



A PHARMACY CONTINUING EDUCATION PROGRAM

W-F Professional Associates, Inc. 400 Lake Cook Rd., Suite 207 Deerfield, IL 60015 847-945-8050

August 2009 "Review of Parkinson's Disease" #707-000-09-008-H01-P



THIS MONTH
"Review:
Parkinson's Disease"

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HAVE YOU RECENTLY MOVED? PLEASE NOTIFY US.

Parkinson's disease is a chronic condition that is considered to be a "movement disorder." It responds to symptomatic drug therapy; therefore, our goal in this lesson is to review the medication options. This lesson provides 1.25 hours (0.125 CEUs) of credit, and is intended for pharmacists in all practice settings. **The program ID # for this lesson is 707-000-09-008-H01-P. Pharmacists completing this lesson by August 31, 2012 may receive full credit.**

To obtain continuing education credit for this lesson, you must answer the questions on the quiz (70% correct required), and return the quiz. Should you score less than 70%, you will be asked to repeat the quiz. Computerized records are maintained for each participant.

If you have any comments, suggestions or questions, contact us at the above address, or call toll free 1-800-323-4305. (In Alaska and Hawaii phone 1-847-945-8050). **Please write your ID Number (the number that is on the top of the mailing label) in the indicated space on the quiz page** (for continuous participants only).

The objectives of this lesson are such that upon completion the participant will be able to:

1. Differentiate between the classes of Parkinson's disease.
2. Discuss the mechanism of action of dopaminergic drugs & their adverse effects.
3. Define "on-off" effect of levodopa.
4. Describe the use of carbidopa in the management of PD.
5. List the most common dopamine agonists.

All opinions expressed by the author/authors are strictly their own and are not necessarily approved or endorsed by W-F Professional Associates, Inc. Consult full prescribing information on any drugs or devices discussed.

INTRODUCTION

Parkinson's disease (PD) is a chronic condition that is considered to be a "movement disorder." These diseases affect motor skills and speech, and are characterized by degeneration in the central nervous system. Such degenerative processes result in muscle rigidity, resting tremors, bradykinesia (slow physical movement) or kinesia (absence of physical movement). Parkinson's disease affects approximately 120 – 180 per 100,000 people who are older than 40 years of age. The disease has been known since the 1800s. Originally it was known as paralysis agitans. Later on, a new term, Parkinson's disease, was coined after James Parkinson described the symptoms of the disease. The biochemical process that results in its emergence was recognized in the 1950s.

CAUSES

The vast majority of PD sufferers have idiopathic PD, which means there is no identified cause. However, there are certain factors that may cause PD including: genetic factors, toxins, insecticides, intake of heavy metals and head trauma.

PATHOPHYSIOLOGY

Symptoms of PD occur as a result of loss of dopamine secreting cells which are located in the extrapyramidal system and basal ganglia corpus striatum, globus pallidum and substantia nigra (black substance). The resultant loss of dopamine can disrupt the activity of the neurotransmission within the basal ganglia that controls movement.

There are two major transmitters released in this area during transmission:

1. An inhibitory transmitter, dopamine (DA), and
2. An excitatory transmitter, acetylcholine (ACh).

Dopamine tends to inhibit discharge of striatal cholinergic neurons. In a healthy person, the excitatory effect of ACh is balanced by the inhibitory activity of DA, and normal body movement results. In PD, the involuntary movement, as well as other symptoms, results from an imbalance between the inhibitory effects of DA and the opposing excitatory effect of ACh. Degeneration of the neurons that provide DA in the extrapyramidal system and basal ganglia results in diminished production or absence of DA. Thus, the excitatory action of ACh predominates. The resultant increase in ACh-mediated excitation leads to the characteristic involuntary PD movements. Tremors are probably due to lack of inhibitory impulses in the lower ganglia. These enhance feedback in the globus pallidus-thalamus-cortical cycle, resulting in oscillation.

Essential tremor should be differentiated from PD. The only symptom that is common between the two disorders is the tremor. Essential tremor lacks all other signs and symptoms present in PD. The tremor may diminish after the intake of Beta blockers.

CLASSIFICATION

PD can be classified into three etiologic classes:

1. Primary,
2. Secondary, and
3. Parkinsonism-plus disorders.

Primary PD is due to neurodegenerative processes that mainly occur in the substantia nigra and locus ceruleus, without any apparent systemic cause even though genetic mutation may play a causative role. The gene responsible for the disorder has not been determined.

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August 2009

Secondary PD may be caused by environmental factors such as toxins (carbon monoxide, exposure to heavy metals, pesticides and smoking), infection (postencephalitic Parkinsonism), vascular effects (subdural hematoma, tumors, infarcts involving the midbrain or basal ganglia) and drugs (antipsychotics and reserpine). Except for thioridazine, which possesses strong anticholinergic activity, the antipsychotics may cause extrapyramidal side effects resulting from blockage of DA receptors in the striatum. These drug-induced symptoms may be corrected by using antiparkinsonian drugs. Reserpine tends to deplete DA in the neurons of the brain's basal ganglia. Extrapyramidal side effects produced by these drugs include:

1. Akathisia (restlessness),
2. Dystonic reactions (impaired muscle tone), and
3. Tardive dyskinesia (dopamine antagonist-induced involuntary, repetitive movements).

Parkinsonism-Plus or multisystem atrophy, occurs when PD exists along with other disorders such as progressive supranuclear palsy, corticobasal degeneration and dementia. This type may progress at a faster rate than the primary PD.

SIGNS AND SYMPTOMS

Onset of symptoms of PD may be abrupt, but the disease usually begins insidiously in 50 to 85% of patients. Clinical manifestations normally start after the age of 40, and the incidence increases as the patient becomes older. The disease is more common among males than females, and there are indications that it is hereditary. It is not directly life threatening, since there is no degeneration in the nerves that supply the heart and other organs.

As stated earlier, PD affects body movements. Tremor, which is the first symptom of PD to appear, is a fine "pill-rolling", resting tremor in the hands (rubbing together of thumb and forefinger), which becomes less intense during voluntary movement. This usually disappears during sleep. About 30% of patients experience little perceptible tremors. However, they may become more prominent during fatigue and emotional stress. Within the course of the disease, the tremors spread from the hands to the arms and legs. Other parts of the body that may be affected include the jaw, tongue and eyelids. Tremors may be limited initially to one limb or two limbs on one side. The face becomes expressionless with the mouth open. Speech is slow and drooling of saliva may occur. These symptoms are followed by gradual, progressive rigidity, bradykinesia (slow movements) and akinesia (absence of movement). Muscular pain may be experienced. Presence of tremor, rigidity and bradykinesia lead to difficulty in initiating walking, which is usually in small, shuffling steps. The head becomes bowed, body bent forward (stooped) and knees slightly bent. In later stages of PD, depression, memory loss and a decline in thought processes may occur. Other symptoms include slow reaction time, dementia, delusion, paranoia, insomnia, disturbances in REM sleep, muscular and joint pain and weight loss.

TREATMENT

Recovering from PD rarely, if ever, occurs. Drugs used in treatment provide only symptomatic relief. With one possible exception, none of the current drugs are capable of slowing progression. Thus, the therapeutic objective is to improve the patient's quality of life. Choice of treatment and dosage is usually determined by the severity of the symptoms.

Since PD symptoms occur as a result of imbalance between the neurotransmitters DA and ACh (low level of DA and high level of ACh), therapy should be focused on restoring the neurochemical balance. To achieve this, two therapeutic approaches are employed:

1. Stimulation of DA receptors (dopaminergic agents); and
2. Suppression of ACh receptors (anticholinergics). Surgery may be attempted. In addition to pharmacological therapy, family and patient education, general health improvement, maintenance of good nutrition and regular exercise and physiotherapy may assist in the management of the disease and its symptoms.

DOPAMINERGIC AGENTS

The pharmacologic basis for using these drugs is to increase the level of dopamine in the brain. This can be achieved by mechanisms such as enhancement of dopamine synthesis, direct activation of dopamine receptors, and pre-

vention of the breakdown of dopamine.

Levodopa: Levodopa (L-Dopa) is transformed into DA in the dopaminergic neurons by L-aromatic amino acid decarboxylase (formerly known as dopadecarboxylase). It is chemically identical to the naturally occurring dihydroxyphenylalanine (DOPA) and is the metabolic precursor of DA. It reduces PD symptoms by promoting synthesis of dopamine.

Levodopa is well absorbed from the GI tract; however, about 90% of the dose is converted by the enzyme L-aromatic amino acid decarboxylase, which is found in the intestinal mucosa, to dopamine. Since dopamine does not cross the blood-brain barrier well, only 10% of the levodopa dose reaches the dopaminergic neurons. Thus, the vast majority of the dose is wasted, since it does not reach the desired site of action. Because of this, the drug must be given in very large doses, causing side effects such as nausea, palpitation and flushing due to the presence of metabolized L-dopa in other parts of the body. L-dopa may result in a reduction in the formation of endogenous L-dopa. The simultaneous administration of the peripheral dopa-decarboxylase inhibitor, carbidopa, will result in increasing the effectiveness of levodopa and in the reduction of the administered dose. As a result, a significant decrease in the side effects of levodopa occurs. Levodopa is administered routinely with benserazide and carbidopa. When administered alone, carbidopa has no antiparkinsonian activity. However, when given concurrently with levodopa, it blocks the peripheral conversion of levodopa to dopamine and enhances penetration of levodopa through the blood-brain barrier. Furthermore, it increases plasma levodopa concentration, resulting in a more effective dose with lesser side effects. Levodopa is the drug of choice for PD. It is effective in managing bradykinesia and rigidity. Frequently there is a significant reduction in intensity of tremor. When compared to other dopaminergic drugs, levodopa is more effective and less expensive. It has been shown that levodopa can improve life expectancy, and in mild cases, patients may return to almost normal lifestyle. It is employed for all types of PD except those that are drug-induced. The usual starting dose alone is 0.5 g and 1 gram, given in two or more divided doses, two to five hours apart, with or after a meal. The dose should be gradually increased every three to seven days by up to 0.75g daily as tolerated, until maximum benefit is reached. This procedure tends to minimize side effects.

When given in combination with carbidopa, treatment is begun with single 10:100 or 25:100 tablets, three times daily, or every other day according to tolerance, until maximum benefit is attained. A minimum daily dose of carbidopa is required to minimize peripheral side effects. Levodopa or a combination of levodopa and carbidopa is not recommended for children.

The common initial side effects are nausea, vomiting, postural hypotension and cardiac arrhythmias. Other side effects, which may occur during therapy, are usually dose-related. They include dyskinesia (abnormal movements) in the form of oral-facial or limb dystonia, akathisia (inability to sit still) and confusion. One of the complications of levodopa therapy is the appearance of the "on-off", effect. This phenomenon, which occurs in about 50% of patients, emerges after two to five years of treatment. It is characterized by fluctuation in the patient's response to levodopa due to transient wearing-off of the dose shortly before the next dose is due. This "on-off" phenomenon is transient, but abrupt, and occurs without warning. This can result in alternating periods of intense akinesia to greater hyperactivity. Such swings can be reduced by using the lowest effective dose and a dosing interval as short as every one to two hours.

Despite the fact that levodopa is beneficial, it does not resolve many of the symptoms of PD. Motor features such as speech and balance do not normally respond to levodopa treatment and tend to worsen over time. Additionally, it has no positive effect on nonmotor Parkinsonian symptoms such as hallucination, cognitive impairment and orthostatic hypertension.

DOPAMINE AGONISTS

Dopamine agonists are not as effective as levodopa. However, when administered with levodopa, the combination tends to reduce the time of the "on-off" effect in patients who experience this phenomenon. Their activity is due to stimulation of dopamine receptors in the striatum.

Bromocriptine (Parlodel[®] ; Cytoset[®]): Bromocriptine is usually used as an adjunct to levodopa due to its direct activation of the dopamine receptors in the basal ganglia. It is beneficial when used in the later stages of PD, when the

effect of levodopa diminishes or when the "on-off" phenomenon emerges. As an adjunct to levodopa, bromocriptine can prolong the therapeutic response. The adverse effects of the drug are dose-related and may be encountered in 30 - 50% of patients. They include: nausea, confusion, hallucinations, agitation, nightmares and paranoid delusions. Bromocriptine can cause dyskinesia (difficulty to perform voluntary movements—may include development of a tic or spasm) and postural hypotension. The initial dose is 1.25 mg once or twice daily, administered with meals for one week, and 2.5 mg daily for the next week. Depending on response and tolerance, the daily dose may be increased by 2.5 mg increments every two weeks. Maintenance dosage ranges from 10 to 40 mg daily when administered concurrently with carbidopa and levodopa. It is important to assess the patient's response every two weeks to ensure that the lowest effective dosage is being employed. If bromocriptine is to be withdrawn, this should be done gradually.

Pergolide (Permax[®]): The mechanism of action, uses and adverse effects of pergolide are similar to those of bromocriptine. It is used as an adjunct to carbidopa-levodopa. It prolongs symptomatic control and reduces fluctuations in motor activity as well as the incidence of levodopa-induced dyskinesia. Its adverse effects are: nausea, postural hypotension, confusion, hallucinations, sedation and delusions. These psychological effects are more common, and dyskinesia is less common, than with levodopa. The recommended initial dose is 0.05 mg daily for two days; then increased gradually by 0.1 to 0.15 mg daily every third day over the next 12 days; then increased by 0.25 mg daily every three days until a therapeutic dose is achieved. The usual daily maintenance dose is 3 mg. The effectiveness of dosage greater than 5 mg daily has not been determined. The titration of pergolide should be accompanied by gradual reduction in the carbidopa-levodopa dosage.

Pramipexole (Mirapex[®]): Pramipexole possesses high affinity to binding and stimulating dopamine receptors in the striatum. When used concurrently with levodopa, the dose of levodopa may be decreased gradually as tolerated. Side effects include orthostatic hypotension, especially during the dose increase, hallucination, exacerbation of pre-existing dyskinesia or dyskinesia potentiated by levodopa, nausea, confusion and amnesia. The starting dose is 0.375 mg daily given with meals in three divided doses every five to seven days. This dose should be increased gradually to a daily dose of 4.5 mg. Maintenance doses usually range from 1.5 to 4.5 mg daily, administered in equally divided doses, three times per day, with or without concurrent levodopa. Discontinuation of the drug should be done gradually over a period of one week.

Ropinirole (Requip[®]): Ropinirole has similar mechanism of action, uses and adverse effects as those of pramipexole. The recommended initial dose is 0.25 mg, three times daily. Titration of the drug should be done on a weekly increment. In the first week, the recommended dose is 0.25 mg, three times daily; in the second week, 0.5 mg, three times daily; in the third week, 0.75 mg, three times daily; and in the fourth week, 1 mg, three times daily. In some cases, the dose may be increased to 1.5 mg daily after the fourth week.

MONOAMINE OXIDASE- B INHIBITORS (MAO-B)

Selegiline (Zelapar[®]; Eldepryl[®]; Emsam[®]): Selegiline's antiparkinsonian activity is due to inhibition of the enzyme MAO-B, which activates dopamine. As a monotherapy, selegiline can improve motor symptoms, and delay for several months the need for employing levodopa. However, the drug is used as an adjunct to carbidopa-levodopa treatment. As such, it can delay destruction of dopamine derived from levodopa, thereby prolonging the effect of levodopa and decreasing the fluctuations in motor control. This beneficial effect is transient as it declines in 12 to 24 months. It is metabolized in the liver, resulting in two CNS stimulant metabolites: amphetamine and methamphetamine. The adverse effects of selegiline include: nausea, insomnia, dizziness, fainting, confusion, abdominal disturbances, hallucinations, orthostatic hypotension, depression, bradykinesia and akinetic involuntary movements. The usual dose is 5 mg taken with breakfast and lunch.

CATHECOL-O- METHYLTRANSFERASE (COMT) INHIBITORS

Entacapone (Comtan[®]) has replaced tolcapone (Tasmar[®]) and is the sole COMT inhibitor available on the market. Tolcapone has been suspended in many countries, and its use is markedly restricted in the U.S. because of hepatotoxicity.

Entacapone is a reversible inhibitor of COMT. It improves levodopa therapy by reducing conversion of levodopa to 3-O-methyldopa, and by acting as a competitor to levodopa for active carrier mechanism. Additionally, it makes the brain more available for levodopa action. It is used as an adjunct to carbidopa-levodopa therapy, especially in patients who experience the "on-off" effect. When given concurrently with carbidopa and levodopa, it increases and prolongs the level of levodopa in the brain. This results in more constant stimulation of dopamine receptors in the brain. However, the increased level of levodopa may intensify the adverse effects, which include: abdominal distress, hallucination, dyskinesia and rhabdomyolysis (destruction of skeletal muscle). The recommended dose of entacapone is 200 mg administered concurrently with each carbidopa-levodopa dose to a maximum of eight times daily. Entacapone has no antiparkinsonian effect unless it is administered with carbidopa-levodopa. It may be taken with or without meals. The drug should not be rapidly discontinued.

NONDOPAMINERGIC MEDICATIONS

Amantadine (Symmetrel[®]) is an antiviral drug that possesses moderate antiparkinsonian properties. Its mechanism of action is unknown, but it is believed to relieve symptoms of PD by promoting the release of dopamine from the dopaminergic terminals in the striatum. However, it has been reported recently that amantadine acts as a weak glutamate antagonist that blocks N-methyl-aspartate (NMDA) receptors. Symptomatic improvement occurs within two to three days, but its activity is less profound than levodopa. In addition, its effectiveness begins to decline within three to six months. It may be used alone in the early stages of PD, but in later stages of the disease, it is employed concurrently with other antiparkinsonian medications. The adverse effects of amantadine include: confusion, lightheadedness, anxiety, lower extremity edema and livedo reticularis (mottled purplish discoloration of the skin). The condition is transient and disappears following discontinuation of amantadine. The usual dose when used alone is 100 mg twice daily. Such dose should be reduced in patients with kidney impairment, since the drug is excreted mainly through the kidneys. When used alone, patients may experience decreased effectiveness within several months. Benefits can be restored by either increasing the dose, or by temporary interruption of treatment for several weeks.

CENTRALLY ACTING ANTICHOLINERGICS

Anticholinergic drugs were the only available option for treating PD before dopaminergic agents. They are used to treat all forms of PD, but most often alone or with amantadine in the early stages of the disease when the symptoms are mild. In addition, they may be used later or along with levodopa. Anticholinergic drugs tend to maintain their therapeutic effect after long-term use, and there appears no need for dosage adjustment. As the disease progresses, these drugs alone are not capable of controlling symptoms. They are often used in treating mild cases in younger patients.

The mechanism of action of anticholinergics is not fully known. As stated earlier, symptoms of PD are triggered by stimulation of striated cholinergic receptors. The excess acetylcholine activity, together with lack of dopamine, produces an excitatory effect on the CNS, resulting in tremor. The anticholinergic drugs act by blocking acetylcholine from stimulating cholinergic receptors.

Compared to levodopa, the adverse effects of anticholinergic drugs are not serious. Because of their ability to block cholinergic receptors in the periphery, anticholinergics cause dry mouth, blurred vision, photophobia, urinary retention, constipation and tachycardia. The magnitude of these effects is dose limiting. Anticholinergics are contraindicated in the presence of glaucoma. It is recommended that the intraocular pressure be checked periodically. Side effects involving the CNS such as confusion, hallucinations and depression may be encountered in the elderly. Commonly used anticholinergics include benztropine, trihexyphenidyl, procyclidine, biperiden, and ethopropazine.

Benzotropine (Cogentin[®]) is used alone or in combination with other antiparkinsonian agents for all forms of PD. The therapeutic effect becomes noticeable in two to three days. Care must be exercised not to increase the dose until the drug takes effect. The usual adult dose is 0.5 mg to 1 mg daily, preferably as a single dose for the first few days. The dose may be increased by 0.5 mg daily (maximum 6 mg per day), until the desired effect is attained. A maintenance dose of 1

to 2 mg should be instituted.

Trihexyphenidyl (Artane®) is used for initial or adjunct treatment of PD. Better therapeutic effect can be Achieved when used with other antiparkinsonian drugs. The initial dose is 1 mg daily, increased by 2 mg every three to five days, until the desired effect is achieved, or a maximum dose of 10 mg is reached. When combined with levodopa, the dose is 3 to 6 mg daily in divided doses.

Procyclidine (Kenadrine®) is indicated for initial or adjunct therapy of all forms of PD. It is more effective in reducing muscle rigidity and tremor. In fact, it may potentiate tremor during early therapy. The dose is determined based on the patient's age, therapeutic response, tolerance and the type of PD. The drug is better tolerated by younger patients than the elderly, and consequently a larger dose may be given to younger patients. A larger dose may be needed to control drug-induced PD symptoms. Initially, 2.5 mg twice or three times daily may be given after meals. The dose may be gradually increased to 5 mg three times daily, if the drug is tolerated by the patient. For drug-induced extrapyramidal symptoms, the initial dose is 205 mg daily. The dose should be increased in increments of 205 mg daily until the patient obtains symptomatic relief. A good therapeutic response is usually obtained with 10 to 20 mg daily.

Biperiden (Akineton®) is chemically related to trihexyphenidyl, and consequently, it is not likely to be used for individuals who did not obtain symptomatic relief from trihexyphenidyl. Biperiden may be used for all types of PD either alone or as adjunct treatment. The usual dose for idiopathic PD is 2 mg, three or four times daily, and for drug-induced symptoms, 2 mg, 1 to 3 times daily, adjusted based on the patient's requirements and tolerance for adverse reactions.

MISCELLANEOUS ANTIPARKINSONIAN DRUGS

In selected cases, a few drugs may be used instead of, or in conjunction to, dopaminergic and anticholinergic drugs.

Ethopropazine is a phenothiazine derivative that can be used as adjunct treatment for all forms of PD. It possesses a marked anticholinergic activity. It is distinct from other phenothiazine drugs that worsen PD symptoms. The usual initial adult dose is 50 mg once or twice daily, increased gradually to the lowest possible effective dose. A dosage of 100 to 400 mg daily may be used for mild to moderate cases and 500 to 600 mg daily for severe symptoms. Because of its adverse effects (extrapyramidal reactions), the usefulness of ethopropazine is limited.

Diphenhydramine (Benadryl®) is an antihistamine that acts as an H₁ receptor antagonist. It may be used in managing all forms of PD. In addition to its antihistaminic activity, it acts as an anticholinergic drug and tends to inhibit dopamine reuptake. It may be used alone by elderly patients who cannot tolerate anticholinergic drugs, and for those with insomnia. Diphenhydramine can be used in adjunct therapy. The main side effect of the drug is sedation. Initially, dose is 25 mg three times daily, increased gradually to 25 - 50 mg three or four times daily, depending on the patient's response and tolerance.

Orphenadrine (Norflex®) is a centrally acting skeletal muscle relaxant that causes central and peripheral anticholinergic action. It is used as an adjunct for all forms of PD, especially when rigidity predominates. Additionally, it reduces pain caused by musculoskeletal disorders, and may produce CNS stimulation resulting in mild euphoria or improved mood. As a result, it may be of use for patients who experience depression. The initial dose consists of 50 mg, three times daily, increased gradually up to 250 mg daily, according to the patient's response and tolerance.

SURGICAL TREATMENT

Surgery may be used in severe cases, where pharmacological intervention is of no benefit, or poorly tolerated. Results have been mixed.

SUMMARY

Parkinson's disease is a chronic disorder that affects persons mainly after the age of 40. The disease is debilitating and characterized by slowing physical movement, muscle rigidity and tremors. The symptoms become more intense as the disease progresses. The causes of the disease are not known even though certain factors, such as genetics, trauma, toxins and insecticides may contribute. The symptoms are due to degeneration in the central nervous system which results in loss of dopamine secreting cells located in the extrapyramidal system and basal ganglia. Loss of dopamine will result in

disruption of the activity of neurotransmission within the ganglia that controls movement. There is no cure for PD, but there are a number of medications that can control the symptoms. These include: levodopa, combination of levodopa and carbidopa, anticholinergics and dopaminergic agents.

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Remaining Topics for 2009

Herbals Hormone Replacement Therapy MRSA

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LESSON EVALUATION

Please fill out this section as a means of evaluating this lesson. The information will aid us in improving future efforts. Either circle the appropriate evaluation answer, or rate the item from 1 to 7 (1 is the lowest rating; 7 is the highest).

1. Does the program meet the learning objectives?

Differentiate between the classes of PD	Yes	No
Discuss the mechanism of action of dopaminergic drugs & their adverse effects	Yes	No
Define "on-off" effect of levodopa	Yes	No
Describe the use of carbidopa in the management of PD	Yes	No
List the most common dopamine agonists	Yes	No

2. Was the program independent & non-commercial

	Yes	No					
	Poor	Average	Excellent				
3. Relevance of topic	1	2	3	4	5	6	7

4. What did you like most about this lesson? _____

5. What did you like least about this lesson? _____

Please Select the Most Correct Answer

- | | |
|--|--|
| <p>1. Cause of idiopathic PD is:</p> <p>A. Unknown</p> <p>B. Inflammation in the CNS</p> <p>C. Damage to the skeletal muscle system</p> <p>D. Excess in intake of vitamins</p> <p>2. Loss of dopamine in the brain will cause:</p> <p>A. CNS stimulation</p> <p>B. CNS depression</p> <p>C. Disruption of neurotransmission in basal ganglia</p> <p>D. Vertigo</p> <p>3. Which of these is correct regarding PD?</p> <p>A. Symptoms may occur abruptly</p> <p>B. L-dopa is administered alone</p> <p>C. Dose of L-dopa ranges from 2 to 3 grams daily</p> <p>D. L-dopa is administered only parenterally</p> <p>4. L-dopa is effective for nonmotor Parkinsonian symptoms.</p> <p>A. True B. False</p> <p>5. The antiparkinson drug amantadine is also used as an:</p> <p>A. Antiemetic</p> <p>B. Antifungal</p> <p>C. Antidepressant</p> <p>D. Antiviral</p> | <p>6. Bromocriptine:</p> <p>A. Is capable of preventing dyskinesia & postural hypotension</p> <p>B. Causes "on-off" effect</p> <p>C. Is considered a monoamine oxidize inhibitor</p> <p>D. Maintenance dose is 150-500mg daily</p> <p>7. Which of these is a COMT inhibitor?</p> <p>A. Selegiline</p> <p>B. Entacapone</p> <p>C. Ropinirole</p> <p>D. Benztropine</p> <p>8. Akathisia is a term for:</p> <p>A. Sedation</p> <p>B. Impaired muscle tone</p> <p>C. Abnormal movement</p> <p>D. Restlessness</p> <p>9. Which of these is considered a centrally acting muscle relaxant?</p> <p>A. Trihexyphenidyl</p> <p>B. Ethopropazine</p> <p>C. Biperiden</p> <p>D. Orphenadrine</p> <p>10. A side effect of tolcapone is:</p> <p>A. Hallucinations</p> <p>B. Bradycardia</p> <p>C. Hepatotoxicity</p> <p>D. Insomnia</p> |
|--|--|

Contributing Author

Farid Sadik, Dean Emeritus
University of South Carolina
College of Pharmacy
Columbia, SC

Executive Editor

William J. Feinberg,
BS Pharm, MBA



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