

Parkinson's Disease: Non-Pharmacological Interventions

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Goal. The goal of this lesson is to provide an overview of non-motor symptoms, risk factors, and non-pharmacological management of Parkinson's Disease.

Objectives. At the completion of this activity, the participant will be able to:

1. identify key features of Parkinson's Disease (PD);
2. recognize non-motor manifestations of PD;
3. demonstrate an understanding of surgical intervention and deep-brain stimulation for PD;
4. state the types and benefits of non-pharmacological management of PD; and
5. list risk and attenuating factors for PD.

Background

Parkinson's Disease (PD) is a progressive neurodegenerative disorder of unknown etiology defined pathologically as degeneration of dopaminergic neurons in the substantia nigra, a region of the midbrain. Following Alzheimer's disease, PD is the most common neurodegenerative disorder in the United States. Available data estimate PD's prevalence rate of approximately 1 percent in persons 60-years-old. Prevalence rates increase with advancing age with

the greatest reported occurrence of approximately 2.5 to 4 percent at age 80 years and over.

This chronic, progressive disease presents with bradykinesia (abnormal slowness of movement), rigidity, resting tremor, and postural instability. These symptoms, and additionally the non-motor effects, contribute to a significantly decreased quality of life.

Patients with PD face increasing severity of symptoms as the disease progresses, which translates into escalating direct costs of medical treatment for pharmacotherapy and non-pharmacological therapies, hospitalizations, and institutional care. The Parkinson's Disease Foundation estimates that the current combined direct and indirect costs of PD in the United States are \$25 billion annually, with medication costs for an individual averaging \$2,500 a year. Because of the nation's rising population of elderly persons, this portends an astounding escalating economic burden for patients and payers.

Although the nation's population of older adults is increasing, the number of clinicians at the primary care level responsible for managing the health needs of this population is not increasing. It is important that allied healthcare personnel, at all levels of practice, familiarize themselves thoroughly with diseases prevalent in the aging population, such as PD. As pharmacists continue to learn more

about Parkinson's Disease, treatment, and patient management, they will better serve this patient population.

Most literature and textbooks concentrate on motor symptoms of PD, rather than on the non-motor aspects. This lesson provides an overview of non-motor challenges of PD, and discusses surgical intervention and nondrug therapy for treating PD. An overview of the disease and the latest reports that review suspected risk and attenuating factors are also provided.

Non-Motor Manifestations

While dopamine neuron loss from within the substantia nigra area of the brain remains the most prominent pathologic feature of PD, this loss eventually spreads throughout the CNS over the course of the disease. This extensive broad-range pathology is likely responsible for the non-motor features of PD that can be classified under four main categories: neuropsychiatric, sleep disturbance, autonomic symptoms, and sensory manifestations (Table 1). As therapy for the motor features has improved, the non-motor aspects contribute more to disabling mobility and mortality. Death frequently results from complications of immobility, including aspiration pneumonia or pulmonary embolism.

Recognition and control of non-motor symptoms have become an area of major challenge to current clinical management of PD.

Table 1
Non-motor symptoms of Parkinson's Disease*

Neuropsychiatric

- Psychosis (hallucinations, delusions)
- Depression
- Apathy
- Anhedonia
- Attention deficit
- Impulsive and compulsive behaviors
- Panic attacks
- Cognitive impairments[‡]
- Dementia

Sleep disorders

- Rapid eye movement (REM) sleep behavior disorder (loss of atonia)[‡]
- Vivid dreaming
- Restless legs syndrome
- Excessive daytime somnolence
- Insomnia

Autonomic symptoms

- Orthostatic hypotension (related falls)
- Gastrointestinal dysfunctions (constipation, fecal incontinence)[‡]
- Bladder disturbances (urgency, frequency)
- Nausea
- Vomiting
- Drooling
- Increased sweating
- Sexual dysfunctions (hypersexuality, erectile dysfunction)
- Dysphagia/choking

Sensory deficits

- Anosmia (olfactory deficits)[‡]
- Ageusia (taste deficits)[‡]
- Pain
- Paresthesia

*Adapted from: Smith Y, Wichmann T, Factor SA, DeLong MR. *Neuropsychopharmacology Rev.* 2012;37:213-246.

[‡]Non-motor symptoms that often precede PD motor dysfunction.

There have been a small number of placebo-controlled trials related to treatment for specific non-motor symptoms, but this field remains largely unexplored and will deserve significant attention in years to come. Treatment of psychosis (hallucinations, etc.) has received extensive attention because these conditions appear in such a large number of patients with PD. Many

non-motor symptoms, which may be unresponsive to, exacerbated by, or induced by conventional PD therapy, are prevalent and affect the majority of patients over the course of their disease. Some non-motor symptoms might complicate the diagnosis and treatment because their appearance can precede the expression of motor symptomatology, sometimes by many years. Furthermore, non-motor symptoms contribute to the severity of disability and may lead to institutionalization.

Non-motor features are of critical importance to individuals with PD since they may have an even greater impact on quality of life than motor symptoms. A study that evaluated the impact of non-motor symptoms on quality of life in Parkinson patients found that these symptoms, especially sleep disorders, fatigue, and apathy, have an impact on this parameter. Non-motor symptom progression contributes to further decline in quality of life. These studies confirm that the total burden of non-motor symptoms is likely more important than motor symptoms in many to most patients with PD in determining quality of life across all stages of their disease.

A study reported on the types and extent of non-motor problems experienced by a group of 149 patients with PD followed for 15 to 18 years. Occurrence rates of selective symptoms were falls, 81 percent (with 23 percent suffering fractures); cognitive decline, 84 percent (48 percent fulfilling criteria for dementia); hallucinations, 50 percent; depression, 50 percent; choking, 50 percent; symptomatic postural (orthostatic) hypotension, 35 percent; and urinary incontinence, 41 percent.

Non-motor symptoms can remain under-recognized by clinicians at all levels of practice. In an extreme case for example, neurologists, who focus primarily on the motor aspects of PD, may not readily realize that patients' non-motor symptoms are connected with the disease. A study was designed to

evaluate the diagnostic accuracy of neurologists for a variety of behavioral symptoms commonly associated with PD. One hundred-one patients with PD completed a brief screening questionnaire for depression and anxiety followed by the administration of diagnostic tests. Standardized testing showed evidence of depression in 44 percent of patients, anxiety in 39 percent, fatigue in 42 percent, and sleep disturbance in 43 percent. The prevalence of these conditions identified by the neurologists was lower: 21 percent with depression, 19 percent with anxiety, 14 percent with fatigue, and 39 percent with sleep disturbance. The diagnostic accuracy of the neurologists was 35 percent for depression, 42 percent for anxiety, 25 percent for fatigue, and 60 percent for sleep disturbance. The study demonstrated that during routine office visits, these neurologists failed to identify the presence of depression, anxiety, and fatigue more than half of the time, and failed to recognize sleep disturbance in 40 percent of patients.

Of further importance is that patients with PD may not always report non-motor symptoms to their physician because they do not understand that these symptoms can be related to PD or to the drug treatment to control motor symptoms. To determine the proportion of PD patients who failed to declare their symptoms, 242 patients representing all age groups and disease stages were recruited to complete a validated, self-completed questionnaire listing 30 items. Undeclared non-motor symptoms ranged from 31.8 percent (diplopia) to 65.2 percent (delusions). The most frequently nondeclared symptoms were delusions, daytime sleepiness, intense and vivid dreams, and dizziness. For many patients, appropriate treatment for undeclared non-motor symptoms was started only after symptoms were recognized following completion of the questionnaire.

Therapy should be individualized for each patient, as treatment

for motor symptoms can improve some non-motor symptoms while aggravating others. In many cases, symptom-specific treatments are available and necessary to control non-motor symptoms of PD.

Surgical Intervention of Parkinson's Disease

Most patients with PD will eventually develop disabling symptoms despite optimal pharmacotherapy. Transplantation of dopaminergic tissue into the brain as a means to substitute lost dopamine neurons with new ones was first explored in the United States and Sweden in the 1970s. The future of this approach remains questionable due to lack of effectiveness and onset of unexpected adverse effects detected in patients enrolled in a number of large controlled clinical trials.

As with transplantation attempts, there is significant current interest in the use of stem cells as a treatment for PD. At the time of this writing, implantation of stem cells in humans remains limited by safety concerns and regulatory issues.

Approved surgical interventions to treat motor complications that cannot be controlled satisfactorily with drugs include lesional therapies (thalamotomy and pallidotomy) that ablate a target region to achieve either tremor control or to lessen dyskinesia (involuntary muscle movements), and deep-brain stimulation (DBS). Procedures that create brain lesions can be effective for treating selective symptoms of PD, but they are irreversible and a frequent cause of adverse effects, especially when lesions are bilateral. As a result, current practice favors DBS when surgery is contemplated.

Deep-Brain Stimulation.

Developed in the 1990s, DBS has received widespread attention and generated enthusiastic responses from patients and practitioners. The procedure is reversible, and is now the state-of-the-art neurosurgical treatment for motor impairments of PD, with more than 30,000 individuals worldwide who

have undergone the procedure.

During DBS, electrodes are surgically inserted into the targeted brain region. A device called an impulse generator, which is similar in function to a cardiac pacemaker, is implanted under the collarbone to provide continuous electrical stimulation to the area of the brain involved in motor function. The patient has an external programmer, which permits him/her to check the battery and turn the impulse generator on or off. A battery lasts about three to five years and is relatively easy to replace under local anesthesia.

FDA has approved unilateral and bilateral DBS of the subthalamic nucleus or the globus pallidus interna for controlling PD motor symptoms. Either target can be effective for alleviating tremor, bradykinesia, and rigidity; increasing the amount of *on* time (good motor control with unimpeded motor function), reducing wearing-off episodes; and decreasing dyskinesia compared with best medical management. Factors that predict a favorable response to surgery for advanced PD include good presurgical response to levodopa, few comorbidities, disabling symptoms in the *off* state, uncontrollable drug-induced dyskinesia, absence of cognitive impairment, and absence of (or well-controlled) depression. A good understanding of potential benefits and risks of the operative procedure and evaluation, ability to give informed consent, as well as a strong emotional support network of family and friends are vital.

In a randomized, controlled trial, 255 patients with PD received bilateral DBS in either the globus pallidus interna or subthalamic nucleus, or "best medical therapy" (pharmacologic and nonpharmacologic therapies [i.e., physical, occupation, and speech therapy] to achieve best symptom control). The primary outcome was time spent in the *on* state without troubling dyskinesia, as reported by patient diaries, at six months. Patients who received DBS gained, on average, 4.6 hours/day of *on* time without

troubling dyskinesia. Those managed with best medical therapy gained zero hours/day *on* time. Seventy-one percent of individuals in the surgical group and 32 percent of patients in the best medical therapy group showed clinically meaningful improvement in motor function. PD quality of life scores also improved significantly in the DBS group compared with patients in the best medical therapy group.

Several other randomized, controlled trials have demonstrated that DBS improves motor symptom control, reduces motor fluctuations and improves quality of life in people with advanced PD, and provides sustained motor benefit persisting over 10 years. Oftentimes dopaminergic drug therapy can be significantly reduced following DBS, which is of particular benefit when the patient has difficulty tolerating the drugs.

The presence of dementia and untreated depression are contraindications to DBS. Strict criteria are in place to qualify for DBS therapy, and only a small percent of patients with PD are suitable candidates. Risks of surgery include intracranial hemorrhage; stroke; infection; lead migration, misplacement, or fracture; and death.

Non-Pharmacological Management

Physical, Occupational, and Speech Therapy. Based largely upon patient reporting, but substantiated by clinical experience, physical, occupational, and speech therapy, as well as exercise, have a substantial benefit for PD patients in terms of maintaining the status quo and improving their quality of life. In a meta-analysis of 29 trials, significant benefit was reported for nine of 18 outcomes assessed. Of potential clinical significance were benefits in speed and activities of daily living. Unfortunately, only a few methods have been tested in high-standard studies and demonstrate efficacy. Describing them individually would extend beyond the scope of this lesson. Ultimately, there is no evidence

that shows a single form of physical therapy is better than another. Large randomized, controlled trials are needed to further assess the benefits of physiotherapy for PD, both for clinical efficacy and cost effectiveness.

Although there is less evidence that occupational therapy is beneficial, it may help patients maintain family, social, and work roles, improve safety and motor function, and can be offered to those having difficulty performing tasks of daily living. Tai chi, a martial art involving slow, controlled movements and the maintenance of various postures, has emerged as a popular intervention for PD. Tai chi seems to benefit gait, balance, and functional mobility, and participants may experience fewer falls.

Many PD patients develop dysarthria (motor speech disorder making it difficult to talk and/or pronounce words), with hypophonia (low speech volume), decreased pitch, and pronunciation difficulties. Speech therapy, particularly exercises aimed at improving the volume of speech, is effective. Lee Silverman voice training is an established technique that has been extensively studied and proven to improve voice quality and audibility in PD patients.

Exercise. There is general agreement that regular exercise can help people with PD maintain and improve mobility and balance. A meta-analysis of 18 randomized controlled trials involving 901 patients with PD demonstrated that aerobic exercise significantly improved motor action, balance, and gait including gait velocity, stride/step length, and walking ability in these patients. There was no evidence to support or refute the value of aerobic exercise in improving quality of life in patients with PD compared with other therapies.

Results of a recent study (July 2014) further supported evidence that aerobic exercise benefits people with PD. Sixty subjects, average age 65 years, walked outdoors at a speed of 2.8 miles per hour, for 45 minutes three times a week for

six months, while continuing their usual PD medications. Researchers assessed participants at the beginning and at regular intervals on aerobic fitness, cognition, severity of PD movement symptoms, and quality-of-life issues including fatigue and depression. Study results showed that the subjects improved on measures of quality of life and on their ability to focus and filter out distracting information, a familiar problem in persons with PD. Also shown was that real-world, easily accessible activity – brisk walking – has the potential to improve fatigue, depression, and quality of life. Aerobic walking was also safe, with no serious adverse events reported.

Diet. Major alterations in the diet are usually not necessary. It is important, however, that the diet include adequate fiber and hydration because constipation is often an issue. The diet should also include sufficient calcium and vitamin D to prevent osteoporosis, because osteopenia is common in patients with PD. Additionally, low-protein diets may help some patients who experience the *on/off phenomenon*, [which is an unpredictable shift from mobility (*on*) to a sudden inability to move (*off*)] that may be worsened with intake of neutral amino acids, which inhibit absorption of levodopa from the gastrointestinal tract.

Risk and Attenuating Factors

The past decade has been characterized by a remarkable acceleration in identifying possible risk factors that cause and/or accelerate onset of PD. In some cases, specific factors have been identified that seem to protect against disease onset.

Age. As is its association with many neurodegenerative diseases, age is a clear risk factor for PD. This correlation was understood nearly two centuries ago when James Parkinson published his *Essay on the Shaking Palsy*. More recent epidemiologic evidence has confirmed that in both men and

women there is a growing risk for PD that correlates with increasing age.

Although the correlation of age to incidence of PD seems self-evident, the underlying physiological process by which advancing age may confer risk for PD remains obscure. The age association suggests that there is an intrinsic factor in the aging process that predisposes an individual to development of overt PD, or that some accumulated exposure or mechanism for genetic malfunction needs to have occurred before onset of pathogenesis that leads to the clinical syndrome of PD. The relationship with age needs to be factored into our understanding of the ultimate causal pathogenesis.

Gender. Men are at greater risk of PD than women; the age-adjusted ratio for men:women is 1.5:1. Numerous studies have demonstrated heterogeneity in this ratio, with a higher male/female ratio in older populations. The explanation for these observations is uncertain. The most simplistic interpretation of PD is that neuroprotection is extended by estrogen, and that testosterone has a neutral or potentially harmful effect. Limited evidence that supports a protective role for estrogen is that women with PD are more likely to have undergone a hysterectomy or oophorectomy than those who did not develop PD. On the other hand, if estrogen were the sole factor, the male:female ratio would likely be higher in younger, rather than older, populations and there would be no expected heterogeneity across cultures. Another relatively simplistic possibility is that there is a recessive susceptibility gene on the male chromosome, which confers enhanced risk among men. The male lifestyle may also predispose to other exposures that are suspected risk factors for PD. The gender finding is considered an association, rather than a causal factor *per se*, because its relationship to the cause of PD is unknown.

Tobacco. Cigarette smoking appears to reduce the risk of PD by

approximately 36 percent. In one review of case-controlled studies, virtually all outcomes demonstrated reduced risk in individuals who smoked cigarettes. The overall relative risk for smokers to develop PD, compared to nonsmokers, was approximately 0.6. Most studies have shown a dosage effect since persons with more pack-years of smoking seem to have an even lower risk of PD than those with fewer pack-years. Although the strongest association appears to be between cigarette smoking and PD, other tobacco exposures also appear to decrease risk for PD. Potential explanations for this observation include the hypothesis that nicotine exposure promotes dopaminergic neuron cell survival. Or, it may decrease production of other metabolic toxins.

There are also potential non-causal explanations for this tobacco observation. For example, noncausal explanations include the fact that increased mortality in smokers means they do not have the same age-related risk for developing PD, that subclinical PD decreases the likelihood of smoking because of decreased sense of taste and smell, or that nicotine has subtle anti-parkinsonian effects that mask the clinical manifestation of PD.

Caffeine. Caffeine, the world's most widely used psychomotor stimulant, appears to decrease the risk of PD in "ever-coffee" drinkers versus "never coffee" use by approximately 33 percent. One study reviewed eight case-controlled and five cohort studies, which demonstrated decreased relative risk of PD in coffee drinkers that was similar to that for cigarette smokers. As with tobacco exposure, there was also a dosage effect with coffee intake. Individuals classified as "heavy" coffee drinkers seemed to have a risk of PD that was even lower than for those who drank less or ingested no caffeine. In a study that looked at the dose effect of coffee consumption by gender and smoking status, the study authors found a protective effect of

coffee drinking, even in individuals who were never-smokers. Also, while both men and women had a relative reduction in risk, the reduction was somewhat higher in men. Consumption of decaffeinated coffee does not appear to modify onset of PD.

Genetics. At present, only a small percentage of individuals with PD have known genetic variations attributed to disease risk. Nonetheless, investigators are vigilant that an underlying genetic defect, or a genetic variation that confers risk for development of symptoms, will soon be identified in the majority of persons with PD. Indeed, many candidate genes have been investigated as potential risk factors and at least three large meta-analyses show there are positive associations between certain genes and pathogenesis of PD. However, even in circumstances where there appears to be a genetic cause of PD, there is often incomplete penetrance. In other words, individuals with a mutation associated with PD do not necessarily develop the illness. This suggests that there is substantial interplay between these genetic variations with either other genetic or epigenetic factors, or with the environment. Additionally, environmental exposures have long been considered as a possible cause of, or risk factor for, PD. Identifying specific environmental risk factors for PD using genetic information has provided emerging evidence regarding possible environmental influence on PD.

Pesticides. There appears to be an approximately three-fold increased risk of PD with occupational pesticide exposure, but this risk varies substantially depending on the specific pesticide used. Most pesticides that have been identified as suspected causative factors share common features, namely the ability to induce oxidative stress, mitochondrial dysfunction, α -synuclein fibrillization, and neuronal cell loss. In general, rotenone, paraquat, permethrin, and maneb have a somewhat higher hazard ratio for PD develop-

ment in persons who are exposed to these pesticides. Overall, increasing frequency, duration and cumulative exposure to pesticides were associated with PD in a dose-dependent manner. However, one complication to understanding this and other potential environmental exposures is the observation that some studies fail to show that pesticides increase risk in people with a family history of PD.

Occupation. Occupation has been investigated as a potential risk factor for PD with variable results. In general, farmers and others with agricultural or rural occupations appear to have a higher risk. It is uncertain whether these occupations are merely a proxy for pesticide exposure or some other environmental factor. Metal welding, mining, and dry battery manufacturing are other occupations frequently linked to PD. Evidence linking welding with PD is limited and relationships do not appear to be consistent. Occupation may be blamed in lieu of studying another exposure. For example, farming may be used to assess pesticide exposure.

Urate. High blood urate levels have been associated with decreased risk of PD. In a meta-analysis of cohort studies of PD, persons with elevated blood urate were noted to have a decreased risk of PD. In subsequent follow-ups of patients with PD, the rate of disease progression also seemed to be associated with the extent of blood urate levels, in that those with the highest blood urate levels appeared to have the slowest PD progression. This remains an intriguing observation, but it is unknown whether there is any reasonable mechanistic explanation. One possible explanation is that oxidative stress might play a critical role in PD, and because urate has antioxidant activity, urate might ameliorate the effects of neurodegeneration and serve as a neuroprotectant for PD.

Nutritional Supplements. There is little conclusive scientific information that nutritional

Table 2
Websites for further
information on
Parkinson's Disease

- National Parkinson Foundation:
www.parkinson.org
- National Institute of Neurological Disorders and Stroke:
www.ninds.nih.gov
- Parkinson's Disease Foundation:
www.pdf.org
- Michael J. Fox Foundation for Parkinson's Research:
www.michaeljfox.org

supplements benefit persons with PD, although the popular press continues to extol their virtues. Some of these supplements include coenzyme Q₁₀; vitamins C, D, and E; creatine; and glutathione. Meanwhile, researchers continue to examine them to evaluate potential effectiveness on slowing PD progression and/or managing its symptoms. While some studies imply limited therapeutic promise for these and dozens of other supplements, others have refuted the popular claims. Further evaluation is needed to address dosing, administration, gender and genetic influences, adverse effects, and long-term risks.

Nonsteroidal Anti-inflammatory Drugs. Modest reductions in PD risk are associated with selective nonsteroidal anti-inflammatory drugs (NSAIDs). Benefit regarding this apparent reduction in risk can be observed in those who regularly use certain non-aspirin NSAIDs and the effect is greater with a longer duration of use. Neither aspirin nor acetaminophen appear to have an effect on PD risk. One study found that ibuprofen conferred decreased risk of PD, but that the effect of other NSAIDs was similar to aspirin and acetaminophen. There remain many conflicting conclusions based on which NSAIDs were evaluated and how the studies were undertaken. The mechanistic explanation for

this response may be due simply to reduction of inflammation. Chronic low-level inflammation has been found in areas of neurodegeneration in PD, but it is unknown whether this pathology is causative or a response to brain injury.

Traumatic Brain Injury.

Traumatic brain injury, such as occurs with professional athletes, has been shown in many, but not all, epidemiologic studies to correlate positively with occurrence of PD, giving rise to speculations about modifying factors. It has been shown that the number of head injuries and duration of concomitant unconsciousness are associated with increased risk of PD. Individuals remaining unconscious for greater than five minutes appear to have a two-fold increased risk. Over the past two decades, many, but not all, studies of traumatic brain injury have linked the injury with or without loss of consciousness to PD. It is theorized that traumatic brain injury may require additional risk or susceptibility factors in order to cause the pathology. This has been referred to as the "multiple hit hypothesis" of PD. Recently, an animal study demonstrated that while a single mild traumatic brain injury was sufficient to cause a progressive loss of dopaminergic neurons, the number of neurons lost during a specified time frame was increased when traumatic brain injury was accompanied by exposure to paraquat at a dose that by itself did not cause a significant loss of dopaminergic neurons. Specifically, an insult such as traumatic brain injury may simply increase the susceptibility of dopaminergic neurons to additional insults, including toxic agents such as pesticides. Again, the mechanistic cause of this apparent association remains unclear.

Risk Summary. Discussed herein is only a brief sampling of associations that have been reported to modify the risk one way or the other for developing PD. Despite the large number of factors that have been investigated, only

age, caffeine, and cigarette smoking appear to provide sufficiently strong evidence of either a causal effect or of an association. At this time, all other factors, when considered independently, have limited evidence of an association. There is however, increasing evidence that the risk for PD pathogenesis is likely to be multifactorial, implying that genetic-environmental interactions contribute more to the onset of PD than a genetic or environmental factor alone.

Overview and Summary

With the aging of the Baby Boomer generation, we can expect an increasing number of patients with Parkinson's Disease. While the benefits of pharmacotherapy and surgery are well understood and documented, the use of non-pharmacologic therapy also plays a vital role in treating the patient. Recognition of risk factors and non-motor symptoms will help detect the disease earlier. Use of non-pharmacologic interventions, such as physical, occupational, or speech therapy, will afford the patient increased quality of life. The websites listed in Table 2 include information, resources, support groups, research, etc. in the treatment of Parkinson's Disease.

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The authors, the Ohio Pharmacists Foundation and the Ohio Pharmacists Association disclaim any liability to you or your patients resulting from reliance solely upon the information contained herein. Bibliography for additional reading and inquiry is available upon request.

This lesson is a knowledge-based CE activity and is targeted to pharmacists in all practice settings.

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Parkinson's Disease: Non-Pharmacological Interventions

1. All of the following are characteristics of Parkinson's Disease (PD) EXCEPT:

- a. the most prominent pathologic feature is dopamine neuron loss in the brain.
- b. the extensive broad-range pathology is likely responsible for non-motor features.
- c. psychotic non-motor symptoms of PD appear in a small number of PD patients.

2. All of the following are true about PD non-motor symptoms EXCEPT:

- a. they may have a greater impact on quality of life than motor symptoms.
- b. they do not lead to institutionalization.
- c. they can remain under-recognized by clinicians at all levels of practice.

3. A study of 101 PD patients revealed that during routine office visits neurologists failed to recognize sleep disturbance in:

- a. 10% of patients.
- b. 20% of patients.
- c. 30% of patients.
- d. 40% of patients.

4. In a group of 242 PD patients who completed a validated questionnaire that listed 30 non-motor symptoms, which of the following was the most often undeclared?

- a. Delusions
- b. Diplopia
- c. Depression
- d. Dysphagia

5. Anosmia is listed within which of the following categories of non-motor symptoms of PD?

- a. Neuropsychiatric
- b. Sleep disorders
- c. Autonomic
- d. Sensory deficits

6. All of the following are true about deep brain stimulation (DBS) in PD EXCEPT:

- a. the procedure is irreversible.
- b. it is the state-of-the-art neurosurgical treatment for motor impairments of PD.
- c. FDA has approved both unilateral and bilateral DBS.

Completely fill in the lettered box corresponding to your answer.

- | | | |
|--------------------|---------------------|---------------------|
| 1. [a] [b] [c] | 6. [a] [b] [c] | 11. [a] [b] [c] [d] |
| 2. [a] [b] [c] | 7. [a] [b] [c] [d] | 12. [a] [b] [c] [d] |
| 3. [a] [b] [c] [d] | 8. [a] [b] [c] | 13. [a] [b] [c] |
| 4. [a] [b] [c] [d] | 9. [a] [b] [c] [d] | 14. [a] [b] [c] [d] |
| 5. [a] [b] [c] [d] | 10. [a] [b] [c] [d] | 15. [a] [b] [c] |

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1. Rate this lesson: (Excellent) 5 4 3 2 1 (Poor)
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7. All of the following are risks of DBS surgery EXCEPT:

- a. stroke.
- b. intracranial hemorrhage.
- c. lead migration.
- d. myocardial infarction.

8. All of the following are true about physical or occupational therapy in PD patients EXCEPT:

- a. they improve quality of life.
- b. benefit is based largely on patient reports.
- c. benefit remains unsubstantiated.

9. Lee Silverman technique is proven to improve:

- a. muscle strength.
- b. voice quality.
- c. restful sleep.
- d. walking speed.

10. Because constipation is often an issue in PD patients, adequate intake of which of the following is recommended?

- a. Protein
- b. Fiber
- c. Carbohydrates
- d. Fats

11. Which of the following has been associated with a decreased risk of PD?

- a. Male gender
- b. Aspirin usage
- c. Increasing age
- d. High blood urate levels

12. Studies have shown cigarette smoking appears to reduce the risk of PD by approximately:

- a. 15 percent.
- b. 24 percent.
- c. 36 percent.
- d. 44 percent.

13. All of the following are true about caffeine EXCEPT:

- a. decaffeinated coffee does not appear to modify PD onset.
- b. heavy coffee drinkers have a lower risk of PD compared to those who ingest little or none.
- c. PD risk reduction with caffeine consumption is somewhat higher in women.

14. All of the following increase the risk of PD for persons exposed to them EXCEPT:

- a. azomite.
- b. maneb.
- c. permethrin.
- d. rotenone.

15. All of the following are true about traumatic brain injury on the risk of PD EXCEPT:

- a. this injury may require additional risk or susceptibility factors to cause PD.
- b. unconsciousness for one minute causes a two-fold increased PD risk.
- c. it may increase the susceptibility of dopamine neurons to additional insults.

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