

Parkinson's Disease

Challenges, Progress and Hope

Parkinson's Disease:
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About the Author

Ellen Steinbart, RN, MA began her career as a medical-surgical nurse at the Hospital of the Albert Einstein College of Medicine in New York, NY. She then worked as an intensive-care burn unit nurse at the University of Michigan Medical Center in Ann Arbor, MI. After finishing graduate studies at the University of Washington in Seattle, WA, she taught medical-surgical nursing at the University of Washington School of Nursing. She then turned to research, coordinating a diabetes research project at the Children's Hospital in Seattle. Finally, for the last 25 years, she worked as a research nurse at the University of Washington, coordinating research projects on the role of genetics in dementia and other neurological diseases, including Parkinson's disease.

Purpose and Goals

The purpose of this course is to provide nurses and other healthcare professionals with current information on Parkinson's disease and its management. Understanding the epidemiology, pathophysiology, risk factors, clinical features, diagnostic process, treatments and complications of Parkinson's disease provides a foundation for nursing care of the patient with Parkinson's disease.

Instructional Objectives

1. Review approximately how many people in the US have Parkinson's disease.
2. Explain the pathophysiology of Parkinson's disease.
3. Recognize risk factors for developing Parkinson's disease.
4. Name the four major motor symptoms of Parkinson's disease.
5. Name non-motor features of Parkinson's disease.
6. Explain the progression of Parkinson's disease.
7. Summarize how the diagnosis of Parkinson's disease is made.
8. Describe medications used to treat Parkinson's disease.
9. Explain nursing responsibilities and patient education related to the administration of medications for Parkinson's disease.
10. Describe who would be a good candidate for deep brain stimulation surgery for Parkinson's disease.
11. Review potential complications of Parkinson's disease.
12. Review nursing assessment of patients with Parkinson's disease.
13. Identify nursing diagnoses for patients with Parkinson's disease.
14. Explain nursing interventions to manage the symptoms of Parkinson's disease.
15. Recognize community resources for those with Parkinson's disease and their families.

Introduction

Parkinson's disease was first described almost 200 years ago. In 1817, Dr. James Parkinson, a London surgeon, published "An Essay on the Shaking Palsy", in which he described the disease that would later bear his name. He presented a series of six cases of "shaking palsy" and wrote a classic description of the disease. "Involuntary tremulous motion, with lessened muscular power, in parts not in action and even when supported; with a propensity to bend the trunk forwards, and to pass from a walking to a running pace: the senses and intellects being uninjured."



Today, we recognize Parkinson's disease as a chronic, progressive movement disorder associated with dopamine deficiency in the brain. It has characteristic motor symptoms of resting tremor (trembling of hands, arms, legs, jaw, or head), bradykinesia (slowed movement), rigidity (stiffness of limbs and trunk) and postural instability (impaired balance and coordination). The motor symptoms affect gait, posture, facial expression, automatic movements (blinking, arm swing, gesturing), speech, motor dexterity, chewing and swallowing. Parkinson's disease is now known to also have significant non-motor features, including autonomic, cognitive, psychiatric, sensory, fatigue, sleep and pain problems.

In the United States, Parkinson's disease currently affects approximately 1,000,000 people. Each year, approximately 50,000-60,000 people in the United State are newly diagnosed with Parkinson's disease. Parkinson's disease is the second most common neurodegenerative disease, after Alzheimer's disease. Parkinson's disease usually develops after age 50, but can occur in younger adults but rarely in children. The average age at onset is 60 years. Onset after age 50 is considered late onset Parkinson's disease; onset before age 50 is considered early-onset Parkinson's disease; onset before age 20 is considered juvenile-onset Parkinson's disease. Parkinson's disease is more common in men than women. Parkinson's disease affects people worldwide. Parkinson's disease is generally thought to be caused by a complex interaction between genetic and environmental factors.

There is presently no cure for Parkinson's disease, but treatments are available to help manage the symptoms. Parkinson's disease causes progressive disability. Although Parkinson's disease itself is not fatal, the Centers for Disease Control ranked complications from Parkinson's disease as the 14th leading cause of death in the US in 2011. The Parkinson's disease Foundation estimates the annual cost of Parkinson's disease in the US at $25 billion (direct and indirect costs, including treatments, lost income from inability to work, and social security payments). Research is being done to better understand Parkinson's disease and to develop more effective treatments, with the ultimate goals of cure and prevention of Parkinson's disease.

Other medical conditions may have clinical features that are similar to Parkinson's disease, referred to as "parkinsonian" or "secondary parkinsonism". These other medical conditions will not be discussed here. This continuing education program will focus exclusively on Parkinson's disease.

Pathophysiology

The major pathology of Parkinson's disease is the loss of dopamine producing neurons in the substantia nigra of the brain. For unknown reasons, these neurons gradually degenerate and die. Dopamine is a chemical messenger which normally transmits signals to the corpus striatum, which is involved in producing smooth voluntary movement. When dopamine producing cells

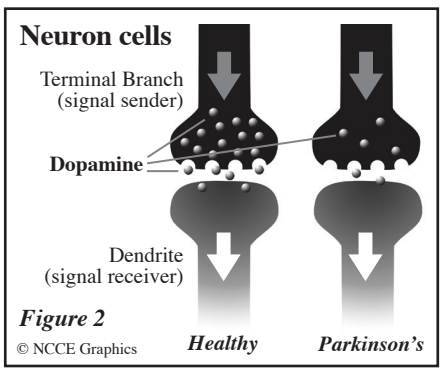

Figure 2

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die, there is a depletion of dopamine in the corpus striatum. The balance between the inhibitory neurotransmitter dopamine and an excitatory neurotransmitter acetylcholine is disrupted. The result is impaired movement, the motor symptoms of Parkinson's disease. When the motor symptoms of Parkinson's disease first appear, more than 50% of dopamine producing cells in the substantia nigra have already been lost, and there is a 50-70% decline in striatal dopamine (***Figure 2***).

In Parkinson's disease, Lewy bodies (named after Friedrich Lewy who first described them in 1912) develop in the remaining neurons in the substantia nigra and also in other areas of the brain. Lewy bodies are unusual deposits of protein, most notably alpha-synuclein. These microscopic Lewy bodies in the substantia nigra are a neuropathological hallmark of Parkinson's disease.

It is thought that the pathological changes of Parkinson's disease do not begin in the substania nigra. Rather, the changes appear to start in the olfactory bulb (involved with the sense of smell) and lower brainstem, followed by ascending brainstem involvement, then including the basal forebrain, amygdala, substantia nigra, and finally extending further to the thalamus and cortex. This process could explain why people with Parkinson's disease often report non-motor symptoms first, such as loss of sense of smell, sleep problems, mood disorders or constipation, before motor symptoms appear.

Other neurotransmitters in the brain may also be involved in Parkinson's disease. Some studies have shown a loss of nerve endings that produce the neurotransmitter norepinepherine. Reduced norepinepherine may contribute to some non-motor symptoms of Parkinson's disease, such as blood pressure control.

Risk Factors

Several risk factors for Parkinson's disease have been identified, including age, gender, family history, race/ethnicity and environmental factors.

Age

Increasing age is a risk factor for developing Parkinson's disease. The average age of onset for Parkinson's disease is about 60 years. About 1% of those over age 55 are affected with Parkinson's disease, and by age 85 more than 4 % are affected. About 5-10 % of those with Parkinson's disease have early-onset Parkinson's disease (onset before age 50). Early onset Parkinson's disease is often, but not always, inherited. The very rare juvenile-onset Parkinson's disease (onset before age 20) is also usually an inherited form of Parkinson's disease.

Gender

Parkinson's disease affects more men than women. The reason for this is not known.

Genetic

Most cases of Parkinson's disease appear to be sporadic, meaning only one individual in a family has Parkinson's disease.

Recent studies have noted that 10-25% of individuals with Parkinson's disease report a family history of at least one other relative with Parkinson's disease. This familial Parkinson's disease suggests that genetics is involved in some cases of Parkinson's disease. However, the role of genetics in Parkinson's disease is not well understood.

Research on families with two or more affected individuals have found two types of genes that can contribute to Parkinson's disease. Susceptibility genes and causal genes. Susceptibility genes are "associated" with Parkinson's disease; variations in these genes appear to increase the risk of developing Parkinson's disease. So far, four susceptibility genes have been identified. Having a variation in a susceptibility gene associated with Parkinson's disease does not mean that an individual will definitely develop Parkinson's disease, but his/her risk is somewhat increased compared with the general population. How variations in these susceptibility genes lead to an increased risk for Parkinson's disease is not known.

Causal genes have been identified in a small number of families, and mutations (changes) in these genes are actually responsible for the development of Parkinson's disease. These inherited forms of Parkinson's disease are very rare, representing approximately 2% of all Parkinson's disease.

So far, three genes have been identified in which mutations cause autosomal dominant Parkinson's disease (SNCA, LRRK2, PARK3). In autosomal dominant Parkinson's disease, each child of an affected parent is born at 50% risk for inheriting the gene mutation, and if he/she inherits the mutation, he/she would very likely develop Parkinson's disease. The first of these genes to be discovered was the alpha-synuclein gene (SNCA) on chromosome 4 in 1997. Mutations in this gene result in abnormal accumulation of alpha-synuclein protein in Lewy bodies in the brain, one of the neuropathological hallmarks of Parkinson's disease. In addition to gene mutations causing autosomal dominant Parkinson's disease, three other genes (Parkin, DJ-1 and PINK1) have been discovered in which mutations cause autosomal recessive Parkinson's disease. In autosomal recessive Parkinson's disease, two copies of the gene mutation are necessary for the disease to develop. Both parents must carry a copy of the Parkinson's disease gene mutation. If a child inherits one copy of the gene mutation from each parent, he/she would very likely develop Parkinson's disease. These rare forms of autosomal dominant or autosomal recessive inherited Parkinson's disease often, but not always, result in an early onset disease.

Individuals often ask "what is my risk for developing Parkinson's disease?. Excluding families with rare forms of inherited Parkinson's disease in which gene mutations causing the disease have been identified, if an individual has a close relative (parent, sibling) with Parkinson's disease, his/her risk for developing the disease is about 2-5%, just slightly greater than the risk for those without a family history of Parkinson's disease.

Race and Ethnicity

In the United States, African Americans and Asian American appear to have lower incidence of Parkinson's disease than Caucasians.

Environmental factors

Environmental factors associated with an increased incidence of Parkinson's disease include head injury, exposure to toxins or pesticides, and drinking well water. Rural living used to be considered a risk factor for Parkinson's disease, but a recent study found no difference between rural and urban living in the United States. The reasons for these associations are not clear. Some studies have found a higher incidence of PD in developed countries, possibly due to increased exposure to toxins.

Some environmental factors have been associated with a lower incidence of Parkinson's disease. Cigarette smokers appear to have a 40% lower risk for developing Parkinson's disease. Coffee drinkers appear to have about a 30% lower risk for Parkinson's disease compared with those who do not drink coffee. The use of non-steroidal anti-inflammatory drugs and estrogen replacement in post-menopausal women has been associated with a lower risk for Parkinson's

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disease. The reasons these environmental factors are associated with a lower risk for Parkinson's disease are unknown.

Clinical Manifestations

Parkinson's disease is characterized by the cardinal motor symptoms of resting tremor, bradykinesia, rigidity and postural instability ***(Figure 3)***. Initially, symptoms may be very mild. a slight resting tremor in one hand, stiffness. Gradually symptoms increase. Typically, one side of the body is affected first, but over time symptoms become bilateral. The side of the body first affected may continue to have more significant symptoms.

Tremor at rest is usually an early symptom of Parkinson's disease, present in approximately 85% of patients with Parkinson's disease at the time of diagnosis. If not present at the onset of Parkinson's disease, most individuals develop a tremor as the disease progresses, but a few may not. The tremor usually affects one side of the body initially, beginning in the hand. Occasionally the foot or jaw is affected first. It is a coarse rhythmic tremor with a frequency of 4-6 hertz (cycles per second). It is sometimes described as "pill rolling", because the index finger and thumb make a circular movement together, as if rolling a small pill around. The resting tremor may also affect the eyelids, legs, lips, tongue and jaw. The tremor can increase when the person is stressed. The tremor disappears with voluntary movement or sleep.

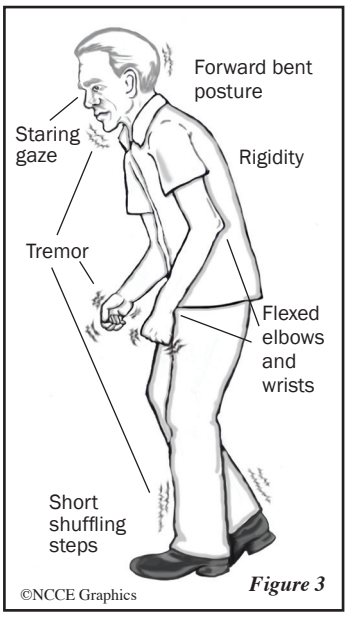

Figure 3

Bradykinesia, meaning slow movement, is the reduction of spontaneous movement. People with Parkinson's disease have difficulty initiating movement. Bradykinesia can be the most difficult problem in Parkinson's disease because it interferes with almost all activities of daily living: walking, rising from a chair, turning in bed, dressing, facial expression, speaking, writing, chewing, and swallowing.

As voluntary movement becomes gradually more impaired, people with Parkinson's disease may sit, lie or stand in one position for a long time.

The face develops a mask-like appearance due to diminished facial expression. There is decreased eye blinking and a staring gaze. Speech becomes soft, muffled, monotone and hesitant. Words may be repeated or slurred. There may also be an uncontrollable acceleration of words, resulting in rapid stammering speech.

Individuals with Parkinson's disease use less gesturing in communication. There is decreased manual dexterity, and handwriting becomes very small (micrographia), cramped and shaky. Chewing and swallowing problems can lead to eating problems, drooling and choking.

Rigidity results from increased muscle tone, the excessive and continuous contraction of muscles, which causes stiffness and increased resistance to movement in joints. It involves the limbs, neck and trunk and impairs active and passive movement. During passive range of motion around a joint, there may be brief, regular interruptions of this resistance, a ratchet-like movement, referred to as "cogwheel rigidity".

Postural instability results from the loss of postural reflexes. It is generally not an early sign of Parkinson's disease, but is common in more advanced disease and can become very debilitating. There are problems with balance and posture.

The gradual change in posture can include flexion of the head, stooping shoulders and upper body, leaning forward, and an inability to maintain an upright trunk position when sitting or standing. There is a tendency to hold arms in a flexed position when walking. People with more advanced Parkinson's disease have difficulty making postural adjustments, righting themselves, if they begin leaning or falling. As the reflexes needed to maintain upright posture are gradually lost, there is an increased risk of falls and injury.

Gait disturbances result from the bradykinesia, rigidity and postural instability of Parkinson's disease. Short shuffling steps are used to help maintain upright posture when walking and decreased arm swing. There is also a tendency to turn en bloc, as a unit, with neck and trunk rigid and taking many small step to turn. A festinating gait can develop with Parkinson's disease. stooped posture, imbalance and short steps that tend to accelerate in an effort to "catch up" with the body's center of gravity, a stumbling run potentially ending in a fall. In more advanced disease, there may be gait "freezing" episodes, a sudden inability to move feet and legs, lasting a few seconds or longer. It may occur at the onset of walking (start hesitation), or when changing directions, or when walking through a narrow space like a doorway.

Muscle cramps and dystonia can occur in Parkinson's disease. Muscle cramps commonly affect legs and toes. The pain of aching muscles and joints is thought to be due to the rigidity and abnormal postures of Parkinson's disease. Dystonia is an abnormal prolonged muscle contraction that causes an involuntary movement or twisted positioning, involving an arm, leg or the torso.

Although rare, Parkinson's disease may cause a severe unexplained burning or stabbing pain. This "central pain" is thought to originate in the brain.

It is now recognized that Parkinson's disease includes many non-motor problems, including mood disorders, psychotic symptoms, cognitive impairment and dementia, sleep disturbances, sensory abnormalities, autonomic function impairment, pain, and fatigue. These are thought to be the result of widespread involvement of many areas of the brain in Parkinson's disease, including brainstem, olfactory, thalamic and cortical areas. Some of these clinical features may be present even before motor signs and symptoms appear.

Mood disorders are common in Parkinson's disease. It is estimated that half of all Parkinson's disease patients have depression, which can occur during any phase of the illness. It is thought to be part of the illness, not just a reaction to having a chronic disease. Anxiety can also be a problem for some people with Parkinson's disease. It can occur alone, or together with depression.

It is estimated that up to 40% of those with Parkinson's disease experience visual hallucinations. These can occur early in the disease or later. Depression and dementia are risk factors for hallucinations. Hallucinations can sometimes be triggered

by medications used to treat Parkinson's disease. Some individuals with Parkinson's disease may experience delusions, usually later in the disease.

Cognitive impairment affects many individuals with Parkinson's disease. One study found 19% of patients had some cognitive impairment at the time of diagnosis. Cognitive problems can present as slow thinking or difficulty with complex tasks, long-term planning, memorizing and retrieving new information.

In some cases of Parkinson's disease, cognitive impairment progresses to dementia. Dementia refers to a decline in mental abilities severe enough to interfere with daily life. It is estimated that 20-30% of patients with Parkinson's disease have dementia. Dementia usually occurs late in the disease, after age 70, often 10-15 years or more after the diagnosis of Parkinson's disease.

Symptoms of Parkinson's disease dementia can include impaired memory, disorientation, distractibility, confusion, poor judgment, language problems, and difficulty processing information and making decisions. People with hallucinations and severe muscle and motor symptoms appear more likely to develop dementia.

Dementia is thought to be due to increasing alpha-synuclein pathology in several areas of the brain (frontal lobes, forebrain, hippocampus, and amygdala), along with the possible development of Alzheimer's disease, which is common in the elderly.

Sleep disorders are common in Parkinson's disease. Patients may have problems falling asleep and may awaken frequently during the night. They may have a restless sleep and experience nightmares. They may have drowsiness during the day. Bradykinesia and rigidity make it difficult to turn and change position in bed. Tremors, involuntary movements, restless leg problems or muscle aches may interrupt sleep. REM (rapid eye movement) Sleep Behavior Disorder. which causes limbs to move abnormally, suddenly and violently during REM sleep. sometimes precedes the motor signs of Parkinson's disease.

People with Parkinson's disease can experience fatigue and loss of energy, especially late in the day. This may be related to muscle stress, difficulty initiating and carrying out movement, poor sleep, or depression.

The autonomic nervous system is affected by Parkinson's disease, leading to problems in several areas. The autonomic nervous system regulates smooth muscle activity. In Parkinson's disease, the GI tract functions more slowly. Diminished peristalsis, along with limited physical activity and problems eating and drinking normally all contribute to constipation.

Some patients with Parkinson's disease may have problems with urination, such as urinary hesitation, inability to empty the bladder or urinary urgency which may occur with little warning. Because of mobility problems, the patient may not be able to get to the bathroom in time, as a result incontinence may develop.

Improper functioning of the autonomic nervous system can cause skin problems. Seborrhea can develop, a scaly, flaky, itchy, redness on the face or similar to dandruff on the scalp.

Some patients experience excessive sweating. Orthostatic hypotension, thought to be due to impaired vasomotor reflexes and sympathetic denervation of the heart, may cause dizziness or light-headedness when changing from a lying to standing position.

Sexual problems can occur with Parkinson's disease. Loss of desire and dissatisfaction with sex life can occur for both men and women with Parkinson's disease. Problems may be related to depression. Tremor (which increases with excitement), rigidity, bradykinesia, impaired fine motor control and dyskinesias may interfere with one's usual sexual life.

The course of Parkinson's disease can span 10-20 years or more. It can begin with subtle changes such as loss of sense of smell, constipation, depression, fatigue, muscle pain or REM sleep behavior disorder years before any motor symptoms are noted. As motor symptoms progress, there is increasing disability. The rate of progression of motor symptoms varies from one individual to the next. Non-motor symptoms affect most people with Parkinson's disease, but they also vary from one individual to the next.

Stages of Parkinson's disease can generally be described as mild, moderate or advanced.

In the mild stage: motor symptoms are inconvenient but the activities of daily living are not affected; symptoms are unilateral; medications are effective in controlling motor symptoms.

In the moderate stage: symptoms are bilateral; movement is slow; "freezing" can occur; balance and coordination is impaired; medications to control motor symptoms may "wear off"; medications may cause side effects such as dyskinesias.

In the advanced stage. patients may be in a wheelchair or bed most of the day because of difficulties walking; they need help with activities of daily living and are unable to live alone; they may have significant cognitive problems; medication benefits and side effects may be difficult to balance.

Life expectancy for people with Parkinson's disease may be decreased, especially with early onset Parkinson's disease. The leading cause of death for people with Parkinson's disease is pneumonia.

Diagnosis

Parkinson's disease can be difficult to diagnose initially. Early symptoms may appear similar to changes that occur with normal aging. Physicians may need to observe patients over time to see how early symptoms evolve. Other neurological problems with parkinsonian features that need to be ruled out include essential tremor, multiple system atrophy, Lewy body dementia, progressive supranuclear palsy, corticobasal degeneration, Alzheimer's disease, spinocerebellar ataxias, Huntington's disease, prion disease, frontotemporal dementia, Wilson's disease, vascular parkinsonism, and normal pressure hydrocephalus.

Early symptoms may appear similar to changes that occur with normal aging

Generally, the diagnosis of Parkinson's disease is made if a person has two or more of the following symptoms: resting tremor, bradykinesia, or rigidity. Approximately 85% of people with Parkinson's disease have tremor at the time of diagnosis. A diagnosis of Parkinson's disease may be difficult to make if tremor is absent initially. Gradual onset of symptoms and unilateral symptoms support a diagnosis of Parkinson's disease.

Early symptoms may include masked facial expression, decreased eye blinking, and shuffling gait with small steps, decreased arm swing, stooped posture, soft voice, and small handwriting. Postural instability typically appears later in Parkinson's disease as does problems with balance and chewing/swallowing.

The diagnosis of Parkinson's disease is based on clinical findings. There is no laboratory test or brain scan that can diagnose

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Parkinson's disease. To diagnose Parkinson's disease, a detailed medical history is taken and a thorough physical examination is done.

The physical examination includes a careful neurological evaluation. Resting tremor, agility of arms and legs, muscle tone, gait and balance are assessed. **The Unified Parkinson's Disease Rating Scale** or the **Hoehn and Yahr Scale** may be utilized in evaluating symptoms. The Hoehn and Yahr Scale rates the progression of the motor symptoms of Parkinson's disease on a scale of 1-5, with 1 being mild unilateral involvement only, and 5 being wheelchair-bound or bedridden unless aided.

The Unified Parkinson's Disease Rating Scale and the Hoehn and Yahr Scale also evaluates cognition, behavior, mood, activities of daily living, independence, complications of therapy, and has a clinician scored motor examination.

The medical history includes a description of all symptoms and how long they have been occurring. In addition to motor symptoms, people with Parkinson's disease may report early vague feelings of weakness, fatigue, pain, and loss of sense of smell.

A family history is important to identify any possible genetic factors that may be involved in the disease.

Questions about brain trauma, stroke or infection are asked, as these may result in symptoms similar to Parkinson's disease.

A history of exposure to toxins can be important, as some, such as heavy metals and carbon monoxide, can cause symptoms similar to Parkinson's disease.

A detailed drug history, including both current and past medications and any recreational drug use, is taken to determine if a prescribed medication or illicit drug may be contributing to the Parkinson's disease-like symptoms. Long term use of medications such as haloperidol, phenothiazines, metoclopramide, methyldopa, reserpine or chlopromazine can cause symptoms similar to Parkinson's disease. Recreational drugs such as "ecstasy" and the heroin-like substance MPTP can cause parkinsonian symptoms.

Even though there are no laboratory tests that are diagnostic of Parkinson's disease, blood work may be ordered to rule out other problems such as B12 deficiency, hypothyroidism, testosterone deficiency and vitamin D deficiency.

In the rare instance of a strong family history of Parkinson's disease, genetic testing may be considered, within the context of formal professional genetic counseling. This could provide a definitive diagnosis of Parkinson's disease if a known genetic mutation causing Parkinson's disease is identified in a symptomatic individual. It would also help define the genetic risk for developing Parkinson's disease in other family members.

CT and MRI brain scans of people with Parkinson's disease are typically normal. Brain scans may be useful in some cases to rule out other problems such as stroke, tumor, or normal pressure hydrocephalus. Research is investigating the possible use of positron emission tomography (PET) and single Photon computed emission tomography (SPECT) as diagnostic tools for Parkinson's disease.

When a diagnosis of Parkinson's disease is considered likely, a trial with a medication that increases dopamine in the brain is often done to confirm the diagnosis. An improvement of symptoms with this trial medication can help confirm the diagnosis of Parkinson's disease.

Treatments

There is presently no cure for Parkinson's disease. The goals of treatment are to relieve the symptoms of Parkinson's disease and to keep the individual with Parkinson's disease as functional and independent as possible. The primary treatment is medication to manage the symptoms of Parkinson's disease, both motor and non-motor symptoms. In some cases of more severe Parkinson's disease, when motor symptoms can no longer be managed with medication therapy, brain surgery (deep brain stimulation) may be done to help manage the motor symptoms. Supportive therapy, including physical therapy, exercise programs, speech and swallowing therapy, nutritional guidance, and occupational therapy can be helpful with mobility, communication, swallowing, diet and managing the activities of daily living.

Medications

Several different types of medications are available to help manage the motor symptoms of Parkinson's disease. The choice of medications for an individual depends on many factors, such as age, symptoms, medication side effects, other health issues and other medications. Below are general descriptions of the types of medications used in the treatment of Parkinson's disease. Full information about indications, dosage, adverse effects, contraindications and patient teaching should be obtained from the package insert for each specific medication.

Dopamine precursors

Carbidopa/levodopa (Sinemet)

Parkinson's disease results from the loss of dopamine producing cells in the substantia nigra and subsequent depleted levels of dopamine in the corpus striatum. Dopamine itself cannot be given as a treatment for Parkinson's disease because it does not cross the blood-brain barrier. However levodopa, a metabolic precursor that is converted into dopamine, does cross the blood brain barrier. Levodopa can be used to replenish the brain's dwindling supply of dopamine. When taken orally, levodopa is absorbed from the gut, and much is quickly converted into dopamine in the periphery before it reaches the brain. Carbidopa inhibits the conversion of levodopa to dopamine before it reaches the brain. Adding carbidopa to levodopa allows a much lower dose of levodopa to be used, with fewer side effects.

Carbidopa/levodopa is the most effective drug for managing the motor symptoms of Parkinson's disease. Therapy typically begins with a low dose, which is gradually increased (typically weekly) as necessary to manage symptoms. People with Parkinson's disease can have a dramatic improvement in symptoms when starting carbidopa/levodopa. Over a period of years, responsiveness to carbidopa/levodopa typically declines, and doses must be increased to control motor symptoms.

Carbidopa/levodopa can be used early in Parkinson's disease or later when other medications cannot control the symptoms. Long term use of carbidopa/levodopa can cause several problems. Dyskinesias (involuntary movement such as twitching, twisting or writhing) are common in people who take large doses over an extended period of time. The effectiveness of each dose may begin to shorten, called "end-of-dose wearing off". Individuals may develop muscle spasms as doses wear off. There may be sudden, unpredictable changes in condition, from "on" when the patient is relatively mobile to "off" when the patient is quite immobile, called "on-off motor fluctuations" or "on-off response.

If problems with dyskinesias, "wearing off", or "on-of. motor fluctuations" occur, medication dose or schedule may need to be adjusted, or other medications added, to find a tolerable balance between the benefits and side effects of carbidopa/levodopa.

Carbidopa/levodopa should be taken at the scheduled times to obtain maximum symptom relief. Initially, it may take some time (weeks)

to get the full effect of the medication. Carbidopa/levodopa is best taken approximately 30 minutes before a meal, to maximize absorption and effectiveness of medication. If nausea is a problem, the medication can be taken with non-protein food. Protein-rich foods and pyridoxine (vitamin B6) may diminish the effectiveness of levodopa.

In early Parkinson's disease, side effects of carbidopa/levodopa include nausea, low blood pressure, dry mouth and dizziness. Later in Parkinson's disease, motor side effects develop, including commonly dyskinesias and dystonia. Other side effects may include mental disturbances (depression, hallucinations, and psychosis), drowsiness, sudden sleep onset, and dysrhythmias. Occasionally there may be a dark color (red, brown) to urine saliva or sweat. A rare side effect of levodopa is oculogyric crisis, the eyes becoming fixed in one position for several hours.

Because carbidopa/levodopa can cause drowsiness and sudden onset of sleep during daily activities, sometimes without warning, patients should use caution when driving or operating machinery, or avoid these activities if they have experienced such sleepiness or "sleep attacks".

Patients should never stop taking carbidopa/levodopa without consulting their physician. A sudden discontinuation or rapid withdrawal of levodopa can precipitate a condition similar to neuroleptic malignant syndrome. Symptoms include fever, rigidity, cognitive changes, and sometimes tremor, tachycardia, tachypnea, diaphoresis, dystonia and chorea. Neuroleptic malignant syndrome is potentially life threatening and requires immediate emergency care and restarting of levodopa.

Carbidopa/levodopa is available in several different preparations and strengths, including immediate release tablets, long-acting tablets, and tablets that dissolve in the mouth without water. Because there are several different formulations of carbidopa/levodopa, it is important for people to be aware which preparation they take. Care should be taken to check each prescription renewal, because accidental substitution of a different formulation of carbidopa/levodopa could cause significant problems of overdose or underdose. Many other medications may interact with carbidopa/levodopa, so it is important for the patient to check with the physician before taking any new medications.

Dopamine Agonists

- Pramipexole dihydrochloride (Mirapex)
- Ropinirole (Requip)
- Rotigotine transdermal system (Neupro Patch)
- Apomorphine hydrochloride (Apokyn)
- Bromocriptine mesylate (Parlodel)

Dopamine agonists are compounds that activate dopamine receptors in the brain, mimicking dopamine, thus helping to produce smooth voluntary movement. Dopamine agonists can be used alone to treat Parkinson's disease, or in combination with levodopa. They can be used in the early stages of Parkinson's disease when symptoms are mild, to delay beginning carbidopa/levodopa. Later in the disease when dopamine agonists are combined with carbidopa/levodopa, they can enhance the therapeutic effects of levodopa and reduce "wearing off" or "on-off motor fluctuations" that some patients experience with long term use of carbidopa/levodopa.

Side effects of dopamine agonists include nausea, drowsiness, sudden sleep onset without warning ("sleep attacks"), orthostatic hypotension, and hallucinations. Hallucinations are more common in elderly patients. Because of drowsiness and the danger of falling asleep (sometimes without warning), patients should avoid driving or operating machinery until they know how the medication affects them.

Dopamine agonists have been associated with compulsive behaviors such as uncontrollable gambling, shopping, eating or sexual urges in a subset of patients. These compulsive behaviors are generally reversible when the medication is stopped.

Pramipexole and ropinerole, oral tablets, are the most commonly prescribed dopamine agonists. Patients are started on a low dose of medication, which is increased gradually (typically weekly) as necessary to achieve a therapeutic effect. Pramipexole and ropinerole can be taken with or without food.

Rotigotine transdermal system is a medication patch applied once daily to the skin. Patients begin on a low dose of medication, which is increased gradually as necessary to achieve a therapeutic effect. The transdermal patch was recalled in 2008, but after improvements it was re-approved and re-introduced in 2012. In addition to the general side effects of dopamine agonists, the rotigotine transdermal system may application site reactions.

Apomorphine hydrochloride is an injectible (subcutaneous) medication, used as a short-acting "rescue" drug for acute intermittent treatment of severe hypomobility ("off" episodes) in people with advanced Parkinson's disease. It is a fast-acting drug that can relieve symptoms within minutes. It comes in pre-filled syringes. In addition to the general side effects of dopamine agonists, apomorphine hydrochloride causes severe nausea and vomiting. An antiemetic is usually taken with it. Apomorphine hydrochloride may also cause dyskinesias, injection site reactions, chest pain, dysrhythmias and spontaneous erections in some men.

Bromocriptine (Parlodel) is an oral tablet that is not often used because of it can potentially cause pulmonary fibrosis.

Monoamine oxidase B (MAO-B) Inhibitors

- Selegiline hydrochloride (Eldepryl)
- Rasagiline mesylate (Azilect)

The enzyme monoamine oxidase B (MAO-B) breaks down dopamine in the brain. MAO-B inhibitors prevent this breakdown, thus increasing the amount of dopamine in the brain and reducing the motor symptoms of Parkinson's disease.

MAO-B inhibitors used to treat the symptoms of Parkinson's disease, either alone or with levodopa. In early Parkinson's disease, they can delay the need for starting levodopa. Later in the disease, they can be given with levodopa, to enhance and prolong the effects to levodopa, thus reducing the "wearing off" phenomenon. When taken in combination with levodopa, it may allow the dose of levodopa to be reduced.

Side effects of MAO-B inhibitors include nausea, dizziness, headache, confusion, postural hypotension, hallucinations and insomnia. Confusion and hallucinations are more common in the elderly.

MAO-B inhibitors should not be used with meperidine or other opioids because of potential life-threatening drug interactions at high doses. MAO-B inhibitors at high doses could also potentially precipitate a hypertensive crisis if very large amounts of tyramine containing foods (cheese, yogurt, beer, red wine) are eaten. Serious drug interactions have been reported with MAO-B inhibitors and some antidepressants.

Selegiline and azilect are available are available as standard oral tablets. An oral disintegrating selegiline tablet (Zelpar) is also available for people who have difficulty swallowing tablets.

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Catecholamine-O-methyltransferase (COMT) inhibitors

- Entacapone (Comtan)
- Tolcapone (Tasmar)
- Carbidopa/levodopa/entacapone (Stalevo)

Catecholamine-O-methyltransferase (COMT) is an enzyme that helps to break down levodopa. For Parkinson's disease patients taking levodopa, inhibiting its breakdown results in increased levels of dopamine in the brain. The effects of the levodopa are prolonged. This can help alleviate end-of-dose "wearing off" and make it possible to reduce the dose of levodopa. COMT inhibitors are only effective in improving motor symptoms when used with levodopa. For individuals not taking levodopa, COMT inhibitors provide no benefit.

Adverse effects of COMT inhibitors include dyskinesias, orthostatic hypotension, nausea, diarrhea, sleep disturbance, urine discoloration (brown/orange) and hallucinations. COMT inhibitors may exaggerate some of the side effects of levodopa, such as dyskinesias, confusion, hallucinations and somnolence. Tolcapone has in rare cases caused liver failure, so patients taking tolcapone need regular blood tests to monitor liver function.

Entacapone is taken with each dose of levodopa. Tolcapone is generally taken three times per day, no matter how many doses of levodopa are taken daily. Carbidopa/levodopa/entacapone is a tablet that combines these three medications. It comes in several formulations.

Anticholinergics

- Trihexythenidyl (Artane)
- Benztropine mesylate (Cogentin)
- Biperiden hydrocholoride (Akineton)
- Procyclidine hydrochloride (Kemadrin)
- Ethopropazine (Parsidol)

Anticholinergics are the oldest group of medications used to treat Parkinson's disease, dating back to the 1900's. Anticholinergics decrease the activity of the neurotransmitter acetylcholine. In Parkinson's disease, the balance between dopamine and acetylcholine is disrupted. Interfering with the production or uptake of acetylcholine can help restore this balance. Anticholinergics can help reduce tremors, muscle rigidity and drooling in some patients and may also help ease dystonias, but they often do not help with bradykinesia. Anticholinergics can be used early in the course of the disease or later along with levodopa. They are generally most helpful in younger patients with Parkinson's disease whose major problem is tremor. Not all people with Parkinson's disease show improvement with anticholinergics. It is estimated that about half are helped with these medication, and the improvement may be limited.

General adverse effects of anticholinergics include blurred vision, dry mouth, constipation, urinary retention, photophobia, tachycardia, heat stroke, psychological side effects (nervousness, anxiety, confusion, depression, delusions, and hallucinations), and memory impairment. Anticholinergics should not be used with angle-closure glaucoma. Older individuals are more susceptible to confusion, hallucinations when taking anticholinergics, so these should be avoided in elderly patients. Serious reactions can occur if anticholinergics are taken with antihistamines, phenothiazines, haloperidol, or other anticholinergic medications. Patients taking anticholinergics should consult their physicians before taking any new medications, including over the counter medications. When stopping anticholinergics, they should be tapered off slowly

The two most commonly used anticholinergics for treatment of Parkinson's disease are trihexyphenidyl and benztropine, both oral tablets.

Motor symptom medications

- Amantadine hydrochloride (Symmetrel)

Amantadine is an anti-viral drug that was developed in the 1960's to treat influenza. It was found coincidentally to decrease the symptoms of Parkinson's disease and levodopa-induced dyskinesias. It is not clear how amantadine works to improve the symptoms of Parkinson's disease. It can be used alone in the early stages of Parkinson's disease, especially to help those with tremor, or combined with anticholinergic drugs or levodopa. Amantadine seems most beneficial for people with Parkinson's disease who have prominent tremor or levodopa-induced dyskinesias. The effectiveness of amantadine seems to wear off after several months for about half the patients taking this drug.

Side effects of amantadine include nausea, light-headedness, insomnia, confusion, hallucinations, anxiety, mottled skin (livedo reticularis, a lacy purple discoloration of the skin on the legs), dry mouth, constipation, orthostatic hypotension, headache and possible heart failure (noted by edema, weight gain, and shortness of breath). It should not be used on patients with angle-closure glaucoma. Patients taking amantadine should consult their physicians before taking any new medications, including over the counter medications. When stopping amantadine, it should be tapered off slowly. Stopping amantadine abruptly could precipitate a parkinsonian crisis similar to neuroleptic malignant syndrome (NMS). Symptoms include fever, rigidity, cognitive changes, and sometimes tremor, tachycardia, tachypnea, diaphoresis, dystonia and chorea. Neuroleptic malignant syndrome is potentially life threatening and requires immediate emergency care and restarting of dopaminergic medications.

Non-motor symptom medications

Medications are available to help control some of the non-motor symptoms of Parkinson's disease. Depression is a common problem in people with Parkinson's disease. Antidepressants can be used to treat depression in Parkinson's disease.

Psychosis, including hallucinations and delusions, can occur with Parkinson's disease. Psychotic symptoms can also be a side effect of some medications prescribed to treat the motor symptoms of Parkinson's disease. Reducing, stopping, and changing Parkinson's disease medications may help the psychosis. If this is not successful, physicians may prescribe atypical antipsychotics, such as clozapine and quetiapine. Clozapine can cause agranulocytosis, so blood tests must be done to monitor for this blood disorder. People with Parkinson's disease can have problems with anxiety. Benzodiazepines can be used to help manage anxiety.

Some people with Parkinson's disease develop dementia. Rivastigmine (Exelon) has been approved by the FDA for use in the treatment of dementia in Parkinson's disease.

Parkinson's disease can cause orthostatic hypotension. Some medications used to treat Parkinson's disease also have the side effect or orthostatic hypotension. Orthostatic hypotension may be managed by reducing anti-hypertensive medications if the patient is taking these drugs. Reducing, stopping,

and changing Parkinson's disease medications may help the orthostatic hypotension. If this is not successful, physicians may prescribe medications such as fludrocortisone.

Daytime sleepiness can be a problem for some people with Parkinson's disease. Somnolence can also be a side effect of some Parkinson's disease medications. Modafinil (Provigil), a drug used for narcolepsy, may help with daytime sleepiness related to Parkinson's disease.

Erectile dysfunction can occur in men with Parkinson's disease. Medications for impotence may be helpful, although they can have the side effect of orthostatic hypotension.

Nursing Responsibilities

Nursing responsibilities related to Parkinson's disease medication management include ensuring that medications are taken as prescribed, monitoring effectiveness of medications, reporting any adverse reactions or side effects, and reviewing all medications (including non-prescription) as needed with physician and pharmacist to avoid potentially dangerous drug interactions.

Management of Parkinson's disease using medications is very individualized. No two people respond exactly the same to a specific medication at a particular time in the course of the disease.

Medications may not completely alleviate the symptoms of Parkinson's disease. Patients, caregivers and health care professionals need to work together to monitor drugs and their effectiveness, and balance this against side effects of the medications. It takes time, effort and patience to find the best dose of the most appropriate medicine to manage the symptoms of Parkinson's disease.

As the disease progresses and symptoms or medication side effects change, the medication management must be continually re-evaluated. For the individual with Parkinson's disease, understanding medications, taking them as scheduled, and keeping track of symptom improvement and side effects can be very challenging. However, this effort will help people with Parkinson's disease derive the most benefit from their medications.

Medications used to treat Parkinson's disease may interact with other medications, vitamins, herbal supplements and over-the-counter cold medicines. It is important that people with Parkinson's disease talk with their physician when considering starting any new medication or supplement to avoid potentially dangerous interactions. It would be advantageous to have all prescriptions filled at the same pharmacy, so the pharmacist can review medications, to help avoid potential interactions between medications.

Medications taken to treat Parkinson's disease should not be stopped abruptly. When medications are discontinued, it is usually done by gradually tapering the dose lower. Patients should not change the dose of a Parkinson's disease medication without first discussing it with their physician. Abrupt withdrawal of medications can cause serious side effects, including the possibility of neuroleptic malignant syndrome. Symptoms include fever, rigidity, cognitive changes, and sometimes tremor, tachycardia, tachypnea, diaphoresis, dystonia and chorea. Neuroleptic malignant syndrome is potentially life threatening and requires immediate emergency care and restarting of dopaminergic medications.

Recent studies have implicated several Parkinson's disease medications, most notably dopamine agonists, in the development of compulsive behaviors in some patients. The patients develop uncontrollable urges such as compulsive gambling, shopping, eating and sexual urges. Families and caregivers should be alerted to this possibility, so they can monitor behavior and report any such adverse effects. Medications can then be adjusted, and these behaviors are usually reversible.

Supportive Therapies

Supportive therapies, including physical therapy, an exercise program, gait training, speech therapy and occupational therapy can be beneficial for people with Parkinson's disease. Physical therapy and an exercise program can improve strength, mobility and flexibility, helping with gait and balance. Exercise can also improve emotional well-being. Exercise can be accomplished through either a structured program or through individual physical activities such as walking, swimming, calisthenics, exercise machines, gardening, etc. People with Parkinson's disease should check with their physicians before starting any new exercise program. Gait training can present new methods of standing, walking and turning that help maintain balance. Speech therapy can address voice disorders and swallowing problems. Occupational therapy can help with the activities of daily living as Parkinson's disease progresses. Physical therapy and occupational therapy can help with adaptive equipment and assistive devices as needed. Dieticians or nutritionist may be helpful if there are problems with inadequate nutrition or fluids.

No special diets or natural foods have proven to slow down the progression of Parkinson's disease. Herbal remedies and dietary supplements, which are not regulated by the Food and Drug Administration, could interact with Parkinson's disease medications and cause serious problems for people with Parkinson's disease. They should not be added to the diet without first discussing them with the physician.

Studies of complimentary therapies such as massage therapy, yoga, tai chi, meditation, music therapy, hypnosis, acupuncture and Alexander technique are very limited. They suggest possible mild benefits for some people with Parkinson's disease, but there is no scientific proof that they improve symptoms of Parkinson's disease.

Surgery

In the 1950's-60's, brain surgeries were done on people with severe Parkinson's disease to try to reduce symptoms. Small lesions were made in the globus pallidus (pallidotomy) or thalamus (thalamotomy), areas of the brain that were thought to be overactive in Parkinson's disease. With the introduction of the medication levodopa in the 1960's to control the symptoms of Parkinson's disease, pallidotomy and thalamotomy surgeries were largely stopped.

In the 1990's a new procedure called deep brain stimulation (DBS) was developed for the treatment of advanced Parkinson's disease with motor fluctuations and dyskinesias. In DBS, electrodes are surgically placed into a specific area of the brain (usually subthalamic nucleus or globus pallidus), guided by magnetic resonance imaging (MRI) brain scans and neurophysiologic mapping.

The procedure is done either unilaterally, if motor symptoms effect only on one side of the body, or more often bilaterally. A battery operated pulse generator, similar to a cardiac pacemaker, is surgically implanted under the clavicle (collarbone). The brain electrodes and the pulse generator are connected by a wire placed under the skin. Electrical impulses from the generator travel to the brain and stimulate the targeted cells.

It is thought that this electrical stimulation blocks the brain's abnormal electrical signals in the targeted area that cause the motor symptoms of Parkinson's disease. The pulse generator can be programmed externally by the physician to deliver the electrical impulses that best control the motor symptoms

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of the individual patient. The battery for the pulse generator typically lasts 3-5 years and can be replaced under local anesthetic.

Deep brain stimulation is not appropriate for all people with Parkinson's disease. It is usually considered only when a Parkinson's disease patient has tried medical therapies for at least five years and motor symptoms cannot be controlled with medications. Parkinson's disease patients with intractable tremors, significant drug-induced motor fluctuations or serious dyskinesias may be candidates for DBS. Motor symptoms must be responsive to levodopa for DBS to be beneficial; those patients whose symptoms were never responsive to levodopa would likely not benefit from DBS. Although DBS can be very effective for tremor, bradykinesia and rigidity, it does not help with the non-motor features of Parkinson's disease. Patients with dementia or psychiatric problems are not appropriate candidates for surgery, as confusion, cognitive decline and mood problems can worsen with DBS surgery.

Most (70%) Parkinson's disease patients undergoing DBS show improvement in motor symptoms. Sometimes the improvement can be dramatic. Tremors, "wearing off", and drug-induced "on/off" motor fluctuations and dyskinesias can be decreased. Most patients need to continue Parkinson's disease medications, but at a lower dose, which helps reduce motor fluctuations and dyskinesias caused by long-term use of high doses of levodopa. The benefits of DBS are reported to last 5-10 years.

Potential complications of DBS include increased intracranial pressure, stroke, seizures, hemorrhage, infection and equipment malfunction. In addition, cognitive impairment and psychiatric problems have been reported in some patients undergoing DBS. A decline in verbal fluency is the most often reported cognitive problem. Mood changes appear to be relatively rare, but there have been reports that DBS may worsen depression and increase the risk of suicide.

It is important to remember that DBS does not cure Parkinson's disease. Rather, it is a tool to help manage the motor symptoms of this chronic, progressive neurological disease when medications are no longer effective and side effects of the medications are presenting serious problems. For some people with advanced Parkinson's disease, DBS can significantly improve their quality of life for many years.

Research into new surgical therapies for Parkinson's disease is ongoing. Neurotransplantation studies, transplanting dopamine producing neurons into the brain striatum of Parkinson's disease patients, met with disappointing results. Although there were initial mild benefits in some young patients, these improvements disappeared after a year, and patients developed severe dyskinesias, even off all medicine. Other areas of research that may present new surgical therapies for Parkinson's disease in the future include stem cell research and gene therapy research.

Complications

Complications of Parkinson's disease include those that result from Parkinson's disease progression, those that are experienced as side effects of treatments for Parkinson's disease, and those that result from the disabilities caused by Parkinson's disease.

As Parkinson's disease progresses, both motor symptoms and non-motor symptoms may become more problematic. Individuals with advanced Parkinson's disease may be unable to walk, unable to feed themselves, and have severe difficulties with communication. They may have dystonia, pain, urinary and bowel problems, depression, dementia, or psychiatric problems.

Acute akinesia is an uncommon but potentially life threatening complication of Parkinson's disease. Acute akinesia is a sudden deterioration in motor function, a freezing or inability to move that lasts more than 48 hours despite treatment with medications. It can be triggered by illnesses, infections, surgery or medication changes.

People with Parkinson's disease have a higher risk for developing melanoma compared to the general population. Why this occurs is unknown.

Over years of treatments, Parkinson's disease medications can lose their efficacy, while the side effects or complications increase. Dyskinesias and "on/off" motor fluctuations can become more problematic. Some medications can cause hallucinations, compulsive behaviors, daytime drowsiness and sleep attacks. It is estimated that 50% of Parkinson's disease patients experience treatment complications after five years of treatment, and 80% after 10 years of treatment. Complications of medication therapy are continually addressed by adjusting medication type, dose, scheduling and sometimes surgery.

Advancing Parkinson's disease presents new potential complications due to disabilities that it creates. Because of decreased mobility, people with Parkinson's disease, both men and women, are at risk for osteoporosis, especially if they have problems walking. Because of problems with gait and balance, they are at high risk for falls and broken bones. Because of difficulties chewing and swallowing, they are at increased risk for malnutrition, dehydration, constipation and choking. There is an increased risk for aspiration, which together with immobility increases the risk for pneumonia. Some individuals with Parkinson's disease may have problems with urination, which together with decreased fluid intake can increase the risk of urinary tract infections. People with advanced Parkinson's disease may difficulty changing positions in bed or chair. This, together with poor nutrition and possible urinary incontinence can lead to skin breakdown and pressure ulcers. Infection is a common cause of death in people with Parkinson's disease.

Nursing Assessment and Interventions

Managing Parkinson's disease is a collaborative effort involving the patient, the patient's family or caregiver, and the health care team. On the health care team, the role of the nurse is to provide education about Parkinson's disease, to help monitor medication management, and to help the patient maintain optimal functioning with as much independence as possible.

Parkinson's disease is a complex multisystem disease. Symptoms vary from one person to the next. For each individual, symptoms change over time as the disease progresses. The nursing assessment is an ongoing process to monitor the symptoms of the disease and the effectiveness of treatment in managing these symptoms.

Assessment includes a history of symptoms; a physical assessment; and a specific review of medication effectiveness or side effects.

History of symptoms includes the following possible problems: mobility problems, fatigue, sleep problems, eating/swallowing problems, weight gain or loss, falls, episodes of fainting or feeling light-headed, pain, muscle cramps, constipation, urinary problems, sexual dysfunction, excessive sweating, skin problems, restless leg syndrome, depression or other mood changes, hallucination or delusions, and behavior changes.

A nursing physical assessment includes evaluation of overall appearance, weight, affect, facial expression, drooling, tremor (presence or absence, and parts of body affected), muscle rigidity, posture, gait, coordination, speech, skin, scalp, mental status.

A medication review addresses the effectiveness of the specific medication in controlling the symptoms of Parkinson's disease, including wearing off, on-off motor fluctuations, and any side effects of medications.

Based on the nursing assessment and medication review, nursing diagnoses are made and appropriate nursing interventions initiated, tailored to the individual patient's specific needs. Some of the more common nursing diagnoses and interventions are discussed below.

Nursing Diagnosis

Knowledge deficit related to Parkinson's Disease

Assess the patient's knowledge of Parkinson's disease. Provide pertinent information about Parkinson's disease and its treatment as the patient and family are ready to learn. Helping patients and their families understand Parkinson's disease enables them to help manage their illness.

Knowledge deficit related to anti-Parkinson medications

When a medication is prescribed for treating the symptoms of Parkinson's disease, assess the patient's and caregiver's knowledge of the medication. Review the purpose, the dose and schedule for taking the medication, the side effects and any special considerations regarding the specific medication. See Treatments section, Medications for a review of medications used in Parkinson's disease.

Impaired physical mobility

In Parkinson's disease, the nursing assessment may note tremors, rigidity, and bradykinesia; problems with walking, coordination, posture and balance; and limited joint range of motion. Patients can be taught techniques to help with standing up, walking and turning. Recommend patients sit in chairs with backs, arms and high seats. To get out of the chair, it can be helpful to sit toward the edge of the seat, place hands on the arm supports, move feet back somewhat, lean slightly forward, rock forward and back, and then stand.

In the bathroom, raised toilet seats can be helpful. If initiating walking is a problem ("freezing"), several techniques can be suggested. rocking side to side, marching in place, singing/humming a marching song, tapping the leg/hip to be moved, bending at the knees and then straightening up, or raising the arms in a quick short motion.

Patients should never be pushed. If the patient is unable to walk forward, stepping back or to the side first can sometimes initiate movement, and the patient can then walk forward. To improve walking, the patient can be taught to use a wide-based gait, swing arms at sides, lengthen the stride, intentionally pick up feet with each step, and lift toes so the heel of the foot touches the floor first.

When turning, suggest that the patient walk in a wide arc, as twisting and pivoting to turn can be difficult. Encourage an upright posture, looking ahead and not down.

Encourage regular activity, exercise and ambulation. Suggest referral to physical therapy, to develop an individualized exercise program. Exercise can help maintain muscle tone, strength, flexibility and balance, which can improve gait. Specific gait training can be done. Exercise can also contribute to emotional well being. Schedule and pace activities so there are rest periods in between, as people with Parkinson's disease may become easily fatigued.

When patients are no longer able to exercise independently, caregivers can help patients ambulate and can provide range of motion exercises to prevent contractures.

Suggest mobility aids (such as canes, walkers) as needed to help facilitate movement and maintain balance.

Impaired verbal communication

With Parkinson's disease, speech volume diminishes and muscular control declines. Nursing assessment may note speech that is quiet, slow, slurred, stammering, poorly articulated, and monotonous tone.

Allow patients with Parkinson's disease enough time to express themselves, do not hurry them. Suggest referral to a speech therapist, who can develop an individualized program of face, tongue and breathing exercises to improve speech.

Allow patients with Parkinson's disease enough time to express themselves

Face exercises may include opening and closing the mouth slowly, then fast; smiling widely and holding, then puckering mouth and holding; pressing lips tightly together. Tongue exercises may include sticking the tongue out and holding it, moving it side to side, moving it in a circle, moving it in and out of the mouth quickly; stretching the tongue to try to touch nose, chin and each cheek.

Encourage patients to read or sing out loud, practicing increased voice volume. As Parkinson's disease progresses and communication becomes more difficult, suggest caregivers develop an individualized method of communicating, such as flash cards, pointing to objects, etc.

Impaired swallowing

With Parkinson's disease, swallowing problems develop. The nursing assessment may note drooling, choking, and regurgitation through the nares. The assessment should consider swallowing problems with solid food, liquids, and pills. An upright position when eating and drinking reduces the risk of choking and aspiration. Small bites of food may be easier to swallow. If thin liquids are difficult to swallow, thickened fluids may be easier to swallow. During meals, allow sufficient time to chew and swallow food. For drooling, encourage an upright head position and active swallowing. Speech therapy exercises (above) for the face, tongue, lip and jaw can also help swallowing. A swallowing consultation, usually an adjunct of speech therapy, can develop an individualized swallowing regimen with techniques and exercises that improve swallowing and help prevent choking and aspiration.

Imbalanced nutrition, less than body requirements

In Parkinson's disease, tremors, bradykinesia, and impaired chewing and swallowing contribute to imbalanced nutrition, with fewer calories than the body requires. The nursing assessment may note declining food intake and weight loss.

Monitoring weight weekly is recommended. The ongoing nursing assessment also evaluates the patient's ability to feed himself/herself, helping to determine when family/caregiver supervision as needed.

The patient should be in a sitting position when eating or drinking. A good general diet high in fiber is recommended, if the patient is able eat such foods. Soft or pureed foods may be easier to swallow.

Recommend taking small bites of food, then swallowing twice or more after each

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bite. Thin liquids may be difficult to swallow and cause aspiration; thickened liquids may be easier. Allow sufficient time for meals. Small frequent meals may be beneficial.

Teach patients and caregivers eating techniques that may decrease tremor (such as holding a piece of bread in the hand not holding the eating utensil). Using stabilized dishes and utensils or other adaptive equipment for eating can be helpful, and occupational therapy may be a good resource for patients and families.

High calorie supplements between meals may be appropriate. A referral to a dietician may be helpful for recommendations on meeting nutritional and caloric requirements and providing food of the most appropriate consistency. A swallowing consultation may be helpful if the patient has swallowing problems (see Impaired swallowing).

Constipation

Parkinson's disease can cause decreased gastric motility. This, along with mobility problems and difficulty swallowing predisposes patients to constipation. The nursing assessment identifies the typical bowel habits for individual patients.

Suggest that the patient or family monitor bowel habits. Help establish a regular bowel routine by encouraging a diet that includes fresh fruits and vegetables, supplemental fiber if necessary and adequate fluids, generally 2 liters per day unless the patient has a fluid restriction for some other health reason.

Because decreased mobility can contribute to constipation, exercise and activity are encouraged.

Stool softeners or laxatives are sometimes prescribed to aid bowel elimination, but long term laxative use is not generally recommended.

Disturbed sleep pattern

Both Parkinson's disease itself and some of the medications used to treat Parkinson's disease can contribute to a disturbed sleep pattern. In the nursing assessment, review the patient's sleep history to understand what kind of sleep problems the patient may be experiencing, such as problems falling asleep or frequent nighttime awakening.

Difficulty changing position in bed can make falling asleep difficult. Pain, anxiety or depression can also interfere with sleep.

Suggest limiting caffeine and alcohol, as they can interfere with sleep. Recommend that patients avoid napping during the day, especially in the afternoon or evening.

Exercise during the day can help aid sleep at night, but avoid strenuous activity in the evening.

Review the patient's medication schedule to see if any evening medications might be interfering with sleep, and review such medications with the physician, to see if schedule changes are possible that would help promote sleep.

Suggest nighttime practices that may promote sleep, such as drinking a glass of milk before bed to help fall asleep more quickly; taking a warm bath or massage to help relax and induce sleep; ensuring the bedroom is dark and quiet.

If the individual with Parkinson's disease has a sleep disorder, other family members may also have disrupted sleep, which can lead to increased family stress. Encourage family members whose sleep is impacted by the Parkinson's disease patient to be part of the discussion on improving sleep.

Risk for falls and injury

With Parkinson's disease, bradykinesia, rigidity and tremors can lead to gait problems and a risk for falls and injury.

The nursing assessment includes evaluating the patient's movement and gait and understanding his/her home environment. Improving movement and gait can reduce the risk of falls (see Impaired Mobility).

To promote safety and avoid falls, suggest removing potential hazards in the home, such as loose rugs or obstacles in pathways. Recommend handrails on both sides of stairways, non-skid surfaces and grab rails in showers and tubs, and raised toilet seats. Suggest that patients with Parkinson's disease wear appropriate shoes with smooth, non-slippery soles. Encourage the family or caregiver to supervise ambulation, as needed. A bedside urinal or commode may be helpful for some patients to prevent nighttime falls.

Some patients with Parkinson's disease experience orthostatic hypotension. If patients become dizzy when moving from a lying to a standing position, teach them to rise slowly, to remain seated for a few minutes with legs dangling over the side of the bed and flex feet upward, to then stand but sit back down immediately if they feel dizzy. Recommend having someone with them when they stand up.

Review medications, as some anti-Parkinson's drugs may cause dizziness; these can be discussed with the physician to see if alternative medications might be beneficial.

Self care deficiency (bathing, dressing, grooming, toileting)

The motor symptoms of Parkinson's disease interfere with all activities of daily living. The nursing assessment includes monitoring the patient's ability to carry out activities of daily living. Allow sufficient time for the patient with Parkinson's disease to carry out his/her activities of daily living. Encourage the patient to be as independent as possible.

As Parkinson's disease progresses, suggest a consultation with occupational therapy for patient and caregiver, to address techniques and adaptive devices for bathing, dressing, grooming, eating and toileting which allow the patient maximum independence.

Ineffective coping

Coping with chronic, progressive illness such as Parkinson's disease is very challenging for patients and families. The nursing assessment identifies the patient's and the family's coping skills and resources. Encouraging active participation in the health management can help empower the patient and family. Teach patients and families can about Parkinson's disease and its management (see Knowledge deficit). Encourage the patient and family to set realistic goals for symptom management.

Suggest not only following the medication schedule, but also maintaining a diary of medication effectiveness and side effects, to help achieve optimal symptom management. Encourage patients and families to report any new symptoms or problems.

Help patients and families build a good working relationship with their health care team, to best address and resolve new issues that arise with the progression of Parkinson's disease. Encourage open communication about all aspects of Parkinson's disease, including their feelings about the disease.

Evaluate both patient and caregiver stress. Provide emotional support and encouragement. Refer patients and caregivers to community Parkinson's disease resources that can provide information about Parkinson's Disease and reinforce what other patients and families have learned from their health care providers. Several community resources are listed at the end of this course.

Resource organizations can also help connect patients and families with Parkinson's disease support groups. Support groups share information, experience, and the emotional support of those who are living

with the same disease, which can also help families cope with Parkinson's disease. In some situations, individual and family counseling may be helpful.

Additional nursing diagnoses

Nursing diagnoses for Parkinson's disease are developed based on the individual situation of each patient. With the progression of Parkinson's disease and a change in the patient's clinical status, additional nursing diagnoses with appropriate assessments and interventions may be developed. Some potential additional nursing diagnoses include:

- Deficient knowledge related to deep brain stimulation
- Disturbed body image
- Chronic low self esteem
- Urinary retention or incontinence
- Altered sexuality pattern
- Chronic confusion
- Social isolation
- Ineffective airway clearance, risk for aspiration and pneumonia
- Impaired skin integrity

There is much research in progress on Parkinson's disease. Scientists are trying to better understand the cause of Parkinson's disease and develop new therapies to manage its symptoms, with the goal of eventually curing Parkinson's disease. As the research moves forward, nurses will be involved in educating patients and families about new findings, monitoring the effectiveness of new therapies, and continuing to help people with Parkinson's disease maintain optimal functioning with as much independence as possible.

Community Resources

National Institute of Neurological Disorders and Stroke, National Institutes of Health (NIH)
PO Box 5801
Bethesda, M. 20824
http://www.ninds.nih.gov/disorders/parkinsons_disease
Tel. 301-496-5751, 800-352-9424

American Parkinson Disease Association
135 Parkinson Avenue
Staten Island, N. 10305-1425
http://www.apdaparkinson.org
Tel. 718-981-8001, 800-223-2732, Young Onset Center 877-223-3801

National Parkinson Foundation
1501 NW 9th Avenue
Bob Hope Road
Miami, F. 33136-1494
http://www.parkinson.org
Tel. 305-243-6666, 800-327-4545

Parkinson Disease Foundation
1359 Broadway, Suite 1509
New York, NY 10018
http://www.pdf.org
Tel: 212-923-4700, 800-457-6676

Parkinson Alliance
PO Box 308
Kingston, N. 08528-0308
http://www.parkinsonalliance.org
Tel: 609-688-0870, 800-579-8440

Michael J Fox Foundation for Parkinson's Research
Grand Central Station, PO Box 4777
New York, N. 10163
http://www.michaeljfox.org
Tel. 212-509-0995

Parkinson's Action Network (PAN)
1025 Vermont Ave. NW, Suite 1120
Washington, DC 20005
http://www.parkinsonsaction.org
Tel: 800-850-4726, 202-638-4101

The Parkinson's Institute and Clinical Center
675 Almanor Avenue
Sunnyvale, CA 94085
http://www.thepi.org
Tel: 408-734-2800, 800-655-2273

Parkinson's Resource Organization
74-478 Highway 111, No. 102
Palm Desert, CA 92260
http://www.parkinsonsresource.org
Tel: 760-773-5628, 877-775-4111

WE MOVE (Worldwide Education & Awareness for Movement Disorders)
5731 Mosholu Avenue
Bronx, NY 10024
http://www.wemove.org
Tel: 347-843-6132

Bachmann-Strauss Dystonia and Parkinson Foundation
551 Fifth Avenue, Suite 520
New York, NY 10176
http://www.dystonia-parkinsons.org
Tel: 212-682-9900

Davis Phinney Foundation
4676 Broadway
Boulder, CO 80304
http://www.davisphinneyfoundation.org
Tel: 866-358-0285, 303-733-3340

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Amantadine; Drugs.com; http://www.drugs.com/pro/amantadine.html; accessed 6/26/2013.

Apokyn; Drugs.com; http://www.drugs.com/pro/apokyn.html; accessed 6/25/2013.

Azilect; Drugs.com; http://www.drugs.com/pro/azilect.html; accessed 6/24/2013.

Bromocriptine; Drugs.com; http://www.drugs.com/pro/bromocriptine.html; accessed 6/24/2013.

Cogentin; Drugs.com; http://www.drugs.com/pro/cogentin.html; accessed 6/24/2013.

Comtan; Drugs.com; http://www.drugs.com/pro/comtan.html; accessed 6/24/2013.

Eldepryl; Drugs.com; http://www.drugs.com/pro/eldepryl-capsules.html; accessed 6/24/2013.

Kemadrin; Drugs.com; http://www.drugs.com/pro/kemadrin.html; accessed 6/25/2013.

Mirapex; Drugs.com; http://www.drugs.com/pro/mirapex.html; accessed 6/23/2013.

Neupro Patch; Drugs.com; http://www.drugs.com/pro/neupro-patch.html; accessed 6/24/2013.

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Sinemet; Drugs.com; http://www.drugs.com/pro/sinemet.html; accessed 6/18/2013.

Stalevo; Drugs.com; http://www.drugs.com/pro/stalevo.html; accessed 6/24/2013.

Tasmar; Drugs.com; http://www.drugs.com/pro/tasmar.html; accessed 6/24/2013.

Trihexyphyeidyl hydrochloride (Artane); Drugs.com; http://www.drugs.com/ppa/trihexyphenidyl-hydrochoride.html; accessed 6/24/2013

Suggested Readings

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Notes