Parkinson Disease: Systemic and Orofacial Manifestations, Medical and Dental Management

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ABSTRACT

Background. More than 1.5 million Americans have Parkinson disease (PD), and this figure is expected to rise as the population ages. However, the dental literature offers little information about the illness.

Types of Studies Reviewed. The authors conducted a MEDLINE search using the key terms "Parkinson's disease," "medical management" and "dentistry." They selected contemporaneous articles published in peer-reviewed journals and gave preference to articles reporting randomized controlled trials.

Results. PD is a progressive neurodegenerative disorder caused by loss of dopaminergic and nondopaminergic neurons in the brain. These deficits result in tremor, slowness of movement, rigidity, postural instability and autonomic and behavioral dysfunction. Treatment consists of administering medications that replace dopamine, stimulate dopamine receptors and modulate other neurotransmitter systems.

Clinical Implications. Oral health may decline because of tremors, muscle rigidity and cognitive deficits. The dentist should consult with the patient's physician to establish the patient's competence to provide informed consent and to determine the presence of comorbid illnesses. Scheduling short morning appointments that begin 90 minutes after administration of PD medication enhances the patient's ability to cooperate with care. Inclination of the dental chair at 45°, placement of a bite prop, use of a rubber dam and high-volume oral evacuation enhance airway protection. To avoid adverse drug interactions with levodopa and entacapone, the dentist should limit administration of local anesthetic agents to three cartridges of 2 percent lidocaine with 1:100,000 epinephrine per half hour, and patients receiving selegiline should not be given agents containing epinephrine or levonordefrin. The dentist should instruct the patient and the caregiver in good oral hygiene techniques.

Key Words. Parkinson disease; local anesthetics; saliva.
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Parkinson disease (PD) is a progressive, disabling neurodegenerative disorder that is characterized by tremors, slowness of movement (bradykinesia), muscle rigidity, postural instability and gait disturbance.[1-3] These symptoms negatively affect the patient's quality of life, often resulting in loss of employment, inability to drive a car and impairment in activities of daily living, including the ability to perform bodily and oral hygiene adequately.

PD affects more than 1 million Americans,[4] and the associated neuromuscular and cognitive deficits enhance the progression of dental disease, impair home care regimens and encumber in-office dental treatment. To acquaint dentists with the latest medical advancements in the treatment of the disorder and dental management techniques, we conducted a literature review. We conducted a MEDLINE search using the key terms "Parkinson's disease," "medical management" and "dentistry." We derived the

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information we present in this article from contemporaneous articles published in peer-reviewed journals, giving preference to articles reporting the results of randomized clinical trials.

PARKINSON DISEASE: SYMPTOMS, ETIOLOGY AND EPIDEMIOLOGY

Symptoms. ***Motor symptoms.*** Parkinsonian tremors generally start in a hand and often appear as if the person is slowly "rolling a pill" between the thumb and fingers. The tremors then may spread to involve the leg, face, tongue and mandible. These typically are resting tremors, seen in the relaxed state, with the amplitude decreasing during purposeful movements. In addition to bradykinesia, patients also evidence a relative inability to initiate voluntary and involuntary movements (akinesia). Akinesia is seen as reduced facial expression, blinking and swallowing; difficulty with dressing, bathing and arising from a chair; and an overall sense of weakness or fatigue. Rigidity of skeletal muscles arising from increased tone is expressed as the resistance to passive movement of an extremity around a joint such as the wrist and elbow. Resistance may be smooth or ratchetlike (evoking the action of a cogwheel).

Postural instability is expressed as a feeling of imbalance. Gait disturbances are apparent, with a stooped posture, head leaning forward on the trunk, knees and hips bent and a reduced arm swing. Patients with PD find it difficult to initiate walking, and their steps are short and shuffling. However, once they begin to walk, they tend to walk uncontrollably faster to keep from falling (festination). Turning may decompose into several steps instead of being a single pivoting maneuver. Postural instability and gait difficulties ultimately may be the most disabling aspect of PD, leading to falls that cause injury (including injury to orofacial structures) and jeopardize the patient's independence.[5]

Nonmotor symptoms. Patients with PD also have a wide spectrum of nonmotor symptoms. Autonomic dysfunction, noted in more than one-half of these patients, manifests itself as variations in blood pressure, particularly orthostatic hypotension; cardiac dysrhythmias; excessive sweating; and bladder, bowel (constipation) and sexual dysfunction.[6] Sleep disorders also are common and include insomnia, sleep apnea and sleep fragmentation with resultant daytime drowsiness.

Behavioral symptoms. PD also produces a wide range of behavioral disturbances: depression, cognitive impairment and dementia arising from the primary illness, as well as psychosis resulting from the use of dopaminergic PD medications.[7] Depression of some degree (major depression, minor depression or dysthymia) affects approximately one-half of patients but is often difficult to diagnose because slowness, lack of facial expression (sometimes interpreted as sadness), sleeplessness, weight loss and loss of energy are signs of both depression and PD. Almost all patients display mild cognitive deficits; these often begin early in the disease process. Late in the disease process, 10 to 30 percent meet the criteria for dementia, which manifests as problems with planning, sequencing, visual motor skills, visuospatial skills, recall of verbal and nonverbal material, and verbal fluency.[8] Medication-induced psychosis affects approximately one-quarter of patients and usually begins with visual hallucinations (often of a stranger sitting quietly and observing the patient). The psychosis may then progress to include paranoid delusions, often involving spousal infidelity and money loss. Persistent psychotic symptoms are associated with greater burdens on caregivers and nursing home placement.[9]

Diagnosis and etiology. ***Diagnosis.*** The diagnosis of PD is based on careful history taking, physical examination and, in some instances, a positive sustained response to dopaminergic medications.[10] Laboratory tests and imaging studies are not used routinely.[11] A definitive diagnosis requires postmortem confirmation. The Hoehn and Yahr[12] scale (Table 1), which is based on the extent of motor symptoms, can assist clinicians in staging the disorder.

Etiology. The motor dysfunction seen in PD arises from damage to and loss of between 60 and 70 percent of the neurons that store and release dopamine in the substantia nigra.[13] This loss of neurons results in the depletion of the neurotransmitter in the striatum (caudate and putamen) of the basal ganglia, where it is needed to produce smooth and coordinated body movement.

The behavioral manifestations of PD are due to varied patterns of degeneration in dopaminergic and nondopaminergic (noradrenergic and seroton-

ABBREVIATION KEY. MAOI: Monoamine oxidase inhibitor. **PD:** Parkinson disease.

TABLE 1

Hoehn and Yahr scale*: five stages of Parkinson disease.

STAGES	SYMPTOMS
I (Mild/Early Disease)	Only one side of the body is affected (for example, tremor of one limb), usually with minimal or no functional impairment
II	Both sides of the body are affected but posture and balance remain normal
III (Moderate Disease)	Both sides of the body are affected, and there is mild imbalance when standing or walking; however, the person remains independent
IV (Advanced Disease)	Both sides of the body are affected, and there is disabling instability while standing or walking; the person in this stage requires substantial help and cannot live alone
V	Severe, fully developed disease is present; the person often is cachectic, restricted to bed or wheelchair unless aided

* Source: Hoehn and Yahr.[12]

ergic) neuronal systems located in the hippocampus and the amygdala.[14,15] Autonomic nervous system symptoms arise because of parasympathetic cholinergic failure (dry mouth, constipation, urinary retention, erectile dysfunction), sympathetic cholinergic failure (decreased sweating) and sympathetic noradrenergic failure (orthostatic hypotension).[16,17]

The causes of neuronal cell death are poorly understood; thus, the disorder occasionally is termed "idiopathic PD" or "sporadic PD." Although most cases do not result from simple inheritance patterns, genetic abnormalities have been identified. However, it is hypothesized that PD results from a complex interaction between multiple predisposing genes and uncertain personal factors (for example, head injury) and environmental factors (for example, exposure to pesticides or the neurotoxin 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine).

Of specific interest is the identification of factors responsible for the development of intraneuronal cytoplasmic fibrillar aggregates of the protein α-synuclein. These aggregates, found in the surviving neurons and known as "Lewy bodies," are believed to be involved in the process that damages and destroys the neurons.[18] The possible neuroprotective effects provided by caffeine in coffee and tea, nicotine in cigarettes and use of nonsteroidal anti-inflammatory drugs have been linked to a reduced occurrence of the disorder.[19-21]

Epidemiology. The National Parkinson Foundation[4] reported that in 2009, in the United States, approximately 1.5 million people have the disease and that an estimated 60,000 new cases are diagnosed each year. While the condition usually develops in people older than 65 years, 15 percent of those diagnosed are younger than 50 years. PD affects men and women in almost equal numbers. Although therapeutic regimens can ameliorate the symptoms, the disease continues to be associated with progressive disability, albeit at a rate that varies greatly from one person to another. Increased mortality depends on disease duration and often results from aspiration pneumonia, pressure sores and urinary tract infections.[22]

MEDICAL MANAGEMENT OF PARKINSON DISEASE

Administration of medications (Table 2) usually is initiated when symptoms interfere with the patient's level of functioning.[23] Therapy, however, has not been proved to alter the underlying progression of PD.

Orally administered levodopa crosses the blood-brain barrier, is taken up by the remaining neurons of the substantia nigra and is transformed into dopamine, thereby facilitating synaptic transmissions. The medication almost always is administered in combination with carbidopa and entacapone, which prevent levodopa from being converted to dopamine in the systemic circulation and, thus, increase the amount of levodopa available to enter the brain.[24]

Long-term (five years or longer) administration of levodopa results in 50 to 75 percent of patients' becoming partially unresponsive to the medication, with fluctuations in symptoms often seen during a 24-hour cycle. The switch between relative freedom from symptoms with good motor function (the "on" period, when the medication is working) to periods of severe immobility (the "off" period, when the medication is not working) is common. Within five years of beginning levodopa treatment, 50 to 75 percent of patients also develop levodopa-induced dyskinesias, which manifest as abnormal, involuntary movements such as purposeless dancelike movements (chorea) of the extremities, trunk, head (wagging), face (grimacing) or tongue (lingual-labial

TABLE 2

Medications used to treat Parkinson disease: mechanism of action and adverse systemic reactions.

GENERIC NAME	TRADE NAME (MANUFACTURER)	MECHANISM OF ACTION	ADVERSE SYSTEMIC REACTIONS
Amantadine	Symmetrel (Endo Pharmaceuticals, Chadds Ford, Pa.)	Amantadine is an antiviral agent also featuring pharmacological properties that increase dopamine release, inhibit dopamine reuptake, stimulate dopamine receptors and possibly exert central anticholinergic effects	Confusion, hallucinations and a worsening of glaucoma and congestive heart failure; elderly patients are more susceptible to these adverse side effects
Benztropine	Cogentin (Merck & Co., West Point, Pa.)	Benztropine is an anticholinergic medication useful in treating tremor but less useful in alleviating muscle rigidity and bradykinesia; it is effective because it counteracts the cholinergic sensitivity that arises in response to dopamine depletion	Confusion, blurred vision, worsening of glaucoma, urinary retention, cardiac dysrhythmia; elderly patients are more susceptible to these adverse side effects
Cabergoline	Dostinex (Pharmacia & Upjohn, Peapack, N.J.) (off-label use)	Cabergoline is a dopamine agonist that bypasses the depleted neurons in the substantia nigra and provides long-lasting direct stimulation of dopamine receptors	Orthostatic hypotension, nausea, vomiting, confusion, and hallucinations; elderly and cognitively impaired patients are more susceptible to these adverse effects; cabergoline, an ergot-related dopamine agonist, has been associated with the development of stiff fibrotic heart valve leaflets, which are unable to close completely, thereby possibly increasing the risk of endocarditis
Levodopa	Numerous names and multiple manufacturers	Levodopa is a dopamine precursor, which is taken up by the remaining neurons in the substantia nigra and transformed into dopamine, thereby facilitating synaptic transmissions; the medication ameliorates bradykinesia and muscle rigidity, but postural instability and gait disturbances are somewhat less responsive	Nausea, orthostatic hypotension; levodopa treatment results in 50 to 75 percent of patients' becoming less responsive to the medication within five years of starting treatment, as well as developing new involuntary movements (dyskinesias)
Levodopa and Carbidopa	Sinemet (DuPont Pharmaceuticals, Wilmington, Del.)	Carbidopa inhibits the decarboxylase enzyme that converts levodopa to dopamine in the systemic circulation and liver, thus increasing the amount of levodopa available to cross the blood-brain barrier; carbidopa also decreases the adverse systemic effects of levodopa	None of major significance
Levodopa and Carbidopa and Entacapone	Stalevo (Novartis, East Hanover, N.J.)	Entacaptone reduces O-methylation of levodopa in the gastrointestinal tract, thereby increasing the amount of levodopa available to cross the blood-brain barrier	None of major significance
Pramipexole	Mirapex (Pharmacia & Upjohn)	Pramipexole is a dopamine agonist, which bypasses the depleted neurons in the substantia nigra and provides long-lasting direct stimulation of dopamine receptors	Orthostatic hypotension, nausea, vomiting, confusion, hallucinations; elderly and cognitively impaired patients are more susceptible to these adverse effects
Rasagiline	Azilect (Teva Neuroscience, Kansas City, Mo.)	Rasagiline blocks monoamine oxidase B from metabolizing dopamine in the brain, thereby potentiating and prolonging the effects of the often coadministered levodopa; the metabolism of this medication does not result in the production of amphetamine byproducts	Less than 10 percent of patients develop dyskinesia and headache
Ropinirole	Requip (GlaxoSmithKline, Research Triangle Park, N.C.)	Ropinirole is a dopamine agonist that bypasses the depleted neurons in the substantia nigra and provides long-lasting direct stimulation of dopamine receptors	Orthostatic hypotension, nausea, vomiting, confusion, hallucinations; elderly and cognitively impaired patients are more susceptible to these adverse effects
Selegiline	Eldepryl (Somerset Pharmaceuticals, Tampa, Fla.)	Selegiline blocks monoamine oxidase B from metabolizing dopamine in the brain, thereby potentiating and prolonging the effects of the often coadministered levodopa; selegiline is metabolized in the liver to l-methamphetamine and l-amphetamine	Cardiac dysrhythmias and possible sensitivity to sympathomimetics because of the presence of the amphetamine byproducts
Trihexyphenidyl	Artane (Lederle Laboratories, Pearl River, N.Y.)	Trihexyphenidyl is an anticholinergic medication useful in treating tremor but less useful in alleviating muscle rigidity and bradykinesia; it is effective because it counteracts the cholinergic sensitivity that arises in response to dopamine depletion	Confusion, blurred vision, worsening of glaucoma, urinary retention, cardiac dysrhythmias; elderly patients are more susceptible to these adverse side effects

dyskinesia) or sustained abnormal contractions (dystonia) of the feet and the muscles of mastication.[25,26] These movements expend large amounts of energy and, when combined with chewing and swallowing problems, may result in significant weight loss.[27]

Selegiline often is used in combination with levodopa, and it inhibits the metabolism of dopamine in the brain, thereby prolonging the effect of levodopa.[28] The medication, however, is metabolized in the liver to l-methamphetamine and l-amphetamine, both of which are implicated in causing cardiac dysrhythmias and sensitivity to sympathomimetics.[29] Rasagiline similarly blocks the metabolism of dopamine in the brain but is not associated with amphetamine metabolic byproducts.[30]

The continued loss of neuronal cells that metabolize levodopa to dopamine and the complications associated with levodopa use have led to the development of dopamine agonists. These drugs bypass the depleted nigral neurons and stimulate dopamine receptors directly. These medications can be used alone as first-line agents or in combination with levodopa. Most frequently administered are pramipexole, ropinirole and cabergoline. Of specific concern to dentists is cabergoline, which is associated with the development of stiff fibrotic heart valve leaflets that are unable to close completely, possibly increasing the risk of endocarditis.[31] Amantadine, a medication with both dopamine-agonist and antiviral properties, is prescribed by some clinicians because of its effectiveness in the treatment of tremor, hypokinesia and postural instability.

Anticholinergic agents (for example, trihexyphenidyl and benztropine) are used by some clinicians to treat tremors, but these medications are less useful in alleviating rigidity and bradykinesia. Older patients frequently are intolerant of these anticholinergics.[32] Anticholinergic agents' adverse effects likewise limit the drugs' effectiveness in older patients.[33]

Surgery for medically refractory disease consists of deep-brain stimulation of the motor components of the subthalamic nucleus. Electrodes connected to a pulse generator are passed through surgically created burr holes in the skull and into the subthalamic nucleus. Electrical stimulation results in inhibition of movement and sustained improvements in tremor, bradykinesia, muscle rigidity, levodopa-related motor complications and decrease in the required dose of dopaminergic medication.[34-36]

Management of associated symptoms. Depression is treated with selective serotonin reuptake inhibitors, tricyclic antidepressant agents, venlafaxine or bupropion. Hallucinations and paranoid delusions arise frequently secondary to dopaminergic therapy, which causes overactivity in the pathways to the limbic system and cerebral cortex. These psychotic symptoms are managed initially by reduction in the dosage of medication.[37] Persistent symptoms are managed by administering atypical neuroleptic agents (such as quetiapine). Orthostatic hypotension may arise as a consequence of the dopaminergic medication regimen. Dosage modification, use of support stockings and increased intake of fluids and sodium often are effective in ameliorating the problem.

Parkinsonian tremor and rigidity of the orofacial musculature may induce orofacial pain, temporomandibular joint discomfort, cracked teeth and dental attrition.

OROFACIAL FINDINGS AND INTERDISCIPLINARY MANAGEMENT

The orofacial complex exhibits numerous signs of PD. They occasionally are seen early in the disease process and, at that point, usually respond to dopaminergic medication; however, they usually are not seen until later in the disease process, when they are refractory to treatment. The usual blink rate of 12 to 20 per minute is markedly reduced, there is limitation in upward gaze, and a masklike, impassive facial appearance arises because of a reduction in the movements of the small facial muscles (hypomimia).[38,39] Parkinsonian tremors are seen in the forehead, eyelids and lip and tongue musculature and in involuntary mandibular movements. Tremor and rigidity of the orofacial musculature may induce orofacial pain, temporomandibular joint discomfort, cracked teeth and dental attrition and may create difficulties in controlling and retaining dentures.[40]

Initially the voice of a patient with PD softens (hypophonia) and speech seems hurried, monotonous and mumbling. As the disease progresses, the voice becomes less audible and, finally, the patient only whispers. These vocal problems arise from rigidity and hypokinesia (that is, reduced

frequency and amplitude of movement) of the respiratory, phonatory and articular apparatus.[41]

People with PD tend to have a gaunt facial appearance and be underweight because of excessive energy expenditures resulting from involuntary muscle movements and an inadequate intake of food.[42] A disease-related loss in the sense of smell (hyposmia or anosmia), possibly arising from a loss of neurons in the anterior olfactory nucleus, and a PD-associated impairment in taste have been implicated in patients' loss of interest in ingesting food.[43,44] Compounding this latter issue is an alteration in taste that commonly results from using many of the PD medications.[45] Further complicating this issue is the prolonged time that it takes patients with PD to consume food because of manipulative difficulties in transporting it to the mouth, slowness in chewing (bradykinesia), reduced tongue movement with consequent loss of bolus formation and propulsion of the food to the back of the oral cavity, and difficulty in swallowing (dysphagia) because of pharyngeal motor deficits.[46-48] To stem the often-noted weight loss, speech therapists teach patients various strategies to compensate for the slowed reflexes. Dietitians recommend cutting food into smaller pieces, as well as altering its texture and consistency to facilitate swallowing.[27,49-52]

To provide safe therapeutic strategies, the dentist must consult with the patient's physician to identify any need for modifications of typical treatment practices.

In a small (three-subject) uncontrolled study, use of dental implant–supported prostheses was associated with marked subjective improvement in chewing ability and an average weight gain of 5 pounds.[53] The authors concluded that implant-supported overdentures overcome some of the eating difficulties encountered by edentulous patients who wear conventional complete dentures and have PD-associated deficits in their oropharyngeal musculature, tremors and rigidity of orofacial musculature and changes in quality and quantity of saliva.[54]

Drooling of saliva from the corners of the mouth—with the often-associated angular cheilosis, skin irritation and odor—is apparent in approximately 75 percent of people with PD. Most researchers believe that this drooling probably results from a PD-related inability to swallow efficiently and with normal frequency, an inability to close the mouth fully and an anteriorly flexed head position.[55-62] Treatment, however, is directed toward decreasing salivary flow and includes administration of anticholinergic medications (such as glycopyrrolate and benztropine), injection of botulinum toxin type A into the salivary glands at approximately five-month intervals, transposition of salivary gland excretory ducts and salivary gland excision and irradiation.[62-68]

Burning mouth syndrome—manifesting as discomfort of the tongue, floor of the mouth, lips and cheek—is a complaint in one-quarter of people with PD. Although the burning sensation has been attributed to a variety of factors (for example, xerostomia, parafunctional purposeless chewing activity, depression and levodopa therapy), the exact cause or causes remain obscure.[69]

The extent of dental caries in patients with PD is also somewhat controversial. Results of two studies indicate that the caries rate among patients with PD does not appear to be greater than that in like-aged patients without PD.[61,70] However, other researchers have claimed that there is an increased incidence of root caries in patients with PD.[71] Further complicating this issue is the increased craving for sweets among patients with PD, as well as a greater frequency of *Streptococcus mutans* noted in plaque samples obtained from this population.[72] Researchers have reported that the extent of periodontal disease in patients with PD appears to be significantly greater than that among control subjects and that it may stem from impaired oral hygiene owing to compromised manual dexterity, which results from loss of fine motor movements.[73] The extent of edentulism also is significantly greater among those with PD than among control subjects, possibly because of advanced periodontal disease.[74]

The U.S. Food and Drug Administration medication package insert accompanying each of the medications used in treating PD and an analysis of the current medical literature reveals that these medications cause numerous adverse orofacial reactions of concern to dentists (Table 3[75-78]). Parkinsonian tremors of the orofacial musculature and the use of levodopa-containing medications may cause bruxism; therefore, the dentist should examine the dentition of a patient with PD

TABLE 3

Adverse orofacial reactions to medications used to treat Parkinson disease.*

GENERIC NAME (TRADE NAME†)	XEROSTOMIA‡	SIALADENITIS	DYSGEUSIA	STOMATITIS	GINGIVITIS	GLOSSITIS
Amantadine (Symmetrel)	+	0	0	0	0	0
Benztropine (Cogentin)	+	0	0	0	0	0
Cabergoline (Dostinex)	+	0	0	0	0	0
Levodopa With Carbidopa (Sinemet)	+	0	+	0	0	+
Levodopa With Carbidopa and Entacapone (Stalevo)	+	0	+	0	0	+
Rasagiline (Azilect)	+	0	0	0	0	0
Ropinirole (Requip)	+	0	0	0	+	+
Selegiline (Eldepryl)	0	0	+	0	0	0
Trihexyphenidyl (Artane)	+	0	0	0	0	0

* Sources: Physicians' Desk Reference,[75] McEvoy,[76] Wynn and colleagues,[77] "Sublingual selegiline: new formulation—new risk of oral adverse effects."[78]
† Manufacturers are listed in Table 2. Trade names given are examples only.
‡ Plus sign (+) indicates "yes"; zero (0), "no."

for excessive loss of tooth structure.[79,80] Interventions may include consultation with the patient's physician to discuss the possibility of changing the medication to one not associated with causing bruxism or, if necessary, fabricating a prosthetic appliance to protect the dentition.[81]

The ergot-derived dopamine agonist cabergoline has been implicated in damaging heart valves and possibly predisposing patients to endocarditis.[82] (Another ergot-derived dopamine agonist, pergolide, has been removed from the market.) The dentist should query the patient, his or her caregiver and his or her physician as to use of these medications. People who have bacterial endocarditis or a prosthetic cardiac valve meet the American Heart Association criteria requiring an antibiotic prophylaxis regimen during dental procedures that involve manipulation of gingival tissue or the periapical region of teeth or perforation of the oral mucosa.[83]

DENTAL TREATMENT

To provide safe therapeutic strategies, the dentist must consult with the patient's physician to identify any need for modifications of typical treatment practices (Table 4). The necessary information includes disease stage, the patient's cognitive ability to provide consent for treatment, disease prognosis, drug regimen and identification of other medical conditions (for example, a history of endocarditis) that may influence treatment. Caregivers should be involved in the consent process even when patients are competent, because their informational support enhances patient comprehension and recall of information contained in the consent form.[84] In the event the physician believes the patient incapable of providing consent, the patient's legal guardian must provide it instead.

The dentist should devise a realistic treatment plan for a patient with PD. Restoration of oral health is best completed as early as possible in the PD process, because the patient's ability to cooperate diminishes as functional and cognitive abilities decline.[85] The plan also must take into account the patient's prognosis in relation to immediate versus long-term dental needs (for example, giving greater weight to removing a jagged nonrestorable tooth that is damaging the adjacent mucosa than to repairing early recurrent caries surrounding a crown), the patient's desires, if expressible, and the caregiver's desires, if reasonable.

Patients with PD should be scheduled for short appointments (no longer than 45 minutes) in the early morning when they usually are least bothered by their symptoms and when their medication is most effective. The peak effectiveness of most PD medications begins within 60 to 90 min-

TABLE 3 (CONTINUED)

TONGUE EDEMA	COATED TONGUE	BRUXISM	MISCELLANEOUS
0	0	0	Nasal dryness
0	0	0	Vomiting, throat and nasal dryness
0	0	0	Toothache, rhinitis, throat irritation, periorbital edema
0	0	+	Glossodynia, trismus, sialorrhea, "dark" saliva, pigmentation of teeth, dysphagia, bruxism, trismus
0	0	+	Sialorrhea, "dark" saliva, pigmentation of teeth, bruxism, trismus
0	0	0	Neck pain, vertigo, rhinitis, conjunctivitis
+	0	0	Falling asleep in dental chair (secondary to sedation), toothache, headache, pharyngitis, tinnitus
0	0	+	Sublingual oral ulcerations, burning lips, burning mouth, facial grimacing, supraorbital pain
0	0	0	Suppurative parotitis secondary to excessive dryness of mouth

utes after administration. Before entering the dental operatory, these patients should empty their bladders, because PD often is associated with urinary urgency and incontinence.[86] Assistance into and out of the dental chair is critical in light of mobility issues. The dentist should raise the dental chair slowly, allowing the patient to adjust to the upright sitting position, to accommodate PD-associated autonomic dysfunction; this dysfunction frequently gives rise to orthostatic hypotension and the possibility of a syncopal episode and resulting injury.[87,88]

A subgroup of patients—often those with a history of younger age at disease onset, higher intake of dopaminergic drugs, greater use of experimental drugs and higher intake of alcohol—occasionally may make inappropriate sexual advances or remarks to members of the dental office staff. This behavior appears to be caused by "spillover" from the effects of dopamine on the brain's reward system.[89,90]

Patients with cognitive deficits can become frustrated and irritable when confronted with unfamiliar circumstances or with questions, instructions or information that they do not understand. However, with the appropriate psychological set on the part of the dentist and the ancillary staff, most routine care can be provided with only minor modification. Routine procedures such as the taking of blood pressure or the application of a rubber dam may arouse intense anxiety or irrational behavior in a patient who has not been forewarned. The dentist should begin each conversation with self-identification ("I am Dr. Smith, your dentist."); use questions that require simple yes/no responses; explain procedures before performing them ("I would like to examine your teeth."); use simple words, short sentences and nouns rather than pronouns; and limit use of the face mask as much as possible. Smiling, direct eye contact and gently touching help convey appropriate cues. The patient's caregiver can sit next to him or her in the operatory to help alleviate stress and anxiety, provide a distraction and hold the patient's hand if needed.

Drugs: interactions and adverse reactions. Dentists must be aware of a number of potentially significant adverse interactions that may occur when prescribing dental therapeutic agents to patients receiving medication for PD. Practitioners must take precautions when administering local anesthetic agents containing epinephrine in patients being treated with levodopa-containing medications and medications containing entacapone, because these patients may experience an exaggerated effect on blood pressure and heart rate.[91] Therefore, it is prudent to administer no more than 0.05 milligrams of epinephrine—as is found in three cartridges of 2 percent lidocaine with 1:100,000 epinephrine—per 30-minute period, with careful aspiration to avoid intravascular administration. Monitoring of the patient's vital signs also is recommended.[92] Entacapone is excreted via bile, so the dentist should be cautious when prescribing erythromycin and ampicillin, both medications known to interfere with biliary excretion.

Patients being treated with the monoamine

TABLE 4

Dental implications and treatment modifications for patients with Parkinson disease.

COMPONENTS OF DENTAL TREATMENT	MODIFICATION, IF INDICATED
Patient's First Visit and Recall Visits	Consult with patient's physician to determine Parkinson disease (PD) stage, cognitive ability to provide consent, and presence of comorbid illnesses requiring treatment modification
Treatment Planning	Major dental interventions should be accomplished early in the course of PD because of the disease's progressive nature; in later stages of disease, a conservative approach is appropriate
Initiation of Treatment	Appointments should be short (45 minutes) and should begin approximately 90 minutes after administration of PD medication; after patient empties bladder, assist him or her into dental chair, use bite prop to keep mouth open, incline chair at 45° and use rubber dam and high-volume oral evacuation to protect airway
Administration of Local Anesthetic Agent	For patients receiving levodopa and/or entacapone, limit administration to three cartridges of 2 percent lidocaine with 1:100,000 epinephrine per 30-minute period to avoid tachycardia and hypertension; avoid prescribing erythromycin and ampicillin so as not to interfere with biliary excretion of entacapone For patients receiving selegiline, do not administer agents containing epinephrine or levonordefrin because of adverse interaction, which may result in severe hypertension
Administration of Pain Medication	For patients receiving selegiline or rasagiline, do not prescribe or administer meperidine hydrochloride because of a severe interaction that may result in severe hyperthermia, hypertension and tachycardia; prescribe or administer other narcotic analgesics at one-half usual dosage
Prevention of Oral Disease	The patient, as well as the caregiver, should be instructed in good oral hygiene techniques because cognitive impairment and loss of motor skills during the disease process may hamper the patient's ability to comply autonomously

oxidase inhibitor (MAOI) rasagiline can receive local anesthetic solutions containing levonordefrin or epinephrine because MAOIs do not potentiate the pressor or cardiac effects of these direct-acting catecholamines. However, the MAOI selegiline is unique in that it undergoes extensive first-pass metabolism to l-methamphetamine and l-amphetamine, and interactions with levonordefrin or epinephrine may result in severe hypertension.[93] Therefore, it is prudent to use a local anesthetic agent devoid of a vasoconstrictor agent. Dentists should not prescribe meperidine hydrochloride to patients being treated with the MAOIs selegiline and rasagiline because of a potentially toxic interaction in which severe hyperthermia, hypertension and tachycardia may develop.[94] MAOIs also increase the potency of other narcotic analgesic agents, so the dentist would be prudent to prescribe only one-half the usual dosage of the narcotic agent.

Practical aspects of treatment. Dental treatment of a patient with PD often is hampered by the patient's inability to keep his or her mouth open, manage saliva, restrict tongue movements and keep his or her head from moving. Placement of an extraoral ratchet-type prop or intraoral rubber bite block will assist in keeping the mouth open. To facilitate the patient's swallowing, the dentist should avoid inclining the dental chair more than 45°. An aspirator tip placed under a rubber dam and stabilized by an assistant (while he or she simultaneously steadies the patient's head) will assist the patient in managing saliva while the dam prevents contamination of the restoration placement. The dam also protects the tongue from injury; patients with PD often have weakness and rigidity of the tongue that prevents them from keeping it in a safe position.[95,96] Lastly, the dam also provides protection of the airway, which is vital in patients with PD because they are in danger of aspiration secondary to a diminished cough reflex.[97] However, the dental team must be vigilant when using the rubber dam and have a high-volume oral evacuation system at hand because saliva may build up excessively in the mouth behind the dam. Four- to six-handed dentistry may be required to provide safe care for these patients.

Glass ionomers and resin-modified glass ionomers are most appropriate for the restoration of carious lesions in patients with PD, including lesions that involve the root surfaces, because they bond to both dentin and cementum and release fluoride.

In the late stages of PD, the patient may be unable to cooperate during most forms of treatment. For these patients, care is best provided in the dental office with the patient's being under

intravenous sedation administered by a trained anesthesiologist or under general anesthesia in the operating room of a surgical center or hospital.

Oral hygiene. Maintenance of good oral hygiene is paramount for people with PD. Patients with no cognitive impairments should be given instructions in proper toothbrushing and flossing methods that maximize removal of dental plaque. However, dental professionals must recognize that these patients may have subtle cognitive deficits as well as depression, which may impair their ability to perform all aspects of personal hygiene, and that toothbrushing and flossing may be particularly difficult because of these deficits as well as because the disease often precludes repetitive movements.[98-100] Use of the Collis-Curve toothbrush (Collis-Curve Toothbrush, Brownsville, Texas) and mechanical toothbrushes, as well as caregiver assistance with brushing, may help these patients maintain their teeth. Caregivers should receive oral and written instructions in proper toothbrushing and flossing methods and how to apply topical sodium fluoride (5,000 parts per million) to the patient's teeth with a toothbrush or sponge applicator. Oral-rinse topical agents such as chlorhexidine gluconate may not be appropriate, because many patients with PD may not be able to swish and expectorate to minimize ingestion. Lastly, artificial salivary products should be prescribed for patients showing signs of xerostomia.

The dentist should provide a clinical examination, oral prophylaxis and application of topical fluorides, including a 5 percent fluoride varnish, to the patient with PD at follow-up visits every three months.[101] He or she also should address defects in the natural dentition or prostheses during these recall visits.

CONCLUSION

PD represents a growing burden on the health care system because of its occurrence among the increasing proportion of elderly people in the United States. Dentistry, in concert with medicine, has much to offer patients with this disease. Dentists familiar with the manifestations of the illness and its medical management can confidently offer these patients appropriately timed dental treatment options. ■

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