments for Non-Motor Symptoms of PD**
People with PD commonly experience non-motor symptoms, including orthostatic hypotension, cognitive difficulties, hallucinations, depression, anxiety, sleep problems, or other difficulties. If you are experiencing any of these problems, be sure to discuss them with one or more members of your healthcare team. Additional therapies or medications (beyond those that treat the motor symptoms of PD) may be useful and can be taken in conjunction with your PD medications.

**Medications Approved for Non-Motor Symptoms of PD**
- **Droxidopa** (Northera™) is specifically indicated to treat the orthostatic hypotension of neurologic diseases like PD. It should be used with caution in people with cardiac disease, as it may aggravate certain cardiac conditions. Common side effects include headache, dizziness, nausea, hypertension, fatigue, fever, and confusion. In addition, high dosages of carbidopa-levodopa may interfere with the activity of droxidopa. Other medications used to treat orthostatic hypotension are fludrocortisone (Florinef®) and midodrine (ProAmatine®).
- **Pimavanserin** (Nuplazid™) is approved to treat hallucinations and delusions that may develop in advanced PD. It can cause swelling of the ankles, constipation, and confusion. Those with heart rhythm disturbances should avoid taking this medication.
- **Rivastigmine** (Exelon®) is approved for dementia associated with PD. It can cause nausea, vomiting, and loss of appetite.
Complementary Therapies for PD

A vast array of complementary treatments for PD are available including meditation, massage, acupuncture, music therapy, and art therapy. Many of these therapies have been studied formally in PD, although usually in only small groups of people. Therefore, much is unknown about whether these therapies are truly effective and if so, how they work and how to maximize their benefit. Nevertheless, you may find them beneficial in addition to evidence-based medicines approved for PD. You should discuss the possible pros and cons of these options with one or more members of your healthcare team.

Many states have legalized medical marijuana and some PD patients have tried it for relief of select motor and non-motor symptoms of PD. There is limited research however, investigating which PD symptoms respond to medical marijuana, as well as what type, delivery system, and dose should be used. There are also side effects of medical marijuana that must be considered including dizziness and low blood pressure. You should discuss the use of medical marijuana with your healthcare team as you would any other medication.

Research in PD is ongoing, and new treatments are on the way. The final chapter discusses the latest in research, potential treatments on the horizon, and clinical trials. Also provided are additional resources for people with PD and their families.

RESEARCH AND RESOURCES

There is a great deal of ongoing research to better understand the cause of PD, confirm the clinical diagnosis of PD, and find new therapies for the symptoms of PD. In addition, investigations are underway to identify treatments that will delay the progression of PD and ultimately cure it.

Along with new formulations or new combinations of existing medications, several novel medications are in late-phase study for the treatment of PD. New levodopa delivery systems are being developed to reduce motor fluctuations and improve “on” time. Drugs are being investigated for their potential to protect nerve cells and prevent their loss. Researchers are looking for ways to target known gene mutations that cause PD or contribute to PD risk. The search for a biomarker to reliably diagnose and follow PD disease course is ongoing. Wearable technology is being perfected that detects motor symptoms of PD and can be used to diagnose and manage PD as well as follow patients during clinical trials of new treatments. Finally, DBS is continuously being studied and refined to determine the best way to deliver stimulation and the best part of the brain to be targeted for stimulation.
Caring for Those With PD

Remember that PD not only affects the person who has been diagnosed with this disease; it also affects family members, friends, and co-workers. People caring for those with PD also need support. If you are a care partner or you know someone who is, it is important to remember that the care partner must pay attention to his or her own physical and mental health. Many times, care partners overlook their own health, because so much time is spent caring for the person with PD.

If you are a care partner, consider contacting a care partner support group. Access to these groups is available through many local APDA Chapters as well as the Information & Referral (I&R) Center(s) (apdaparkinson.org/community/). These groups can provide up-to-date information on PD, treatment strategies, and tips to assist care partners. Through these groups, care partners can also meet others who have had similar experiences in caring for an individual with PD. Your state’s Department of Aging may also provide information, resources, and support for PD care partners.

Living to Your Fullest Potential

At APDA, we want you to achieve the best quality of life right now and to live to your fullest potential. It is possible to have an active and fulfilling life with PD. We hope that this handbook has been a helpful first step for you or someone you know with PD to achieve a life filled with meaning, hope, and optimism. Please contact APDA for further information and use APDA’s additional resources at apdaparkinson.org.
Resources

APDA is here to help you to live with Parkinson’s disease (PD). The APDA network provides information and referrals, education and support programs, health and wellness activities, and events to facilitate a better quality of life for the PD community. Search the APDA website by state to connect to an Information & Referral (I&R) Center or APDA Chapter in your community at: apdaparkinson.org/community/ (800) 223-2732

APDA Rehabilitation Resource Center at Boston University was established to help people with PD access information on exercise recommendations. This center provides callers an opportunity to speak with a licensed physical therapist who can answer questions about exercise and resources in the caller’s area. Find out more at: www.bu.edu/neurorehab/resource-center (888) 606-1688

For information about services provided through the Veterans Health Administration for military veterans with PD, call: (800) 223-2732 apdaparkinson.org/resources-support/national-veteran-resources

A Closer Look Blog, by Dr. Rebecca Gilbert, APDA Vice President and Chief Scientific Officer, aims to address both timely and timeless topics related to PD. In addition, the blog focuses on practical, take-home tips that can be gleaned from the information discussed. apdaparkinson.org/doctor-blogs/a-closer-look/

APDA provides free online publications on a variety of topics. apdaparkinson.org/resources-support/download-publications

GLOSSARY

Action tremor: a tremor that occurs with intentional movement

Alpha-synuclein: a protein that builds up in certain nerve cells in certain brain regions of people with PD and related conditions

Amantadine: medication originally used to treat influenza, that is now used to ease the tremor and rigidity of PD

Anorexia: decreased appetite

Anosmia: loss of the sense of smell

Anticholinergics: a class of drugs often used for the management of PD, typically as adjunct medications to other, standard PD therapies; used to reduce the tremor of PD or ease the problems associated with the wearing off of levodopa therapy

Antioxidants: substances found in certain foods and supplements that may remove toxic free radicals from the body

Biomarker: measurable characteristic in the body which indicates that disease is present

Bradykinesia: slowness of movement; a common motor symptom of PD

Carbidopa-levodopa: a combination medication commonly used to treat PD; intended to increase dopamine levels in the brain

Care partner: a person, such as a close family member or friend, who supports an individual with a chronic medical condition

Clinical trials: studies conducted in humans, often involving a drug or some other type of treatment

Cognitive: pertains to thought processes, such as memory, attention, concentration, and judgment
Cognitive behavioral therapy (CBT): a form of psychotherapy that focuses on challenging unrealistic thoughts and replacing them with more realistic ones.

COMT inhibitors: drugs that block catechol-O-methyltransferase (COMT), an enzyme that breaks down dopamine and levodopa; used in PD to prevent the breakdown of levodopa therapy before it reaches the brain.

DaTscan: FDA-approved imaging test used to detect dopamine function in the brain; can help differentiate essential tremor from idiopathic PD and other disorders that cause tremor.

Deep brain stimulation (DBS): involves the use of implantable pulse generators to improve the motor symptoms of PD, often allowing for a reduction in medication; surgical option for people with advanced PD who have tried a number of different medication regimens for their motor symptoms.

Dopamine: a brain chemical (neurotransmitter) that enables movement; brain levels of dopamine are decreased in certain brain regions in people with PD.

Dopamine agonists: drugs that mimic the action of dopamine.

Droxidopa: FDA-approved drug used to treat the orthostatic hypotension of neurologic diseases like PD.

Dyskinesias: fragmented or jerky movements of the limbs or torso; often apparent at peak times of levodopa therapy in more advanced PD.

Dysphagia: difficulty swallowing.

Essential tremor: a neurologic movement disorder in which tremor is the major symptom. Tremor is typically an action tremor, rather than the rest tremor of PD.

Evidence-based medicine: use of the best scientific evidence from clinical research to optimize clinical decision-making.

Festination: an abnormal walking pattern associated with PD, in which steps shorten and become more rapid.

Fludrocortisone: medication used to treat orthostatic hypotension.

Focused ultrasound: a procedure that uses targeted ultrasound waves to heat up and lesion a precise location in the brain.

Free radicals: toxic substances that are produced by cells in the body during injury or stress.

Freezing: involuntary inability to move; frequently “freezing of gait,” where the person with Parkinson’s wants to walk forward but their feet feel stuck to the ground.

Gait: pattern of walking.

General neurologist: a physician who is trained to diagnose and treat neurologic disorders.

Hypomimia: a reduced or mask-like expression of the face.

Hyposmia: reduced sensitivity to odors.

Idiopathic: of unknown cause.

Impotence: the inability to maintain or achieve an erection.

Internal tremor: sensation of vibration inside the body.

Lewy bodies: clumps of protein (alpha-synuclein) found in the nerve cells in certain brain regions of people with PD and related conditions.

Libido: sexual desire.

Magnetic resonance imaging (MRI): imaging technique that allows physicians to see the structure of the brain.

Micrographia: small handwriting.

Midodrine: medication used to treat orthostatic hypotension.

Motor complications: disease- and treatment-related complications, including “off” periods, dyskinesias, and other phenomena, that develop after several years of treatment.
Motor symptoms: symptoms that primarily involve movement

Movement disorder: a neurological condition that affects movement

Movement disorder specialist: a physician, typically a neurologist, who has undergone further training to diagnose and treat movement disorders

Neurodegenerative disease: a disease in which neurons (brain cells) die. Neurodegenerative diseases include Parkinson’s disease and Alzheimer’s disease

Neurons: nerve cells, which form the structure for interconnected, intercommunicating networks in the brain

Neurotransmitter: a brain chemical that allows neurons to communicate with one another

Non-motor symptoms: symptoms that do not primarily involve movement

“Off” time: periods when treatments are not providing control of symptoms

Orthostatic or postural hypotension: the body’s inability to quickly regulate blood pressure, particularly when sitting from a lying position or standing from a sitting position

Oxidative stress: a destructive condition in which free radicals damage cells; associated with cell loss and aging

Parkinsonian syndromes: movement disorders that are not idiopathic PD but have some overlapping symptoms, such as rigidity and slowness of movement (bradykinesia)

Positron emission tomography (PET): imaging technique that can measure certain brain functions

Primary insomnia: the inability to fall asleep

Rapid eye movement (REM) sleep behavior disorder: a particular type of sleep disorder that may be associated with talking or thrashing in one’s sleep

Resting tremor: a tremor that occurs when a body part is not being held against gravity and is not being moved; a hallmark of PD

Retropulsion: the tendency to fall backward

Rigidity: stiffness of the muscles

Secondary insomnia: the inability to stay asleep

Selective MAO-B inhibitors: drugs that selectively block the enzyme monoamine oxidase B (MAO-B) in the brain; MAO-B breaks down dopamine

Substantia nigra: meaning “black substance” in Latin, a region in the base of the brain that contains dopamine-producing neurons, which appear dark under a microscope; people with PD experience cell loss in this region

Tai Chi: a form of exercise developed in ancient China that can help with posture and balance

Tremor: a form of rhythmic shaking

Urinary frequency: the need to urinate often

Urinary hesitancy: difficulty initiating a urine stream

Urinary urgency: the feeling that one must urinate right away, even if the bladder is not full

Wearable technology: devices worn by a patient that capture movement information
Introducing an easier way to track your symptoms and manage your care.

Download the free APDA Symptom Tracker mobile app today.
American Parkinson Disease Association would like to acknowledge Medtronic for supporting the education mission of this program with an unrestricted financial contribution. Medtronic has no input over any educational content.