

## Parkinson's Syndrome

### Goals & Objectives

#### Course Description

"Parkinson's Syndrome" is an asynchronous online continuing education program for occupational therapists and occupational therapy assistants. The course presents an updated comprehensive review of Parkinson's Syndrome including etiology, epidemiology, diagnosis, treatment, pharmacology, therapeutic interventions, and caregiver strategies.

#### Course Rationale

Parkinson's Syndrome continues to afflict a significant percentage of the population. The effects of this disease can profoundly alter the quality of life of an individual. By better understanding the pathology and the various care options available, it is possible for occupational therapists and occupational therapy assistants to have a positive impact on both the patient and their family.

#### Course Goals and Objectives

Upon completion of this course, the participant will be able to:

1. identify the possible etiological factors that cause Parkinson's Syndrome.
2. recognize the epidemiology and risk factors of Parkinson's Syndrome
3. identify the clinical signs of Parkinson's Syndrome
4. differentiate the pharmacological treatment options available to treat Parkinson's syndrome.
5. identify the rehabilitation needs and therapeutic interventions of individuals with Parkinson's Syndrome
6. identify and utilize biometric scales to quantify the health status of individuals with Parkinson's Syndrome
7. recognize the many secondary conditions associated with Parkinson's syndrome.
8. recognize and understand the challenges faced by the caregiver.

**Course Provider** – Innovative Educational Services

**Provider Contact Information** – [information@cheapceus.com](mailto:information@cheapceus.com)

**Course Instructor** – Michael Niss, DPT

**Financial/Non-financial Disclosure** - Neither the Provider nor the instructor have any financial or non-financial conflict of interest related to the presentation of this CE program.

**Target Audience** – Occupational Therapists, Occupational Therapy Assistants

**OT Scope of Practice** – OT Service delivery (evaluation, intervention, outcomes); Foundational knowledge (Human body diagnoses and conditions)

**Course Educational Level** – This course is applicable for introductory learners.

**Course Prerequisites** – None

**Method of Instruction** – Distance Learning – Independent; Asynchronous online text-based home study

**Location** - Cheapceus.com

**Date** – Continuously available on-demand

**Course Completion Requirements / Criteria for Issuance of CE Credits** – Completion of instructional materials and a score of 70% or greater on the course post-test.

**Continuing Education Credits** – Four (4) contact hours / .4 AOTA CEUs / NBCOT 5 PDU's

**Course Fee** - \$39.95

**Registration Information** – No pre-registration required; available on-demand at Cheapceus.com

**Special Needs Requests** – Email: [information@cheapceus.com](mailto:information@cheapceus.com) or phone: 954-663-4101

**Cancellation by the Learner** – Learners may cancel their participation at any time and receive a full refund of all paid fees.

**Cancellation by the Provider** – Registrants for the cancelled activity will be contacted by an IES staff member via phone call or email within 12 hours of the cancellation decision. If the decision to cancel is made less than 14 hours prior to the event, each participant will be contacted no later than 2 hours prior to the scheduled start time of the activity. Registrants will receive a full refund if an activity is cancelled by the Provider. The refund will be processed back to the Registrant within 12 hours of the cancellation.

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**Refund Policy** - Unrestricted 100% refund upon request. The request for a refund by the learner shall be honored in full without penalty or other consideration of any kind. The request for a refund may be made by the learner at any time without limitations before, during, or after course participation.



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# PARKINSON'S SYNDROME

## Parkinson's Syndrome

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## Overview

Parkinson's Syndrome (PS), also called paralysis agitans, is a chronic movement disorder with distinct symptoms. The term Parkinsonism refers to the group of symptoms that can occur with this movement disorder. In the past, Parkinson's Disease was the term used for this movement disorder when the cause of the biochemical disorder in the part of the brain leading to the symptoms was unknown (as opposed to the movement disorder being due to a brain injury or as a side effect of drugs or poisons, etc.). Because of the bad connotation of the word "disease," and for other reasons, the phrase Parkinson's Syndrome (PS) is now being used by some groups instead of the phrase Parkinson's Disease. When Parkinsonism symptoms occur as a secondary symptom of some brain disorder other than Parkinson's Disease (such as Alzheimer's Disease), this is called a Parkinsonism-plus syndrome or Parkinson's Plus syndrome.

PS is characterized by three main symptoms — tremor (shaking), rigidity (muscle stiffness), and bradykinesia (difficulty in starting movement and slowing down of voluntary movement). Intelligence is not affected. It does not cause paralysis. PS results from a loss of a specific type of brain cells (nigrostriatal cells) deep within a midbrain region known as the "black substance" (Substantia Nigra). The cells in this area of the brain control the movement of our body parts by making neurotransmitters called dopamine that are sent to other brain cells to tell them how to control our movements. When 80% or more of the cells in the SN die, no matter what mechanism destroyed them, Parkinsonism is the result. When the cause of Parkinsonism cannot be determined, it is called idiopathic Parkinson's Disease (PD), and such people form the largest subgroup of people with Parkinson's Syndrome (PS).

Parkinson's Plus Syndrome (PPS) is a mimic of PS, and can take various forms. There may be a disproportionate involvement of gait with few limb signs. This is sometimes called "lower half Parkinsonism," and may be due to small strokes in the brain. While 10 to 20% of people with Parkinson's Disease either lack a tremor or have more postural than rest tremor, this is even more common in other brain disorders mimicking Parkinson's Disease such as PPS. And there are other clues in such things as balance changes, eye movement changes, functioning of the autonomic nervous system, and the early onset and progression of a decline in cognitive functioning that may suggest that a person has a Parkinson's Plus Syndrome and not Parkinson's Syndrome. The distinction is important because those with PPS respond only minimally and sometimes only briefly to the usual medications used to treat PS.

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Parkinson's disease occurs when neurons, in the substantia nigra die or become impaired. Normally, these neurons produce dopamine. Dopamine is a chemical messenger responsible for transmitting signals between the substantia nigra and the next "relay station" of the brain, the corpus striatum, to produce smooth, purposeful movement. Loss of dopamine results in abnormal nerve firing patterns within the brain that cause impaired movement. Studies have shown that most Parkinson's patients have lost 60 to 80 percent or more of the dopamine-producing cells in the substantia nigra by the time symptoms appear. Recent studies have shown that people with PS also have loss of the nerve endings that produce the neurotransmitter norepinephrine. Norepinephrine, which is closely related to dopamine, is the main chemical messenger of the sympathetic nervous system, the part of the nervous system that controls many automatic functions of the body, such as pulse and blood pressure. The loss of norepinephrine might help explain several of the non-motor features seen in PS, including fatigue and abnormalities of blood pressure regulation.

Many brain cells of people with PS contain Lewy bodies – unusual deposits or clumps of the protein alpha-synuclein, along with other proteins. Researchers do not yet know why Lewy bodies form or what role they play in development of the disease. The clumps may prevent the cell from functioning normally, or they may actually be helpful, perhaps by keeping harmful proteins "locked up" so that the cells can function.

Scientists have identified several genetic mutations associated with PD, and many more genes have been tentatively linked to the disorder. Studying the genes responsible for inherited cases of Ps can help researchers understand both inherited and sporadic cases. The same genes and proteins that are altered in inherited cases may also be altered in sporadic cases by environmental toxins or other factors. Researchers also hope that discovering genes will help identify new ways of treating PS.

### **Genetic Causes**

Several genes have now been definitively linked to PS. The first to be identified was alpha-synuclein. In the 1990s, researchers studied the genetic profiles of a large Italian family and three Greek families with familial PS and found that their disease was related to a mutation in this gene. They found a second alpha-synuclein mutation in a German family with PS. These findings prompted studies of the role of alpha-synuclein in PS, which led to the discovery that Lewy bodies from people with the sporadic form of PS contained clumps of alpha-synuclein protein. This discovery revealed a potential link between hereditary and sporadic forms of the disease.

Researchers studying inherited PS discovered that the disease in one large family was caused by a triplication of the normal alpha-synuclein gene on one copy of chromosome 4. This triplication caused people in the affected family to

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produce too much of the normal alpha-synuclein. This study showed that an excess of the normal form of the protein could result in PS, just as the abnormal form does.

Other genes linked to PS include parkin, DJ-1, PINK1, and LRRK2. Parkin, DJ-1, and PINK-1 cause rare, early-onset forms of PS. The parkin gene is translated into a protein that normally helps cells break down and recycle proteins. DJ-1 normally helps regulate gene activity and protect cells from oxidative stress. PINK1 codes for a protein active in mitochondria. Mutations in this gene appear to increase susceptibility to cellular stress.

LRRK2, which is translated into a protein called dardarin, was originally identified in several English and Basque families and causes a late-onset form of PS. Subsequent studies have identified this gene in other families with PS as well as in a small percentage of people with apparently sporadic PS.

Researchers are continuing to investigate the normal functions and interactions of these genes in order to find clues about how PS develops. They also have identified a number of other genes and chromosome regions that may play a role in PS, but the nature of these links is not yet clear.

### **Environmental Toxins**

Although the importance of genetics in PS is increasingly recognized, most researchers believe environmental exposures increase a person's risk of developing the disease. Even in familial cases, exposure to toxins or other environmental factors may influence when symptoms of the disease appear or how the disease progresses. There are a number of toxins, such as 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine, or MPTP (found in some kinds of synthetic heroin), that can cause parkinsonian symptoms in humans. Other, still-unidentified environmental factors also may cause PS in genetically susceptible individuals.

### **Viral**

Viruses are another possible environmental trigger for PS. People who developed encephalopathy after the 1918 influenza epidemic were later stricken with severe, progressive Parkinson's-like symptoms. A group of Taiwanese women developed similar symptoms after contracting herpes virus infections. In these women, the symptoms, which later disappeared, were linked to a temporary inflammation of the substantia nigra.

### **Oxidative Stress**

Several lines of research suggest that mitochondria may play a role in the development of PS. Mitochondria are the energy-producing components of the

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cell and are major sources of free radicals — molecules that damage membranes, proteins, DNA, and other parts of the cell. This damage is often referred to as oxidative stress. Oxidative stress-related changes, including free radical damage to DNA, proteins, and fats, have been detected in brains of PS patients.

Other research suggests that the cell's protein disposal system may fail in people with PS, causing proteins to build up to harmful levels and trigger cell death. Additional studies have found evidence that clumps of protein that develop inside brain cells of people with PS may contribute to the death of neurons, and that inflammation or overstimulation of cells (because of toxins or other factors) may play a role in the disease. However, the precise role of the protein deposits remains unknown. Some researchers even speculate that the protein buildup is part of an unsuccessful attempt to protect the cell. While mitochondrial dysfunction, oxidative stress, inflammation, and many other cellular processes may contribute to PS, the actual cause of the dopamine cell death is still undetermined.

### **Epidemiology**

About 50,000 Americans are diagnosed with PS each year, but getting an accurate count of the number of cases may be impossible because many people in the early stages of the disease assume their symptoms are the result of normal aging and do not seek help from a physician. Also, diagnosis is sometimes difficult and uncertain because other conditions may produce symptoms of PS and there is no definitive test for the disease. People with PS may sometimes be told by their doctors that they have other disorders, and people with PS-like diseases may be incorrectly diagnosed as having PS.

The frequency of occurrence of Parkinson's Syndrome rises from 1 per 1,000 in the general population to 1 per 100 in those between the ages of 55 and 65. More people have PS than other movement disorders combined (including multiple sclerosis).

PS strikes about 50 percent more men than women, but the reasons for this discrepancy are unclear. While it occurs in people throughout the world, a number of studies have found a higher incidence in developed countries, possibly because of increased exposure to pesticides or other toxins in those countries. Other studies have found an increased risk in people who live in rural areas and in those who work in certain professions, although the studies to date are not conclusive and the reasons for the apparent risks are not clear.

One clear risk factor for PS is age. The average age of onset is 60 years, and the incidence rises significantly with increasing age. However, about 5 to 10 percent of people with PS have "early-onset" disease that begins before the age

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of 50. Early-onset forms of the disease are often inherited, though not always, and some have been linked to specific gene mutations. People with one or more close relatives who have PS have an increased risk of developing the disease themselves, but the total risk is still just 2 to 5 percent unless the family has a known gene mutation for the disease. An estimated 15 to 25 percent of people with PS have a known relative with the disease.

### **Symptomology**

Early symptoms of PS are subtle and occur gradually. Affected people may feel mild tremors or have difficulty getting out of a chair. They may notice that they speak too softly or that their handwriting is slow and looks cramped or small. They may lose track of a word or thought, or they may feel tired, irritable, or depressed for no apparent reason. This very early period may last a long time before the more classic and obvious symptoms appear.

Friends or family members may be the first to notice changes in someone with early PS. They may see that the person's face lacks expression and animation (known as "masked face") or that the person does not move an arm or leg normally. They also may notice that the person seems stiff, unsteady, or unusually slow.

As the disease progresses, the shaking or tremor that affects the majority of Parkinson's patients may begin to interfere with daily activities. Patients may not be able to hold utensils steady or they may find that the shaking makes reading a newspaper difficult. Tremor is usually the symptom that causes people to seek medical help.

People with PS often develop a so-called parkinsonian gait that includes a tendency to lean forward, small quick steps as if hurrying forward (called festination), and reduced swinging of the arms. They also may have trouble initiating movement (start hesitation), and they may stop suddenly as they walk (freezing).

PS does not affect everyone the same way, and the rate of progression differs among patients. Tremor is the major symptom for some patients, while for others; tremor is nonexistent or very minor.

PS symptoms often begin on one side of the body. However, as it progresses, the disease eventually affects both sides. Even after the disease involves both sides of the body, the symptoms are often less severe on one side than on the other. The three primary symptoms of PS are bradykinesia, tremor, and rigidity.

## **Bradykinesia**

It is characterized by a delay in starting all movements, slowness and poverty of all movements, and the arrest of ongoing movements. PS often produces acute difficulty or slowness in movements such as rising from the table or the bed or toilet, reaching for objects, turning, or getting out of bed. It is also seen as difficulty swallowing, constipation, decreased facial expression ("masked facial features"), and reduced blink frequency. Loss of spontaneous movement occurs as a diminished ability to carry out movements such as arm-swinging while walking, reduced emotional expressiveness, and may create a somewhat stony stare (with infrequent blinking). As PS progresses, difficulties with balance are frequent, as are falls.

## **Tremor**

It is absent in as many as 25% of people with PS. Not necessarily the first, but often the clearest sign of PS, and one of its more annoying symptoms. At first a mere intermittent "tapping" of the hand (when hands are resting or supported on the lap, better known as a resting tremor), the shaking usually subsides when the hands are moved or during sleep, but increases with stress. It may also appear in the feet. Later, often years later, the tremor may spread to the head or neck or face or lips or jaw or legs. In some people, the tremor may increase when the hands are stretched out in front, and this is called a sustentation or postural tremor. If the tremor increases when the hands are moving, it is called an action tremor. Sustentation and action tremors usually do not respond to anti-parkinsonian drugs.

## **Rigidity**

Rigidity, or a resistance to movement, affects most people with PS. A major principle of body movement is that all muscles have an opposing muscle. Movement is possible not just because one muscle becomes more active, but because the opposing muscle relaxes. In PS, rigidity comes about when, in response to signals from the brain, the delicate balance of opposing muscles is disturbed. The muscles remain constantly tensed and contracted so that the person aches or feels stiff or weak. The rigidity becomes obvious when another person tries to move the patient's arm, which will move only in ratchet-like or short, jerky movements known as "cogwheel" rigidity.

## **Other Symptoms**

### **Gait disturbance**

The gait disturbance is often referred to as "festination" which means the inability to walk slowly and a tendency to walk with small quick shuffling steps. The person may develop a forward tilt or a tilt to one side, which further impairs balance. There is difficulty in turning, and there may be abrupt freezing spells.

### **Psychiatric disorders**

Depression (occurs in as many as 50%), mood swings, and insomnia may occur with PS and may require treatment with medications. Depression may be associated with anxiety and agitation, a so-called "agitated depression."

### **Sleep disturbances**

Can include an inability to fall asleep or an inability to stay asleep, with frequent nighttime awakenings. Some people experience a reversal of their sleep patterns, sleeping during the day and being awake at night. People may have vivid dreams, and rarely, nightmares. Bed partners often report that people speak in their sleep and have jerking, involuntary movements of their limbs (myoclonus) during sleep. For some people, difficulty in sleeping is related to their depression. Some sleeping difficulties, especially vivid dreaming and myoclonus, are related to L-dopa. Readjustment of the dose of L-dopa, and eliminating the evening dose (if possible) may improve the person's sleep. On the other hand, some people require L-dopa to sleep because a lack of medication makes them so rigid that they cannot turn in bed. Standard sleeping medications are occasionally helpful, but must be used with caution—particularly for depressed people.

### **Intellectual impairment or dementia**

This occasionally occurs in some people, usually those who are elderly and in the late stages of PS. It can include memory failure, problems with recognition, reduced problem solving ability, problems with calculations, visuospatial abnormalities, and slow thought. Some develop dementia in later stages with confusion and disorientation. Anti-Parkinsonian drugs such as L-dopa, the anticholinergic drugs, amantadine, and the dopamine agonists may cause delusions, confusion, paranoia, or hallucinations on their own, but when the drug is stopped, the symptoms clear (it may take several days). The appearance of drug-induced mental changes in a person who is not senile does not mean that the person will become senile.

### **Forced eyelid closure (blepharospasm)**

The inability to open the eyelids, or once opened, to keep them open. Rarely, blepharospasm results in the inability to open the eyes at all. Such people cannot read, watch television, or carry on many other daily activities. Antiparkinsonian drugs alleviate the blepharospasm for some people, though they may worsen this condition for others. To alleviate this, surgical treatment of the eyelid muscles may be necessary, or it may be possible to inject a small amount of a paralytic drug into the muscles that close the eyes.

### **Drooling (sialorrhea)**

This usually results from the person's inability to swallow saliva leading to its accumulation in the throat. This may be evident only at night when people are reclining and lose gravity's assistance in swallowing. In a few people, drooling

may result from an overproduction of saliva. Drooling usually improves with the use of medications, especially the anticholinergic drugs that decrease the production of saliva.

### **Speech problems**

Usually, a mild impairment that consists of a change in voice volume, phonation, or articulation. Generally, the volume change is the first such symptom and the person speaks "more softly." The person's voice may be loud at the beginning of a sentence and then fade towards the end. The voice may also become monotonous, lacking variation and feeling. In others, the voice may sound breathy, tremulous, high-pitched, hoarse, or strident. Words may become slurred and indistinct, word endings may be omitted, final consonant sounds, such as the "k" in look may be unclear, syllables and words may be crowded and run together, and words may be accelerated towards the end of a sentence. Occasionally, speech difficulties arise early in the disease, and initially these problems may be evident only when the person uses the telephone (which filters out some of the normal frequencies). Specific measures such as speech therapy, amplification devices, and medications may be helpful.

### **Difficulty in swallowing (dysphagia)**

Most often occurs late in PS, and is rarely one of the early symptoms. Sometimes people are unable to swallow pills, and if not reported by the person, this will only become apparent after other PS symptoms worsen. People may experience this difficulty with both solids and liquids, and it may be due to three problems:

1. In some people, the swallowing difficulty arises from an inability to force the food down the throat and an inability of the voluntary muscles of the throat and esophagus to contract. This results in pooling of food in the throat. These people complain of food getting stuck in their throat. To alleviate the problem, people should place small portions of food in their mouth and chew and swallow slowly and carefully. They should always completely swallow one morsel before putting other food into the mouth.
2. In some PS people, pooling of food in the throat may cause food to go into the lungs (aspiration). Frequent episodes of this may appear as a cough or pneumonia. This usually only occurs in people with advanced disease.
3. Other swallowing difficulties may result from the failure of the valve mechanism that allows food to pass from the gullet into the stomach to work properly. This will allow acid and chemicals from the stomach to come back up into the esophagus and burn it (and the lungs if there is aspiration also). The symptoms include indigestion, heartburn, burning

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in the throat or chest, and may occasionally resemble the pain of a heart attack. These symptoms should be evaluated by a doctor.

### **Sexual Problems**

A decrease in the desire for sex may result from the non-specific effects of a chronic illness, fear of being unable to perform satisfactorily, depression, and medications. Men who note the inability to achieve or maintain an erection need to be evaluated for causes other than PS. People who seem to have increased sexual function after being started on L-dopa do so because of their general improvement, not because of a direct effect of L-dopa.

### **Breathing Problems (Dyspnea)**

Shortness of breath after minimal physical activity should be evaluated to rule out heart or lung disease. If the results are negative, shortness of breath may be due to rigidity, or bradykinesia of the chest wall muscles which prevents the lungs from expanding, and increasing the anti-parkinsonian medications may help. Some people on L-dopa may experience shortness of breath and abnormal grunting respirations because L-dopa may cause uncoordinated involuntary movements of the diaphragm, chest wall muscles, and upper throat muscles. This usually improves if the L-dopa is decreased.

### **Weight Loss**

Weight loss up to 30 pounds is not uncommon. It may be related to difficulty with swallowing, the increased energy used up by the tremors, or decreased appetite.

### **Urinary Dysfunction**

This is a problem, which may consist of urgency (a strong desire to void even with little urine in the bladder), frequency (passing urine frequently, and if this happens at night it is called nocturia), hesitancy in starting to void, difficulty in completing voiding, and incomplete voiding with dribbling. Rarely a person may be unable to void. PS may cause difficult voiding because the muscles of the bladder become rigid and bradykinetic, thus decreasing the ability of the bladder to contract and expel urine. Some antiparkinsonian drugs, such as the anticholinergics and Symmetrel, may increase the voiding difficulty. PS people who experience difficulty in voiding must be checked for the other diseases and problems that can mimic these symptoms (prostate problems in men, vaginal laxness in women, diabetes, infection, etc.)

### **Peripheral Edema**

Swelling in the extremities may occur toward the end of the day after a person has been standing and disappears when the person lies down. When this occurs, the person has to be evaluated for heart failure, but the swelling of the feet usually results from gravity holding fluid in the feet, and the inability of the rigid leg muscles to massage fluid from the feet back to the heart. Drugs such as Symmetrel and some of the dopamine agonists may result in swelling of the feet. If the swelling is mild, it may not require any treatment, but if it becomes more

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pronounced, a number of measures may be used, including elevating the feet, elastic support stockings, and less often, water pills.

### **Dizziness Upon Standing (Postural or Orthostatic Hypotension)**

This is the result of a drop in blood pressure upon standing. Some antiparkinsonian drugs (L-dopa and the dopamine agonists) may cause dizziness on standing, but it usually disappears once the body has adjusted to the drugs. If it doesn't, a decrease in the dose of the drugs may be needed, water pills must be stopped, and elastic stockings may be needed. The person may also have to take drugs (Florinef, Indocid) or table salt, which cause the body to retain fluid. Dizziness may also be caused by other drugs which some people take for different medical conditions (i.e., high blood pressure, heart failure, and depression), and can also occur due to dehydration, malnutrition, diabetes, or other illnesses.

### **Stooped Posture**

The stooped posture arises, in part, from rigidity of the neck and back muscles. It is helpful for such people to participate in exercises that are designed to lessen this rigidity of the back muscles. Occasionally, the stooped posture may respond to treatment with L-dopa or one of the dopamine agonists.

### **Other**

Diminutive handwriting (micrographia), extreme fatigue, increased body secretions (saliva, perspiration, and skin oil), constipation, muscle cramps, and a susceptibility to bladder and chest infections (partly due to immobility). Some people will have vague strange sensations and pain in the shoulders, trunk, low back, and lower limbs.

## **Diagnosis**

An accurate diagnosis depends on a careful history (including a history of any drug or medication use), and a physical exam. There are currently no blood or laboratory tests that have been proven to help in diagnosing sporadic PS. Therefore, the diagnosis is based on medical history and a neurological examination. The disease can be difficult to diagnose accurately. Early signs and symptoms of PS may sometimes be dismissed as the effects of normal aging. The clinician may need to observe the person for some time until it is apparent that the symptoms are consistently present. Since many other diseases have similar features but require different treatments, making a precise diagnosis as soon as possible is essential so that patients can receive the proper treatment.

The abundance of guidelines for PS diagnosis is reflective of the difficulty in diagnosing this condition. One relatively straightforward list of research criteria for probable PD includes:

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1. Evidence of disease progression.
2. Presence of at least two of the three cardinal features of parkinsonism (tremor, rigidity, bradykinesia)
3. Presence of at least two of the following:
  - a. Marked response to L-dopa (functional improvement or dyskinesia)
  - b. Asymmetry of signs
  - c. Asymmetry at onset
4. Absence of clinical features of alternative diagnosis
5. Absence of etiology known to cause similar features

Other diagnostic guidelines incorporate requirements pertaining to disease duration, and more specifics regarding tremor and response to dopaminergic agonists.

A more recent variation of clinical guidelines for PS diagnosis describes an adult-onset, slowly progressive motor disorder combining two or more of: rest tremor, bradykinesia, limb rigidity, and gait instability (late), with dramatic and sustained response to L-dopa. Accepted associated phenomena include depression (early or late), cognitive decline (late), and limited autonomic involvement, such as constipation. Some proposed diagnostic criteria for PS categorize patients as having definite, probable, or possible PS, based on the number of criteria they meet.

The pathologic hallmark of PS is substantia nigra depigmentation and the presence of Lewy bodies, which are neuronal eosinophilic cytoplasmic inclusions. They are not, however, specific for PS, and may be seen in small numbers in other neurodegenerative diseases.

While autopsy provides the pathological gold standard, no clinical gold standard diagnostic test for PD has been identified. Comparisons of clinical and pathological diagnoses have shown that up to 25 percent of patients with clinical diagnoses of PD are found to have different pathological diagnoses at autopsy.

Structural imaging modalities, such as computerized tomography (CT) and magnetic resonance imaging (MRI) have a limited role in diagnosing PS. Increased iron concentration in the substantia nigra causes decreased signal intensity on T2 weighted images, but these changes are not sufficient to reliably distinguish PS patients from healthy controls. These technologies are more useful for ruling out other conditions than for diagnosing PS.

Olfactory deficits occur early in PS, and do not improve with L-dopa treatment. The University of Pennsylvania Smell Identification Test (UPSIT) is a multiple choice "scratch and sniff" test that is used to evaluate olfactory function.

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Olfaction is impaired in other neurodegenerative diseases, such as Huntington's Disease and Alzheimer's dementia

A myriad of other tests have been proposed to diagnose and evaluate patients with PS. Depletion of cerebrospinal fluid (CSF) homovanillic acid (HVA) levels indicate dopamine deficiency, but this test has not been shown to reliably discriminate healthy controls from PS patients. Studies of handwriting, tremor analysis, personality, reaction times, and movement velocities have shown differences between patients with PS and normal controls; however, the overlap between the two groups does not enable these tests to reliably diagnose PS in individual patients.

### **Other Diseases Resembling Parkinson's**

A number of disorders can cause symptoms similar to those of PS. People with symptoms that resemble PS but that result from other causes are sometimes said to have parkinsonism. "Red flags" that suggest a diagnosis other than PS include early dementia or apraxia, early instability and falls, prominent autonomic impairment, oculomotor disturbances, and cerebellar signs.

Some of the disorders that resemble Parkinson's Syndrome are listed below.

#### **Postencephalitic Parkinsonism**

Just after the first World War, a viral disease, encephalitis lethargica, attacked almost 5 million people throughout the world, and then suddenly disappeared in the 1920s. Known as sleeping sickness in the United States, this disease killed one third of its victims and led to post-encephalitic parkinsonism in many others. This resulted in a particularly severe form of movement disorder that appeared sometimes years after the initial illness. (In 1973, neurologist Oliver Sacks published *Awakenings*, an account of his work in the late 1960s with surviving post-encephalitic patients in a New York hospital. Using the then-experimental drug levodopa, Dr. Sacks was able to temporarily "awaken" these patients from their statue-like state). In rare cases, other viral infections, including western equine encephalomyelitis, eastern equine encephalomyelitis, and Japanese B encephalitis, have caused parkinsonian symptoms.

#### **Drug-induced Parkinsonism**

A reversible form of parkinsonism sometimes results from use of certain drugs, such as chlorpromazine and haloperidol, which are prescribed for patients with psychiatric disorders. Some drugs used for stomach disorders (metoclopramide), hypertension (reserpine), and epilepsy (valproate) may also produce parkinsonian symptoms. Stopping the medication or lowering the dosage of these medications usually causes the symptoms to go away.

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### **Toxin-induced Parkinsonism**

Some toxins — such as manganese dust, carbon disulfide, and carbon monoxide — can cause parkinsonism. The chemical MPTP also causes a permanent form of parkinsonism that closely resembles PS. Investigators discovered this reaction in the 1980s when heroin addicts in California who had taken an illicit street drug contaminated with MPTP began to develop severe parkinsonism. This discovery, which showed that a toxic substance could damage the brain and produce parkinsonian symptoms, caused a dramatic breakthrough in Parkinson's research: for the first time, scientists were able to simulate PS in animals and conduct studies to increase understanding of the disease.

### **Arteriosclerotic Parkinsonism**

Sometimes known as pseudoparkinsonism, vascular parkinsonism, or atherosclerotic parkinsonism, arteriosclerotic parkinsonism involves damage to the brain due to multiple small strokes. Tremor is rare in this type of parkinsonism, while dementia — the loss of mental skills and abilities — is common. Antiparkinsonian drugs are of little help to patients with this form of parkinsonism.

### **Post-traumatic Parkinsonism**

Also known as post-traumatic encephalopathy or "punch-drunk syndrome," parkinsonian symptoms can sometimes develop after a severe head injury or frequent head trauma that results from boxing or other activities. This type of trauma also can cause a form of dementia called dementia pugilistica.

### **Essential tremor**

Essential tremor, sometimes called benign essential tremor or familial tremor, is a common condition that tends to run in families and progresses slowly over time. The tremor is usually equal in both hands and increases when the hands are moving. The tremor may involve the head but usually spares the legs. Patients with essential tremor have no other parkinsonian features. Essential tremor is not the same as PS, and usually does not lead to it, although in some cases the two conditions may overlap in one person. Essential tremor does not respond to levodopa or most other PS drugs, but it can be treated with other medications.

### **Normal Pressure Hydrocephalus**

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Normal pressure hydrocephalus (NPH) is an abnormal increase of cerebrospinal fluid (CSF) in the brain's ventricles, or cavities. It occurs if the normal flow of CSF throughout the brain and spinal cord is blocked in some way. This causes the ventricles to enlarge, putting pressure on the brain. Symptoms include problems with walking, impaired bladder control leading to urinary frequency or incontinence, and progressive mental impairment and dementia. The person also experiences a general slowing of movements or may complain that his or her feet feel "stuck." These symptoms may sometimes be mistaken for PS. Brain scans, intracranial pressure monitoring, and other tests can help to distinguish NPH from PS and other disorders. NPH can sometimes be treated by surgically implanting a CSF shunt that drains excess cerebrospinal fluid into the abdomen, where it is absorbed.

### **Progressive Supranuclear Palsy**

Progressive supranuclear palsy (PSP), sometimes called Steele-Richardson-Olszewski syndrome, is a rare, progressive brain disorder that causes problems with control of gait and balance. Patients with PSP generally present with postural instability, often coming to medical attention due to frequent falls. The hallmark of PSP is vertical gaze paralysis. Other symptoms include other visual disturbances, dysarthria, mental changes, speech difficulties, bradykinesia, nuchal dystonia, rigidity, and postural tremors, although resting tremors are uncommon. PSP patients often show alterations of mood and behavior, including depression and apathy as well as mild dementia. The symptoms of PSP are caused by a gradual deterioration of brain cells in the brainstem. It is often misdiagnosed because some of its symptoms are very much like those of PD, Alzheimer's disease, and other brain disorders. PSP symptoms usually do not respond to medication. PSP is progressive, and usually leads to death within five to seven years after diagnosis.

### **Corticobasal Degeneration**

Corticobasal degeneration results from atrophy of multiple areas of the brain, including the cerebral cortex and the basal ganglia. Initial symptoms may first appear on one side of the body, but eventually affect both sides. Symptoms are similar to those found in PS, including rigidity, impaired balance and coordination, and dystonia. Other symptoms may include cognitive and visual-spatial impairments, apraxia (loss of the ability to make familiar, purposeful movements), hesitant and halting speech, myoclonus (muscular jerks), and dysphagia (difficulty swallowing). Unlike PS, corticobasal degeneration usually does not respond to medication.

### **Multiple System Atrophy**

Multiple system atrophy (MSA) refers to a set of slowly progressive disorders that affect the central and autonomic nervous systems. MSA may have symptoms

## PARKINSON'S SYNDROME

that resemble PS. It also may take a form that primarily produces poor coordination and slurred speech, or it may have a mixture of these symptoms. Other symptoms may include breathing and swallowing difficulties, male impotence, constipation, and urinary difficulties. The disorder previously called Shy-Drager syndrome refers to MSA with prominent orthostatic hypotension — a fall in blood pressure every time the person stands up. MSA with parkinsonian symptoms is sometimes referred to as striatonigral degeneration, while MSA with poor coordination and slurred speech is sometimes called olivopontocerebellar atrophy.

### **Dementia with Lewy Bodies**

Dementia with Lewy bodies is a neurodegenerative disorder associated with abnormal protein deposits (Lewy bodies) found in certain areas of the brain. Symptoms can range from traditional parkinsonian symptoms, such as bradykinesia, rigidity, tremor, and shuffling gait, to symptoms similar to those of Alzheimer's disease. These symptoms may fluctuate, or wax and wane dramatically. Visual hallucinations may be one of the first symptoms, and patients may suffer from other psychiatric disturbances such as delusions and depression. Cognitive problems also occur early in the course of the disease. Levodopa and other antiparkinsonian medications can help with the motor symptoms of dementia with Lewy bodies, but they may make hallucinations and delusions worse.

### **Parkinsonism Accompanying Other Conditions**

Parkinsonian symptoms may also appear in patients with other, clearly distinct neurological disorders such as Wilson's disease, Huntington's disease, Alzheimer's disease, spinocerebellar ataxias, and Creutzfeldt-Jakob disease. Each of these disorders has specific features that help to distinguish them from PS.

## **Assessment**

One of the first questions many PS people and their families ask about is the severity of the disease. Since people are frequently at their “best” in their doctor's office, decisions regarding treatment are not based solely on office observation. Scales rating the severity of PS are based on an evaluation of the symptoms. There are several different scales, and they differ in which symptoms are evaluated, and the value assigned to each symptom.

### **Scales**

The most common scales used to assess PD severity are the Hoehn & Yahr Disability Scale (H&Y), Schwab & England (S&E) Activities of Daily Living (ADL)

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scale, Unified Parkinson Disease Rating Scale (UPDRS), Webster scale, Columbia University Rating Scale (CURS), and Northwestern University Disability Scale (NUDS).

Scales to measure the status of a person with PS have several purposes. Some are used to follow a person's progress, such as the Hoehn and Yahr Staging Scale, Schwab and England Activities of Daily Living Scale. Others such as the Unified Parkinson Disease Rating Scale are often used for research studies to objectively determine the effectiveness of a new treatment.

### **The Hoehn and Yahr Scale**

The H&Y scale divides patients into stages, based on their levels of clinical disability.

Stage 0 patients have no signs of disease. Stage I patients have unilateral involvement, with minimal or no functional impairment. Stage II patients have bilateral or midline involvement, without balance impairment. Stage III patients have impaired equilibrium, unsteadiness, and significant slowing of body movements. Stage IV patients have severe symptoms, are still able to walk and stand unassisted, but are extremely incapacitated and unable to live alone. Stage V patients are confined to bed or wheelchair, and require constant nursing care.

The Hoehn and Yahr Scale:

**Stage zero** - no visible disease.

**Stage one** - mild unilateral tremor and some rigidity with minimal or subtle bradykinesia.

**Stage two** - moderate bilateral tremor and rigidity, moderate bradykinesia, and mild difficulties with daily activities.

**Stage three** - more severe bilateral tremor and rigidity, more severe bradykinesia, gait disturbances and instability, and impaired postural reflexes (dizziness when getting up from lying or sitting).

**Stage four** - severe disability with risk of falling spontaneously. The person may be mobile with a walking aid. The person may experience multiple freezing episodes, and may have periods of complete immobility, and a mild dementia.

**Stage five** - means complete immobility with an inability to walk, and there is often a moderate dementia and an inability to function independently.

Stages zero to two are mild disease, stage three is moderate disease, and stages four and five are marked or advanced disease. There are grey areas between the successive stages. Another way of staging the disease is to divide

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it into an early (or honeymoon) stage, a “predictable wearing off stage” about 3 to 5 yrs. into the disease, and an “unpredictable motor fluctuations stage,” which may occur 5 to 10 yrs. into the disease.

Complementing these rating scales are functional disability scales that rate the person's ability to perform activities of daily living. These scales assign a weighted value to a particular activity, such as walking, eating, dressing, hygiene, and so on, and then grade the person's degree of disability. For people who experience daily oscillation changes in performance, clinicians assess the daily number of hours people spend in their “on” (good) periods and the number of hours they spend in their “off” (bad) periods. Such a determination is made by instructing people with these oscillations (or their care-givers) to keep a daily diary of both the number of hours they are “on” and the number “off”. Through such a diary, the clinician gains a better understanding of the person's overall performance. Since reports on the degree of the person's disability may differ between the person and the family, both may need to keep a diary for comparison purposes.

### **Schwab and England Activities of Daily Living**

The Schwab & England ADL scale has ratings from 0 to 100 percent, where 0 is bedridden with no swallowing, bladder, or bowel function, and 100 percent is completely independent.

Rating can be assigned by rater or by patient.

- 100% - Completely independent. Able to do all chores w/o slowness, difficulty, or impairment.
- 90% - Completely independent. Able to do all chores with some slowness, difficulty, or impairment. May take twice as long.
- 80% - Independent in most chores. Takes twice as long. Conscious of difficulty and slowing
- 70% - Not completely independent. More difficulty with chores. 3 to 4X along on chores for some. May take large part of day for chores.
- 60% - Some dependency. Can do most chores, but very slowly and with much effort. Errors, some impossible
- 50% - More dependent. Help with 1/2 of chores. Difficulty with everything
- 40% - Very dependent. Can assist with all chores but few alone
- 30% - With effort, now and then does a few chores alone or begins alone. Much help needed
- 20% - Nothing alone. Can do some slight help with some chores. Severe invalid
- 10% - Totally dependent, helpless
- 0% - Vegetative functions such as swallowing, bladder and bowel function are not functioning. Bedridden.

### **Unified Parkinson Disease Rating Scale (UPDRS)**

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## PARKINSON'S SYNDROME

In the UPDRS, points are assigned for a comprehensive list of PD symptoms. Patients may receive a total of 199 points, with 0 representing no disability, and 199 representing total disability. The total score is composed of four major subscales:

- Mentation, Behavior, and Mood (range 0-16),
- ADL (range 0-104),
- Motor Exam (range 0-56), and
- Complications of therapy over the past week (range 0-23).

Each of these subscales is broken down into further subscales, which range from 0 (normal) to 4 (maximum severity). Each UPDRS score may be reported in the "off" and "on" state, which refer to presence or absence of L-dopa effectiveness. Practically-defined "off" scores are measured approximately 12 hours after the last dose of L-dopa, although in actual clinical practice, "off" scores often indicate periods when the patients feel their medication is not working. "On" scores are measured shortly after a dose, or when patients feel their medication is working. The UPDRS scales are validated tools that are useful in following the progression of disease and response to interventions.

### **Mentation, Behavior, Mood**

#### **Intellectual Impairment**

- |   |   |
|---|---|
| 0 | none  |
| 1 | mild (consistent forgetfulness with partial recollection of events with no other difficulties)  |
| 2 | moderate memory loss with disorientation and moderate difficulty handling complex problems      |
| 3 | severe memory loss with disorientation to time and often place, severe impairment with problems |
| 4 | severe memory loss with orientation only to person, unable to make judgments or solve problems  |

#### **Thought Disorder**

- |   |  |
|---|--|
| 0 | none   |
| 1 | vivid dreaming   |
| 2 | "benign" hallucinations with insight retained  |
| 3 | occasional to frequent hallucination or delusions without insight, could interfere with daily activities |
| 4 | persistent hallucination, delusions, or florid psychosis.  |

#### **Depression**

- |   |   |
|---|---|
| 0 | not present   |
| 1 | periods of sadness or guilt greater than normal, never sustained for more than a few days or a week |
| 2 | sustained depression for >1 week  |
| 3 | vegetative symptoms (insomnia, anorexia, abulia, weight loss)                                       |
| 4 | vegetative symptoms with suicidality  |

#### **Motivation/Initiative**

- |   |  |
|---|--|
| 0 | normal   |
| 1 | less of assertive, more passive                                      |
| 2 | loss of initiative or disinterest in elective activities             |
| 3 | loss of initiative or disinterest in day to day (routine) activities |
| 4 | withdrawn, complete loss of motivation                               |

### **Activities of Daily Living**

#### **Speech**

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## PARKINSON'S SYNDROME

- 0 normal
- 1 mildly affected, no difficulty being understood
- 2 moderately affected, may be asked to repeat
- 3 severely affected, frequently asked to repeat
- 4 unintelligible most of time

### Salivation

- 0 normal
- 1 slight but noticeable increase, may have nighttime drooling
- 2 moderately excessive saliva, may have minimal drooling
- 3 marked drooling

### Swallowing

- 0 normal
- 1 rare choking
- 2 occasional choking
- 3 requires soft food
- 4 requires NG tube or G-tube

### Handwriting

- 0 normal
- 1 slightly small or slow
- 2 all words small but legible
- 3 severely affected, not all words legible
- 4 majority illegible

### Cutting Food/Handing Utensils

- 0 normal
- 1 somewhat slow and clumsy but no help needed
- 2 can cut most foods, some help needed
- 3 food must be cut, but can feed self
- 4 needs to be fed

### Dressing

- 0 normal
- 1 somewhat slow, no help needed
- 2 occasional help with buttons or arms in sleeves
- 3 considerable help required but can do something alone
- 4 helpless

### Hygiene

- 0 normal
- 1 somewhat slow but no help needed
- 2 needs help with shower or bath or very slow in hygienic care
- 3 requires assistance for washing, brushing teeth, going to bathroom
- 4 helpless

### Turning in Bed/ Adjusting Bed Clothes

- 0 normal
- 1 somewhat slow no help needed
- 2 can turn alone or adjust sheets but with great difficulty
- 3 can initiate but not turn or adjust alone
- 4 helpless

### Falling-Unrelated to Freezing

- 0 none
- 1 rare falls
- 2 occasional, less than one per day
- 3 average of once per day
- 4 >1 per day

### Freezing When Walking

- 0 normal
- 1 rare, may have start hesitation
- 2 occasional falls from freezing,
- 3 frequent freezing, occasional falls
- 4 falls from freezing

### Walking

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## PARKINSON'S SYNDROME

- 0 normal
- 1 mild difficulty, day drag legs or decrease arm swing
- 2 moderate difficulty requires no assist
- 3 severe disturbance requires assistance
- 4 cannot walk at all even with assist

### Tremor

- 0 absent
- 1 slight and infrequent, not bothersome to patient
- 2 moderate, bothersome to patient
- 3 severe, interfere with many activities
- 4 marked, interferes with many activities

### Sensory Complaints Related to Parkinsonism

- 0 none
- 1 occasionally has numbness, tingling, and mild aching
- 2 frequent, but not distressing
- 3 frequent painful sensation
- 4 excruciating pain

### Motor Exam

#### Speech

- 0 normal
- 1 slight loss of expression, diction, volume
- 2 monotone, slurred but understandable, mod. impaired
- 3 marked impairment, difficult to understand
- 4 unintelligible

#### Facial Expression

- 0 Normal
- 1 slight hypomymia, could be poker face
- 2 slight but definite abnormal diminution in expression
- 3 mod. hypomymia, lips parted some of time
- 4 masked or fixed face, lips parted, complete loss of expression

#### Tremor at Rest Face

- 0 absent
- 1 slight and infrequent
- 2 mild and present most of time
- 3 moderate and present most of time
- 4 marked and present most of time

#### Right Upper Extremity (RUE)

- 0 absent
- 1 slight and infrequent
- 2 mild and present most of time
- 3 moderate and present most of time
- 4 marked and present most of time

#### Left Upper Extremity (LUE)

- 0 absent
- 1 slight and infrequent
- 2 mild and present most of time
- 3 moderate and present most of time
- 4 marked and present most of time

#### Right Lower Extremity (RLE)

- 0 absent
- 1 slight and infrequent
- 2 mild and present most of time
- 3 moderate and present most of time
- 4 marked and present most of time

#### Left Lower Extremity (LLE)

- 0 absent
- 1 slight and infrequent
- 2 mild and present most of time
- 3 moderate and present most of time

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## PARKINSON'S SYNDROME

4 marked and present most of time

### Action or Postural Tremor RUE

0- absent  
1- slight, present with action  
2- moderate, present with action  
3- moderate present with action and posture holding  
4- marked, interferes with feeding

### Action or Postural Tremor LUE

0 absent  
1 slight, present with action  
2 moderate, present with action  
3 moderate present with action and posture holding  
4 marked, interferes with feeding

### Rigidity Neck

0 absent  
1 slight or only with activation  
2 mild/moderate  
3 marked, full range of motion  
4 severe

### Rigidity RUE

0 absent  
1 slight or only with activation  
2 mild/moderate  
3 marked, full range of motion  
4 severe

### Rigidity LUE

0 absent  
1 slight or only with activation  
2 mild/moderate  
3 marked, full range of motion  
4 severe

### Rigidity RLE

0 absent  
1 slight or only with activation  
2 mild/moderate  
3 marked, full range of motion  
4 severe

### Rigidity LLE

0 absent  
1 slight or only with activation  
2 mild/moderate  
3 marked, full range of motion  
4 severe

### Finger taps Right

0 normal  
1 mild slowing, and/or reduction in amp.  
2 moderate impaired. Definite and early fatiguing, may have occasional arrests  
3 severely impaired. Frequent hesitations and arrests.  
4 can barely perform

### Finger taps Left

0 normal  
1 mild slowing, and/or reduction in amp.  
2 moderate impaired. Definite and early fatiguing, may have occasional arrests  
3 severely impaired. Frequent hesitations and arrests.  
4 can barely perform

## PARKINSON'S SYNDROME

### **Right Hand Movements (open and close hands in rapid succession)**

- 0 normal
- 1 mild slowing, and/or reduction in amp.
- 2 moderate impaired. Definite and early fatiguing, may have occasional arrests
- 3 severely impaired. Frequent hesitations and arrests.
- 4 can barely perform

### **Left Hand Movements (open and close hands in rapid succession)**

- 0 normal
- 1 mild slowing, and/or reduction in amp.
- 2 moderate impaired. Definite and early fatiguing, may have occasional arrests
- 3 severely impaired. Frequent hesitations and arrests.
- 4 can barely perform

### **Right Rapid Alternating Movements (pronate and supinate hands)**

- 0 normal
- 1 mild slowing, and/or reduction in amp.
- 2 moderate impaired. Definite and early fatiguing, may have occasional arrests
- 3 severely impaired. Frequent hesitations and arrests.
- 4 can barely perform

### **Left Rapid Alternating Movements (pronate and supinate hands)**

- 0 normal
- 1 mild slowing, and/or reduction in amp.
- 2 moderate impaired. Definite and early fatiguing, may have occasional arrests
- 3 severely impaired. Frequent hesitations and arrests.
- 4 can barely perform

### **Right Leg Agility (tap heel on ground)**

- 0 -normal
- 1 mild slowing, and/or reduction in amp.
- 2 moderate impaired. Definite and early fatiguing, occasional arrests
- 3 severely impaired. Frequent hesitations and arrests.
- 4 can barely perform

### **Left Leg Agility (tap heel on ground)**

- 0 normal
- 1 mild slowing, and/or reduction in amp.
- 2 moderate impaired. Definite and early fatiguing, may have occasional arrests
- 3 severely impaired. Frequent hesitations and arrests.
- 4 can barely perform

### **Arising From Chair (patient arises with arms folded across chest)**

- 0 normal
- 1 slow, may need more than one attempt
- 2 pushes self up from arms or seat
- 3 tends to fall back, may need multiple tries but can arise without assistance
- 4 unable to arise without help

### **Posture**

- 0 normal erect
- 1 slightly stooped, could be normal for older person
- 2 definitely abnormal, mod. stooped, may lean to one side
- 3 severely stooped with kyphosis
- 4 marked flexion with extreme abnormality of posture

### **Gait**

- 0 normal
- 1 walks slowly, may shuffle with short steps, no festination or propulsion
- 2 walks with difficulty, little or no assistance, some festination, short steps
- 3 severe disturbance, frequent assistance
- 4 cannot walk

### **Postural Stability (retropulsion test)**

- 0 normal
- 1 recovers unaided
- 2 would fall if not caught
- 3 falls spontaneously
- 4 unable to stand

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## PARKINSON'S SYNDROME

### Body Bradykinesia/ Hypokinesia

0	none
1	minimal slowness, could be normal, deliberate character
2	mild slowness and poverty of movement, definitely abnormal
3	moderate slowness, poverty, or small amplitude
4	marked slowness, poverty, or amplitude

### Prognosis

Parkinson's Syndrome is not by itself a fatal disease, but it does get worse with time. The average life expectancy of a PS patient is generally the same as for people who do not have the disease. However, in the late stages of the disease, PS may cause complications such as choking, pneumonia, and falls that can lead to death. Fortunately, there are many treatment options available for people with PS.

The progression of symptoms in PS may take 20 years or more. In some people, however, the disease progresses more quickly. There is no way to predict what course the disease will take for an individual person.

### Treatment

Treatment is tailored to the person's needs and life situation, and must be tempered by the realization that long-term therapy may be associated with problems that eventually cause as much difficulty for the person as the basic disease itself does. The benefit of treatment for most people is improvement of symptoms with the ability to maintain adequate physical activity, and the ability to continue working. No specific treatment may be necessary when PS is first diagnosed except developing a regular exercise routine, and learning about PS. A major dimension of therapy involves helping the person and family adapt to the reality of a chronic, progressive, disabling disease, and methods to help preserve the person's independence assume top priority. Everyone should be encouraged to maintain social contacts and family and occupational responsibilities to counter the tendency toward immobility and apathy. Support groups can help with general information and answers to specific questions that arise in daily living, while providing a supportive social environment. People with early stage PS can be generally be divided into two groups: those in whom tremor is the major symptom, and those in whom slowness and stiffness predominate. People in whom tremor has become troublesome are candidates for the "anticholinergic drugs," while people with stiffness and slowness are candidates for Amantadine. L-dopa is indicated when Parkinsonian symptoms become socially or occupationally disabling.

### Medications

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## PARKINSON'S SYNDROME

Medications for PD fall into several categories: Dopamine precursors and agonists, drugs affecting neurotransmitters, and drugs to control non-motor symptoms.

### **Dopamine Precursors**

The first category includes drugs that work directly or indirectly to increase the level of dopamine in the brain. The most common drugs for PD are dopamine precursors – substances such as levodopa that cross the blood-brain barrier and are then changed into dopamine. Other drugs mimic dopamine or prevent or slow its breakdown.

### **Levodopa**

The cornerstone of therapy for PD is the drug levodopa (also called L-dopa). Levodopa (from the full name L-3,4-dihydroxyphenylalanine) is a simple chemical found naturally in plants and animals. Levodopa is the generic name used for this chemical when it is formulated for drug use in patients. Nerve cells can use levodopa to make dopamine and replenish the brain's dwindling supply. People cannot simply take dopamine pills because dopamine does not easily pass through the blood-brain barrier, a lining of cells inside blood vessels that regulates the transport of oxygen, glucose, and other substances into the brain.

Usually, patients are given levodopa combined with another substance called carbidopa. When added to levodopa, carbidopa delays the conversion of levodopa into dopamine until it reaches the brain, preventing or diminishing some of the side effects that often accompany levodopa therapy. Carbidopa also reduces the amount of levodopa needed.

Levodopa is very successful at reducing the tremors and other symptoms of PD during the early stages of the disease. It allows the majority of people with PD to extend the period of time in which they can lead relatively normal, productive lives.

Although levodopa helps most people with PD, not all symptoms respond equally to the drug. Levodopa usually helps most with bradykinesia and rigidity. Problems with balance and other non-motor symptoms may not be alleviated at all.

People who have taken other medications before starting levodopa therapy may have to cut back or eliminate these drugs in order to feel the full benefit of levodopa. People often see dramatic improvement in their symptoms after starting levodopa therapy. However, they may need to increase the dose gradually for maximum benefit. A high-protein diet can interfere with the absorption of levodopa, so some physicians recommend that patients taking the

## PARKINSON'S SYNDROME

drug restrict their protein consumption during the early parts of the day or avoid taking their medications with protein-rich meals.

Levodopa is often so effective that some people may temporarily forget they have PD during the early stages of the disease. But levodopa is not a cure. Although it can reduce the symptoms of PD, it does not replace lost nerve cells and it does not stop the progression of the disease.

Levodopa can have a variety of side effects. The most common initial side effects include nausea, vomiting, low blood pressure, and restlessness. The drug also can cause drowsiness or sudden sleep onset, which can make driving and other activities dangerous. Long-term use of levodopa sometimes causes hallucinations and psychosis. The nausea and vomiting caused by levodopa are greatly reduced by combining levodopa and carbidopa, which enhances the effectiveness of a lower dose.

Dyskinesias, or involuntary movements such as twitching, twisting, and writhing, commonly develop in people who take large doses of levodopa over an extended period. These movements may be either mild or severe and either very rapid or very slow. The dose of levodopa is often reduced in order to lessen these drug-induced movements. However, the PD symptoms often reappear even with lower doses of medication. Doctors and patients must work together closely to find a tolerable balance between the drug's benefits and side effects. If dyskinesias are severe, surgical treatment may be considered. Because dyskinesias tend to occur with long-term use of levodopa, doctors often start younger PD patients on other dopamine-increasing drugs and switch to levodopa only when those drugs become ineffective.

Other troubling and distressing problems may occur with long-term levodopa use. Patients may begin to notice more pronounced symptoms before their first dose of medication in the morning, and they may develop muscle spasms or other problems when each dose begins to wear off. The period of effectiveness after each dose may begin to shorten, called the *wearing-off effect*. Another potential problem is referred to as the *on-off effect* — sudden, unpredictable changes in movement, from normal to parkinsonian movement and back again. These effects probably indicate that the patient's response to the drug is changing or that the disease is progressing.

One approach to alleviating these side effects is to take levodopa more often and in smaller amounts. People with PD should never stop taking levodopa without their physician's knowledge or consent because rapidly withdrawing the drug can have potentially serious side effects, such as immobility or difficulty breathing. Fortunately, physicians have other treatment choices for some symptoms and stages of PD. These therapies include the following:

### **Dopamine Agonists**

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These drugs, which include bromocriptine, apomorphine, pramipexole, and ropinirole, mimic the role of dopamine in the brain. They can be given alone or in conjunction with levodopa. They may be used in the early stages of the disease, or later on in order to lengthen the duration of response to levodopa in patients who experience wearing off or on-off effects. They are generally less effective than levodopa in controlling rigidity and bradykinesia. Many of the potential side effects are similar to those associated with the use of levodopa, including drowsiness, sudden sleep onset, hallucinations, confusion, dyskinesias, edema (swelling due to excess fluid in body tissues), nightmares, and vomiting. In rare cases, they can cause compulsive behavior, such as an uncontrollable desire to gamble, hypersexuality, or compulsive shopping. Bromocriptine can also cause fibrosis, or a buildup of fibrous tissue, in the heart valves or the chest cavity. Fibrosis usually goes away once the drugs are stopped.

### **MAO-B inhibitors**

These drugs inhibit the enzyme monoamine oxidase B, or MAO-B, which breaks down dopamine in the brain. MAO-B inhibitors cause dopamine to accumulate in surviving nerve cells and reduce the symptoms of PD. Selegiline, also called deprenyl, is an MAO-B inhibitor that is commonly used to treat PD. Studies supported by the NINDS have shown that selegiline can delay the need for levodopa therapy by up to a year or more. When selegiline is given with levodopa, it appears to enhance and prolong the response to levodopa and thus may reduce wearing-off fluctuations. Selegiline is usually well-tolerated, although side effects may include nausea, orthostatic hypotension, or insomnia. It should not be taken with the antidepressant fluoxetine or the sedative mepiridine, because combining selegiline with these drugs can be harmful. Another MAO-B inhibitor, rasagiline, was approved by the FDA for use in treating PD.

### **COMT inhibitors**

COMT stands for catechol-O-methyltransferase, another enzyme that helps to break down dopamine. Two COMT inhibitors are approved to treat PD in the United States: entacapone and tolcapone. These drugs prolong the effects of levodopa by preventing the breakdown of dopamine. COMT inhibitors can decrease the duration of "off" periods, and they usually make it possible to reduce the person's dose of levodopa. The most common side effect is diarrhea. The drugs may also cause nausea, sleep disturbances, dizziness, urine discoloration, abdominal pain, low blood pressure, or hallucinations. In a few rare cases, tolcapone has caused severe liver disease. Because of this, patients taking tolcapone need regular monitoring of their liver function.

### **Anticholinergics**

Another category of PD drugs affects other neurotransmitters in the body in order to ease some of the symptoms of the disease. For example, anticholinergic drugs interfere with production or uptake of the neurotransmitter acetylcholine. These

## PARKINSON'S SYNDROME

drugs help to reduce tremors and muscle stiffness, which can result from having more acetylcholine than dopamine.

These drugs, which include trihexyphenidyl, benztropine, and ethopropazine, decrease the activity of the neurotransmitter acetylcholine and help to reduce tremors and muscle rigidity. Only about half the patients who receive anticholinergics are helped by it, usually for a brief period and with only a 30 percent improvement. Side effects may include dry mouth, constipation, urinary retention, hallucinations, memory loss, blurred vision, and confusion.

When recommending a course of treatment, a doctor will assess how much the symptoms disrupt the patient's life and then tailor therapy to the person's particular condition. Since no two patients will react the same way to a given drug, it may take time and patience to get the dose just right. Even then, symptoms may not be completely alleviated.

### **Medications for Non-Motor Symptoms**

The last category of drugs prescribed for PD includes medications that help control the non-motor symptoms of the disease, that is, the symptoms that don't affect movement. For example, people with PD-related depression may be prescribed antidepressants.

Doctors may prescribe a variety of medications to treat the non-motor symptoms of PD, such as depression and anxiety. For example, depression can be treated with standard anti-depressant drugs such as amitriptyline or fluoxetine (however, as stated earlier, fluoxetine should not be combined with MAO-B inhibitors).

Anxiety can sometimes be treated with drugs called benzodiazepines. Orthostatic hypotension may be helped by increasing salt intake, reducing antihypertension drugs, or prescribing medications such as fludrocortisone

Hallucinations, delusions, and other psychotic symptoms are often caused by the drugs prescribed for PD. Therefore, reducing or stopping PD medications may alleviate psychosis. If such measures are not effective, doctors sometimes prescribe drugs called atypical antipsychotics, which include clozapine and quetiapine. Clozapine also may help to control dyskinesias. However, clozapine also can cause a serious blood disorder called agranulocytosis, so people who take it must have their blood monitored frequently.

### **Other Medications**

#### **Apomorphine**

Apomorphine is a drug with anti-parkinsonian properties that are qualitatively similar to those seen with L-dopa. Although used in the 1950s, apomorphine was not widely prescribed because of the need for administration by injection and

adverse reactions such as nausea, vomiting, low blood pressure, and sedation. In Europe it is being used as a continuous infusion with the aid of a mini-pump to deal with “off period” disabilities such as pain, bladder dysfunction, dystonia, and gastrointestinal symptoms.

### **Chelation**

There is no evidence that iron chelation therapy helps PS, and it is not recommended for it. Additionally, there is little evidence to suggest that giving iron helps PS, and iron is not a recommended treatment.

### **Dextromethorphan**

Dextromethorphan (DM) a common ingredient in over-the-counter cough syrups may help tremor and rigidity, but it has to be taken at 120 to 180 mg per day (the usual dose for coughs is up to 90 mg per day).

### **Immunoglobulin (IGIV)**

There have been some reports of intravenous Immunoglobulin (IGIV) treatments helping those with PS, but it is unclear if such treatments are of any benefit.

### **NADH (nicotinamide adenine dinucleotide hydrogen)**

NADH, also called Coenzyme 1 or Q1, is being promoted for treatment of PS. Note that this is not NAD, but rather “reduced NAD or NADH.” There are no clinical studies showing its usefulness for PS, but there are reports of it helping some symptoms and of PS people being able to reduce their dose of L-dopa while on it. It may have some potential to do harm if too much is taken, one of the common side effects being insomnia.

### **Neuroprotective Treatments**

Investigations on neuroprotection are at the forefront of PD research. Several molecules have been proposed as potential treatments. However, none of them have been conclusively demonstrated to reduce degeneration. Agents currently under investigation include anti-apoptotics (TCH346, CEP-1347), antiglutamatergics, monoamine oxidase inhibitors (selegiline, rasagiline), promitochondrials (coenzyme Q10, creatine), calcium channel blockers (isradipine) and growth factors (GDNF). Preclinical research also targets alpha-synuclein.

### **Complications of Long-Term Levodopa Use**

Unfortunately drug side effects from long-term use of medications may become worse than the symptoms they're meant to suppress:

### **Dyskinesia**

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This means bizarre, involuntary, jerky movements of the head, tongue, and extremities, and is a particularly troublesome side effect of L-dopa. The abnormal movements can gradually become incapacitating unless the L-dopa dosage is reduced. Small doses of L-dopa, stopping anticholinergic therapy, and using bromocriptine with the L-dopa may help to control this.

### **“Frozen State”**

A wearing off response to medication, with a return of symptoms can produce an awkward, “frozen” state that may persist until more medication is absorbed, although people sometimes spontaneously “unfreeze” without more medication. Freezing episodes, especially when starting to walk, run, or change direction, may become frequent as the effect of the drugs wear off. For some people taking more medication can bring on bad side effects, but not taking it may mean a worsening of the Parkinsonian symptoms.

### **End of Dose Deterioration**

As each dose of medication wears off, symptoms may return with varying “good” and “bad” times through the day. More frequent L-dopa doses, sometimes with bromocriptine may help, and Sinemet CR may help.

### **Addiction to L-dopa**

People who have had a problem with drug addiction (alcohol, street drugs) may become addicted to L-dopa, and experience withdrawal effects when it has to be stopped.

### **The “On-Off” Phenomenon**

Sudden spells of immobility, apparently unrelated to drug doses, may occur several times a day and last from minutes to hours. Drugs do not help this type of parkinsonian immobility, but lowering the L-dopa dosage (and sometimes adding bromocriptine) may help these.

## **Surgery**

Surgery, once a mainstay of treatment, was abandoned in the 1960s, when Ldopa therapy was introduced, and because of a high incidence of complications. However, improved techniques, as well as the limitations of drug treatment, have prompted a return to surgical treatments. Of the four procedures available, the two most established — thalamotomy and pallidotomy — require the destruction of certain brain cells that interfere with normal movement. These cells can be identified by using electricity to stimulate various areas of the brain

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(a procedure called brain mapping). The technique is also used to identify cells that control sight and other senses, so that they won't be inadvertently destroyed during surgery.

Candidates must be relatively young (usually under age 65 or 70), in otherwise good health, and have the specific symptoms a particular technique is designed to address. When appropriate, surgery often relieves symptoms and decreases the need for medication (but may not eliminate the need for medications altogether), but it's not a cure — nor does it stop progression.

Surgery can be a reasonable choice, but only if you have the specific symptoms for which a procedure was developed, and only when medication produces intolerable side effects or can no longer provide adequate control of symptoms. The four surgical options are:

### **Thalamotomy**

The oldest type of surgery (thalamotomy) affects only tremor on the opposite side of the body to the surgery, and in the past was associated with significant postoperative deficits such as weakness, numbness, and disturbances of memory and language. New brain mapping techniques limit the risk of serious permanent complications (such as paralysis, loss of sensation, and stroke) to less than 1% now, with confusion and balance problems sometimes occurring. In this technique, the surgeon destroys a specific group of cells in the thalamus, the main relay center of the brain. Thalamotomy is appropriate for the 5 to 10% of people who have a disabling tremor of the hand or arm, and few other symptoms. Improvement is immediate, with 80 to 90% of people experiencing a significant reduction or even elimination of tremor.

The procedure is performed with the person conscious, under local anesthesia and sometimes sedation. The hospital stay is usually two days, with full recuperation in six weeks. Risks include temporary balance disturbances and numbness around the mouth and in the hand.

### **Pallidotomy**

Pallidotomy is also done by using "brain mapping" techniques. Microelectrodes are implanted by a surgeon in a specific group of cells within the globus pallidus, a portion of the brain's movement center, and a current is passed through them. This causes lesions in the part of the brain that cause dyskinesias which usually improves them.

Although the criteria for pallidotomy have not been firmly established, the technique seems to be most effective for slow movement, with improvement in off-period motor scores, and is especially useful to ameliorate the side effects of medication such as severe dyskinesia and widely fluctuating symptoms. It may

not help gait disturbances and falling, and sometimes does not help tremor much.

Like thalamotomy, pallidotomy is performed with the person conscious, and requires a similar recuperation period. Risks and benefits are also similar, with the additional possibility of damaging peripheral vision. Careful brain mapping limits the risk of visual impairment to between 2 and 5%. PS medication has to be continued after the surgery, and this treatment does not stop the progression of PS, but can give relief from dyskinesias for three to five years.

### **Deep Brain Stimulation**

Deep brain stimulation, or DBS, uses an electrode surgically implanted into part of the brain. The electrodes are connected by a wire under the skin to a small electrical device called a pulse generator that is implanted in the chest beneath the collarbone. The pulse generator and electrodes painlessly stimulate the brain in a way that helps to stop many of the symptoms of PD. DBS has now been approved by the U.S. Food and Drug Administration, and it is widely used as a treatment for PD.

DBS can be used on one or both sides of the brain. If it is used on just one side, it will affect symptoms on the opposite side of the body. DBS is primarily used to stimulate one of three brain regions: the subthalamic nucleus, the globus pallidus, or the thalamus. However, the subthalamic nucleus, a tiny area located beneath the thalamus, is the most common target. Stimulation of either the globus pallidus or the subthalamic nucleus can reduce tremor, bradykinesia, and rigidity. Stimulation of the thalamus is useful primarily for reducing tremor. DBS usually reduces the need for levodopa and related drugs, which in turn decreases dyskinesias. It also helps to relieve on-off fluctuation of symptoms. People who initially responded well to treatment with levodopa tend to respond well to DBS. While the benefits of DBS can be substantial, it usually does not help with speech problems, "freezing," posture, balance, anxiety, depression, or dementia.

One advantage of DBS compared to pallidotomy and thalamotomy is that the electrical current can be turned off using a handheld device. The pulse generator also can be externally programmed.

Patients must return to the medical center frequently for several months after DBS surgery in order to have the stimulation adjusted by trained doctors or other medical professionals. The pulse generator must be programmed very carefully to give the best results. Doctors also must supervise reductions in patients' medications. After a few months, the number of medical visits usually decreases significantly, though patients may occasionally need to return to the center to have their stimulator checked. Also, the battery for the pulse generator must be surgically replaced every three to five years, though externally rechargeable

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batteries may eventually become available. Long-term results of DBS are still being determined. DBS does not stop PD from progressing, and some problems may gradually return. However, studies up to several years after surgery have shown that many people's symptoms remain significantly better than they were before DBS.

DBS is not a good solution for everyone. It is generally used only in people with advanced, levodopa-responsive PD who have developed dyskinesias or other disabling "off" symptoms despite drug therapy. It is not normally used in people with memory problems, hallucinations, a poor response to levodopa, severe depression, or poor health. DBS generally does not help people with "atypical" parkinsonian syndromes such as multiple system atrophy, progressive supranuclear palsy, or post-traumatic parkinsonism. Younger people generally do better than older people after DBS, but healthy older people can undergo DBS and they may benefit a great deal.

As with any brain surgery, DBS has potential complications, including stroke or brain hemorrhage. These complications are rare, however. There is also a risk of infection, which may require antibiotics or even replacement of parts of the DBS system. The stimulator may sometimes cause speech problems, balance problems, or even dyskinesias. However, those problems are often reversible if the stimulation is modified.

Researchers are continuing to study DBS and to develop ways of improving it. They are conducting clinical studies to determine the best part of the brain to receive stimulation and to determine the long-term effects of this therapy. They also are working to improve the technology used in DBS.

### **Fetal or Neural Tissue Implantation or Transplantation**

The goal of this experimental technique is to restore brain function by replacing damaged tissue in the dopamine-producing area of the brain with brain tissue that will produce dopamine. Because of the ethical concerns surrounding use of fetal tissue, it's likely that genetically engineered cells may one day be used. While neuroprotein systems are being developed that will provide protection from breaking down for the neurons that produce dopamine naturally, within the last years, scientists have discovered how to make "cultured" brain cells grow into the specialized nerve cells that are lost in PS. This discovery may make brain cell transplants less controversial and more widely available.

Additionally, the implantation of some types of animal cells such as pig brain cells is being studied and early results are encouraging. Specific criteria and optimal techniques have yet to be determined for transplanting cells into the brain, and most people undergo the procedure as part of a study.

### **Physical and Occupational Therapy**

The symptoms of PS can keep people from using their muscles adequately. Although prescribed medication will help relieve the symptoms, it is as important for people to keep as physically active as possible as it is for them to take medication. For most people, a simple at-home exercise program is strongly recommended, and must be done as regularly as taking medication. All physical activity is beneficial, from taking long walks to self-feeding, bathing, dressing and doing chores at home. No task should be undertaken to the point of fatigue. When fatigue sets in, the person should rest. Swimming and walking are particularly good for maintaining fitness and muscle tone.

People with balance problems should avoid walking alone. Individuals with a tendency to freeze in their movements should be supervised carefully and should only do exercises when sitting or lying. Individuals with fluctuations in the effectiveness of their medication should not be pressured to exercise during "off" periods when the medication is not working. Forcing people to walk or exercise during their "off" or "bad" periods will not "loosen them up." Such exercises can be painful, as well as dangerous. When people emerge from an "off" period, most are so relieved that they will start moving about on their own, and encouragement and assistance at this time will motivate them even further.

Those who are unable to exercise for themselves should receive daily passive range-of-motion exercises to prevent permanent contractures of limbs. Tai Chi has been taken up by many with PS with great benefit.

Physical therapists and occupational therapists can help initiate a program of home therapy and can provide advice about appropriate adaptations of household furnishings. Gait training, strength training, and balance training can help with balance and help to prevent falls. Railings, and specially designed toilet seats, eating utensils, dishes, dressing aids, garments, shoes, canes and walkers, and easy chairs can all be of help depending on the need for them. Speech therapy and even speech amplification devices can help if speech is affected. Using an electric razor may become necessary. Shoes without laces such as loafers may be best (and they may need non-slip soles). Close attention should be paid to keeping the mouth and teeth clean. And, physical exercise is very important for a caregiver, and must not be forgotten.

### **Stretching**

Stretching is important because Parkinson's can cause muscles to feel stiff and tight, especially when medications are wearing off. Sometimes muscles ache with PD. Muscles can become weak and joints may stiffen. Gradually people

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with Parkinson's can become stooped and shuffle. Even being able to breathe deeply may be difficult.

There are many, many different stretches, but with PD the most useful stretches are those for the neck, chest, anterior shoulder, the back of the legs and hands.

### **Strengthening**

Parkinson's does not directly cause weakness of muscles. But it can have a marked impact on muscle strength if it progresses to the point that everyday tasks are affected.

In mild to moderate Parkinson's muscle strength can be maintained by keeping up an active life – walking, cycling, jogging, golf, bowls, swimming – any physical activity that the person enjoys. When Parkinson's starts to limit these activities, then muscle strength must be maintained in other ways that are structured and safe. (i.e., weights, resistance tubing, balls and springs). Individuals may need extra prompts or cues to 'kick-start' exercises. Exercising to music or counting out loud are excellent ways of overcoming the slowness of Parkinson's.

Muscles to focus on:

- The large muscles of the arms and legs.
- The muscles of the trunk – abdominals and back extensors.
- The muscles of the upper back and neck.

### **Fall Prevention**

Falls are common in people aged 65 years and older and are the leading cause of injury in this age group. It has been estimated that up to 40% of people with Parkinson's have problems with balance and falling.

Parkinson's affects the automatic actions of the brain - like balance control. Loss of balance and falling can be a common feature of Parkinson's caused by a variety of factors:

- slowness and weakness of the legs
- dizziness due to lower blood pressure
- painful joints
- cataracts

Research has shown that exercise can enhance balance and significantly reduce falls.

The following are some precautions and tips for patients to prevent accidents and falling.

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- Avoid carrying objects in both hands while walking.
- Avoid standing for long periods with feet too close together, this can increase the risk of falls.
- Increase stride length when walking. Falls may result when individuals shuffle their feet, or as a result of foot drag, which is common among people with Parkinson's disease.
- Swing arms when walking. This helps maintain balance and decreases fatigue.
- If feet feel 'frozen to the floor' when walking is initiated, attempt to step over an imaginary obstacle, or initiate the transfer of weight by rocking from side to side.
- Do not wear rubber-soled shoes because they grip the floor and may cause you to trip.
- If rising from a seated position is difficult, place feet directly under the knees, lean forward and stand up firmly in a smooth movement.
- Rather than using the hands and arms when attempting to stand up, use the thigh muscles. This exercise also helps strengthen the large muscles of the legs, maintaining strength and walking ability.
- If feeling lightheaded, move slowly when changing positions. When getting out of bed, sit up slowly, remain seated on the side of the bed for 15 to 30 seconds, and then stand in one place for 15 to 30 seconds before walking. This technique can prevent falls.
- Do not use throw rugs or wax the floors in the home. Ensure that rooms are well lit, and use night-lights in hallways and bathrooms. Keep the home free of clutter to prevent tripping.
- Install grab bars to get into and out of the bath and shower. A raised toilet seat with arm rails may also be a helpful.

### **Walking**

Walking becomes a task which cannot be performed without considerable attention. The automatic arm swing and weight transference become progressively diminished. Some people become stooped and their footsteps get smaller.

Some patient tips to improve gait:

- Visualize taking long steps.
- Think "back straight" and look ahead.
- Tell arms to swing.
- Tell heel to touch first.
- Plan the route.

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- When turning around, walk in a semi-circle, don't swivel on one foot.
- If feeling unsteady, stop and take a step to recover.
- Don't walk and talk at the same time.
- Walking to a regular beat like music works well.
- Try walking with a group.
- Always wear supportive shoes, but not with rubber soled soles as this increases friction making it more difficult to walk.

### **Relaxation**

Living with Parkinson's means that much mental energy is needed to do even simple things. Huge amounts of energy are expended, resulting in mental and physical fatigue. Anxiety is a feature of many people's PD, especially in the 'off' times of medication.

Learning to relax is part of living well with Parkinson's. There are many relaxation techniques that may help. The following are just some techniques that people with Parkinson's and families may wish to try.

#### Relaxation position

- Position self supine with a small pillow under the head.
- Put a small pillow under the knees.
- Take a deep breath in through the nose.
- Breathe out, opening the mouth and sigh a long, loud AAAHHH.
- Repeat 3 – 5 times.
- Be aware of any tension in the face, relax the jaw.
- On the in-breath feel the abdomen rise, then fall on the out-breath.

#### Concluding relaxation

- If feeling deeply relaxed, start to move fingers and toes.
- Breathe in, lifting arms up in the air.
- Breathe out; lift arms above the head, stretching them back towards the floor.
- Hold and stretch for 2 breaths.
- Breathe in and lift arms back up.
- Breathe out, lower arms to sides. Turn over, slowly sit up.

### **Activities of Daily Living**

#### **Fine Motor Skills**

A resting tremor may occasionally interfere with the functional activities that involve the hands. To better manage the tremor, have the person press the affected elbow against their body to stabilize the upper arm, and then perform the desired movement as quickly as they can.

#### **Dressing**

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Dressing and undressing can be very tiring and time consuming. The following tips can help make dressing quicker and easier:

- Put on and take off clothes from the stiffer side first.
- If standing balance is affected, have the person sit over the edge of the bed or in a chair with armrests to dress.
- Use elastic waistbands or Velcro closures instead of buttons or zippers.
- Choose loose, lightweight clothes.
- Choose clothing that closes in the front.
- Use elastic shoelaces or slip-on shoes.
- Use a long-handle shoehorn to assist with shoes

### **Bathing**

The bathroom is usually the most dangerous place for anyone with impaired balance, difficulty in walking, or tremors. When bathroom surfaces are wet, they are extremely slippery. Some preventive measures to avoid accidents are:

- Place a non-slip rubber mat or adhesive anti-slip surfaces at the bottom of the tub or stall shower.
- Remove glass doors from the tub.
- Use a tub bench or shower chair.
- Try a showerhead on a flexible hose.
- Attach soap to a rope which makes retrieving easier.
- Avoid using bathroom fixtures (i.e., towel racks) as grab railings since they are not very strong. Have grab bars installed if more support is required.
- A raised toilet seat makes rising easier and arm rails can be attached to the toilet seat or wall for leverage.

### **Driving**

Many people in the early stages of PS are capable of driving. However, depending on the stage of the disease, and the medications being taken, it may be unwise for an individual to drive, even if they feel they can. PS can affect reaction time, and defensive driving skills may be diminished. People on medications and with the more advanced stages should not be allowed to drive.

### **Speech**

One of the most widely practiced treatments for speech disorders associated with Parkinson's disease is the Lee Silverman voice treatment (LSVT). It focuses on increasing vocal loudness and has an intensive approach of one month.

### **Diet**

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In the early stages of PS, because the tremor and the muscular stiffness burn a lot of calories, an increase in food intake may be needed to maintain weight. The patient should be weighed at least once per week to monitor weight loss. In the middle and late stages of PS, a low protein diet may be necessary (this will aid in the absorption of L-dopa into the body). There is often a compromise between taking L-dopa on an empty stomach (to get full absorption) and taking it with food (to prevent nausea and vomiting). If there is weight loss, PS people should not skip meals. Supplements such as Carnation Instant Breakfast or Ensure or Sustocal or Enercal may be started either to ensure proper nutrition, or to prevent weight loss. Smooth solids and thick liquids are swallowed best. Tucking the chin to the chest when swallowing helps prevent aspiration. The patient should be upright (at 60 to 90 degrees) for meals and should remain sitting upright for 15 to 30 minutes after meals.

Since the conversion of L-dopa to dopamine is aided by an enzyme that requires vitamin B6, it was initially thought that more B6 would increase the conversion of L-dopa to dopamine. However, it is now known that B6 given with L-dopa counteracts the effect of L-dopa. Sinemet and Prolopa do not have this problem. People who are on L-dopa without Sinemet or Prolopa should avoid foods high in B6, such as avocados, lentils, and lima beans.

Because Vitamin E and Vitamin C have anti-oxidant effects, it was hoped that Vitamin E or C might delay the progression of PS, but so far studies have been disappointing. Vitamin E, used at a dose of 2000 IU per day, does not seem to work.

### **Hospital Tips**

The hospitalized person with PS, because of slowness and stiffness, is at increased risk for hospital acquired complications. Pneumonia, aspiration, urinary tract infection, clots (phlebitis) in leg and pelvic veins (that can break off and go to the lungs), and skin ulcers are particular concerns. Mobilization, careful feeding, preventative measures for phlebitis (especially after surgery) and attention to skin care may be lifesaving.

It is also important to ensure that the individual with PS continues to get their medications according to their schedule (and not the hospital's), and the person and their caregivers need to become part of the hospital staff team to ensure that this happens.

### **Comorbidities**

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People with PS often have other medical conditions. Sometimes these conditions, or the drugs that are prescribed for them, influence the treatment of PS. Some of these conditions are:

### **Glaucoma**

Glaucoma may be exacerbated by anticholinergic drugs. These drugs do not necessarily have to be discontinued in people with glaucoma, but should be used with care. People with both conditions should be carefully monitored by an eye doctor.

### **Heart Disease**

People who have had a recent heart attack, or whose heart rhythm is irregular (arrhythmia), may be sensitive to the side effects of some antiparkinson drugs. People who have had a recent heart attack may not be able to tolerate the slight drop in blood pressure caused by some of the antiparkinson drugs. These drugs may not have to be stopped as long as the drugs' benefits exceed their risks.

### **Hypertension**

In rare instances, drugs used to treat high blood pressure may worsen PS symptoms.

Clonidine is such a drug, but the effect is temporary and will disappear when Catapres is stopped. Clonidine should be avoided if another suitable drug is available.

Aldomet (methyldopa) may compete with the carbidopa in Sinemet and may decrease the beneficial effect of Sinemet. This does not always occur, and Aldomet does not have to be stopped in people with PS.

Diuretics are frequently used to treat people with high blood pressure or heart disease. Water pills produce a decrease in the amount of body fluid, which may result in dizziness on standing. This dizziness is more likely to affect PS people, particularly those who take Sinemet or a dopamine agonist.

### **Stomach, Intestinal Diseases**

There have been some isolated reports relating bleeding ulcers to Sinemet and the dopamine agonists. However, no direct cause-and-effect relationship has been established. Sinemet and the dopamine agonists may open the valve like mechanism between the esophagus and the stomach, resulting in gastric reflux and causing an inflammation of the esophagus. This may be followed by nausea and vomiting, which will result in further inflammation. People who have a hiatus hernia are especially vulnerable to this complication. This inflammation may

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result in erosion of the wall of the esophagus, which can lead to bleeding. People who have a hiatus hernia, and take PS drugs, should follow an appropriate diet. In addition, such people may require antacids or other measures to decrease the inflammation. Antacids should not be taken within one hour of other medications since antacids may decrease the absorption of other drugs.

People with a history of liver disease, jaundice, or hepatitis should have "liver function tests" before taking antiparkinson drugs. These tests may have to be repeated periodically.

### **Bladder Conditions**

Drugs such as the anticholinergics, Symmetrel, and some of the antidepressants may cause a temporary inability to void. This is especially likely to occur in men with an enlarged prostate gland. These drugs should be used carefully in such people.

### **Cancer**

In general, the influence of a cancer on PS is not known. There have been a few reports in which the use of L-dopa has been associated with the growth of a melanoma. However, there have also been reports in which L-dopa has been given to people with melanoma who showed no accompanying increased growth in the skin tumor. Although this relationship is still unproven, caution is urged when using L-dopa with PS people who have a history of melanomas. If the person's neurological condition worsens to the point where the PS becomes the main problem, then L-dopa should be used with the knowledge and consent of the person.

## **Sleep Disorders and Parkinson's Syndrome**

Insomnia is now being recognized as a symptom that can have a significant impact on diseases and the quality of life. Fifty percent of PS people have sleep disorders. Insomnia is not a diagnosis. It is a symptom of many disorders and it has to be assessed like any other symptom. A management plan comprises documentation of the nature and duration of the sleep complaint (sleep diary), improvement in sleep hygiene, and possibly intervention with other techniques and medications.

Some sleeping difficulties, especially vivid dreaming and myoclonus, are related to L-dopa. Readjustment of the dose of L-dopa, and eliminating the evening dose (if possible) may improve the patient's sleep. On the other hand, some

patients require L-dopa to sleep because a lack of medication makes them so rigid that they cannot turn in bed.

### **Dementia and Parkinson's Syndrome**

Dementia means an irreversible permanent general mental deterioration including some or all of the following: disturbance of memory, disturbances in learning, reasoning, thinking, calculation, and aptitudes, and disturbances of personality and judgment. For many years, it was thought that intellectual deterioration was not an intrinsic feature of PS. It now appears that people with PS are at high risk (10 times that of age-matched controls) of developing dementia. The incidence of brain cell changes of the Alzheimer type is higher in people with PS than in age-matched controls. Because so many symptoms of dementia in PS people seem to involve a slowing of both mentation and motor activity, it has been suggested that the part of the brain involved in PS (the black cells) may play an important role in mental functioning (which had always been thought to be the function of the grey matter of the brain).

Dementia often, but not always occurs late in the course of PS. It must always be remembered that the drugs used in the treatment of PS can cause mental confusion. When mental confusion occurs in a person with PS, a decision has to be made as to which is worse: movement symptoms of PS left untreated, or the mental confusion.

If the mental confusion is severe enough to be a problem for the person with PS, or their caregivers, then there has to be a withdrawal of drugs to see if the person's mental functioning improves. Anticholinergic drugs are withdrawn first, followed by dopamine agonist medication and lastly levodopa preparations. The progressive withdrawal of medication may need to be done in the hospital because of the risk of complications such as severe rigidity and immobility, heightened risk of falling, aspiration, and rarely a severely elevated temperature. As the effects of the medications wear away, the person with PS can then be assessed to see if changes in mental ability were secondary to drug use, or were going to be present even in the absence of drugs. Currently dementia in PS is treated the same as any other type of dementia (such as Alzheimer's Disease).

Drugs such as Donepezil (Aricept) may improve cognitive and behavioral functioning in PS people, just as it does in some Alzheimer's patients, although there are no clinical studies proving this as yet.

An early symptom of drug toxicity in someone with PS is often nocturnal upset such as nightmares. Various daytime illusions then follow, with constant severe confusion and agitation in the final stage. Almost any drug may adversely affect a person with PS who is developing dementia. Pain killers (even aspirin), minor tranquilizers (Serax or oxazepam, Ativan or lorazepam, Valium or diazepam,

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Rivotril or clonazepam, Lectopam or bromazepam, Xanax or alprazolam, Loftran or ketazolam, sleeping pills containing "benzodiazepams," etc.), antidepressants, and the anti-ulcer drug Cimetidine have all caused problems. The worst offenders are drugs with "anticholinergic" properties such as some of the drugs used to treat PS, many of the drugs used for urinary frequency, and even some of the drugs that are usually used to control symptoms that accompany dementia.

Drug holiday is no longer recommended for the general management of levodopa complications, but occasionally a few days off all medication is necessary to clear severe drug-induced confusion. Some late stage people with PS and dementia may be managed with small, frequent doses of levodopa-carbidopa (50 to 100 mg, four to six times per day or half a tablet of 100/25 every two hours), or plain levodopa tablets (250 to 500 mg) may induce less confusion in these people.

### **Psychosis in Parkinson's Syndrome**

Probably because of news media and movie presentations, for most people, the word "psychotic" usually brings immediate fearful feelings to mind. Medically speaking however, a psychosis is a mental disorder in which a person's mental capacity, emotional responses, and capacity to recognize reality, communicate, and relate to others are impaired enough to interfere with their capacity to deal with the ordinary demands of life. Psychosis occurs in approximately 30% of people with PS within 5 years of starting treatment with L-dopa. Psychosis in a PS person decreases their quality of life, increases caregiver stress, and poses a dilemma for treating physicians. PS people and their caregivers are often reluctant to report psychotic symptoms which delays the diagnosis of them, and doctors often hesitate to treat the symptoms for fear of worsening parkinsonian symptoms.

A hallucination is a false sensory perception without a concrete external source for the perception. Common hallucinations involve sights or sounds, although any of the senses can be involved. A delusion is a false fixed belief that is not in accord with a person's intelligence and life situation. Common delusions include delusions of control which is a false belief that the person is being manipulated by others, delusions of grandeur which is an exaggerated belief of the person's importance, delusions of infidelity which is a false belief that a person's spouse is unfaithful, delusions of persecution which is a false belief that the person is being harassed by others, and paranoid delusions which is an over suspicious belief by the person about the actions of others.

Commonly reported symptoms in PS that are signs of a psychotic condition include: visual hallucinations (which are the most common), auditory hallucinations, sleeping alterations, vivid dreams, delusions, paranoia, altered

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sexual behavior and agitation. Psychotic symptoms can occur when a person seems otherwise clear mentally, but they can also occur with delirium (which medically speaking is actually a psychotic state in which confusion is a major symptom) or with a dementia. As an aside, delirium (a confusional state) is usually acute in onset, but may be chronic if it is mild and goes unrecognized for some time. It is characterized by a fluctuating level of consciousness that often becomes more pronounced as the day progresses ("sundowning" effect). Anticholinergic medications can cause or worsen delirium. There may be several causes for delirium in the same person going on at the same time. Hallucinations in PS often occur when the person seems otherwise clear, and are characterized by images from the person's past. Realistic images of people, familiar or strange, are common. They may involve formed images of people or animals familiar to the person, or may involve dead relatives, absent friends or relatives. The most common type of delusions tends to be those of paranoia, most often involving feelings of persecution, being followed, fears of being injured, poisoned, or controlled.

Determining a cause for a PS person's psychotic symptoms can be helpful in decisions that have to be made regarding treatment of them. Some experts find that a useful classification of psychotic symptoms is based on their timing related to the stage of the PS, or their timing related to the initiation of drug therapy for PS.

"Early onset" of psychosis refers to symptoms that develop within days to weeks of starting anti-parkinsonian therapy. A prior history of a psychotic disorder is a risk factor for early onset psychosis. Late onset of psychosis is usually observed after years of L-dopa therapy, and most PS patients who develop this do not have a previous history of psychiatric disease. PS people with advanced stages of PS and who have a dementia are more at risk for late onset psychosis. This type of psychosis is often but not always related to antiparkinsonian drug therapy. Because there is no definitive test for the cause of a psychosis, the causes must be deduced from the person's current and past history, the history their caregivers give, the relationship of the symptoms to drug therapy, and the quality and type of hallucinations and delusions.

Other than being antiparkinson drug induced, psychosis may also be caused by medical conditions such salt imbalances in the blood, thyroid disease, adrenal gland disease, some types of infections, low oxygen levels in the blood, and also by mental disorders such as depression, dementias, and alcohol or other substance abuse disorders.

Treatment with an antiparkinsonian medication is the most common cause of psychosis in PS, and can result from any of the antiparkinsonian drugs from any class (MAO inhibitors such as Selegiline, dopaminergic agents such as L-dopa, amantadine, and the anticholinergic agents such as Bzotropine). L-dopa can

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cause many neuropsychiatric problems in PS, including depression, crying spells, very low energy, mood swings, euphoria, manic or "hyper" states, increase in libido or loss of sexual inhibitions, anxiety, panic attacks, social avoidance, agitation, restlessness, nightmares, night terrors, vivid dreams, delusions, confusion, delirium, visual or sensory (or both) hallucinations, personality alterations, and paranoid feelings or thoughts.

Diagnosis of psychosis in PS is often delayed because PS persons and their caregivers may view these symptoms as embarrassing and are reluctant to report them to their doctors, or, the PS person may not be able to remember episodes of psychotic symptoms so that they go unreported. Once recognized, some investigations may need to be done to help sort out the cause, and then the issue becomes treatment.

Psychotic symptom treatment for PS persons with drugs is difficult as the optimal treatment for the psychotic symptoms may mean worse motor symptoms, and increased risk for falls and bowel problems for example. Thus part of the treatment of psychotic symptoms involves "behavioral interventions." The PS person and their family may have to learn to accept and cope with the presence of hallucinations. Families need to be educated regarding symptoms and encouraged to provide a calm stable environment. Paranoid patients are less likely to behave aggressively if family members avoid showing alarm. A variety of psychologic interventions may be helpful for the PS person and their caregivers - supportive or expressive psychotherapy may help the patient cope better with issues of loss, impaired functioning, depression or disrupted relationships, and marriage or family counseling may help.

Drugs called neuroleptic drugs that are often used to treat psychotic symptoms in people without PS often worsen PS. The newer neuroleptics such as Clozapine are expensive, and may injure the bone marrow. Thus initial steps in psychotic symptom treatment may involve adjusting the dose or withdrawing antiparkinsonian drugs other than L-dopa. If the symptoms persist, then the L-dopa dose may have to be adjusted. It should be noted that the effects of dosage reduction may take weeks to become apparent. In some, there may be a "threshold" dose of Ldopa, below which there are no psychotic symptoms, and above which the psychotic symptoms recur. In some, higher doses of L-dopa may be tolerated later on without recurrence of their symptoms. Unfortunately for many PD people, it may be impossible to find a dose that gives acceptable control of the motor symptoms without causing psychotic symptoms. If the psychotic symptoms are not relieved, then treatment with a "neuroleptic" drug can be tried. Many experts think that currently Clozapine is the drug of choice as it does not usually worsen motor symptoms and adjustment of L-dopa dosage is not necessary. However, it does have potential side effects, such as sedation, and bone marrow injury, and it can lower blood pressure significantly leading to falls or black outs. It occasionally paradoxically worsens the psychotic symptoms instead of improving them because of its anticholinergic activity. Another

medication that can be used is called Risperidone, although it is more likely to worsen motor symptoms than Clozapine. Olanzapine at 2.5 mg to 10 mg per day is less likely to worsen motor symptoms than Risperidone, although more likely to lower blood pressure. Ondansetron is another medication that has been used in this situation. If the PD person has depression, ETC. treatment may help both the depressive and psychotic symptoms.

### **Caring for the Caregiver**

“When one is sick, two need help.” Ms. Paula Goldberg, R.N., has explained the meaning of this quotation in talking about the roles a caregiver should fill in the life of the person with PS and in their own life. There are positives to caregiving, such as increased closeness and better communication between the person and the caregiver. There are also negatives, and it is important to eliminate or control as many negatives as possible. Caregivers face multiple problems related to day-to-day aspects of life such as financial problems, career planning, change of life situation, and loss of hobbies and activities. Caring for a person with PS means dealing with their psychological and physical problems in addition to the caregiver’s own problems. The responsibility for the partner’s life can be overwhelming. Feelings of guilt, insufficiency, anger, and aggression commonly occur. The spouse often behaves as if the disease has stricken the couple as a “team,” which in many respects is true.

Studies suggest that as many as 60% of PS people are depressed, but about 30% of their caregivers are also depressed. It is estimated that less than 10% of PS people and their caregivers are being treated for depression, as it often goes unrecognized.

People and their spouses should remember that PS need not be as incapacitating as it appears. People and their spouses should do as many things together as possible. A spouse can participate in doctor’s visits and help report on the person’s progress, difficulties, and needs. Spouses should encourage PS people to go out socially. If embarrassment from a disability arises, there are methods to minimize psychological distress. For example, when expected for a social engagement, people can arrive early and leave late, or in a restaurant, a waiter can be asked to specially prepare the food before the person is served.

Warning signs of caregiver stress include denial of the disease and its effect on the person with Parkinson’s, anger toward the Parkinson’s person or others because currently available treatments may not be perfectly effective or because no cures currently exist, social withdrawal from friends and activities that once brought you pleasure, anxiety about facing another day and what the future holds, depression affecting your ability to cope, exhaustion making it nearly

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impossible to complete necessary daily tasks, sleeplessness caused by a never-ending list of concerns, irritability leading to moodiness and triggering negative responses and reactions from others, lack of concentration making it difficult to perform your usual tasks, health problems that begin to take a toll, both mentally and physically.

Here are some tips for the caregiver:

- Don't compare yourself to others; others may look like saints to you, but they have shortcomings too, even if they don't show. Instead take talents from your past life and apply them to caregiving.
- Decide what absolutely must be done, and assign priorities, avoiding busy work. Learn to say no. Subdivide tasks into 15 minute sections, and keep a victory list of what you've accomplished.
- Reward yourself. Do something you enjoy that is unrelated to your caregiving routine.
- Humor and exercise are two coping mechanisms that are helpful for both you and the person you give care to.

The assessment of someone with PS isn't over until it has been determined how the caregiver is coping. Most people with PS are seen every three months by a doctor, and the caregiver should always be seen with them. It is important for the caregiver to have adequate means of coping when difficult situations arise. PS can be variable in its daily presentation, and even minor changes in medication levels can affect the person drastically. It is the caregiver who, in the first instance, has to learn to cope with these sudden unexpected changes.

To provide the best care for the person with PS, the caregiver will need outside support. That support can be met by family, friends, and professionals. The caregiver should make the situation clear to family and friends, get a community resources list, join a support group, and be sure to keep the doctor and his staff informed.

### Resources

#### **American Parkinson Disease Association**

135 Parkinson Avenue  
Staten Island, NY 10305-1425  
apda@apdaparkinson.org  
<http://www.apdaparkinson.org>

#### **Parkinson's Disease Foundation (PDF)**

1359 Broadway  
Suite 1509  
New York, NY 10018  
info@pdf.org  
<http://www.pdf.org>

#### **National Parkinson Foundation**

1501 N.W. 9th Avenue  
Bob Hope Road  
Miami, FL 33136-1494  
contact@parkinson.org  
<http://www.parkinson.org>

#### **Parkinson Alliance**

P.O. Box 308  
Kingston, NJ 08528-0308  
admin@parkinsonalliance.org  
<http://www.parkinsonalliance.org>

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### Supplemental Information

[Rehabilitation of Patients Suffering from Parkinson's Disease by Normotensive Therapy](#)

Orgeret, G. (2011). Rehabilitation of Patients Suffering from Parkinson's Disease by Normotensive Therapy. In J. Dushanova (Ed.), *Diagnostics and Rehabilitation of Parkinson's Disease*: InTech. CC BY 3.0

[Electrical Stimulation of Primary Motor Cortex for Parkinson's Syndrome](#)

Tani, N., & Saitoh, Y. (2011). Electrical Stimulation of Primary Motor Cortex for Parkinson's Syndrome. In J. Dushanova (Ed.), *Diagnostics and Rehabilitation of Parkinson's Disease*: InTech. CC BY 3.0

[Joint Replacement Surgery in Parkinson's Disease](#)

Gheiti, A. J. C., Baker, J. F., & Mulhall, K. J. (2011). Joint Replacement Surgery in Parkinson's Disease. In J. Dushanova (Ed.), *Diagnostics and Rehabilitation of Parkinson's Disease*: InTech. CC BY 3.0

[Sleep Disturbances in Patients with Parkinson's Disease](#)

Suzuki, K., Miyamoto, T., Miyamoto, M., Numao, A., Sakuta, H., Fujita, H., . . . Hirata, K. (2014). Sleep Disturbances in Patients with Parkinson's Disease. In A. Q. Rana (Ed.), *A Synopsis of Parkinson's Disease*: InTech. CC BY 3.0

[Parkinson's Disease and Peripheral Neuropathy](#)

Podgorny, P., & Toth, C. (2014). Parkinson's Disease and Peripheral Neuropathy. In A. Q. Rana (Ed.), *A Synopsis of Parkinson's Disease*: InTech. CC BY 3.0

[Update in Parkinson's Disease](#)

Carrillo, F., & Mir, P. (2012). Update in Parkinson's Disease. In J. Dushanova (Ed.), *Mechanisms in Parkinson's Disease - Models and Treatments*: InTech. CC BY 3.0

[Timing Control in Parkinson's Disease](#)

Almeida, Q. J. (2012). Timing Control in Parkinson's Disease. In J. Dushanova (Ed.), *Mechanisms in Parkinson's Disease - Models and Treatments*: InTech. CC BY 3.0

[Nonmotor Features in Parkinson's Disease: What Are the Most Important Associated Factors?](#)

Kadastik-Eerme, L., Muldmaa, M., Lilles, S., Rosenthal, M., Taba, N., & Taba, P. (2016). Nonmotor Features in Parkinson's Disease: What Are the Most Important Associated Factors?. *Parkinson's Disease, 2016*. CC BY 4.0

[Mindfulness for Motor and Nonmotor Dysfunctions in Parkinson's Disease](#)

Dissanayaka, N. N., Idu Jion, F., Pachana, N. A., O'Sullivan, J. D., Marsh, R., Byrne, G. J., & Harnett, P. (2016). Mindfulness for Motor and Nonmotor Dysfunctions in Parkinson's Disease. *Parkinson's Disease, 2016*. CC BY 4.0

[Herbal Medicines for Parkinson's Disease: A Systematic Review of Randomized Controlled Trials](#)

Kim, T. H., Cho, K. H., Jung, W. S., & Lee, M. S. (2012). Herbal medicines for Parkinson's disease: a systematic review of randomized controlled trials. *PLoS One, 7*(5), e0122469. CC BY

[Exploring music-based rehabilitation for parkinsonism through embodied cognitive science](#)

Schiavio, A., & Altenmüller, E. (2015). Exploring music-based rehabilitation for parkinsonism through embodied cognitive science. *Frontiers in neurology, 6*. CC BY

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## Parkinson's Syndrome

### Post-Test

1. Which of the following is NOT a main symptom of Parkinson Syndrome? (p. 3)
  - A. Tremor
  - B. Rigidity
  - C. Bradykinesia
  - D. Cognitive impairment
2. Dopamine is produced in the \_\_\_\_\_. (p. 3)
  - A. substantia nigra
  - B. corpus callosum
  - C. Broca's area
  - D. Amygdala
3. Parkinson's occurs in approximately 50% more women than men. (p. 6) A. True B. False
4. Which of the following symptoms is NOT typically associated with one of the primary symptoms of Parkinson's Syndrome? (p. 8)
  - A. Masked facial features
  - B. Intermittent hand tapping
  - C. Increased muscle tone in limbs
  - D. Bladder incontinence
5. Which of the following is best for diagnosing Parkinson's Syndrome? (p. 12)
  - A. Dopamine Agonist Assay
  - B. EEG
  - C. Triphasic CSF profile
  - D. History and physical examination
6. Apraxia, autonomic impairment, and cerebellar signs are all red flags that suggest a diagnosis other than Parkinson's syndrome. (p. 14) A. True B. False
7. Which disease has primary symptoms similar to those of PS, but also includes visual-spatial impairment, apraxia, myoclonus, and dysphagia? (p. 14-17)
  - A. Normal pressure hydrocephalus
  - B. Progressive Supranuclear Palsy
  - C. Corticobasal degeneration
  - D. Dementia with Lewy bodies
8. An individual is experiencing bilateral finger tapping, moderate slowness of gait, and mild difficulty with grooming tasks. According to the Hoehn and Yahr Scale, they are in which stage of Parkinson's? (p. 18-19)
  - A. Stage 2
  - B. Stage 3
  - C. Stage 4
  - D. Stage 5

## PARKINSON'S SYNDROME

9. Which of the following is NOT accurately scored utilizing the Unified Parkinson Disease Rating Scale (UPDRS)? (p. 19-24)
- A. Severe memory loss with disorientation to time and often place, severe impairment with problems (3 points)
  - B. Can cut most foods; some help needed (2 points)
  - C. Monotone speech, slurred but understandable (3 points)
  - D. Unable to arise from chair without help (4 points)
10. Parkinson's syndrome is a fatal disease. (p. 25) A. True B. False
11. The benefit of treatment for most people is improvement of symptoms with the ability to maintain adequate physical activity, and the ability to continue working. (p. 25)
- A. True B. False
12. The cornerstone of therapy for PD is the drug \_\_\_\_\_. (p. 26)
- A. Acetylcholine
  - B. MAO-B inhibitors
  - C. COMT inhibitors
  - D. Levodopa
13. Anticholinergics help to reduce \_\_\_\_\_. (p. 28-29)
- A. Festination
  - B. Orthostatic hypotension
  - C. Tremors and muscle rigidity
  - D. Bradykinesia
14. Which of the following is NOT a complication of long-term levodopa use? (p. 30-31)
- A. Dyskinesia
  - B. "Frozen state"
  - C. "On-Off" phenomenon
  - D. Sundowning
15. Which region of the brain is the most common target for DBS? (p. 33)
- A. Subthalamic nucleus
  - B. Amygdala
  - C. Hypothalamus
  - D. Red nucleus
16. Stretching of which muscles are typically indicated for individuals with PS? (p. 35)
- A. Quadriceps
  - B. Thoracic paraspinals
  - C. Hamstrings
  - D. Triceps
17. Strengthening exercises are important because Parkinson's syndrome directly causes muscle weakness. (p. 36) A. True B. False
18. It now appears that people with PS are at high risk (10 times that of age-matched controls) of developing \_\_\_\_\_. (p. 43)
- A. Hypertension
  - B. Dementia
  - C. Hepatomegaly
  - D. Kidney damage

## PARKINSON'S SYNDROME

19. Psychosis occurs in approximately 30% of people with PS within 5 years of starting treatment with L-dopa. (p. 44) A. True B. False
20. Approximately 30% of the caregivers of people with PS experience \_\_\_\_\_. (p. 47)
- A. headaches
  - B. depression
  - C. back pain
  - D. arthritis

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