Parkinson's Disease: Early Diagnosis and Management

Matthew B. Stern, MD
Philadelphia, Pennsylvania

Parkinson's disease is a common neurodegenerative disorder affecting approximately 1% of the population over the age of 50 years. There is no known cure for Parkinson's disease, but research gains over the last two decades have been substantial, resulting in improved medications and therapeutic strategies for managing early symptoms and delaying the onset of serious disability. Particularly promising is current research suggesting the possibility of neuroprotective therapies that may ultimately be capable of slowing disease progression. Early and accurate diagnosis is especially important to optimize the benefits of new therapies.

Key words. Parkinson disease; drug therapy; levodopa, dopaminergic agents; selegiline. (*J Fam Pract 1993; 36:439-446*)

Parkinson's disease is a chronic, progressive neurologic disorder that was first described in 1817 by an English physician, James Parkinson, who documented cases of what he called a "shaking palsy."[1] Parkinson's disease is relatively common; it affects approximately 1% of the United States population over the age of 50 years, and is a major cause of neurologic disability. It is estimated that there are currently over 500,000 diagnosed cases in the United States; 50,000 new cases are reported annually.[2]

Diagnosing Parkinson's disease, especially at an early stage, is challenging because the cardinal manifestations—resting and postural tremor, bradykinesia, rigidity, and postural instability—are not immediately distinguishable from a spectrum of syndromes and diseases that make up a large and varied symptom complex now known as parkinsonism. Consequently, it is estimated that there are an additional 500,000 Americans who are in the early stages of Parkinson's disease but who are undiagnosed or misdiagnosed, and remain untreated.[2]

Early and accurate diagnosis of Parkinson's disease has taken on new urgency in light of current research that is leading both to improvements in current pharmacologic therapies and, possibly, to the ability to slow progression of the disease itself.

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From the Parkinson's Disease and Movement Disorders Center, The Graduate Hospital, University of Pennsylvania, Philadelphia. Requests for reprints should be addressed to Matthew B. Stern, MD, Department of Neurology, Suite 900, The Graduate Hospital, 1 Graduate Plaza, Philadelphia, PA 19146.

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Parkinson's Disease: An Overview

Parkinson's disease accounts for approximately 75% of all cases of parkinsonism. The average age of onset is 60 years of age, although 5% of patients are under age 40. The precise cause of Parkinson's disease is not yet clear, but the disease is linked to the degeneration of dopamine-producing cells in the substantia nigra, a pigmented region in the ventral midbrain. The disease may have a lengthy preclinical course, perhaps 30 years or more, before the appearance of symptoms.[3] It has been estimated that up to 80% of dopaminergic neurons are lost before the cardinal signs and symptoms of Parkinson's disease first appear.[4]

The primary and secondary features of Parkinson's disease are listed in Table 1. Primary signs and symptoms include tremor, rigidity, bradykinesia, and postural instability.

Tremor. Tremor is the presenting complaint for approximately 75% of patients. It is usually observed at rest with a frequency of 4 to 5 cycles per second and diminishes with voluntary activity or with sleep, but may be exacerbated by stress. Tremor in the hand is common, and is characterized by a "pill-rolling" type of movement between the thumb and forefinger. Early in the course of the disease, the tremor is generally unilateral, affecting one or more limbs; however, it may also be seen in the jaw, lips, and lower facial muscles.

Rigidity. "Cogwheel" or "ratchety" resistance to passive movement is often detected in the limbs of Parkin-

Table 1. Manifestations of Parkinson's Disease

Cardinal Manifestations
Tremor
Rigidity
Bradykinesia
Postural instability
Secondary Manifestations
Incoordination
Micrographia
Blurred vision
Impaired upward gaze
Blepharospasm
Glabellar reflex
Dysarthria
Dysphagia reflex
Sialorrhea
Masked facies
Hand and foot deformities
Dystonia
Edema
Scoliosis
Kyphosis
Pain and sensory symptoms
Seborrhea
Constipation
Urinary urgency, hesitancy, and frequency
Loss of libido
Impotence
Freezing
Dementia
Depression

From Stern MB, Hurtig HI, eds. The comprehensive management of Parkinson's disease. *New York: PMA Publishing, 1988. Reprinted with permission.*

Table 2. A Classification of Parkinsonism

Primary (ideopathic, Lewy body, nigral degeneration)
Secondary (symptomatic)
Infectious (postencephalitic, luetic, Creutzfeldt–Jakob disease)
Vascular (lacunar state)
Drug-induced (phenothiazines, butyrophenones, reserpine, metaclopramide)
Toxins (MPTP, manganese, carbon disulphide, cyanide, carbon monoxide)
Metabolic
Wilson's disease
Hepatocerebral degeneration
Hallervorden–Spatz disease
Hypoparathyroidism
Structural
Brain tumors
Hydrocephalus (normal pressure hydrocephalus)
Head trauma
Degenerative
Progressive supranuclear palsy
Multiple system atrophy
Striatonigral degeneration
Olivopontocerebellar atrophy
Shy–Drager syndrome
Spinocerebellar-nigral degeneration
Corticonigral degeneration with neuronal achromasia
Parkinson-dementia complex or Guam (with or without motor neuron disease)
Parkinsonism with amyotrophy
Senile gait apraxia
Alzheimer's disease

From Stern MB, Hurtig HI, eds. The comprehensive management of Parkinson's disease. *New York: PMA Publishing, 1988. Reprinted with permission.*

son's disease patients. There may be muscle discomfort or stiffness, and occasionally pain, if rigidity has become severe.

Bradykinesia. Slowness in initiating movement is one of the most disabling symptoms of Parkinson's disease. It is associated with facial masking, difficulty in rising from a chair, impaired manual dexterity, and difficulty in performing sequential or repetitive motor activities.

Postural instability. Patients with mild symptoms and unilateral disease may have only a slight reduction in arm swing (associated movement) and may drag their leg on the affected side. As the disease progresses, steps become short and shuffling (festination), associated arm movements are lost, and sudden "freezing" impairs turning and initiation of gait. Loss of postural reflexes leads to backward falls (retropulsion) or forward falls (propulsion) without cause. Once postural reflex loss becomes severe, the patient may ultimately be confined to a wheelchair or bed.

Challenges in Early Diagnosis

The early diagnosis of Parkinson's disease is often difficult because early symptoms can be nonspecific and appear gradually. Years before classic clinical manifestations appear, there may be subtle changes in personality; patients may feel slightly depressed or tire easily. Mild slowing, changes in handwriting and coordination, a feeling of weakness or nonspecific pain may be attributed to normal aging or other causes.

In addition, both primary and secondary manifestations of Parkinson's disease are easily confused with other parkinsonian syndromes and various neurological disorders associated with aging or other pathologic conditions (Table 2). For example, dementia, as defined by the criteria in the *Diagnostic and Statistical Manual of Mental Disorders*, Third Edition (DSM-III), is estimated to occur in approximately 20% of patients with Parkinson's disease[5] and is difficult to distinguish from the dementia of Alzheimer's disease. The dementia of Parkinson's disease, however, usually occurs later in the course of illness. Parkinsonism can also be induced by drugs such as neuroleptics and metoclopramide, neurotoxins, infections, structural lesions, and metabolic disorders; by degenerative diseases; and by other diseases associated with aging. Some parkinsonian conditions that are commonly mistaken for Parkinson's disease include the following:

Essential tremor. This is the condition most often

misdiagnosed as Parkinson's disease. Essential tremor is faster than parkinsonian tremor (6 to 9 cycles per second), is primarily an action tremor, and is usually bilateral. In contrast, the tremor of early Parkinson's disease usually occurs at rest and is frequently unilateral. While cogwheel rigidity is occasionally found in patients with essential tremor, other parkinsonian signs and symptoms are absent. Furthermore, patients with essential tremor frequently report a family history of tremor, and symptoms do not respond to antiparkinsonian medications.

Progressive supranuclear palsy (PSP). This is a degenerative disease that causes, in addition to parkinsonian symptoms, a distinctive abnormality in ocular motility, particularly in the patient's ability to voluntarily look downward. Other features of PSP include speech impairment, nuchal and axial rigidity, and marked postural instability. Early signs and symptoms of PSP, however, are very similar to those of Parkinson's disease, although tremor is rare. Unresponsiveness to antiparkinsonian medication may be the only clue to diagnosis.

Striatonigral degeneration (SND). This condition manifests with signs and symptoms very similar to those of Parkinson's disease, except that tremor is less common and dementia is not a feature. Antiparkinsonian medications are generally ineffective, and SND steadily progresses over a course of about 5 years.

Olivopontocerebellar atrophy (OPCA). Patients with OPCA will commonly present with many parkinsonian signs and symptoms, including rigidity, resting tremor, bradykinesia, masked facies, and shuffling gait, all of which may mask subtle signs of cerebellar degeneration. Patients will also fail to respond to antiparkinsonian medications, and diagnosis may depend on magnetic resonance imaging to detect brain stem and cerebellar atrophy. OPCA can occur as a familial disease.

Parkinsonism-autonomic failure (Shy–Drager syndrome [SDS]). This disorder combines the primary features of Parkinson's disease with autonomic failure, the signs of which include orthostatic hypotension, urinary urgency and retention, anhydrosis, and impotence. Patients may respond transiently to antiparkinsonian medications, although dopaminergic agents may induce or aggravate hypotension. Shy–Drager syndrome, SND, and OPCA are referred to as "the multisystem atrophies."

Metabolic disorders. Parkinsonian features may also accompany certain diseases with metabolic causes, including Wilson's disease, an autosomal recessive disorder of copper metabolism with accompanying liver dysfunction, frequently presenting in the second or third decade of life. Young patients who present with parkinsonism should therefore be evaluated for Wilson's disease. A number of other rare neurodegenerative disorders with parkinsonism, including Hallervorden–Spatz syndrome, may also be associated with a systemic metabolic defect.

Diseases of aging. Alzheimer's disease has some symptoms in common with Parkinson's disease, including stooped posture, shuffling gait, generalized slowness, loss of affect, and expressionless facies. Its primary feature is dementia, however, and resting tremor rarely occurs. Senile gait apraxias may resemble the gait disorder associated with Parkinson's disease, but other cardinal parkinsonian features are absent. Rheumatoid arthritis can cause stiffness and apparent rigidity, but hand and foot problems are the result of joint deformities, not the striatal posturing seen in Parkinson's disease.

Assessing the Patient with Parkinsonism

The differential diagnosis of Parkinson's disease depends heavily on a thorough neurologic examination and medical history. For example, a careful drug history should include exposure to neuroleptic medications, gastrointestinal drugs (eg, metoclopramide), and certain antihypertensive drugs (eg, reserpine). Drug-induced parkinsonism can be clinically indistinguishable from Parkinson's disease and accounts for the majority of patients with secondary (symptomatic) parkinsonism, or about 7% of cases.[6] An occupational and environmental history should be obtained to exclude exposure to toxins (eg, carbon monoxide, manganese, cyanide, or MPTP [a synthetic narcotic "designer drug"]). It is also helpful to establish a chronology of symptom onset, although patients frequently have difficulty pinpointing the onset of symptoms before meaningful dysfunction. Table 3 lists the differential diagnosis of parkinsonian syndromes.[7]

Neurologic Examination

A thorough neurologic examination is essential in every patient with parkinsonism.

MENTAL STATUS

Results of the mental status examination are usually normal in early Parkinson's disease. Although depression is common in Parkinson's disease, the finding of significant dementia early in the course of illness suggests Alzheimer's disease or one of the other dementing disorders.

CRANIAL NERVES

Extraocular movements are generally normal in Parkinson's disease with the exception of impaired upward

Table 3. Differential Diagnosis of Parkinsonian Syndromes

Early or Predominant Neurologic Sign	Suggested Diagnoses
Tremor	Parkinson's disease
Gait disorder	Multiple-system atrophy, progressive supranuclear palsy (PSP)
Dementia	Alzheimer's disease, multi-infarct state, normal pressure hydrocephalus (NPH)
Autonomic instability (urinary urgency, constipation, loss of libido)	Multiple-system atrophy (Shy–Drager syndrome)
Visual complaints (diplopia, ophthalmoparesis)	PSP, multiple-system atrophy
Ataxia	Olivopontocerebellar atrophy (OPCA), multiple-system atrophy
Apraxia	NPH, senile gait apraxia
Peripheral neuropathy	OPCA, multiple-system atrophy, spinocerebellar-nigral degeneration
Pyramidal tract signs	Multi-infarct state, multiple-system atrophy, cervical myelopathy

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gaze. A disturbance in ocular motility suggests PSP or one of the other multiple system atrophies. Similarly, difficulty with lid opening or closure is a feature of PSP. Nystagmus and slow saccadic movements may be seen in OPCA and the multisystem atrophies.

The patient's sense of smell is often lost in early Parkinson's disease. Analyzing olfactory function may become a useful test for differentiating this disease from essential tremor and some of the other parkinsonian syndromes.

In Parkinson's disease, facial expression and the patient's blink rate are frequently reduced (masked facies). Speech is often hypophonic and muffled.

MOTOR AND EXTRAPYRAMIDAL SYSTEM

Cogwheel rigidity is one of the cardinal manifestations of Parkinson's disease and is demonstrated by passively moving a limb. Early in the disease, rigidity can be brought out by having the patient perform similar movements on the contralateral limb (distracting maneuvers). Rigidity predominates in the limbs in Parkinson's disease, whereas axial rigidity is more prominent in PSP. A slow-frequency, resting tremor is noted in the majority of patients with Parkinson's disease but is rarely a feature of the other parkinsonian syndromes. The lower jaw, hands, arms, and legs may all be affected by tremor. A head tremor is more commonly seen in essential tremor. Cerebellar tremors are also increased during purposeful movements.

Hand and foot posturing (dystonic posture) are also frequently observed in patients with Parkinson's disease.

Hyperreflexia and extensor plantar responses can be observed in the multisystem atrophies. Although reflexes may be increased in Parkinson's disease, hyperactive or pathologic reflexes are not seen. Lower motor neuron signs such as depressed reflexes and muscle atrophy indicate either a peripheral neuropathy or one of the less common parkinsonism-amyotrophy syndromes.

GAIT

Patients with Parkinson's disease typically have a stooped posture with rounding of their shoulders and a forward stoop with arms flexed at the elbow. The arm swing is diminished and steps are frequently short and hesitant. Significant loss of postural reflexes generally occurs later in the course of the disease. A prominent gait disturbance early in the course of disease with postural instability suggests PSP or one of the multiple-system atrophies. Similarly, a wide-based gait is not a feature of Parkinson's disease and suggests cerebellar ataxia, as in OPCA.

SENSORY EXAMINATION

Loss of distal sensation to primary sensory modalities such as pain and temperature is frequently found in older persons. While not an inherent feature of Parkinson's disease, peripheral neuropathies are frequently seen in patients with OPCA.

AUTONOMIC FUNCTION

Prominent autonomic nervous system dysfunction occurs in the multisystem atrophies, although patients with Parkinson's disease often have mild autonomic nervous system involvement. Blood pressure should be recorded in the lying, sitting, and standing positions. A drop of greater than 20 mm Hg of systolic blood pressure after standing for several minutes suggests autonomic impairment. Other tests of autonomic function include the isometric exercise test, Valsalva ratio, and cold-pressor response, all of which evaluate the patient's cardiovascular response to a variety of stimuli.

Diagnostic Studies

Routine laboratory studies should be performed to assess the patient's general health and to identify any potentially complicating conditions. Liver function tests, as well as serum copper and ceruloplasmin levels, should be evaluated if parkinsonism occurs in a young person and Wilson's disease is suspected. Neuroimaging studies may not be necessary for patients with typical drug-responsive Parkinson's disease. Patients with unilateral parkinsonism (hemiparkinsonism), however, should undergo computed tomography or magnetic resonance imaging to

exclude structural lesions such as a tumor or infarct. Moreover, patients with predominant dementia, pyramidal tract dysfunction, ataxia, ophthalmoparesis, or drug resistance should undergo imaging studies to document the presence of cerebrovascular disease, hydrocephalus, or the brain stem and cerebellar atrophy that frequently occurs in patients with the multisystem atrophies.

Managing the Patient with Early Disease

Once Parkinson's disease is diagnosed, the most important issues to decide are whether to initiate some form of drug therapy and which drugs to use. This will depend, in part, on the patient's level of functioning and extent of disability. Early management of Parkinson's disease also requires planning for long-term progression and disability. How can we prevent or delay the future complications of a chronic, degenerative disease that may have to be managed over a period of many years?

Treatment with antiparkinsonian drugs is the primary method of managing the symptoms of Parkinson's disease (although nonmedical rehabilitative strategies also have a very important role to play). In general, antiparkinsonian drugs work to manage symptoms by replacing or maintaining quantities of dopamine in the brain, or by stimulating dopamine receptors directly. Options for drug therapy (Table 4) include levodopa, dopamine agonists, amantadine, and anticholinergic agents.

Table 4. Medications Currently Used in Parkinson's Disease

Drug Classification and Name	Indications	Most Common Adverse Effects
Anticholinergics		
Trihexyphenidyl (Artane)	Tremor, rigidity, drooling	Dry mouth, constipation, blurred vision, confusion, hallucinations
Benztropine (Cogentin)		
Procyclidine (Kemadrin)		
Antihistamines		
Diphenhydramine (Benadryl)	Tremor, rigidity, insomnia	Dry mouth, lethargy, confusion
Dopaminergics		
Amantadine (Symmetrel)	Rigidity, bradykinesia	Leg edema, livedo reticularis
Carbidopa/levodopa (Sinemet)	Tremor, rigidity, bradykinesia	Orthostatic hypotension, nausea, hallucinations, dystonia, dyskinesias
Carbidopa/levodopa (Sinemet CR)		
Dopamine agonists		
Bromocriptine (Parlodel)	Fluctuations of Parkinson's disease (wearing off, dyskinesias, dystonia)	Hallucinations, mental fogginess, orthostatic hypotension, confusion
Pergolide (Permax)	Fluctuations of Parkinson's disease	Orthostatic hypotension, nausea, insomnia
MAO-B inhibitors (deprenyl)	Adjunct to carbidopa/levodopa in patients experiencing fluctuations	Nausea, dizziness, lightheadedness, fainting, abdominal pain

Based on data in Vernon GM. Parkinson's disease. J Neurosci Nurs 1989; 21(5):273–84.

Levodopa

Levodopa, the precursor of dopamine, replaces the dopamine lost to striatonigral cell degeneration and has been the centerpiece of drug therapy for Parkinson's disease since the mid-1960s, when selective dopamine deficiency was first recognized as a feature of the parkinsonian brain. The drug is now commonly administered in a carbidopa/levodopa combination (Sinemet). The addition of carbidopa helps to inhibit the premature decarboxylation of dopamine outside the blood-brain barrier, a problem that can cause a variety of effects including nausea and vomiting.

Levodopa's effects can be dramatic and long-lasting, enabling many disabled patients to regain a substantial degree of motor function. After months or years of chronic levodopa therapy, however, response to the medication starts to deteriorate. Duration of response may then last only a few hours or even less, requiring repeated dosing of medication. Random fluctuations (the "on-off effect") can leave patients suddenly and unpredictably immobilized. Additionally, abnormal involuntary movements (dyskinesias) frequently complicate long-term levodopa administration.

Dopamine Agonists

Dopamine agonists (eg, bromocriptine and pergolide) directly stimulate the postsynaptic dopamine receptor, bypassing the degenerating nigrostriatal neurons. They are more stable than levodopa and have longer half-lives, so that "end-of-dose" fluctuations and dyskinesias are much less pronounced than with levodopa. Dopamine agonists, by themselves, however, are not as effective as levodopa in alleviating parkinsonian symptoms. Nevertheless, the levodopa-sparing effect of dopamine agonists argues in favor of their relatively early use in Parkinson's disease.

Dopamine agonists have been used as monotherapy for Parkinson's disease with some success. It has been

suggested, for example, that bromocriptine (Parlodel) used before levodopa helps to limit the eventual development of dyskinesias and fluctuations in levodopa response.[8,9] These same studies make it clear, however, that only a small proportion of patients (less than one third) can be managed on bromocriptine alone for any length of time because of insufficient therapeutic response or side effects. In the Rinne study,[8] only 58% of patients were still taking bromocriptine alone after 6 months of therapy, and only 28% were still taking bromocriptine alone after 3 years.

Amantadine

Patients with mild symptoms who do not yet need to begin levodopa therapy may benefit from a course of amantadine (Symmetrel), 100 mg twice a day, increasing to 100 mg three times a day if needed. Its mechanism of action is not clear, but it appears to stimulate release of stored dopamine from presynaptic terminals.[3] Side effects are not common but may include restlessness, confusion, depression, and nausea. Amantadine is occasionally useful in patients with more advanced disease.

Anticholinergic Agents

These drugs have mild antiparkinsonian effects; they are helpful in relieving tremor and rigidity, less so in alleviating bradykinesia, masklike facies, or gait disturbances. Their usefulness is limited by their significant side effects, which are more serious in elderly patients and include confusion, urinary retention, palpitations, and visual blurring. There is also concern that these agents may exacerbate or predispose patients to the dementia that afflicts approximately 20% of parkinsonian patients.

The Patient with Minimal Disability

In the early stages of Parkinson's disease, when patients are not yet disabled by tremor, bradykinesia, rigidity, or postural instability, it is usually neither necessary nor desirable to initiate therapy with levodopa or other agents whose effects may "wear off" over time. There does, however, seem to be some scientific justification for early treatment with deprenyl, or selegiline hydrochloride (Eldepryl), a type B–selective monoamine oxidase (MAO-B) inhibitor.

Deprenyl (Selegiline Hydrochloride)

Deprenyl inhibits the metabolizing action of the MAO-B enzyme that is responsible for the breakdown of dopamine in the brain. Deprenyl is currently indicated as adjunctive therapy in patients experiencing fluctuations in response to levodopa, and it may make possible a reduction in levodopa dose.

There is evidence, however, that deprenyl also has a role as monotherapy before beginning a levodopa regimen. In a double-blind, placebo-controlled study in patients with early Parkinson's disease, monotherapy with deprenyl extended the time before levodopa was necessary to alleviate symptoms.[10] More recently, the interim report of a large, multicenter controlled clinical trial (Deprenyl and Tocopherol Antioxidative Therapy of Parkinsonism [DATATOP])[11] of 800 patients with early, untreated Parkinson's disease concluded that development of disability requiring levodopa treatment was delayed for nearly 1 year in subjects who received deprenyl (10 mg per day) alone or in combination with tocopherol (2000 IU per day).

It is not yet clear whether deprenyl alone has subtle symptomatic effects that might explain the results of the DATATOP study, or whether deprenyl has actual neuroprotective properties. Nevertheless, deprenyl may suppress the oxidant stress associated with the increased turnover of dopamine,[12] and thus may help to actually slow progressive nigral degeneration and associated dopamine depletion; DATATOP and other long-term studies are pursuing validation of this hypothesis.

At our center, all newly diagnosed patients with early Parkinson's disease are started on deprenyl (10 mg/d), given in doses of 5 mg twice daily with breakfast and lunch. Side effects in patients taking deprenyl alone are generally not severe, but may include nausea, dizziness or lightheadedness, insomnia, or confusion.

Combination Therapy for the Symptomatic Patient

When the symptoms of Parkinson's disease affect the patient's ability to manage his or her daily routine, further therapy is warranted. The degree of disability for which treatment is needed will vary from patient to patient. The goal of treatment is to achieve symptomatic improvement without long-term drug-related complications (motor fluctuations and dyskinesias). Combination therapy (most frequently, combining carbidopa/levodopa and a dopamine agonist) appears to be a useful strategy for taking advantage of levodopa's more potent antiparkinsonian effects while keeping the dose low and thus delaying the onset of levodopa-related dyskinesias or fluctuations.

Clinical evidence supporting this approach has been provided by Rinne,[8] who found fluctuations occurring in 19% of patients taking only levodopa after 3 years of treatment, compared with only 4% of those treated with

combined levodopa and bromocriptine. Peak-dose dyskinesias occurred in only 21% of those on combined therapy, compared with 57% of those on levodopa alone. Rinne's 5-year follow-up study[13] substantiated his original findings, and other studies have also reported promising results with combined therapy.[14,15]

Patients may be started on carbidopa/levodopa (25 mg of carbidopa and 100 mg of levodopa daily) titrating the dose to about 400 mg of levodopa daily. A controlled-release formulation (Sinemet CR), which is now available, is effective in regulating the response to medication in patients who are experiencing end-of-dose fluctuations. Sinemet CR may prove to be the preferred levodopa preparation from the outset, as its slow release properties more closely resemble the natural physiologic state. If a satisfactory response is not achieved (or clinical fluctuations occur) within this dose range, bromocriptine (Parlodel), 1.25 mg daily, or pergolide (Permax), 0.05 mg daily, may be added and increased slowly (up to 15 to 30 mg daily for bromocriptine and up to 2 to 6 mg daily for pergolide) until adequate control is achieved.

While interest in deprenyl has focused on its potential neuroprotective properties, deprenyl has been shown to increase synaptic dopamine and has a symptomatic effect when used in combination with carbidopa/levodopa. In addition, a recent study by Elizan et al[16] has suggested that initial symptomatic therapy with deprenyl and low-dose carbidopa/levodopa may prolong the time before the onset of major functional disability.

In view of all the options for early management, it is best to tailor therapy to the functional status and needs of individual patients, and these will vary considerably.[17] An active younger patient may require more vigorous symptomatic treatment than an older patient. The pros and cons of each option should be clearly explained to the patient. Furthermore, physicians need to be aware of and sensitive to the cost of medications. Combination drug therapy can be a costly endeavor for patients, especially for those on fixed incomes. Ideally, the physician should strive for a good understanding of the patient's needs and expectations, and provide the patient with a realistic view of the future.

Nonmedical Management

A crucial aspect of managing care for the patient with early stage Parkinson's disease is helping patients cope with the diagnosis emotionally as well as medically. Initially, patients frequently deny the diagnosis or unrealistically hope for a cure. Fear and anxiety are natural responses, and may be considerably exacerbated if patients are poorly informed about the disease. Depression is an ever-present danger to Parkinson's disease patients. Its immobilizing effect can lead to social isolation, physical inactivity, and dependency, all of which may impede medical management and hasten symptomatic progression.

Table 5. Parkinson's Disease Organizations

National organizations

The American Parkinson Disease Association
116 John St
New York, NY 10038
1-800-223-APDA

National Parkinson Foundation
1501 NW 9th Ave
Bob Hope Rd
Miami, FL 33138

United Parkinson Foundation
360 W Superior St
Chicago, IL 60610

National support groups

Parkinson Support Groups of America
11376 Cherry Hill Rd, #204
Beltsville, MD 20705

For other information, call The National Parkinson Foundation, 1-800-327-4545.

As time goes on, patients must be helped to cope with a myriad of challenging psychosocial adjustments, including problems with self-image, self-esteem, and changing roles within the family; employment and financial concerns; and caregiving decisions.

The challenges inherent in managing these issues necessitate a comprehensive approach, one that provides access to a range of medical professionals and support services and one that, to the greatest extent possible, actively involves the patient and family in identifying problem areas and developing coping strategies.[18] Although in the early stages of the disease patients may not need physical, speech, or occupational therapy, each of these disciplines will play a key role in the patient's care as the disease progresses. The physician should be prepared to make appropriate referrals as a patient begins to require additional care in these areas.

Patients and their families will frequently need emotional support as well as medical counsel. The voluntary nonprofit organizations involved with Parkinson's disease can be excellent resources for information and support (Table 5). These organizations can link patients and their families with local support groups and specialized health care services, and provide educational materials on all aspects of the disease including treatments, self-help strategies, and new research.

Conclusions

Before the late 1960s, little could be done for patients with Parkinson's disease. Progression of the disease and its debilitating effects was unrelenting and devastating, and little was known about its cause. Mysteries still remain, but a great deal has been learned—enough, in fact, so that current research is actively pursuing neuroprotective and preventive therapies, improved pharmacologic treatments, and new diagnostic interventions.

Parkinson's disease remains, ultimately, a progressive illness, but there are more options for treatment and a greater chance that those diagnosed with it can look forward to additional productive and satisfying years of life. If a comprehensive approach to the management of Parkinson's disease—one that combines accurate diagnosis and improved medications, rehabilitation strategies, and self-help programs—can be initiated early in the disease process, patients and caregivers will be able to better cope with the disease's troubling manifestations, and patients will sustain the quality of their lives longer.

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