PARKINSON'S DISEASE

INTRODUCTION

Parkinson's disease is a chronic disorder of the brain and the nervous system. It is one of the most common neurological diseases in people over the age of 60. Approximately 1.5 million Americans suffer from Parkinson’s disease, 60,000 new cases are diagnosed each year, and it is the second-most common neurodegenerative disorder after Alzheimer's disease.

Parkinson’s disease is caused by slow, progressive damage to parts of the brain and nervous system that control movement. Parkinson’s disease cannot be detected in the early stages of its development and at this time the cause, or causes of the disease are not known. Treatment can slow the progression of Parkinson's disease but there is no cure. Eventually the patient who has Parkinson’s disease will develop permanent complications and disabilities, and ambulation and self-care will no longer be possible.

_Parkinson's disease is a chronic, progressive disease of the nervous system that affects voluntary and involuntary movement._

OBJECTIVES

When the student has finished this module, he/she will be able to:

1. Identify a basic definition of Parkinson's disease.
2. Correctly identify the basic cause of Parkinson’s disease.
3. Identify the two specific causes of Parkinson's disease.
4. Identify the four most common signs and symptoms of Parkinson's disease.
5. Explain how Parkinson’s disease is diagnosed.
6. Identify three common treatments for Parkinson’s disease.
7. Identify the name of a drug that is commonly used to treat Parkinson's disease.
8. Identify a serious side effect caused by medication used to treat Parkinson’s disease.
9. Name three daily activities you will be responsible for when caring for patients with Parkinson's disease.
10. Identify an eating disorder associated with Parkinson's disease and a serious consequence associated with this disorder.

THE NERVOUS SYSTEM AND PARKINSON'S DISEASE

The nervous system consists of the brain, the spinal cord, and the nerve fibers. The nervous system is responsible for initiating and controlling all of our activities and body functions, conscious and unconscious, voluntary and involuntary.
The brain can be thought of as the command center of the nervous system. The brain sends out nerve impulses to both other areas of the brain and to the organs and muscles. The nerve impulses - which are essentially information or messages - move from one part of the nervous system to another by way of nerve fibers and the nerve impulses are the way that the brain controls both conscious and unconscious behavior. For example, breathing, heart rate, and digestion are controlled by the brain but all of these are done without our conscious control. Muscle movements such as walking are done consciously.

The “messages” that control and regulate conscious and unconscious behavior travel along nerve fibers, and the nerve fibers are similar to wires in an electrical system. Much as electrical wires carry current the nerve fibers carry messages from the brain but unlike electrical wires, nerve fibers are not continuous; this is an important point. At various intervals along the nerve fibers and at the point where nerve fibers meet organs and muscles there is a break, a gap called the synapse.

The nerve impulses by themselves cannot cross the synapse; the transmission must be done by a neurotransmitter. Neurotransmitters are specialized substances that are made and stored in nerve fiber endings. When a nerve impulse reaches a synapse, a neurotransmitter is released into the synapse and effectively “carries” the nerve impulse to the continuation of the nerve fiber or to an organ, ie, the heart, the lungs, or a muscle. There are many different neurotransmitters such as acetylcholine, epinephrine, and serotonin. The neurotransmitter that is involved in Parkinson’s is called dopamine, and Parkinson’s disease causes irreversible damage to specific parts of the brain that depend on dopamine.

WHAT CAUSES PARKINSON'S DISEASE?

Parkinson’s disease is characterized by progressive, irreversible neurologic changes and by two basic pathologic processes: damage to the substantia nigra and the formation of Lewy bodies.

The substantia nigra is an area of the brain that helps controls involuntary and voluntary movement. The substantia nigra uses the neurotransmitter dopamine to transmit the nerve impulses that initiate and inhibit movement, but Parkinson’s disease destroys the cells in the substantia nigra that make dopamine. Because of this absolute or relative lack of dopamine nerve signals from the substantia nigra cannot cross synapses or can only do so weakly and ineffectively, so the intended movements either cannot occur or are weak and ineffective. In a sense, the loss of dopamine in Parkinson’s disease represents a “broken connection” between the part of the brain that helps control movement and the involuntary and voluntary muscles that carry out movement.

The other pathologic process that occurs in Parkinson’s disease is the formation of Lewy bodies. Lewy bodies can best be described as clumps of abnormal proteins that accumulate inside brain cells and interfere with normal cell functioning. The Lewy body protein clumps (Which are also present in other neurological disorders such as Alzheimer’s disease) in patients who have
Both of these pathologic changes have been well documented and although it is not completely understood how they are responsible for the clinical presentation of Parkinson’s disease, it seems clear that damage to the substantia nigra and the formation of Lewy bodies are the primary reasons for the signs and symptoms. However, how this damage happens, why it happens and who is at risk is not clear and at this time the specific cause or causes of Parkinson’s disease are not known. Age is by far the biggest risk factor for Parkinson’s disease and after age 60 the chances of developing the disease increases dramatically. Yet the great majority of the elderly do not develop Parkinson’s disease so other factors are clearly involved, and most of the evidence suggests that a combination of genetic and environmental factors is the cause of Parkinson’s disease.

- Genetic factors: The tendency to inherit Parkinson’s or a susceptibility to the disease can be inherited, but it is not clear how and for whom this is important. It has been estimated that there are over 25 genetic mutations that increase the risk for developing Parkinson’s disease and there are 15 genes that may cause Parkinson’s. However, the exact contribution of genetic factors as a cause for Parkinson’s disease is not known, and cases of Parkinson’s disease that are clearly caused by genetic mutations represent a very small percent of the total number of patients who have the disease. There is evidence that people who develop Parkinson’s disease at a relatively early age and who have a severe form of the disease may have a higher genetic risk profile.

- Environmental factors: Exposure to the heavy metals such lead and manganese has been implicated as a risk factor for Parkinson’s, as has environmental or occupational exposure to solvents and welding fumes, and chronic ingestion of well water. Perhaps the most investigated environmental risk factor is exposure to pesticides. Although it is not known how or why pesticides may increase the risk for developing Parkinson’s disease there are many studies that show an association between pesticide exposure and the disease. In some cases the risk for Parkinson’s disease is dramatically increased when exposure to pesticides is increased.

It is well accepted that Parkinson’s disease is caused by a combination of genetic and environmental factors, but exactly how the genes and the environmental exposures interact to cause the disease is not clear. This is not unusual. Many chronic diseases are thought to be partly inherited and partly the result of an environmental factor and it may be that many of us have susceptibility genes for Parkinson’s disease but we never have an exposure to a causative agent for the disease. Approximately 10% of all cases of Parkinson’s disease have a clear genetic basis but none of the environmental factors that are suspected to contribute to Parkinson’s disease has been proven to be a cause.
THE SIGNS AND SYMPTOMS OF PARKINSON'S DISEASE

The onset of Parkinson's disease is usually at 60 years of age or older and men are 1.5 times more likely to develop Parkinson's disease than women. Early onset (age 40 or younger) is unusual. Parkinson's disease develops slowly. People with Parkinson's disease may initially notice that their sleep is disturbed, that they have a slight tremor in their hands or their coordination has become worse. Depression and a lack of energy are also common complaints. In many patients these initial signs and symptoms can be mild and subtle. People can easily ignore them or decide that these changes are just part of the normal aging process. Early signs and symptoms of Parkinson's disease are also quite different than the later ones that are commonly associated with the disease. Unfortunately, there is evidence that by the time someone with Parkinson's disease begins to develop the characteristic signs and symptoms (Described below) there has already been extensive and irreversible damage to the dopamine-dependent nerve fibers.

The signs and symptoms of Parkinson's disease can be usefully separated into two categories, non-motor and motor (Motor refers to signs and symptoms involving the muscles or movement).

The non-motor signs and symptoms are usually the first to develop but they can also persist as the disease progresses. Examples of non-motor signs and symptoms are listed in Table 1. Many of these are quite common in Parkinson's disease, but signs and symptoms such as constipation, sleep disturbances, and urinary urgency are non-specific: they can be caused by many diseases, and it is the motor signs that are the hallmark of Parkinson's disease. However, as previously mentioned, the non-motor signs and symptoms are no less serious than the motor signs; they are simply less visible and dramatic.

Table 1: Non-Motor Signs and Symptoms of Parkinson's Disease

| Changes in voice, ie, decreased tone and volume |
| Constipation |
| Depression |
| Decreased facial expressions |
| Decreased sense of smell |
| Dementia |
| Excessive salivation |
| Forgetfulness |
| Orthostatic hypotension |
| Sexual dysfunction |
| Sleep disturbances |
| Speech disturbances |
| Sweating |
| Tremor |
| Urinary urgency |
| Weakness |
There are many motor signs associated with Parkinson’s disease, but there are four that are considered to be the cardinal signs of Parkinson’s: bradykinesia, muscular rigidity, postural instability, and resting tremor. Each of these will be explained in detail and the neurological tests that are used to detect their presence and evaluate their severity will be explained, as well.

**Bradykinesia** is a medical term that means slow or decreased speed of movement. Bradykinesia is one of the most common signs of Parkinson’s disease. Patients who have Parkinson’s disease with bradykinesia are unable to quickly and completely make voluntary movements and because of this, the activities of daily living such as eating, dressing, and personal hygiene take much, much longer. In addition, bradykinesia affects the patient’s ability to *complete* voluntary movements *and* many patients have a decreased number of spontaneous movements. The last point is illustrated by imagining yourself sitting in a chair or on a couch. When you do so you are often crossing your legs, shifting from side to side, and adjusting your position but the patient who has Parkinson’s disease has a noticeable lack of these spontaneous adjustments of position and posture.

**Muscular rigidity** is simple to understand: the patient who has Parkinson’s disease has stiff and rigid muscle tone. However, unlike bradykinesia, postural instability, and tremor, muscle rigidity is not a sign that is visible. Muscular rigidity can only be detected by an examiner performing passive range of motion of the patient’s arms and legs.

**Postural instability** is a very dramatic sign of Parkinson’s disease and it is one of the most serious consequences of the disease, accounting for many falls and injuries. Maintaining a stable posture, especially while walking, is an intricate activity that requires coordination of muscles, joints, nerves, and reflexes. Each time you take a step you must temporarily balance on one leg, move the other leg a certain distance forward, plant your foot, shift your weight, bend your knees, flex your ankles, accommodate changes in the walking surface, and hold your spine in a certain position. Walking and maintaining a normal posture seem very simple but when they are closely examined it’s clear they are quite complex and the damage to the substantia nigra from Parkinson’s disease causes postural instability - patients do not have the muscle control to maintain normal posture while walking. Postural instability is a late sign of Parkinson’s disease; once it has developed the patient is typically in an advanced stage.

**Resting tremor** is defined as a rhythmic, involuntary shaking at rest that stops during movement. A patient with Parkinson’s disease who has resting tremor will have typically have the tremor in one arm or hand but over time the resting tremor will happen to other parts of the body. The tremor may only happen when the patient is stressed or tired and it can be very slight or quite exaggerated. One of the resting tremors often associated with Parkinson’s disease is called pill rolling tremor. Pill rolling tremor is characterized by the tip of the thumb and the tip of the forefinger being constantly rubbed together, a motion that is similar to someone who is feeling the texture of something.
Testing for the Presence and Severity of Motor Signs and Symptoms

There are many ways to assess for the presence and severity of Parkinson’s disease. Many clinicians use a series of tests called the Unified Parkinson’s Disease Rating Scale (UPDRS). The UPDRS assesses: 1) a patient’s mental health and mood; 2) her/his abilities at performing activities of daily living, and; 3) the patient’s motor skills. The complete UPDRS is somewhat lengthy and it will not be included here; interested readers can find many websites available at which they can view the UPDRS. Some of the tests from the UPDRS are described below; these are ones that are used to determine the presence and severity of the four cardinal signs of Parkinson’s disease.

- Bradykinesia: There are many tests that are used to detect the presence of bradykinesia and evaluate its severity. Two of these are explained here, and performing them yourself will help you understand how the severity of Parkinson’s disease is assessed and also why it affects the activities of daily living. The first is the rapid alternating hand movement and the second is leg agility. The **rapid alternating movements of the hands** test is done by having the patient hold out both his/her hands, fingers together and with the thumbs pointing up. The patient is then asked to quickly rotate and change the position of his/her hands in the following way: palms facing down, return to the starting position of the hands vertical, palms down again, return to the starting position and so on. You will be able to do this quite rapidly, at least one movement per second. The person who has Parkinson’s disease can only move the hands slowly and with hesitation and occasionally she/he will stop midway through the movement. The **leg agility test** is performed by having the patient sit in a chair, lift one leg up 3 inches from the ground, tap the floor with her/his heel, and repeat. Again, you will be able to perform this test rapidly and easily. The patient who has Parkinson’s disease may not be able to lift the leg 3 inches or the up and down movement will be slow and will start and stop. Obviously these tests are not realistic in what they require the patient to do, but they illustrate how the bradykinesia of Parkinson’s disease prevents normal movements. Bradykinesia is also assessed and evaluated by forming a general impression of the patient’s body movements, paying special attention to see if the movements are slow, hesitant, interrupted, or the range of motion is decreased.

- As mentioned previously muscular rigidity can only be detected by an examiner performing passive range of motion of the patient’s arms and legs. One way of doing this is to hold the patient’s elbow in the palm of the hand, grasp the patient’s wrist with the other and then bend the arm back towards the shoulder - arm flexion. In someone who does not...
have Parkinson’s disease the arm flexion movement will be smooth, fluid, and easy. If the patient has Parkinson’s disease the arm flexion movement will be slow, stiff, and difficult because of muscular rigidity. The results are rated from no difficulty in flexion (Score 0) to severe (Score 4) in which flexion is achieved only with significant difficulty.

- Postural instability is assessed by examining the patient’s gait. It can also be assessed by the pull test. The patient is standing and the examiner is positioned behind the patient. The examiner puts her/his hands on the patient’s shoulders and then pulls backwards, quickly and suddenly on the patient’s shoulders. If the Parkinson’s disease is advanced the patient will lose his/her balance and will not be able to stay erect.

- There is no specific test for resting tremor nor does there need to be one as it can easily be observed. In patients who Parkinson’s disease the tremor can be mild or quite severe. In patients who have advanced disease the tremor can be very significant; the patient’s hands may flap uncontrollably and continually and the tremor will prevent self-care activities such as feeding.

It is not critically important that readers have a detailed knowledge of how Parkinson’s disease is assessed and evaluated. These tests are described so that CNAs will have an understanding of the impairments and limitations caused by Parkinson’s disease. This understanding can be furthered by performing these tests yourself or with a co-worker. This can be very helpful when you are working with a patient who has Parkinson’s disease

**The Progression of Parkinson’s Disease**

Parkinson’s disease is a chronic disease and it is progressive. The patient’s condition slowly worsens over time and the disease is typically described as having five stages.

Stage 1: Patients at this stage have mild signs and symptoms but they are prominent enough that the patient, family, and friends can notice the changes and the activities of daily living may be affected. Tremor is present (But only on one side) there may be voice changes (The patient’s voice is noticeably softer), gait abnormalities are possible, and muscular rigidity and some level of bradykinesia are usually present. However, at this stage the level of impairment is often manageable and the patient may not seek help. Also, because Parkinson’s disease usually affects people who are 60 years and older the first stage of Parkinson’s may be dismissed as normal aging.

Stage 2: The tremor, bradykinesia, and muscular rigidity become worse and bilateral in nature. The patient may have a decreased ability to change facial
expressions, ie, the “mask-like” facial feature that is common to Parkinson’s disease. The patient’s posture may become stooped. Performing activities of daily living becomes more challenging, and walking and maintaining balance are difficult.

Stage 3: The decrease in motor abilities continues. The patient’s movements are noticeably slow. She/he cannot walk in a straight line. Maintaining balance is difficult and at this stage the patient will have an abnormal pull test so falls are possible. However, despite these difficulties the patient should still be able to complete activities of daily living and perform self-care.

Stage 4: At this stage the impairments in motor abilities are so severe that the patient cannot function independently. Walking unassisted is still possible but the patient is not able to live alone.

Stage 5: The patient cannot walk unassisted and cannot perform self-care; he/she is dependent on other for eating, dressing, hygiene, etc. Parkinson’s dementia may begin at this stage and this complication affects approximately 20%-40% of all patients who have the disease. People who have reached this stage of Parkinson’s disease require constant one-on-one care.

There is no cure for Parkinson's disease. The progression of the disease is individualized: some patients deteriorate very quickly while for others the impairments develop slowly. Male gender, an older age at the onset of the disease, early onset of cognitive decline, and a poor response to drug therapy are signs that the patient’s condition may worsen comparatively faster. Most patients will live for 10-20 years after Parkinson’s disease has been diagnosed.

**HOW IS PARKINSON’S DISEASE DIAGNOSED?**

There is no screening test that can help physicians identify people who are at risk for developing Parkinson’s disease. Laboratory tests, x-rays, and diagnostic procedures such as CT scan or MRI may be done to make sure the patient does not have co-morbidities or if the signs and symptoms are non-specific, but there are no tests that can be reliably used to detect and diagnose Parkinson’s disease. Parkinson’s disease is diagnosed by a physician’s examination and by interviewing the patient and her/his family. If the patient has two of the four cardinal signs of the disease, bradykinesia, muscular rigidity, postural impairment, and tremor; if these signs have been progressing in intensity and frequency, and; the signs and symptoms cannot be accounted for by other diseases or illnesses - the patient has Parkinson’s disease.

Once the diagnosis has been made the patient will be evaluated for his/her cognitive abilities and level of mental health, the ability to perform the activities of daily living such as dressing, eating, speech, swallowing, toileting, walking, etc., and motor impairments; these are the three assessment categories of the UPDRS. When assessing the patient’s cognitive abilities and mental health the
clinician should pay particular attention for the presence of dementia and depression. Dementia affects approximately 20%-40% of all patients who have Parkinson’s disease, and the risk of developing dementia is increased 2-6 times by the presence of Parkinson’s disease. Depression and other mood disturbances are also very common in patients who have Parkinson’s disease and the patient should be screened for mood disorders at the time of diagnosis.

TREATMENT FOR PARKINSON’S DISEASE

The treatments for Parkinson’s disease are intended to control the signs and symptoms and maintain quality of life for the patient. Some of the available therapies are unproven and some will only be effective for a small number of patients. None will provide a cure and even the most commonly used treatment, the anti-Parkinson’s medications, will only be effective for a relatively short period of time, 4 to 6 years. Regardless of which therapies are chosen, they must be carefully tailored for each individual and the patient must be carefully monitored for side effects.

The available therapies for treating Parkinson’s disease can be divided into four categories: drug therapy; experimental therapies such as gene therapy, stem cell therapy, and transplant therapy; deep brain stimulation, and; surgery. Drug therapy is by far the most commonly used treatment and it will be discussed in a separate section. The other therapies are used much less often and they will be only briefly discussed.

Surgery

- Surgery: Surgery is an option for treating patients who have Parkinson’s disease and there are three procedures that can be done: pallidotomy, thalamotomy, and subthalamotomy. The names of the procedures describe the specific part of the brain that is operated on, but despite these differences the therapeutic goal is the same for all three procedures: the area that has been damaged by Parkinson’s disease is removed by thermocoagulation (Application of a heated probe) and hopefully this removal will provide symptomatic relief. These procedures are typically used for patients who have advanced Parkinson’s disease and serious impairments and who have not, or are no longer responding to drug therapy. They can be successful, but there are serious post-operative complications and the number of patients who are considered suitable candidates is relatively small. Because of these issues the surgical procedures are seldom done and have largely been replaced with deep brain stimulation.

- Deep brain stimulation: Deep brain stimulation (DBS) was first used in the 1970s. However, performing the procedure with the unsophisticated technology available at the time was very difficult, there were also serious complications, and DBS was essentially abandoned. Today, however, DBS is considered to be one of the most effective treatments for patients
with advanced Parkinson’s disease. DBS involves placing a wire with an electrode into the affected area of the brain. The wire is attached to an electrical pulse generator, and the generator acts in the same way as a pacemaker for the heart. It sends out a regular, intermittent electrical signal that stimulates the particular part of the brain that is no longer functioning properly because of Parkinson’s disease. Although DBS can be quite successful for the right patient it is not exactly understood how it works. However, for some patients it can be very effective and unlike the traditional surgical options it is reversible as brain tissue does not have to be destroyed. DBS is also used because the pulse can be easily adjusted for the patient’s needs, and the wire can be removed if DBS is not working or there is a complication. DBS is very expensive and because it involves surgery, infection is a possibility.

- Experimental therapy: Transplant therapy has been tried as a treatment for Parkinson’s disease, but at this point it hasn’t been very successful. Some researchers have tried to genetic manipulation and stem cell therapy is being investigated, but these approaches are still experimental.

DRUG THERAPY FOR PARKINSON’S DISEASE

Treating patients with medication is still the cornerstone of therapy for Parkinson’s disease. These drugs are very helpful, but they do not change the course of the disease, slow down its progression, or provide a cure. There are several different classes of drugs that are used, but the drugs that are most commonly used and are the most effective are the ones that provide a supply of dopamine or prevent the breakdown of dopamine. (Note: the generic name of each drug is provided and commonly used brand names are in parentheses)

- Dopamine pro-drugs: A pro-drug is a medication that is converted by the body into its active form and the pro-drugs used to treat Parkinson’s disease are converted to dopamine after they have been absorbed. Carbidopa/levodopa (Sinemet) is a dopamine pro-drug, and it is perhaps the most commonly prescribed anti-Parkinson’s medication. The levodopa is converted to dopamine; the carbidopa prevents side effects that can occur from excess conversion of levodopa to dopamine and it allows for a higher brain concentration of the levodopa. The dopamine pro-drugs are the most effective ant-Parkinson’s medication for controlling the signs and symptoms of Parkinson’s disease and they have the fewest side effects.

- Anticholinergic drugs: Anticholinergic drugs such trihexyphenidyl (Artane) and benztropine (Cogentin) work by affecting the activity of the neurotransmitter acetylcholine. The drugs can be effective at reducing the tremor associated with Parkinson’s disease; they are not effective for controlling bradykinesia or muscular rigidity. Dizziness, drowsiness, and dry mouth are common side effects of the anticholinergic drugs.

- MAO-B inhibitors: The MAO-B inhibitors rasagiline (Azilect) and selegiline (Anipryl) decrease the activity of an enzyme that breaks down dopamine
and by doing so they increase the amount of circulating dopamine. These drugs are often used in addition to Sinemet and other drugs of that class when the patient is not responding fully to the dopamine agonists. Dizziness, headache, and nausea are common side effects of these drugs.

- **Dopamine agonists:** The dopamine agonists directly stimulate the parts of the brain and the nerve fibers that use dopamine. Carbidopa/levodopa is considered a dopamine agonist and other commonly used dopamine agonist are ropinirole (Requip), pramipexole (Mirapex), and amantadine.

- **Acetylcholinesterase inhibitors:** Donepezil (Aricept), rivastigmine (Exelon), and galantamine (Razadyne) are acetylcholinesterase inhibitors that decrease the activity of an enzyme that breaks down the neurotransmitter acetylcholine. This effect increases the amount of acetylcholine in the brain and these drugs can improve cognitive functions such as memory and learning. Abdominal cramping and diarrhea are common side effects of the acetylcholinesterase inhibitors.

Patients are usually started on a dopamine pro-drug and other medications are added as needed. Drug therapy is often initially very effective at controlling the signs and symptoms of Parkinson’s disease. However, as mentioned previously the available medications do not slow the progression of the disease or provide a cure. In addition, drug therapy typically works for a relatively short period of time, 4 to 6 years, the side effects of the medications are unpleasant, and patient compliance is an issue. For example, carbidopa/levodopa can cause a serious movement disorder called dyskinesia, abnormal and involuntary muscles movements that can be distressing and uncomfortable. The acetylcholinesterase inhibitors can cause gastrointestinal distress such as abdominal cramping and diarrhea, and the anticholinergic medications can cause dry mouth, dizziness, and drowsiness.

**CARING FOR THE PATIENT WHO HAS PARKINSON'S DISEASE**

Medications and surgery are used to treat people with Parkinson’s disease, but these people need much more. Because of their disabilities, they need various degrees of help with their activities of daily living (ADL) such as eating, bathing, dressing, walking, etc. Physical therapy, occupational therapy, and speech therapy are useful, and you may be asked to accompany the patient to these therapy sessions and to help and encourage them during the sessions. But the day to day responsibility of assisting these people will be your responsibility. You will need to focus on the following areas.

- **Nutrition:** Dysphagia (difficulty swallowing) can be a sign of Parkinson’s disease. Because of this, the patient may have excessive drooling and be unable to swallow. The patient with dysphagia is at risk for malnutrition and can also aspirate food or liquid into the lungs - a very serious
problem. The patient's diet will be ordered by the physician. Following these orders is important; do not deviate from the ordered consistency (e.g., some patients can only eat soft or pureed food) or the size of each bite (some patients can only tolerate very small pieces). Never rush the patient through a meal; this increases the chance of aspiration. Use standard aspiration precautions.

- **Skin care**: People with advanced Parkinson's are less mobile and may be incontinent of urine and/or feces. Because of this, they can develop bed sores. The patient should be encouraged to move, the skin should be kept clean and dry, and if the patient is immobile, he/she needs frequent position changes.

- **Ambulation**: The patient with Parkinson's disease cannot maintain a normal posture; simply standing upright without assistance can be impossible. These people also cannot walk normally; they can only take small steps and they usually shuffle rather than walk. These gait impairments limit their independence and puts them at risk for falls. You will need to determine how much assistance these patient need when ambulating. The physical therapy department will usually provide some direction in terms of how often and for how long the patient should walk, and how to help them. Many patients find it helpful to do some slow, gentle stretching before they walk. Also, many patients with Parkinson's experience a "lag time" with physical activity. The patient may decide to move, but it takes them far longer after the decision to actually move. Help them by letting them know when it is time to ambulate, and then giving them extra time to get ready. When they do ambulate, tell them to focus and to look at each at their feet and legs and "visualize" the activity.