PHARMACY CONTINUING EDUCATION FROM WF PROFESSIONAL ASSOCIATES

"Parkinson's Disease"

May 2017

Parkinson's disease (PD) is a chronic, progressive, degenerative neurologic disease that affects body movement, muscle control and balance. It is considered the most common movement disorder. It is a topic that we revisit every few years because it is the third most common neurologic disease (10 in 1000), and new therapies are often coming into existence.

This lesson provides 1.25 (0.125 CEUs) contact hours of credit, and is intended for pharmacists & technicians in all practice settings. **The program ID # for this lesson is 0798-0000-18-231-H01-P for pharmacists & 0798-0000-18-231-H01-T for technicians.**

Participants completing this lesson by April 30, 2020 may receive full credit. Release date for this lesson is May 1, 2017.

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 1-843-488-5550. **Please write your name, NABP eProfile (CPE Monitor®) ID Number & birthdate (MM/DD) in the indicated space on the quiz page.**

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

For Pharmacists:

1. Define PD & describe its clinical manifestations.
2. List the factors that are believed to contribute to PD.
3. Differentiate between PD & drug induced Parkinson's.
4. List the classes of drugs used in treating PD.
5. Describe the MOAs of the drugs used for treating PD.
6. Discuss the side effects of antiparkinson drugs.
7. Describe effectiveness of DBS, exercise, PT & OT.

For Technicians:

1. List the classes of medications used in treating PD.
2. Describe the MOAs of the drugs used for treating PD.
3. Discuss the side effects of antiparkinson drugs.

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INTRODUCTION

Parkinson's disease (PD) is a chronic, progressive, degenerative neurologic disease that affects body movement, muscle control and balance. It is considered the most common movement disorder. PD is the third most common neurologic disease (10 in 1000), the first being migraine (121 in 1000), followed by Alzheimer's Disease (67 in 1000 elderly) and finally convulsion disorders (7 in 1000). The disease affects about one million diagnosed patients in the U.S. and 10 million worldwide. About 60,000 PD cases are diagnosed in the U.S. annually. This number does not include those who are living with the disease because of misdiagnosis or no diagnosis. PD may occur at any age, but normally the onset is about 40 years or older. It typically affects older patients, and the incidence increases with age. About 2% of sufferers are estimated to be below the age of 40, and 4% develop the disease before age 50. The average age of the onset of the disease is about 60, and normally occurs between the ages of 55 and 75. Approximately 1 in 100 PD patients are 60 years of age and older. Men are 1½ times more prone to the disease than women. Cost-wise, the direct and indirect expenditure in the U.S. including lost wages as a result of disability is estimated to be about $25 billion. The average annual cost of drugs per patient is up to $2500 (cost of surgery may reach up to $100,000).

Typically, PD is not fatal. Premature death usually occurs as a result of complications such as pneumonia or following incidences that result in injuries such as hip fracture. However, the life expectancy of PD patients is shorter than the general population. Patients who develop PD later in life are more vulnerable to psychotic symptoms, dementia and daily malfunctioning. Such patients have a shorter life expectancy than those who had early diagnosis.

CLINICAL MANIFESTATIONS

The clinical course of PD and the symptoms produced as the disease progresses vary from patient to patient. The disease can occur abruptly but usually begins insidiously in 50% - 85% of the patients, and progression of symptoms is gradual. PD is characterized by **primary** and **secondary** motor manifestations.

Primary Manifestations

1. Muscular rigidity and stiffness characterized by tightness and achiness in the arms, legs and trunk.

2. Involuntary tremor or shaking at rest. Such manifestations may occur anywhere in the body but are encountered most commonly in the arms, hands, legs, lower lip and face.

3. Bradykinesia or slow movements.

4. Postural gait, instability and impaired balance. There's a potential to fall when walking or turning around.

Secondary PD manifestations

1. Shuffled walk or dragging feet without lifting them fully.
2. Reduced arm swing.
3. Impaired facial expressions.
4. Slurred speech due to facial muscle rigidity.
5. Low volume, expressionless voice.
6. Expressionless face with the mouth open or drooling saliva.

The manifestations begin with hardly noticeable hand tremor, mainly on one side. The symptoms intensify as the disease progresses over time. The hand tremor usually appears as a back-and-forth rubbing motion of the thumb and forefinger. This occurs even when the hand is at rest. The tremors spread from the hands to the arms and legs. As the disease progresses the head becomes bowed, body bent forward and knees slightly bent. In later stages of PD, depression, anxiety and dementia may occur.

PD symptoms occur as a result of degeneration of neurons in the substantia nigra which produces a neurotransmitter called dopamine (DA) that transmits messages to the part of the brain that controls movement and balance. Over time, the dopamine level gradually decreases, and as a result the symptoms become more intense. There are two major transmitters in the extrapyramidal system and basal ganglia, which are released during transmission: the inhibitory transmitter DA, and an excitatory transmitter, acetylcholine (ACh). DA tends to inhibit discharge of striated cholinergic (copus striatum area of the brain) 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 patients, the involuntary movement, as well as other symptoms, result from an imbalance between the inhibiting effect of DA and the opposing excitatory effect of ACh. Degeneration and death of the neurons that produce DA in the extrapyramidal and basal ganglia results in decrease or absence of DA. Thus, the excitation of ACh predominates resulting in the characteristic involuntary movements that PD patients experience.

ETIOLOGY

The causes of the brain cell degeneration are unknown. However, it is believed that a combination of **genetic** and environmental **factors**, which differ from patient to patient, may contribute to PD.

Genetic Factors

The significance of the genetic components and the role they play in PD is continuously under discussion as the vast majority of cases do not seem to be inherited. Approximately 15% to 25% of patients indicate having a relative suffering from PD. It was reported that individuals whose parent or sibling is affected by the disease have only a 4% to 9% higher likelihood of acquiring the disease than the general population. Thus, the risk seems minimal. There are a small number of families that possess gene mutations that have effects on dopamine producing cells.

Environmental Factors

Toxins, pesticides, injury and rural living may contribute to PD. However, simple exposure to such factors does not result in the disease. There are drugs that can cause PD due to their extrapyramidal side effects. The extrapyramidal system is a neural network that is part of the CNS and plays a role in the control of involuntary movements. The side effects include muscular spasms and contraction, motor restlessness, slowness of movement, tremor and irregular jerky movements.

DRUG-INDUCED PARKINSONISM (DIP)

Parkinsonism is any condition that causes symptoms similar to those encountered in PD. DIP develops following the use of medications for treating other disorders. Patients with idiopathic PD (Not arising as a result of other disorders) may experience worsening of symptoms when taking such medications. Any drug that blocks dopamine receptors (dopamine antagonist) most likely will lead to DIP. Drugs employed in the treatment of psychotic disorders such as schizophrenia, behavioral disturbances experienced in dementia patients, bipolar, and psychotic depression are known to cause extrapyramidal side effects. Typical antipsychotic or first generation antipsychotic drugs such as chlorpromazine, haloperidol, thioridazine and trifluoperazine are used less (due to their high risk of producing extrapyramidal side effects) than the relatively newer atypical antipsychotics or second generation antipsychotic drugs. Atypical antipsychotic drugs such as aripiprazole, clozapine, ziprasidone, risperidone, quetiapine and olanzapine normally produce fewer extrapyramidal side effects.

The antiemetic drug prochlorperazine, which acts as a dopamine receptor antagonist, may cause extrapyramidal side effects. This also may occur with metoclopramide.

The antiarrhythmic drug amiodarone may cause some DIP symptoms such as tremor and lack of coordination.

MANAGEMENT AND TREATMENT

Since there is no cure for PD, treatment is focused on relief of the extrapyramidal symptoms that accompany the disease. However, none of those drugs prevent the slow progression of PD. Therefore, the therapeutic objective is to improve the patient's quality of life and to allow carrying out daily activities such as work, walking, dressing, eating, arising from bed or a chair and getting in and out of a car. Choice of treatment and dosage is usually determined by the severity of the condition. Some of the side effects of PD 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:

- Suppression of ACh receptors (anticholinergic action) to block their activity.
- Stimulation of DA receptors (using dopaminergic agents to provide more DA).

Surgery may be attempted in severe and unresponsive cases. In addition, complementary and supportive therapies such as physical, occupational and speech, as well as diet and exercise are essential for sustaining the quality of life.

ANTICHOLINERGICS

Anticholinergics were once the only available option for treating PD before dopaminergic agents. However, they are used less due to their side effects. They may be used for all types of PD either alone or in combination with other drugs. As the disease progresses, the anticholinergics become less effective. They are often used in treating mild cases in younger patients.

Their precise mechanism of action is not fully known, but it is believed that they block ACh from stimulating cholinergic receptors. Side effects include dry mouth, blurred vision, photophobia, urinary retention, constipation and tachycardia. The anticholinergics are contraindicated in the presence of glaucoma. Side effects involving the CNS such as confusion, hallucination and depression may occur in the elderly. Commonly used anticholinergics include benztropine, trihexyphenidyl, procyclidine and biperiden.

Benztropine

It is used alone or in combination with other antiparkinson drugs. The therapeutic effect becomes noticeable in two to three days. Care must be exercised not to increase the dose until the drug takes effect.

Trihexyphenidyl

Used for initial or adjunct treatment. Better therapeutic effects can be achieved when used with other drugs.

Procyclidine

Indicated for initial or adjunct therapy. It is more effective in reducing muscle rigidity than tremor. It may potentiate tremor during early therapy. The drug is better tolerated by younger patients than older ones. A large dose may be needed to control DIP symptoms.

Biperiden

Used for all forms of PD either alone or concurrently with other PD medications. It is chemically released to trihexyphenidyl and, consequently, it is not likely to be useful for patients who did not obtain symptomatic relief from trihexyphenidyl. It is capable of improving Parkinsonism that may occur following the use of typical and atypical antipsychotic therapy (such as the movement disorder akathisia which is characterized by unintentional motion or restlessness). It also blocks the effect of muscarinic acetylcholine receptors. Side effects include blurred vision, dry mouth, drowsiness, constipation and triggering glaucoma. Contraindications include narrow angle glaucoma.

AMANTADINE

Amantadine is approved by the FDA for use as an antiviral and for relief of PD symptoms. As an antiparkinson drug, it promotes the release of dopamine from the dopaminergic terminals of the striatum of the basal ganglia in the brain. In addition, amantadine is a weak antagonist of N-methyl-aspartate (NMDA) receptors and prevents dopamine reuptake. Symptomatic relief usually occurs within a few days, but its activity is less profound than levodopa. 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, amantadine is taken in combination with other antiparkinson medications. The side effects include confusion, lightheadedness, anxiety, lower extremity edema and livedo reticularis (a benign condition characterized by mottled discoloration of the skin). This is transient and disappears following discontinuation of the intake of the drug.

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 the prevention of breakdown of dopamine.

Levodopa

Levodopa is chemically identical to the naturally occurring dihydroxyphenylalanine (DOPA) and is the metabolic precursor of DA. It reduces PD symptoms by promoting the synthesis of dopamine (DA). Levodopa is well absorbed from the GI tract; however, about 90% of the dose is converted by the enzyme DOPA decarboxylase, which is formed in the intestinal mucosa, to DA. Since DA does not cross the blood-brain barrier well, only 10% of a levodopa dose reaches the basal ganglia. 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.

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 due to allowing a larger proportion of peripheral levodopa to cross the blood-brain barrier. As a result, a significant decrease in the side effects of levodopa is achieved. Levodopa is administered routinely with carbidopa. The combination of carbidopa-levodopa is available in ratios of 10:100 or 25:250 mg. 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 more effective dose with lesser side effects. Levodopa is the drug of choice for PD. It is effective in managing bradykinesia and rigidity, and 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 of levodopa alone is between 0.5 g and 1 gram, given in two or more divided doses, two to five hours apart, with or after meals. The dose should be gradually increased every three to seven days by up to 0.75 g daily as tolerated, until maximum benefit is reached. This procedure tends to minimize side effects of the drug when given in combination with carbidopa. Treatment is begun with single 10:100 or 25:250 mg tablets, three times daily, or every other day, according to tolerance, until a maximum benefit is attained. A minimum dose of carbidopa is required to minimize peripheral side effects,

which may occur during therapy, and are usually dose-related. They include dyskinesia in the form of oral-facial or limb dystonia (involuntary muscle contract ion), akathisia 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. The "on-off" phenomenon is transient, but abrupt, and occurs without warning. This effect can result in alternating periods of intense akinesia or 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 the use of levodopa is advantageous, it does not resolve many of the symptoms of PD. Motor features such as speech, gait, posture and balance do not normally respond to levodopa treatment and tend to worsen over time. Additionally, it has no positive effect on non-motor Parkinsonian symptoms such as hallucination, cognitive impairment and orthostatic hypotension. In fact, levodopa may intensify such symptoms.

CATECHOL-O-METHYLTRANSFERASE (COMT) INHIBITORS

COMT inhibitors are drugs that inhibit the enzyme Catechol-O-Methyltransferase which acts to breakdown catecholamines (transmitters) such as dopamine, epinephrine and norepinephrine. They also prevent the breakdown of levodopa, thereby prolonging the duration of a dose of levodopa. Entacapone and tolcapone are used as adjuncts to levodopa/carbidopa. Both have no antiparkinsonian effect unless they are taken with levodopa/carbidopa.

Entacapone

Approved by the FDA in 1999, it improves levodopa therapy by reducing the conversion of levodopa to 3-O-methyldopa and by making the brain more available to levodopa action. It is used in combination with levodopa and carbidopa, especially in patients who experience the "on-off" effect. However, the resultant increase in levodopa may also intensify the adverse effects. The drug should not be rapidly discontinued. Side effects include dizziness, lightheadedness or fainting, drowsiness, diarrhea, confusion, arrhythmia, brownish orange color of urine, rhabdomyolysis, nausea, vomiting and hallucination.

Tolcapone

This drug was marketed in the U.S. early in 1998 but was suspended late in the same year due to the risk of potentially fatal acute fulminant liver failure. Authorization for its use was renewed in 2009. However, the FDA issued a black box warning and label revision. The drug may be used in PD patients who are taking levodopa/carbidopa who experience "on-off" effect and who are not properly responding to other adjunctive therapies. Patients who are not favorably responding to treatment within three weeks after the start of the treatment should discontinue taking the medication. The medication should not be used for patients with liver problems, history of muscle problems, or are taking monoamine oxidase inhibitors. Side effects include dizziness, drowsiness, light headedness, especially when sitting up, nausea, vomiting, dark urine, pale stool and rhabdomyolysis.

MONOAMINE OXIDASE-B INHIBITORS (MAO-BI)

Monoamines are neurotransmitters chemically. They consist of an amino group attached to an aromatic ring by a chain that contains two carbon atoms. The monoamines include histamine and the catecholamines adrenaline, dopamine and norepinephrine. Monoamine

oxidases (MAO) are a group of enzymes found in the outer membrane of mitochondria in most tissue cells in particular in the brain, in peripheral adrenergic and dopaminergic nerve endings. MAO oxidase function is to catalyze the breakdown of norepinephrine, dopamine and serotonin and to remove them from the brain. As such they regulate the synaptic transmission of monoamines such as norepinephrine, dopamine and serotonin. The selective MOA-A inhibitors are used to treat depression and anxiety (but are now replaced by other antidepressants that are safer and produce less side effects). Selective MOA-B inhibitors are used to treat PD. MAO-B is one of the MAO enzymes which is encoded in humans by the MAO-B gene whereas MAO-A enzyme is encoded by the MAO-A gene. Both are isoforms. MAO in the GI tract and liver are primarily MAO-A to protect the body from exogenous amines such as tyramine which, if absorbed without being broken down, can cause hypertensive crisis or "cheese crisis." Exogenous substances like fermented cheese, wine, herring and others that are rich in tyramine can cause this if taken with MAO-A inhibitors.

MAO oxidase inhibitors (MAOIs) work by preventing the breakdown of transmitters, especially dopamine in PD, by the enzyme MAO, thereby maintaining normal level of transmitters in the brain cells. A newer MAOI used in the treatment of PD is selegiline, which was approved by the FDA in 2006 as the first transdermal patch for treating depression. It is also used for PD.

Selegiline

At usual doses, this drug acts as an MAO-B inhibitor, but at higher doses it inhibits both MAO-A and MAO-B. By inhibiting MAO, it activates dopamine, resulting in an increase in the dopamine level in the brain. As a monotherapy, selegiline can improve Parkinsonian motor symptoms and delay for several months the need for using levodopa. However, the drug is used as an adjunct to levodopa/carbidopa treatment. As such, it can delay destruction of dopamine derived from levodopa, thereby prolonging the effect of levodopa and decreasing the fluctuation in motor control. The beneficial effect is not permanent as decline occurs in 12 to 24 months. Side effects include nausea, dry mouth, insomnia, dizziness, fainting, orthostatic hypotension, hallucination and bradykinesia. Patients need to avoid foods and beverages containing tyramine.

Rasagiline

This MAO-B inhibitor is relatively new. It was approved in the U.S. in 2006 for use as a monotherapy to treat early symptoms of PD or in combination with other drugs such as dopamine agonists or levodopa if the symptoms become intense. Side effects are hypertension or hypotension. In fact, the FDA label includes such a warning. Other adverse effects include flu-like symptoms, headache, fatigue, nausea, joint pain and accidental injuries due to falls.

DOPAMINE AGONISTS

These are drugs that stimulate dopamine receptors in the striatum. They are not as effective as levodopa, but when given with levodopa, the time of the "on-off" effect is positively impacted.

Apomorphine

This medication possesses activity similar to that of dopamine. It is used to treat the wearing off phenomenon experienced by some patients with advanced PD. The drug, which is a non-ergoline dopamine agonist, is given as a subcutaneous injection. Side effects include nausea, vomiting, lightheadedness, dizziness, fainting, confusion and hallucination.

Bromocriptine

Bromocriptine is a potent dopamine agonist that is an ergot alkaloid derivative and typically used as an adjunct to levodopa in treating PD. It directly activates the dopamine receptors in the basal ganglia of the brain. 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 are dose-related and may be encountered in 30% to 50% of patients. They include nausea due to stimulation of the brainstem vomiting centers, orthostatic hypotension, confusion, hallucination, agitation and nightmares.

Rotigotine

This drug was approved by the FDA in 2012 for treating early and advanced stages of PD and Restless Leg Syndrome (RLS) which is a neurological disorder that causes the patient to have a strong desire to move the legs while sitting or lying down. This urge is due to crawling, creeping, throbbing, aching, itching and pins and needles sensation in the legs. It occurs mostly at night. Up to 10% of the population has RLS which affects mostly middle-aged and older people.

Pramipexole

This is a dopamine agonist of the non-ergoline class for treating PD symptoms as well as for RLS. Side effects include drowsiness, nausea, vomiting, fainting and hallucination.

Ropinirole

Ropinirole is used in the treatment of PD and RLS. It is a non-ergoline dopamine agonist. The drug is available in the generic form. Side effects include nausea, vomiting, dizziness, orthostatic hypotension and sudden sleep attacks.

ADJUNCT THERAPY

Surgery may be used in some cases, especially in younger patients, when pharmacological therapy is poorly tolerated or has failed to relieve symptoms such as tremors, slowness of movement, rigidity and imbalance. Deep brain stimulation (DBS) involves the implantation of one end of a hair-thin electrode in certain areas of the brain that produce irregular impulses that cause PD symptoms. The other end of the electrode is connected to a pulse generator, similar to a pace-maker device. Upon activation, the generator will deliver gentle electrical impulses to the brain resulting in correction of the brain's electrical impulses and relief of symptoms. DBS can be performed while the patient is awake or under sedation and usually the level of stimulation is adjustable so that the generator provides the most effective relief with the least number of adverse effects. It is estimated that 40% - 60% of patients who have undergone DBS have experienced some symptomatic relief.

BENEFITS OF EXERCISE AND PT

Reports indicate that regular exercise and physical therapy can improve the quality of the PD patient's life. It reduces stiffness, improves mobility, posture, balance, gait, speech and functional ability. In addition, it increases the amount of oxygen reaching the brain, thereby reducing the magnitude of brain cell damage or death, maintaining cognitive skills and improving muscle control and the prevention or reduction of falls. Exercises such as aerobics, and those that enhance heart and lung functions, improve biomechanics, good posture, trunk rotation, symmetric movement and balance are recommended. Dancing, skipping and cycling, brisk walking, yoga, Tai chi, gentle jumping over low obstacles, treadmilling (at a slow

speed), and swimming with big arm swing. Tai chi is an exercise aimed at achieving slow flowing motions and gentle movements.

SUMMARY

Parkinson's disease is a chronic progressive degenerative disorder that affects one million diagnosed Americans and about 60,000 patients are diagnosed with the disease annually. It can strike insidiously at any age, but usually affects people 40 years of age and older.

PD occurs due to degeneration of the substantia nigra which produces a neurotransmitter called dopamine. Reduction or loss of dopamine results in PD symptoms which are characterized by muscular rigidity and stiffness, involuntary tremor or shaking at rest, bradykinesia or slow movement, and postural, gait instability and impaired balance.

There is no cure for PD, but there are medications that can alleviate the symptoms. However, such medications do not stop the disease progression. The medications used belong to the following classes: anticholinergics, amantadine, dopaminergics, (COMT) Inhibitors, MAO-B inhibitors, and dopamine agonists.

A surgical procedure, deep brain stimulation (DBS), may be used in severe cases where pharmacological therapy is poorly tolerated or has failed to relieve symptoms.

Exercise and physical therapy can improve symptoms and general health.

REFERENCES

1. Sevinbjomsdottir S, "The clinical symptoms of Parkinson's disease," Journal of Neurochemistry, 139: 318 (2016).
2. Kalia LV, Lang AE, "Parkinson's Disease," Lancet, 386 (9996): 896 (2015).
3. Davie CA, "A review of Parkinson's disease," Br. Med. Bull., 86(1): 109 (2008).
4. Olanow CW, Watkins PB, "Tolcapone," Clinical Neuropharmacology, 30(5): 287 (2007).
5. Chen JJ, Swope DM, Dashtipour K, Lyons KE, "Transdermal rotigotine: a clinically innovative dopamine-receptor agonist for the management of Parkinson's disease," P9(12): 1452 (2009).
6. Chen JJ, Swope DM, "Clinical pharmacology of rasagiline: a novel, second generation propargylamine for the treatment of Parkinson's disease," J. Clin. Pharmacol., 45:8): 878 (2005).

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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?

Define PD & describe its clinical manifestations	YES NO
List the factors that are believed to contribute to PD	YES NO
Differentiate between PD & drug induced Parkinson's	YES NO
List the classes of drugs used in treating PD	YES NO
Describe the MOAs of the drugs used for treating PD	YES NO
Discuss the side effects of antiparkinson drugs	YES NO
Describe effectiveness of DBS, exercise, PT & OT	YES NO

2. Was the program independent & non-commercial? YES NO

	Low Relevance						Very Relevant
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 Mark the Correct Answer(s)

1. **Which statement is FALSE about PD?**
   A. PD can be cured.
   B. PD strikes most often in adolescence.
   C. Never a genetic or hereditary relationship.
   D. PD does not effect females.
   E. All are false.

2. **PD occurs due to:**
   A. Reduced epinephrine level.
   B. Damage or death of cells in the substantia nigra.
   C. High level of dopamine.
   D. Inactivity of the pituitary gland.

3. **Which of the following is NOT a symptom of PD?**
   A. Involuntary tremor. B. Slow movement.
   C. Muscular rigidity. D. Muscular atrophy.

4. **Which of the following neurotransmitters Is excitatory?**
   A. Norepinephrine. B. GABA.
   C. ACh. D. Serotonin.

5. **Which drug(s) can induce Parkinsonism (DIP)?**
   A. Levodopa B. Phenobarbital
   C. Risperidone D. Benztropine

6. **Which of the following approaches is employed to treat PD symptoms?**
   A. Reestablishment of balance between dopamine and acetylcholine.
   B. Activation of ACh receptors.
   C. Suppression of dopamine receptors.
   D. Increased blood level of Cortisol.

7. **The MOA of levodopa is:**
   A. CNS depression.
   B. Stimulation of production of ACh.
   C. Stimulation of the synthesis of dopamine.
   D. Blocking mechanism of epinephrine.

8. **Which statement is true about carbidopa?**
   A. A potent dopamine stimulant.
   B. Effective in reducing PD symptoms.
   C. Blocks penetration of levodopa through the blood-brain barrier.
   D. Increases the effectiveness of levodopa.

9. **Which of the following drugs is a COMT-inhibitor?**
   A. Amantadine
   B. Entacapone
   C. Trihexyphenidyl
   D. Rasagiline

10. **Which drug is available as a transdermal patch?**
    A. Rotigotine
    B. Bromocriptine
    C. Selegiline
    D. Benztropine