Movement Disorders in People With Parkinson Disease: A Model for Physical Therapy

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Perspective

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People who are diagnosed with idiopathic Parkinson disease (PD) experience movement disorders that, if not managed, can lead to considerable disability. The premise of this perspective is that physical therapy for people with PD relies on clinicians having: (1) up-to-date knowledge of the pathogenesis of movement disorders, (2) the ability to recognize common movement disorders in people with PD, (3) the ability to implement a basic management plan according to a person's stage of disability, and (4) problem-solving skills that enable treatment plans to be tailored to individual needs. This article will present a model of physical therapy management for people with idiopathic PD based on contemporary knowledge of the pathogenesis of movement disorders in basal ganglia disease as well as a review of the evidence for physical therapy interventions. The model advocates a task-specific approach to training, with emphasis on treating people with PD-related movement disorders such as hypokinesia and postural instability within the context of functional tasks of everyday living such as walking, turning over in bed, and manipulating objects. The effects of medication, cognitive impairment, the environment, and coexisting medical conditions are also taken into consideration. An argument is put forward that clinicians need to identify core elements of physical therapy training that apply to all people with PD as well as elements specific to the needs of each individual. A case history is used to illustrate how physical therapy treatment is regularly reviewed and adjusted according to the changing constellation of movement disorders that present as the disease progresses. [Morris ME. Movement disorders in people with Parkinson disease: a model for physical therapy. *Phys Ther.* 2000;80:578–597.]

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In this article, a model of physical therapy management for people with idiopathic Parkinson disease (PD) is presented. This model is based on current knowledge of the pathogenesis of movement disorders and evaluation of the evidence for specific physical therapy interventions. Parkinson disease is common among older people, affecting more than 1 in every 100 people over the age of 75 years and 1 in every 1,000 people over the age of 65 years.[1] Given that more than 10% of Americans are over the age of 65 years, it can be estimated that at least 1 million US citizens currently have the disease. Approximately 10 people per 1 million in the population are diagnosed in their 30s and 40s; thus, early-onset PD is uncommon.[2] On a worldwide basis, it is thought that around 10 million older people have PD.[2] With a large proportion of the population aging, it can be predicted that by the year 2020 more than 40 million people in the world will have this progressive neurological condition.

Movement disorders are the hallmark of PD and can severely compromise an individual's ability to perform well-learned motor skills such as walking, writing, turning around, and transferring in and out of bed. According to the American Physical Therapy Association's *Guide to Physical Therapist Practice*,[3] the main role of the physical therapist within the multidisciplinary team is to teach people with PD strategies for coping with impairments and disabilities. These strategies, theoretically, will allow them to move more easily, minimize disability, and retain independent living skills. Physical therapists also should be able to assess and measure changes in function, disability, activity, and participation in response to therapy, medication, surgery, and the natural progression of the disease. These roles have been described in detail elsewhere.[3–10] In my opinion, few people initially assessed by physical therapists fit the textbook description of PD,[11] in which the person walks with a forward stooped posture, festinating gait, rigidity, and drooling. In the first 10 years of the disease, I contend, it is more common for people to exhibit slowness of movement, mild gait hypokinesia, resting tremor, micrographic handwriting, and reduced speech volume.[4] In the latter stages, festination, dyskinesia, akinesia, marked hypokinesia, postural instability, and falls are thought to be more of a problem.[11] Because there is considerable variation across individuals in the manifestation of their movement disorders as well as variations in motor performance over time,[4] clinicians should be able to design programs that are tailored to the changing needs of these individuals and their caregivers.

Due to the aging population, by the year 2020 more than 40 million people worldwide may have movement disorders secondary to Parkinson disease.

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Table 1.
Common Movement Disorders in People With Parkinson Disease[a]

Bradykinesia	Reduced movement speed and amplitude; at the extreme, it is known as "hypokinesia," which refers to "poverty" of movement
Akinesia	Difficulty initiating movements
Episodes of freezing	Motor blocks/sudden inability to move during the execution of a movement sequence
Impaired balance and postural control	Difficulty maintaining upright stance with narrow base of support in response to a perturbation to the center of mass or with eyes closed; difficulty maintaining stability in sitting or when transferring from one position to another; can manifest as frequent falling
Dyskinesia	Overactivity of muscles; can manifest as dystonia; wriggling/writhing movements; chorea or rarely athetosis
Tremor	Usually resting tremor; more rarely postural or action tremor
Rigidity	Hypertonicity and hyperreflexia in agonist and antagonist muscle groups in a given limb
Adaptive responses	Reduced activity, muscle weakness, reduced muscle length, contractures, deformity, reduced aerobic capacity

[a] Adapted and reproduced with permission from Morris et al[32] (copyright Kingston Centre).

What Is the Nature of the Deficit?

In recent years, there has been a rapid growth in knowledge about the pathogenesis of the movement disorders that occur in people with PD.[12] The most frequently observed movement disorders are described in Table 1. Of these movement disorders, slowness in the performance of movement sequences (bradykinesia) is the most common and affects around 80% of people with PD.[11] Slowness may be so marked as to result in poverty of movement, which is known as "hypokinesia." People with hypokinesia typically have an expressionless, mask-like face and walk with reduced trunk rotation, short steps, and diminished arm swing, which is more pronounced on one side than the other. Although PD-related movement disorders characteristically occur bilaterally, movement disorders such as bradykinesia are asymmetrical in their severity. This means that physical therapists need to carefully assess the degree of bradykinesia on the right and left sides in addition to comparing their patients' performance with that of people of similar age who are without impairment.

All people with bradykinesia experience difficulty in performing repetitive or sequential movements of the limbs such as alternating pronation and supination of the forearms or repetitive tapping of the feet or fingers.[12] This is because movement size progressively decreases during sequential actions. This diminution of movement is known as "motor instability"[13] and can be clearly seen in people with gait hypokinesia, in whom the footsteps become shorter and shorter the further they walk. Likewise, the handwriting of people with PD is typically miniaturized and becomes both smaller and slower as a paragraph is written.[14] When a person with PD stops a movement sequence, has a short rest, and begins again, the movement size and speed start at values that are close to normal, then again start to reduce as the new sequence is performed.

There is growing evidence that bradykinesia in people with PD results from disruption of the neurotransmitters used in the neural projections from the internal segment of the globus pallidus of the basal ganglia (BG) to the motor cortical regions known as the supplementary motor area (SMA) and the primary motor cortex.[15] The SMA is critical in regulating the increase in neural activity that needs to occur before a movement is executed.[16,17] It also ensures that a movement is terminated at the appropriate time.[16,17] If the preparation for forthcoming movement is disrupted, then movements can be reduced in size and speed (bradykinesia). At the extreme, if there is no activity in the SMA and primary motor cortex, movement fails to occur.

Absence of movement associated with an inability to initiate movement is known as "akinesia."[18] Sudden cessation of movement (motor blocks) partway through an action sequence is known as "freezing" (Tab. 1). Clinical evidence suggests that akinesia and freezing episodes are context dependent.[18] For example, the person may "freeze" when attempting to walk through a narrow doorway or when making a transition from walking on carpet to wooden floorboards, even though he or she can walk quickly without motor blocks across an empty parking lot. Research on primates suggests that spiny neurons in the striatum of the BG play a role in recognizing patterns of input from convergent input from multiple cortical sites.[19] This recognition of behavioral events or environmental contexts from prior experience may then be used for the planning and performance of intelligent behavior.[20] It has been hypothesized that when striatal pattern recognition is defective, motor performance is not ideally matched to task demands.

Some people with PD can also find it difficult to cease actions such as walking, turning around, or speaking,[21] presumably because they have sustained discharge in the SMA, rather than the rapid drop in neural activity in the SMA that normally allows movements to be terminated. Difficulty terminating locomotor actions such as walking, running, or turning during walking is thought to be one of the major factors that predisposes people with PD to slips, trips, and falls.[22]

The neurotransmitter imbalance in the motor cortex-BG-motor cortex feedback loop arises due to a relentless and progressive death of neurons in the substantia nigra pars compacta (SN) of the brain stem.[15] These brain-stem neurons normally secrete the neurotransmitter dopamine that apparently plays a role in allowing people to execute well-learned skilled movements quickly and smoothly. Why cell death occurs in this region of the brain stem is not known, although exposure to environmental toxins coupled with a genetic predisposition to PD is one hypothesis.[23] What is known is that the balance of dopamine, gamma-aminobutyric acid (GABA), enkephalin, glutamate, acetylcholine, and substance P in the BG is normally very finely tuned.[15] In people with bradykinesia, there is a decrease in the excitation of the dopaminergic projections from the SN to the striatum and the internal globus pallidus coupled with a reduction in the inhibitory activity of dopaminergic projections from the SN to the striatum and the external globus pallidus.[15] The net result is excessive inhibitory output from the globus pallidus to the thalamus that leads to reduced movement. In contrast, with Huntington disease, for example, there is a progressive loss of GABA/enkephalin neurons in the striatum that project to the external globus pallidus, and as a result large-amplitude, irregular, involuntary choreiform movements occur.[24]

In a similar way, some people with advanced PD who have been receiving levodopa medication for more than 15 to 20 years develop dyskinesia, which may be associated with relatively excessive amounts of GABA/enkephalin. Dyskinesia manifests as purposeless wriggling or writhing movements as well as dystonic posturing of the feet, hands, trunk, and neck. This condition includes chorea, athetosis, tics, dystonia, and tremor.[22] The term "dystonia" refers to excessive and sustained overactivity of a particular muscle group such as the triceps surae or long finger flexors. The overactivity occurs for periods of minutes to hours and frequently recurs over the course of a day, month, or even years.

Dyskinetic movements are usually most noticeable when a person is sitting upright, standing, or walking, and they disappear when the person is asleep. Dyskinesia can be categorized according to the following characteristics:

- peak dose: typically occurs 1 to 3 hours after medication is taken
- biphasic: peaks twice within the levodopa medication cycle, typically in the half-hour periods at the beginning and end of the dose
- end of dose: commences around 30 minutes prior to the next dose
- nocturnal: occurs only at night time when medication levels are low
- random presentation

Bradykinesia, akinesia, freezing, and dyskinesia are not the only movement disorders in PD. As early as 1967, Martin[25] recognized that balance disorders were also an inherent feature of the disease. The reason why balance is disrupted is unclear, although it appears to be associated with neurotransmitter disturbances in the output projections from the internal globus pallidus to the midbrain and brain-stem regions involved in maintaining upright stance and extensor muscle activity.[21] A balance disturbance in a person with PD is most easily detected by quickly and unexpectedly pulling the person backward at the shoulders while he or she is standing with his or her feet slightly apart.[26] People without PD respond to this "pull test" by dorsiflexing their ankles, lifting the arms forward, and, in some cases, flexing forward at the hips. When the pull is stronger, individuals without PD typically take a step backward to protect them from falling. In people with PD, these postural responses are compromised, and the ankle, hip, arm, and stepping strategies are either absent or diminished in amplitude.[27] They might take several steps backward to recover stability or, in more severe cases, fall rigidly into the therapist's arms. People with a balance disturbance of this type are at high risk for falls. This is particularly the case when they have to respond to an unexpected push or pull or an unexpected movement of the support surface they are standing on, or when they have to make automatic postural adjustments.[27]

Another hallmark of idiopathic PD is rigidity.[28] Rigidity can be detected by slow passive movement of the affected body part while the person focuses his or her attention on a secondary task (such as reciting the days of the week backward to avoid compensating for his or her movement disorder). The examiner assesses the degree of resistance encountered while passively moving the affected body part. The resistance is either "lead pipe" (slow and sustained) or "cogwheel" (where tremor is superimposed on rigidity). There is some evidence that rigidity is due to abnormal activation of long-latency stretch reflexes coupled with an increase in central reflex gain.[28] In addition, muscle stiffness is increased in people with advanced PD due to changes in the peripheral mechanical properties of muscle.[29] Although the effect of rigidity on passive movement can be detected, the neural component of rigidity does not appear to compromise voluntary movement.[17] Moreover, people with PD rarely complain about its presence, even when a clinician rates it as severe.[22] For these reasons, there appears to be little point in directing physical therapy treatment toward reducing the neural component of rigidity, as was suggested in the 1950s and 1960s.[30] A controlled trial of physical therapy using proprioceptive neuromuscular facilitation, the Bobath technique, and the Peto

method (a method of conductive education) to reduce rigidity and increase rotation showed these interventions were ineffective in enhancing walking, decreasing festination, or increasing range of movement.[31]

Resting tremor (4–6 Hz) is also characteristic of idiopathic PD and is often the first symptom reported.[11] It may be due to an altered firing rate of thalamic neurons, although the exact mechanism by which this occurs is not known. Less commonly, action tremor (6–8 Hz) can be observed during the execution of movements, or postural tremor can be observed when the person bears weight through the limb or encounters resistance to movement of the limbs, trunk, head, or neck.[11] Physical therapists rarely need to treat individuals with resting tremor because it disappears during movement and therefore does not interfere with the ability to perform everyday tasks such as walking, writing, or grasping objects. In addition, resting tremor responds well to levodopa. There are anecdotal reports[32] that physical therapy interventions such as relaxation and directing attention toward minimizing tremor may have short-term beneficial effects on the severity of resting tremor. However, these effects are only transient.[32] Tremor severe enough to be considered socially unacceptable by the person with the disease, in my opinion, may be best treated by surgical interventions such as thalamotomy, pallidotomy, and deep brain stimulation.

A Model for Physical Therapy

One of the striking features of PD is that the ability to move is not lost, rather there is an activation problem.[13,25] As a result, people with PD appear to be reliant on cortical control mechanisms to initiate movement.[8,13,19,20,25] There is also evidence of increased reliance on frontal-cortical "attentional" mechanisms to sustain the execution of complex movements, due to defective BG mechanisms subserving movement automaticity.[13,16,17,25] The current model for physical therapy intervention in people with PD is based on the assumption that normal movement can be obtained by teaching patients strategies to bypass the BG pathology. The Figure illustrates the factors taken into account when this model was created and for setting the conditions for training. When planning physical therapy interventions, I believe that therapists should take into account the response of movement disorders to external cues and attentional strategies, knowledge of how interventions can be adapted according to severity of cognitive impairment, the need to analyze functional task performance as a basis for designing task-specific training regimens, and the effects of PD medication on movement. In addition, when designing training programs tailored to the needs of individuals and their caregivers, I contend that physical therapists need to consider the effects of aging, concurrent pathologies, and secondary adaptive changes in the musculoskeletal and cardiovascular systems (Figure).

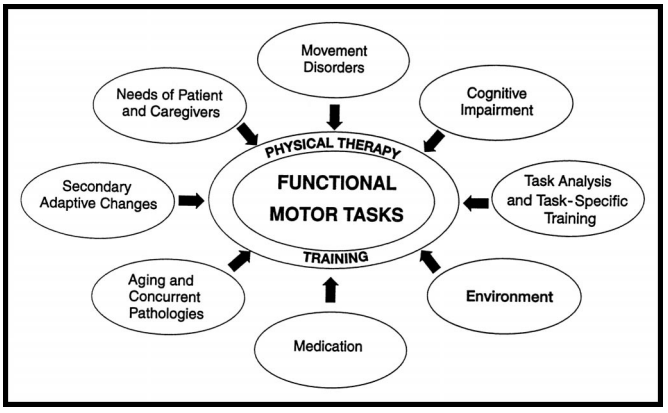

Figure.
Key elements of the model for training people with Parkinson disease to utilize strategies for performing functional motor tasks.

Knowing about the characteristic features of movement disorders in people with PD is, in my opinion, the starting point for designing physical therapy interventions. Despite the troublesome nature of disorders such as hypokinesia, akinesia, and dyskinesia, people with PD have a remarkable capacity to move quickly and with near-normal movement size under certain circumstances.[33–37] For example, when a person with PD performs a simple ballistic task such as pointing to an object or catching a moving ball, the movement size and speed are frequently normal.[33] However, when simple movements are integrated into a long or complex action sequence, they are performed slowly and with much more difficulty.[34] This is presumably because the primary motor cortex, brain stem, and spinal cord are the major anatomical regions involved in the control of simple, ballistic or reflexive movements, whereas more complex actions are regulated by the cerebellar circuits and cortex-BG-cortex feedback loop.[13] The latter is defective in people with PD.[13] Notwithstanding this, performance has the potential to be enhanced by training people with PD to break down long or complex sequences into component parts and to focus their attention on performing each part separately.[32] People with PD also benefit from focusing their attention on performing one task at a time and avoiding dual task performance.[8,38] Presumably, when 2 activities are performed at the same time, one activity is controlled by the faulty BG while attention is focused on the other activity, and the task that runs through the BG reduces in speed and size.[8] Preparing in advance for forthcoming movement by using mental rehearsal and visualization might also be of benefit,[32] although the effects of these strategies have not been documented through controlled clinical trials.

Evidence is accumulating that people with PD can move more easily when external cues are available to guide

their performance.[35–37] External cues can be visual, auditory, or proprioceptive in type. For example, when people with gait hypokinesia are provided with visual cues on the floor set at the appropriate step length for their age, height, and sex, they are able to walk at normal footstep amplitude and speed, provided they do not have severe postural instability.[35–37] In a similar way, lined paper assists people with micrographia to write with larger strokes.[14] Auditory cues appear to be particularly useful for people with gait akinesia and freezing, whereas visual cues are most useful for people with gait hypokinesia.[35–37] Rhythmical sensory cues, such as rocking the body from side to side, may sometimes be useful in assisting the initiation of movements such as walking or rolling over in bed.[10] External cues may assist people with PD to move more easily because they utilize the intact premotor cortex of the brain rather than the defective BG-SMA circuits to control movement.[39] An alternative explanation is that external cues may simply focus the person's attention on critical aspects of the movement that need to be regulated, such as stride length,[35–37] weight transference to unload the leg, or axial motion to assist in turning.[6] Both of these explanations are compatible with the idea that the ability to move is not lost in people with PD, rather the person is dependent on cortical mechanisms to activate and sustain movement.

The presence of external cues is not mandatory for activating neuronal networks in people with PD. In people who are cognitively intact, simply focusing attention on the critical aspect of movement that needs to be controlled can be sufficient to activate movement with near-normal speed and size.[37,40–42] Because cortical regions remain unaffected by the disease in the early stages, the person appears to be able to use "online" frontal-lobe cognitive strategies to compensate for BG insufficiency. Strategies that rely solely on methods such as these, however, may not be effective in people with severe cognitive impairment due to the accumulation of inclusion bodies (Lewy bodies) in neurons throughout the cortical, subcortical, and brain-stem regions in individuals with end-stage PD.[43] Because Lewy bodies impair mitochondrial processes,[23] neural function throughout large regions of the central nervous system becomes compromised. The implication for physical therapy is that training that relies on cortically mediated learning processes and cognitive strategies may not be effective in people with end-stage PD because the capacity for learning new motor skills declines.[42] People with cognitive impairment might well benefit more from external cues, environmental restructuring, and demonstrations or instructions from physical therapists and caregivers, as these strategies appear to be less reliant on complex information processing.

Task analysis and task-specific training are central elements of the model (Figure). In my opinion, knowledge of the biomechanics of movement for a range of everyday tasks can be used in an attempt to ensure that the most efficient strategy is taught. Researchers who have measured biomechanical performance in people with PD in response to different physical therapy treatment strategies have provided data that clinicians might find useful.[24,37,40,44] I further argue that it is important for training to take place within the context of functional tasks of everyday living, such as walking, standing up from a sitting position, turning around, moving around the bed, writing, and dressing. Task-specific training seems, in my view, particularly appropriate, given that movement disorders appear to be context dependent[19] and are most prominent for well-learned, complex motor skills.[33,34] Although there has not yet been a controlled clinical trial comparing the effects of training functional motor tasks with the effects of training isolated movements, the motor skill learning literature indicates that generalization of training is most effective when there is a high degree of similarity between the trained task and new variations of the task.[45] From my perspective, there is little point, for example, in training a person to control dystonia in the foot while lying supine if the training does not generalize to walking, standing up, or obstacle negotiation. It is preferable to train the person to control dystonia while walking, as this is when it is much more disabling.

To further enhance transfer and retention of training, I suggest that physical therapy takes place in the environment where the individual's movement disorders are most troublesome. This is usually inside the person's home, in the bedroom, bathroom, kitchen, or family room, although training the person to use community ambulation skills such as road crossing and negotiation of obstacles (eg, curbs) is frequently also a priority. If the physical therapist is unable to travel to the person's residence, then key aspects of the home environment can be simulated in the physical therapy department so that the person can practice the movement strategies in a similar context. Environmental modifications such as creating open walkways and providing ramps and rails to optimize movement and reduce the risk of falls in people with PD should be considered. However, controlled clinical trials are needed to measure the effects of environmental context on motor performance in people with PD.

The effects of PD medications on movement and functional capacity should not be overlooked (Figure).[4,5,46] In the early stages after diagnosis, patients can have an excellent response to drugs such as levodopa and apomorphine and show very little residual deficit.[11] However, after a number of years, movement disorders again

become commonplace, and motor performance can become highly variable.[4] These variations in performance are known as "motor fluctuations." For this reason, physical therapists need to ensure that they train people with PD to cope with movement disorders during both "off" and "on" periods of levodopa use. The "off" phase is when levodopa levels are low and movements are hypokinetic, typically at the end of the levodopa cycle. The "on" phase is usually at peak dose, when movements are more normal. I contend that, at times, this will necessitate 2 different sets of strategies—one set of movement strategies for when they are at the end of a dose and very hypokinetic and another set of movement strategies for when they are at peak dose and have a different combination of movement disorders.[22,26] For people who have uniphasic, biphasic, peak-dose, end-of-dose, or random presentation dyskinesia, training needs to focus on strategies for coping with the involuntary extra movements whenever they occur during the medication cycle. Moreover, clinical observations suggest that it is advisable for people to perform activities to maintain general strength, range of movement, and fitness as well as task-specific training when they are at peak dose during the medication cycle.[22] For example, the daily routine can be planned so that walking or playing golf occur from 11 AM to 1 PM or from 3 to 5 PM if they are on the typical levodopa schedule where medication is administered at 6 AM, 10 AM, 2 PM, 6 PM, and 10 PM.

Aging, concurrent medical conditions, and secondary adaptive changes in the musculoskeletal and cardiovascular systems, in my view, are also important considerations when devising the physical therapy program. The majority of people with PD are older than 65 years, and many have age-related frailty or concurrent medical conditions and lead a sedentary lifestyle.[32] People with PD, therefore, are at risk of developing weakness, reduced joint range of movement, thoracic kyphosis, and diminished aerobic capacity because they tend to reduce the amount and variety of physical activities they perform.[32] They can also experience reductions in exercise capacity[47,48] and can have diminished force production.[49,50] Shortening of the triceps surae muscle is also a frequent outcome of prolonged hypokinesia[10] and limits power generation at the ankle at the end of the stance phase of gait.[51] The physical therapy assessment needs to differentiate between movement disorders that are due to PD and those that arise from other conditions or disuse (this process is discussed in detail elsewhere[4,9]). Physical therapy treatment can then be tailored to the specific movement disorders found on assessment.[9,10,32]

Finally, I suggest that, to obtain the full benefit of physical therapy intervention, the perceived needs articulated by the patients and their caregivers and utilization of the diverse skills of the multidisciplinary team need to be taken into consideration (Figure).[52] Because PD progresses slowly, patients and their families need to be consulted in developing programs to be implemented over the long-term. This consultation will assist them in taking greater responsibility for the management of their health and well-being.[9,10] The wide range of motor, cognitive, autonomic, and psychosocial problems that can occur in people with PD may be too complex for any single practitioner to manage in isolation.[52] In this regard, the ability of the physical therapist to consult with other health care professionals with specialist skills would appear to be a distinct advantage.

Physical Therapy Strategies to Enhance Performance of Functional Motor Tasks

Walking

Most people with PD experience difficulty walking at some stage during the disease. Unfortunately, however, gait disorders are not always responsive to antiparkinsonian medication, and slowness and small steps can remain despite the best attempts at pharmacotherapy.[46] Gait hypokinesia affects almost everybody with PD and increases in severity with the progression of the disease.[35] The fundamental deficit in gait hypokinesia is a disorder in step length regulation.[35] Because there is a proportional relationship between step length and ground clearance, people with hypokinesia are at considerable risk of tripping over obstacles during the swing phase of gait. This is because people with step lengths less than 1 m can have ground clearances less than 0.8 cm, as compared with the usual value of 1 to 1.3 cm.[53] The risk of tripping together with very slow walking can limit residential and community ambulation. Therefore, physical therapists dedicate considerable time toward teaching people to walk with steps that are appropriate in size for their height and age.

The use of external cues and cognitive strategies are the therapist's main training options for gait hypokinesia. The research literature provides considerable evidence that visual cues (eg, white lines on the floor spaced at step lengths suitable for the person's age and height) normalize the spatial and temporal variables of gait.[7,35–37,51] In addition, Behrman et al[40] showed that attentional strategies, where the person responds to different instructional sets such as instructions to walk with long steps or swinging the arms, are effective in the short-term in enhancing stride length and walking speed. Moreover, 2 experiments have shown that avoiding dual task performance during gait helps people with PD to maintain long strides.[37,38] When people with hypokinesia divert their attention from their footsteps to a second task such as carrying a tray with drinks[38] or talking,[37] the stride length and gait speed immediately

show marked reductions. There have been no investigations, however, to measure the long-term effects of external cues and cognitive strategies. In addition, there has not yet been a fully blind, controlled randomized clinical trial (RCT) on the effects of external cues, attentional strategy training, or unitask performance on walking in people with PD.

Gait akinesia and freezing are most common in end-stage disease and affect fewer than 20% of patients.[11] There are no RCTs on the effects of physical therapy on gait akinesia in people with PD, although it has been shown that, within a single session, auditory cues enhance the ability to begin the walking sequence and avoid episodes of freezing.[54] Thaut, McIntosh, and colleagues[55,56] conducted a series of trials on the effects of auditory cues on gait in subjects with PD, although they did not state whether the subjects had akinesia, freezing, hypokinesia, or a combination of these movement disorders. In one experiment, Thaut et al[55] demonstrated that a 3-week gait training program using audiotapes of rhythmical musical beats enhanced gait speed and stride length as well as altering the electromyographic patterns of the tibialis anterior and vastus lateralis muscles. In another experiment, they showed that rhythmical auditory stimulation normalized the temporal and spatial variables of the footstep pattern, in both the "on" and "off" stages of the levodopa medication cycle.[56] Controlled clinical trials measuring the long-term benefits of these strategies are yet to be conducted.

Dystonic gait is also common in people with PD. Often, this condition manifests as dystonia of the plantar flexors and invertors of one foot, which varies in severity over time and predisposes the person to tripping and falling. Adjustment of the antiparkinsonian medication by the neurologist frequently will resolve the problem. Otherwise, based on a case report,[10] the physical therapist can measure the effects of prolonged stretching of the dystonic muscles prior to functional performance or else teach the person to attend to heel-strike and push-off when walking.

Choreiform dyskinetic gait disorders, in my opinion, are less amenable to physical therapy treatment and are often best managed by adjusting the patient's medication or, in more severe cases, with neurosurgery. Although persistent wriggling, writhing, and flick-like movements can be exhausting when severe, individuals with very mild choreiform dyskinetic gait disorders might not even be aware that they have extra movements. I contend that for short-term relief, which may be necessary in certain social situations, the physical therapist can try compression and resistance. For example, wearing weighted ankle cuffs or teaching the person to squeeze a ball or tightly clasp his or her hands behind the back to dampen down the large-amplitude oscillations can be tried. Data are not available, however, to indicate whether these strategies are effective. A proportion of people also report that relaxation strategies, tai chi, or Feldenkrais methods are helpful,[57] although these methods have not been validated in controlled trials with people who have PD.

Turning Around

Turning around while walking is most problematic for people who experience episodes of freezing or motor instability. Usually when elderly people perform a 360-degree turn during walking, they take fewer than 6 steps to complete the action.[6] In contrast, those with PD and motor instability take up to 20 steps to turn, with each step becoming smaller and smaller until they eventually stop.[6] In addition, people with PD show little movement of the trunk, head, and arms when turning, whereas people without movement disorders turn by moving the head, shoulders, trunk, and legs in a fluid sequence.[6] To overcome episodes of freezing during turns, people with PD can be trained to concentrate on turning in a large arc of movement, using full body movements, rather than focusing on rapidly switching directions.[6] Using this strategy, Yekutiel et al[6] found that 12 people with PD decreased their turning time by a mean of 40% following 3 months of twice-weekly physical therapy. In very small spaces, where turning in a large arc is not possible, the "clock turn" strategy is recommended.[57] For this strategy, the person stands on the spot and then consciously thinks of stepping with the right foot and then the left foot to relevant positions for the task (eg, to make a 180° turn, step to 12 o'clock, 3 o'clock, and 6 o'clock).[57] Attention is directed to lifting the feet clear in a deliberate stepping action, rather than shuffling or swiveling around. Although these recommendations are based on current knowledge and theories, data to indicate their effectiveness are not available.

Standing Up and Sitting Down

Carr and Shepherd[58] have pointed out that to stand up from a sitting position, it is necessary to sequence 4 actions:

- shifting the body forward so that the buttocks are close to the edge of the chair,
- placing the feet flat on the floor so that the heels are well back,
- leaning the trunk forward, and
- standing up quickly while thinking of leaning "forward and up" in an arc of movement.

A common problem is that people with PD fail to lean far enough forward when standing up. As a result, the line of center of gravity falls too far posteriorly in relation to the feet, and the loading moments of force

on the hips and knees are increased.[58] This problem makes rising very difficult. A downward gaze and loss of momentum due to akinesia further increase the difficulty in performing this task. For people with hypokinesia, mental rehearsal of the sequence prior to its performance as well as the use of verbal cues, such as counting or saying the action out loud, may enable this task to be performed more easily.[22] In people with akinesia, the use of proprioceptive cues, such as gently rocking backward and forward prior to the movement, or auditory cues, such as saying "go," can be of use. In a study of the sit-to-stand movement using these strategies, Kamsma and colleagues[59] found that 4 training sessions resulted in reduced errors in the planning and execution of this action sequence in 10 subjects with PD. In addition, 3 patients who were examined 1 year after training showed no deterioration in performance, which is promising given that PD is a progressive neurological condition. Yekutiel et al[6] showed that 12 subjects with PD improved their sit-to-stand time by more than 50% with 3 months of twice-weekly physical therapy that emphasized attention on whole body movements during this action. By increasing the speed of this action, it is likely that it became more energy efficient and easier to perform.

In frail older people or those with marked disability, a chair with a high seat and armrests can be used to enable the person to stand up. By increasing the height of the seat and using armrests, the loading moment of force on the hips, knees, and ankles is reduced,[32,58] making the task easier to perform. Observations suggest that, for some people, it is useful to start with a chair with a high seat and armrests so the sit-to-stand action is performed independently and to gradually reduce seat height (and presence of armrests) over the course of training so the person can eventually perform the action from a standard dining room chair.[32] In addition to assessing the suitability of chair design, the physical therapist should assess the person's ability to get in and out of a chair at a table. This is another sequential task of everyday living that is difficult for people with PD to perform. Caregivers also need to be shown how they can safely assist with this task so as to avoid musculoskeletal injuries, which might occur when they attempt to move a chair while the person with PD is still sitting.

Turning Over and Getting Out of Bed

Another frequent problem reported by people with PD is difficulty in turning over and getting out of bed. There are, in my view, 2 main reasons why this task is difficult to perform. First, it is a complex sequential motor skill that has many subcomponents, which include:

- throwing back the bed covers,
- shifting the pelvis toward the center of the bed so that, when the turn is completed, the body is not too close to the edge,
- turning the head,
- bringing the arm across the body in the direction of rolling,
- swinging the legs over the edge,
- pushing up, and
- adjusting postural alignment to sit upright.

Second, this action is usually performed at night, when levodopa levels are low and hypokinesia and akinesia are at a peak. At night, people often perform this action in near-darkness, which means that they cannot use vision to guide each movement in the sequence. Urinary frequency and urgency are also common in people with PD due to poor control of the detrussor muscle, arising from the effects of PD on the autonomic nervous system.[32] Therefore, these individuals may need to get out of bed and walk to the toilet many times every night. For these reasons, I argue that it is essential to teach the person with PD effective strategies for rolling over, moving around, and getting in and out of bed so that the caregiver is not required to physically assist with this task multiple times every evening.

My observations, together with those of Kirkwood et al,[57] suggest the following strategies can assist with turning over and getting out of bed:

- using a slow-acting levodopa medication (such as Sinemet CR* or Madopar HBS[†]) overnight to increase bed mobility,
- keeping a night-light on so that vision can be used to guide the movement,
- using a lightweight quilt on the bed, which is easier to throw back than heavy blankets and sheet; couple this with satin or silk sheets and nightwear to reduce friction,[56]
- mentally rehearsing the action sequence before it is commenced,
- consciously attending to performing each submovement one at a time and focusing attention on each submovement as it is performed,
- using self-generated or caregiver-generated verbal cues (such as "head, arm, legs, up") to trigger each submovement in the sequence, and
- ensuring that the bed height is not too low, as this makes it difficult to stand up.

* Merck, Sharp and Dohme (Australia) Pty Ltd, 473 Williamstown Rd, Port Melbourne, Victoria, Australia 3207.
† Roche Pharmaceuticals, Roche Laboratories Inc, 340 Kingsland St, Nutley, NJ 07110.

Placement of a commode chair next to the bed also reduces the distance the person needs to walk for toileting. Only one experiment has quantified the effects of cognitive strategies on the ability to turn in bed. In a sample of 10 subjects with PD, Kamsma et al[59] found that repetitive practice of a bed mobility strategy that incorporated mental rehearsal as well as breaking the action sequence down into steps that avoided the need for simultaneous action led to progressive improvements in performance. Gains were greatest for people classified as grade II or III on the Hoehn and Yahr scale[60] and least for people were severely disabled (grade IV) and had limited potential for motor skill learning. However, measurements were obtained only over 4 sessions, and the long-term effects of training were not evaluated.

Preventing Falls

More than 35% of people with advanced PD experience falls, and 18% sustain fractures as a result of falling over.[61] Therefore, falls prevention is a major goal of physical therapy for people with end-stage disease. It is beyond the scope of this article to discuss all of the strategies that can be used to prevent falls, and there is extensive research literature on falls prevention (for reviews, refer to Morris and colleagues[22,32,62] and Lipsitz et al[63]). In broad terms, prevention strategies can be grouped according to whether the person's falls are due to:

- movement disorders and cognitive impairment arising from PD,
- the way that tasks are performed (unitask or multi-task),
- environmental factors,
- adverse effects of medication, or
- individual (non-PD) factors, such as age-related changes in postural control or weakness.[62]

By keeping a falls diary that details the location of falls in the home and describes the task being performed, date, time of day, and time of last medication, people with PD can better inform therapists about the nature of their falls.[64] This information allows an effective prevention program to be tailored to individual needs.

Reaching, Grasping, Manipulating Objects, and Writing

Due to bradykinesia, the ability to reach for, grasp, and manipulate objects is compromised in many people with PD, and sequential tasks such as dressing, grooming, and feeding are performed exceedingly slowly and with movements that are underscaled in size.[65–67] People with PD also generate abnormally high grip forces when performing precision grip tasks such as lifting a peg or a pencil.[66] The scaling disorder in PD appears to be related to defective reflex-gain mechanisms, which are also implicated in the genesis of rigidity.[28,29] In addition, people with PD take longer than usual to lift objects, particularly when the load is very light.[33] The physical therapist often works in conjunction with occupational therapists and caregivers to train people with PD to utilize strategies to enhance reaching, grasping, and manipulation. My experience suggests that it may be valuable to provide the following advice:

- Mentally rehearse the action sequence before it is performed.
- Look at the object to be grasped before and during movement, as the object may act as a "visual cue" that activates more normal upper-limb performance.
- Break prehension movements down into separate parts and concentrate on performing each component separately. For example, to pick up and take a drink from a polystyrene cup filled with water, the person could be trained to concentrate on executing one or more of the following submovements step by step: (1) transporting the hand to the object, (2) opening the hand so that the aperture is a little larger than the object, enabling it to be grasped, (3) closing the hand around the object, (4) gently applying force to grip the cup so that the polystyrene is not distorted, (5) lifting the cup to the mouth without applying excessive grip force, (6) gradually tilting the cup and drinking, (7) returning the cup to the table, and (8) releasing grasp of the cup.
- Verbally cue key components of the task, such as saying "go" to help trigger transportation of the arm or "release" to let go of the object at completion (which many people find to be the most difficult phase of the task due to difficulty terminating actions).
- Avoid attending to distracting stimuli in the environment or performing a secondary task at the same time.

Although people with PD are slow to reach to stationary targets, they are able to reach forward and grasp moving objects (such as a moving ball) at near-normal speed, presumably because the movement of the ball triggers lower-level brain-stem or spinal reflex responses that bypass the defective BG.[33] However, people rarely reach toward moving objects in everyday life.[58] I contend it is much more important to ensure that people with PD practice prehension tasks that are performed routinely, such as grasping and manipulating objects for dressing, eating, grooming, showering, home duties, leisure activities, and work-related tasks.

Even more troublesome than reaching and grasping is handwriting, a task that requires constant attention in people with PD to ensure that the strokes do not progressively diminish in size and speed. A number of experiments have shown that lined paper assists people to write more easily, presumably by acting as an ampli-

Table 2.
Upper-Limb Homework Tasks

- Buttoning, with buttons of different sizes and shapes
- Handwriting (eg, crosswords, writing with lined paper, signatures, filling in forms with multiple boxes)
- Reaching, grasping, and drinking from cups of different sizes, shapes, and weights (eg, china cups, coffee mugs, polystyrene cups), which afford different grasps and grip strengths
- Pouring water from one cup to another (difficult in marked bradykinesia)
- Opening and closing a range of jars with contents inside
- Lifting jars and boxes of different weights onto and off pantry shelves of different heights
- Picking up grains of rice with the thumb and forefinger and placing them in an eggcup
- Picking up a straw between the thumb and forefinger and placing it in a can
- Elements of dressing, such as putting on a sweater while incorporating verbal cues such as "right arm, left arm, head, pull"
- Dialing telephone numbers of family, friends, and work colleagues (while sitting down)
- Paper folding, such as folding napkins and placing letters in envelopes

tude cue to guide this action sequence.[14] My observations suggest that focusing attention on writing with large strokes may also enable some people to overcome micrographia. However, the use of visual cues and attentional strategies appears to have only short-term effects, and the micrographic handwriting returns when the person performs a second task, such as talking on the telephone while attempting to write a message.

In general, I recommend that people with upper-limb involvement due to PD set aside time each week to practice performing a range of prehension tasks that require dexterity, precision, and careful attention to force regulation so that these skills are maintained at an optimal level. It is usually most beneficial for this homework to incorporate functional tasks specific to the individual, such as those listed in Table 2. To enhance generalizability of training, I recommend that a number of variations of each task be practiced, with different goals, object sizes, shapes, textures, and weights as well as variations in movement speed and object distance. Tasks can also be designed to maintain muscle length and force development. For example, hanging clothes on a clothesline can be structured in a way that stretches the shoulder and elbow extensors and long finger flexor muscles at the same time as providing the person with opportunities to practice his or her pincer grip.

Maintaining General Fitness, Muscle Force, Aerobic Capacity, and an Upright Posture

The prevention of muscle atrophy and weakness, restricted range of movement, and reduced exercise capacity is usually the initial aim of physical therapy for people with PD and, in my opinion, should commence as soon as their condition is diagnosed. In the early stages, encouragement to participate in regular physical activities such as walking, swimming, yoga, tai chi, golf, lawn bowling, or cycling on a bicycle track may be all that is needed. Although people with PD have a more rapid drop in physical activity levels than age-matched control subjects,[68] Canning et al[47] suggest that people with mild to moderate PD have the potential to maintain normal exercise capacity with regular aerobic exercise. They also have the potential for improvement of force development and coordination with regular activities such as karate[69] and spinal flexibility exercises.[44,70,71] As the disease progresses, task-specific practice routines incorporating an aerobic element may be beneficial.[44] Examples include practicing standing from a sitting position to enhance quadriceps femoris muscle force development using seats of appropriate height to achieve a training effect and stair climbing or repetitive stepping on and off a small step to improve force development in the triceps surae muscles. Details on how to select the type of task, contraction, load, number of repetitions, and duration of training to obtain context-appropriate force development and range of movement are provided elsewhere.[32,70,71] Programs to increase force development, range of movement, or endurance need to be adapted when movement disorders such as hypokinesia and akinesia are present. Therefore, visual cues and attentional strategies such as breaking down complex tasks into parts and focusing on unitask performance may need to be incorporated into the training program.[72,77]

When a person is found to have postural deformities or malalignment (eg, forward stooped posture), the therapist should assess whether the condition is due to muscular, joint, or skeletal factors. If it is restricted to soft tissue factors, then I recommend visual feedback (in the form of photographs, mirrors, or videotape) coupled with carefully designed strengthening or stretching programs. My observations suggest that lying flat in a supine or prone position for at least 30 minutes every day is advisable in order to maintain muscle length. When the malalignment cannot be volitionally corrected, the provision of orthoses or special seating and bedding may be considered. These interventions await validation with controlled clinical trials.

Evidence for the Efficacy of Physical Therapy Interventions

Throughout this article, reference has been made to the evidence supporting various physical therapy interventions for people with PD. It is useful to classify this evidence according to Sackett's rules[78] (Tab. 3[‡]). According to Sackett,[78] there are 5 main levels of evi-

[‡] References 6, 7, 10, 14, 25, 30, 31, 35–38, 40, 41, 44, 48, 51, 54–57, 59, 69–77, 80–82.

Table 3.
Evidence for Physical Therapy Treatment of Movement Disorders in Parkinson Disease[78]

Level	Description	Investigations on Effects of Physical Therapy
1	Large randomized trial with clear-cut results	General exercises[44]
2	Small randomized trial with uncertain results	General exercises[71]
3	Nonrandomized, contemporaneous controls	Visual cues[7,14,35–37] Auditory cues[54–56,81] Verbal instructional sets[40] Attentional/compensatory strategies[6,37,40,41,57,59,77] Dual-task avoidance[38,57,59] Aerobic exericse[48,69] Karate[69] General exercises[69,72,74,77] Whole-body movements[6] Sensory stimulation[75,82] Stretches[74] Bobath technique[31,a] Peto technique[31,a] Proprioceptive neuromuscular facilitation[31,a] Trunk muscle training[70]
4	Nonrandomized, historical controls	Exercises, heat, vibration[76]
5	No controls, case series only	Combined therapy[10,80] Stretches[10] Relaxation[10] Visual cues[10,25,51,73] Proprioceptive neuromuscular facilitation[30]

[a] No results for effects of physical therapy intervention. The remaining investigations had positive results.

dence for clinical interventions. At the highest level (level 1) are interventions that have been validated with RCTs with low false-positive (alpha) rates and high power. Level 2 is where the intervention is supported by RCTs with high false-positive rates and low power.[78] Level 3 applies when nonrandomized comparisons between concurrent, matched groups have been used. Alternatively, a group may be compared with control subjects or with their own performance at another point in time. Level 4 applies to nonrandomized "historical" group comparisons, such as comparing one group treated according to local hospital procedures with another a group previously treated at the same hospital.[78] This category also includes experimentally controlled single-case time-series designs. Level 5 refers to case series without controls, where information is provided only on the outcome of patients[78] without evidence of experimental design. Case histories can be classified under this heading.

As shown in Table 3, only 2 RCTs have yet been conducted on physical therapy for PD. Schenkman et al[44] found that exercises to enhance recruitment of appropriate muscle synergies as well as relaxation to enhance muscle length and coordination enhanced function in 46 people with PD. Comella et al[71] showed that repetitive exercises done for 4 weeks to improve range of motion, endurance, balance, walking, and fine motor dexterity led to improvements on the motor section of the United Parkinson's Disease Rating Scale in 16 people with PD. Neither of these studies investigated the effects of external cues, cognitive strategies, task-specific training, or environmental modification on movement in people with PD. The effect of these latter interventions still needs to be validated with RCTs. Table 3 shows that most of the evidence for physical therapy for people with PD is still only at levels 1 to 3.

Putting It All Together: Establishing Core Elements of Physical Therapy in Addition to Addressing Individual Needs

In the current health care environment, where physical therapists may need to treat large numbers of patients with few resources and little time, I argue that it is essential to identify the core elements that form the basic unit of physical therapy interventions, to which other elements can be added according to the needs of the individual. Table 4 summarizes the core elements of physical therapy that I recommend be incorporated into the weekly routines of the vast majority of people with idiopathic PD. Because PD slowly progresses over periods of 5 to 30 years, I contend it is necessary to adjust the core routine according to the stage of disability. The Hoehn and Yahr scale[60] provides a useful method of categorizing patients according to their level of disability, and the core routines shown in Table 4 are defined in relation to this scale.

In addition to the basic training routine outlined in Table 4, I recommend that physical therapists teach patients and their caregivers additional strategies for coping with the specific movement disorders and functional problems that each individual encounters. The following case history provides an example of how this may be done. Readers can refer to Schenkman and colleagues[9,10] and Morris and Iansek[52] for further examples of how physical therapy can be tailored to individual needs.

Table 4.
Suggested Core Elements of Physical Therapy According to Hoehn and Yahr Scale[60] Stages of Disability

Hoehn and Yahr stage I: "Unilateral involvement only, usually with minimal or no functional impairment"

Primary physical therapy goals:

1. *Health promotion and maintenance of aerobic fitness, muscle force, and soft tissue extensibility*
2. *Education of the person with PD[a] and caregiver about the disease and ways in which to prevent secondary complications*
3. *Train the person with PD in movement strategies for later use while the person still has intact cognition*

- Maintain regular physical activity; walk at least 3 times a week for 40 minutes, concentrating on maintaining long strides and adequate ground clearance; practice walking over a variety of terrains; practice stepping on and off curbs and stepping over obstacles; continue with activities such as playing golf, lawn bowling, dancing, karate, tai chi, and yoga
- Maintain upright posture by (1) consciously attending to standing upright, (2) checking posture in mirror, and (3) strengthening low back extensors and hip extensor muscles
- Minimize micrographia by writing at least one page of script every day, concentrating on forming large, even characters
- To maintain lower-limb force, practice standing up from seats of different heights; squats; stair climbing
- Practice standing up, turning, walking, and moving from lying supine to sitting up over edge of the bed using cueing and attentional strategies

Hoehn and Yahr stage II: "Bilateral or midline involvement, without impairment of balance"

Primary physical therapy goals:

1. *Train the person with PD in movement strategies for hypokinesia, bradykinesia, akinesia, and dyskinesia, as needed, within the context of tasks of everyday living*
2. *Teach the person with PD and caregiver how to monitor the effects of medication*
3. *Educate person with PD and caregiver about the disease and ways in which to prevent secondary complications and to maintain aerobic fitness, muscle force, and soft tissue extensibility*
4. *Environmental analysis and environmental restructuring to prevent falls and enhance movement*

- Maintain regular physical activity; walk at least 3 times a week for 40 minutes, concentrating on maintaining long strides and adequate ground clearance; practice walking over a variety of terrains; practice stepping on and off curbs and stepping over obstacles (may need supervision); continue with activities such as playing golf, lawn bowling, dancing, karate, tai chi, yoga, and stationary bicycling
- Maintain upright posture by (1) consciously attending to standing upright, (2) checking posture in mirror, and (3) strengthening low back extensors and hip extensor muscles
- Minimize micrographia by writing at least one page of script every day, concentrating on forming large, even characters
- To maintain lower-limb muscle force, practice standing up from seats of different heights; squats; steps
- Practice standing up, turning, walking, and moving from lying supine to sitting up over edge of the bed using cueing and attentional strategies
- Structure the home to prevent falls (eg, create large open walkways, remove loose cords and rugs, install handrails in bathrooms and on stairs, repair uneven pavement)
- Implement muscle stretches and positioning programs (eg, prone lying 30 minutes per day, calf stretches)

Hoehn and Yahr stage III: "First sign of impaired righting reflexes. This is evident by unsteadiness as the patient turns or is demonstrated when he or she is pushed from standing equilibrium with the feet together and eyes closed. Functionally, the patient is somewhat restricted in his or her activities but may have some work potential, depending on the type of employment. Patients are physically capable of leading independent lives, and their disability is mild to moderate."

Primary physical therapy goals:

1. *Train the person with PD in movement strategies for postural instability, hypokinesia, bradykinesia, akinesia, and dyskinesia, as needed, within the context of tasks of everyday living*
2. *Prevent falls*
3. *Teach the person with PD and caregiver how to recognize and respond to "on" and "off" stages of the medication cycle: 2 sets of movement strategies may be needed (one for "on" phase and one for "off" phase of medication)*
4. *Health promotion and maintenance of regular physical activity with aerobic and endurance components*
5. *Teach the caregiver to reinforce physical therapy strategies in the home and community*

- Maintain regular physical activity; walk daily at least 100 m with large strides and more than 1.5 cm of ground clearance using cues or attentional strategies; practice walking over a variety of terrains and negotiating obstacles with supervision from another person; carefully attend to stepping on and off curbs and stepping over obstacles; when possible, continue with activities such as playing golf, lawn bowling, dancing, karate, tai chi, yoga, stationary bicycling, and treadmill walking
- Keep a falls diary, recording the date, time, location, and task performed when falls occur; structure the home to prevent falls; know what factors predispose people with PD toward falls

(continued)

Table 4.
Continued

- Practice strategies (eg, attention, cues, mental rehearsal, unitask performance) for overcoming movement slowness and postural instability when walking, standing up, moving around the bed, turning and reaching, grasping and manipulating objects, and writing
- Maintain upright posture by (1) consciously attending to standing upright, (2) checking posture in mirror, and (3) strengthening low back extensors and hip extensors to maintain lower-limb muscle force, practice standing up from seats of different heights; squats
- Muscle stretches and positioning program (eg, prone lying 30 minutes per day, calf stretches in a standing position using a wedge)

Hoehn and Yahr stage IV: "Fully developed, severely disabling disease; the patient is still able to walk and stand unassisted but is markedly incapacitated"

Primary physical therapy goals:

1. *Train the caregiver in how to reinforce physical therapy strategies for preventing falls and coping with hypokinesia, bradykinesia, akinesia, and dyskinesia within the context of tasks of everyday living*
2. *Train the person with PD and caregiver in what to do if a fall occurs*
3. *Ensure that the person with PD or caregiver can correctly administer medications and monitor the effects of medications, and know how to telephone if movement disorders/cognitive impairment suddenly become severe*
4. *Maintain walking distance and endurance, aerobic fitness, muscle force, and soft tissue extensibility*
5. *Ensure that the person with PD and caregiver are implementing strategies to prevent secondary musculoskeletal sequelae*

- Together with the caregiver/friend/assistant, maintain regular physical activity; walk daily at least 100 m with large strides using cues or attentional strategies (may require wheeled walking frame or assistance from another person); carefully attend to stepping on and off curbs and stepping over obstacles; when possible, continue with physical activities and social outings
- Together with the caregiver, practice strategies (eg, attention, cues, mental rehearsal, unitask performance) for overcoming akinesia, freezing, dyskinesia, and movement slowness when walking, standing up, moving around the bed, turning and reaching, grasping and manipulating objects, and writing
- Keep a falls diary, recording the date, time, location, and task performed when the fall occurred; structure the home to prevent falls; know what factors predispose people with PD toward falls
- Maintain upright posture by (1) consciously attending to standing upright, (2) checking posture in mirror, and (3) strengthening low back extensors and hip extensors
- To maintain lower-limb muscle force, practice standing up from seats of different heights; squats
- Muscle stretches and positioning programs (eg, prone lying 30 minutes per day, calf stretches in a standing position using a wedge; hamstring muscle stretches in a sitting position)

Hoehn and Yahr stage V: "Confinement to bed or wheelchair, unless aided"

Primary physical therapy goals:

1. *Maintain activity and participation and enhance comfort and quality of life*
2. *Prevent falls*
3. *Where possible, reinforce movement strategies to assist with walking, moving around the bed, reaching and grasping, standing up and turning*
4. *Train caregivers and nursing staff in safe practices for lifting/transferring/toileting/showering/dressing/feeding*
5. *Prevent skin breakdown and pressure areas*
6. *Maintain clear airways and vital capacity*
7. *Train the person with PD, caregiver, and nursing staff in positioning in sitting and lying and the need for regular changes in body position*
8. *If appropriate, act as an advocate for the needs and rights of the person with PD*

- Assisted/supervised walking daily, when possible; may need to use a wheeled walker
- Assisted/supervised standing daily, when possible
- If possible, lie supine for 15 minutes twice daily; otherwise, position in side lying with hips, knees, and trunk in a neutral position
- Prevent falls; caregiver/nursing staff to maintain falls diary
- As necessary, prescribe appropriate wheelchairs, chairs, beds, and other assistive devices
- Educate caregivers and nursing staff about safe techniques for lifting and transfers and how to assist with bed mobility and environmental restructuring to promote movement and safety
- Educate the person with PD, caregiver, and nursing staff about the need for regular changes in position, optimal body positioning, and skin care

[a] PD=Parkinson disease.

Case History: Mrs A

In 1988, at the age of 39 years, Mrs A was diagnosed with PD by her family general practitioner after exhibiting a mild resting tremor of her left hand, micrographia, slowing of movement, and occasional loss of balance. Mrs A had previously been well and lived at home with her husband and 16-year-old daughter. She reported home duties as her occupation. Because the movement disorders were initially mild, the general practitioner advised that drug treatment be withheld until symptoms were more apparent. By 1990, Mrs A reported that domestic tasks were becoming increasingly difficult to perform due to slowness of movement, tremor, and loss of balance. She was referred to a neurologist, who prescribed Madopar M† (levodopa benserazide) to be taken 5 times per day, and this medication provided temporary relief of all symptoms (Tab. 5).

By April 1993, the symptoms had re-emerged due to disease progression, despite attempts to adjust medication. Mrs A was referred to the Kingston Centre Movement Disorders Clinic for consultation with the physical therapist, neurologist, occupational therapist, and other team members. Over the first 3 clinic consultations (spaced 1 month apart), the physical therapist conducted an assessment of movement disorders and functional disability and commenced a movement training program. The initial physical therapy assessment in May 1993 revealed the following problems:

- a short-stepped hypokinetic walking pattern,
- mild gait akinesia and occasional freezing in doorways,
- delayed stepping response to external perturbation of the center of mass in steady stance, and
- moderately severe resting tremor of the left (nondominant) hand.

In addition, at the end of dose of each 4-hour medication cycle, there was moderately severe dyskinesia of the head, upper limbs, and trunk. The episodes of dyskinesia were most pronounced in the afternoons, particularly around 3:30. Due to the dyskinesia, driving was restricted to the mornings. The neurologist, therefore, changed the medication regimen by adding Madopar Q† 5 times a day to the Madopar M that Mrs A was already taking.

Because Mrs A attended the clinic as an outpatient, the physical therapy intervention consisted of a home program to overcome movement difficulties. The physical therapist and the occupational therapist attended the home in an attempt to ensure that strategies were being used effectively in that setting and were reinforced by the family. An outdoor mobility course was mapped out in the back garden to enable Mrs A to practice maintaining balance during locomotion. Homework (a home practice routine) was written up in a diary and included strategies for coping with gait hypokinesia (visual cues and attentional strategy training), freezing (counting out loud, rhythmically rocking from side to side, stopping the task and then beginning the task again, and thinking of stepping over a log), postural instability (practicing stepping strategies, mobility course), and extra movements (looking at the part that is moving excessively and thinking about reducing the overactivity of that part; progressive relaxation). For general fitness, she was encouraged to continue with daily half-hour walks and twice-weekly yoga classes. In addition, the physical therapist together with the other team members commenced the process of educating Mrs A and her family about PD and how to best cope with movement disorders. Information was also provided on how to access support groups, such as the Parkinson's Disease Association.

The next contact was in June 1994 when Mrs A was admitted to the inpatient ward for a 3-week period to monitor the effects of medication and provide her with an opportunity for intensive movement training. The main problems noted by the physical therapist at the admission assessment were:

- painful dystonic posturing of the left foot due to overactivity of the gastrocnemius and soleus muscles during walking and standing tasks,
- difficulty rolling over to the left and getting out of bed,
- reduced left-hand dexterity, limiting the ability to sew, prepare food, and tie laces, and
- difficulty writing in the afternoons and evenings.

The goals of physical therapy were modified to include: (1) teaching Mrs A additional strategies for overcoming dystonia, (2) enhancement of bed mobility, and (3) use of visual cues and attentional strategies for improving writing and dexterity of the hands. To temporarily reduce dystonia, Mrs A was shown how to perform a prolonged stretch of the gastrocnemius and soleus muscles in standing. In an effort to enhance bed mobility, she was taught to mentally rehearse the rolling-over sequence, read instructions on a cue card placed on a bedside table, and break the action of rolling over into parts. Mrs A was also trained to deliberately look at the object she was reaching for and to practice a variety of upper-limb tasks, as outlined in Table 2. While an inpatient, Mrs A attended two 40-minute physical therapy sessions 5 days a week for the 3-week period. Over this time, she made considerable gains, and at the time of discharge she was using stretches to temporarily overcome the dystonia. Her stride length had increased, and she was able to walk more than 200 m at a time by

Table 5.
Physical Therapy Assessment Data for Mrs. A Over a Period of 10 Years[a]

	Diagnosis, 1988	Neurologist Referral, 1990	Clinic Referral, May 1993	June 1994	July 1996	February 1998	December 1998
Movement disorder							
Bradykinesia	X	X	X		X		X
Akinesia			X initiation disturbance 20% of time				
Freezing			X Occasional				
Dyskinesia (choreiform movements)			X	X			
Dystonia			X	X L plantar flexors	X L plantar flexors	X L plantar flexors	X L plantar flexors
Tremor (resting)	X	X	X			X	
Postural instability							
Steady stance						X Tandem 7/15 s	
Self-pertubation	X	X			X Step test=6:15 s	X Step test=5:15 s	
External pertubation	X	X	X 3/5	X 3/5	X 3/5	X 4/5	
Functional reach					X 26 cm	X 26 cm	
Functional task							
Walking			X	X	X	X	
Speed			65 m/min	58.5 m/min	77.7 m/min	65 m/min	
Stride length			1.3 m	1.3 m	1.4 m	1.3 m	
Cadence			100 steps/min	90 steps/min	111 steps/min	100 steps/min	
Double-limb support			35% of gait cycle	38% of gait cycle	35% of gait cycle	35% of gait cycle	
Timed Up & Go Test					11.3 s	10.0 s	
Turning over in bed				X 6.8 s			
Standing up							
Handwriting (micrographia)	X	X	X	X	X	X	X
Reaching, grasping, manipulating objects				X Left-hand dexterity decreased		X Left-hand dexterity decreased	
Falls					X 3 in last 6 mo		
Aerobic capacity	P	P	P	P	X Walking distance <80 m	P	X Walking distance <50 m
Disability (Webster Disability Scale)[b]			15	13	15	12	16
Medications	None	Madopar M 100/25, 5 times/d	Madopar M 100/25, 5 times/d Madopar Q 50/12.5, 5 times/d	Madopar M 100/25, 5 times/d Madopar Q 50/12.5, 5 times/d	Sinemet CR (1/2) 5 times/d Madopar Q 50/12.5 Pergolide mesylate 500 μg, 3 times/d	Sinemet CR 200/50, 3 times/d Sinemet CR (1/2) 200/50, 2 times/d Pergolide mesylate 500 μg, 3 times/d	Sinemet CR 200/50, once Pergolide mesylate 750 μg, 3 times/d Madopar M 100/25, 4 times/d

[a] X=problem in this area requiring treatment; P=requiring preventative program.
[b] Kempster PA, Frankel JP, Bovingdon M, et al. Levodopa peripheral pharmacokinetics and duration of motor response in Parkinson's disease. *J Neurol Neurosurg Psychiatry.* 1989;52:718–723.

concentrating on walking with large strides. Upper-limb performance was functional.

Mrs A returned home with her diary revised to include gastrocnemius and soleus muscle stretches, bed mobility activities, and use of attentional strategies during upper-limb performance in addition to the other tasks. She was re-examined at 6 monthly intervals at the Movement Disorders Clinic. She remained stable until July 1996, when she experienced a series of falls. Readmission to the inpatient ward for assessment and reinforcement of movement training was arranged. On admission, mild bradykinesia was evident. During the previous 6 weeks, she had fallen in the garden when weeding, had slipped on the bathroom mat and fallen to the floor, and had fallen again in the garden when turning around to talk to her grandson. There was no evidence of postural hypotension. The physical therapy assessment showed that she could maintain steady standing positions with feet apart, with feet together, during stride stance, and during single-limb stance for the maximum testing time of 30 seconds. However, she performed poorly for her age on the step test and the Pastor Pull Test.[26]

Physical therapy intervention at this stage was targeted at preventing falls. In addition to reinforcing the need to focus attention on maintaining balance when performing mobility tasks in a standing position, she was educated about the risk factors for falls. Another home visit was conducted by the physical therapist and the occupational therapist, with Mr and Mrs A present. This home visit resulted in the removal of loose mats, cords, and a glass-topped coffee table in the center of the family room. The occupational therapist recommended that the concrete path in the back garden be repaired, and a set of handrails was installed at the backdoor steps. A rail was also installed in the shower recess, and a nonslip mat, shower chair, and handheld shower hose were provided for showering. Mrs A was encouraged to maintain regular physical activities, such as walking and yoga, with a partner present. She was provided with a falls diary and trained in how to record the date, time, location, and perceived reason for each fall. After discharge, Mrs A continued with 3 monthly monitoring visits to the Movement Disorders Clinic and continued with the daily homework program and falls diary.

In February 1998, Mrs A was again admitted to the inpatient ward, this time for severe resting tremor in the hands at the end of dose. The neurologist hypothesized that the tremor was due to neurotransmitter imbalance, and her medication regimen was therefore altered to levodopa carbidopa and pergolide (Tab. 5). Before and after alteration of the medication regimen, the physical therapist conducted dose-response trials, which are serial measurements used by physical therapists, to chart the effects of medication on movement disorders and independence (refer to Morris et al[5]). The new medication regimen soon ameliorated the tremor and resolved residual dexterity problems of the left hand. However, mild gait hypokinesia was again apparent. With longer walking sequences, the walking speed slowed even further, and she was finding it difficult to traverse pedestrian crossings with sufficient speed to avoid traffic. Although Mrs A only took 10 seconds to complete the Timed Up & Go Test,[79] her footsteps progressively reduced in size and number of steps during the turning component of the test.

The goals of physical therapy at this stage were to increase her walking speed to 75 m/min and to teach her strategies for avoiding motor instability when walking and turning. Once again, training incorporated the use of mental rehearsal (visualizing walking with long steps before the action), visual cues and attentional strategies, avoidance of secondary task performance when walking and turning, and turning using a large, "whole-body" arc of movement rather than swiveling around on a small base of support. Training was conducted within the context of community ambulation tasks such as road crossing, shopping, and negotiation of curbs, slopes, and rough ground. To continue to promote general fitness and aerobic capacity, Mrs A was encouraged to walk for 30 minutes each day with another person, as well as to continue with her regular yoga classes. By the end of this 3-week admission, Mrs A had achieved her major goal of walking confidently at 75 m/min over a range of surfaces as well as having no residual tremor or difficulties with reaching, grasping, and manipulating objects. She again was discharged home with a home program written up in her diary.

When Mrs A was re-examined in December 1998, micrographia, mild bradykinesia, and dystonia of the plantar flexors were found to be residual problems that were not markedly disabling. Because her aerobic capacity and walking distance had diminished, these problems become the focus of a burst of more intensive physical therapy treatment. She was encouraged to walk at least 3 times a week, concentrating on maintaining long strides, and to participate in yoga and other physical activities.

This case history illustrates how physical therapy intervention was adapted according to the client's needs over the first 10 years that she had PD. The example shows how the signs and symptoms of PD as well as the medication regimen changed over time, requiring frequent adjustment of physical therapy goals and procedures. Some problems persisted, despite attempts at adjusting medication and physical therapy. The persistent problems were dystonia of the gastrocnemius and soleus muscles, micrographia, postural instability, and

mild gait hypokinesia. The most intensive period for physical therapy was in May 1993, when the initial medication regimen and physical therapy training program needed to be established. For this woman, bursts of goal-directed physical therapy provided within a multidisciplinary team setting appeared to be particularly helpful in managing the symptoms of PD.

Conclusion

This article has outlined a model for physical therapy management of people with PD. I have advocated the need to treat movement disorders such as bradykinesia and postural instability within the context of functional tasks of everyday living such as walking, turning over in bed, and manipulating objects. I have also emphasized the need for treatment goals to be regularly reviewed and adjusted by the multidisciplinary team, patient, and caregivers, according to the decline in performance that inevitably occurs. Physical therapy intervention cannot cure movement disorders in people with PD. Rather, it presumably offers symptomatic relief by teaching people strategies for bypassing the defective BG in order to move more easily. Physical therapists can also teach strategies for maintaining the musculoskeletal and cardiovascular systems in optimal condition and for preventing falls. Together with medication, therefore, physical therapy has the potential to reduce disability among people with this disease and to enhance their quality of life. Randomized clinical trials are now needed to evaluate the specific effects of physical therapy and to validate this model of care for people with PD.

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