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Hypokinesia and Movement Challenges in Parkinson's Disease

by Eva Henry, MD and Eugene C. Lai, MD, PhD

Parkinson's disease (PD) is a chronic, progressive neurodegenerative disease characterized by hypokinesia and motor dysfunction. The cardinal symptoms of PD are *bradykinesia, rigidity, postural instability, and tremor.* Associated deficits such as worsening handwriting (micrographia), shuffling gait, speech disturbance, dysphagia, and finger incoordination also occur frequently. In general, the combination of asymmetric presentation of symptoms and clinical improvement with levodopa treatment distinguishes PD from other movement disorders.

PD is caused by dysfunctions in the basal ganglia. The basal ganglia consist of several important nuclei in the subcortical part of the brain and play a major role in initiation and control of normal voluntary movement. They do not regulate strength nor do they communicate with the spinal cord or peripheral limbs directly. They receive their primary input from the cerebral cortex and send the majority of their output, via the thalamus, back to the cortex. Neuronal loss in the substantia nigra, leading to a decrease in the level of dopamine in the striatum, is the pathophysiologic hallmark of PD. The loss of dopamine produces an imbalance of activities in the striatum and causes an increase in inhibitory signals to the thalamus. Because the thalamus is responsible for the activation of the cortical areas involved in generation of movements, the final outcome of dopamine deficiency is poverty or slowness of movements.

Bradykinesia or slowness of movement can be observed in the loss of a patient's automatic movements, such as decreased arm-swing while walking, eye-blinking, facial expression, or swallowing of saliva. Patients also complain of difficulty getting up from a chair, turning over in bed, or putting on a jacket or sweater. They feel restrained in their movements and every action becomes more effortful. There is hesitation in initiating voluntary movement, sluggishness during movement, and then rapid fatigue. Because of basal ganglia dysfunction, there appears to be difficulty in organizing and initiating the proper sequence of cortical neuronal signals to carry out a motor task. Limitations in movement can also be caused by the incoordination of reciprocal agonist and antagonist muscles involved in the movement. All movement requires the cooperation of opposite muscles and a failure in their reciprocal relaxation results in a decrease in the flexibility, speed, and range of the action. Bradykinesia is evident when a patient performs rapid alternating movements, such as pronation and supination of the forearms.

Rigidity or muscle stiffness, characteristically associated with a cogwheel quality (regular, jerky resistance to motion), can be demonstrated in both passive and active movements of the limbs. It also can involve the axial muscles and produce abnormal muscle tone. Muscles are constantly tensed in a state of sustained contractions, typically due to the failure of antagonist muscles to relax during movement. In addition to slower movements, rigidity can cause back and neck pain, cramping, and persistent soreness and a feeling of heaviness in the muscles. It also results in the typical stooped posture with flexed limbs in PD.

Postural instability causes gait imbalance and falls. The maintenance and control of posture requires coordination of many motor and sensory inputs and outputs to and from the brain. In particular, there is a lack of dopamine from the basal ganglia to the brain stem that affects the integrity of postural control. Furthermore, bradykinesia and rigidity cause slowness and inflexibility of muscle responses to rapid postural changes, thus creating imbalance with movements. Postural instability can be demonstrated by an abnormal "pull test" in which the patient takes an extra number of steps or cannot maintain balance when pulled backwards.

Freezing or motor block refers to transient episodes of arrest in movement or inhibition in initiating certain movement. They are often unpredictable and can increase the risk of imbalance and falling. Frequently, freezing episodes occur when the patient attempts to start an action, such as walking, but feels the feet "sticking or glued" to the ground. This is known as "start hesitation." The episodes can also happen when the patient switches from one motor task to another, such as turning or getting up from a chair. In more advanced disease, freezing can occur even in the middle of a motion. Some patients are more symptomatic when they are stressed or anxious, such as being in a crowded place or performing a task under other's observation. Other freezing phenomena can arise during speech, handwriting, eye-lid opening, or teeth-

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Hypokinesia and Movement Challenges *(Cont'd from page 1)*

brushing.

Motor fluctuation is a disabling and distressing symptom associated with more advanced PD. It refers to a fluctuation of immobility (akinesia) and excessive movement (dyskinesia) that is often unpredictable and may be related to disease progression as well as treatment complications. Symptoms include sudden wearing-off, "on-off" yo-yoing, no "on" response, paradoxic kinesis, and beginning or end of dose dyskinesia. Patients can have multiple symptom events throughout the day. They are particularly difficult to manage and may no longer respond to pharmacologic treatments.

About 75% of patients with PD have speech impairment during some part of their disease. Symptoms consist of reduced volume, hypokinetic dysarthria, monotonous pitch and loudness, imprecise articulation, disordered speech rate, and stuttering. They limit verbal communication and cause frustration and embarrassment, thus often restricting a patient's social functioning. Speech deficits are usually not very responsive to medication treatments. Speech therapy with emphasis on prosody and speech loudness may be helpful, but more evidence is needed to demonstrate its efficacy.

Dysphagia can affect 40% of patients. It is often related to disease severity, but not always. Rigidity and incoordination of muscles of the mouth, tongue and pharynx may be responsible for the swallowing impairment. Symptoms are worse during the "off" state. Patients may choke while eating and are at risk for aspiration. Excessive drooling and retention of food and pills in the mouth are also common. Affected patients should be evaluated by a speech pathologist. Swallowing studies may help to define the nature of the dysphagia and the presence and absence of silent aspiration. Eating only during the "on" state and soft diets may improve the swallowing problem.

Amidst the hypokinetic presentations described above, patients with PD can have periodic involuntary hyperkinetic symptoms, such as *tremor and dyskinesia*. Tremor occurs typically at rest and subsides with action or during sleep. It involves the hands most commonly, but can be present in legs, tongue, lips and jaw also. It has a characteristic 3-5 Hz slow, rhythmic pill-rolling appearance. It is often asymmetric and variable. Some patients may describe a quivering or vibrating sensation inside the body. Tremor is worsened by emotional changes, such as stress and anxiety, and can be disabling and embarrassing. Dyskinesia is one of the most difficult to treat motor complications of PD. It can occur at the beginning and/or end of medication dose, or fluctuate unpredictably. Symptoms include chorea, dystonia, stereotypy, or ballism. It is also exacerbated by emotional instability. Severe dyskinesia causes pain, anxiety, irritability, exhaustion, and a much diminished quality of life.

Motor dysfunctions in PD are challenging for clinicians to treat. Fortunately, there are many management choices,

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Non-Motor Complications of PD

Non-motor complications occur frequently in PD, especially in more advanced disease. They include neuropsychiatric manifestations, cognitive impairment, sleep disturbances, autonomic dysfunctions, and sensory phenomena. Oftentimes, they are equally, if not more, distressing to patients and their caregivers, and are challenging to the clinician to treat. Therefore, a thorough understanding of these problems and their successful management will greatly improve patients' quality of life, lessen caregiver burden, and reduce healthcare expenses.

Neuropsychiatric manifestations. *Depression* affects about 40-50% of patients and is characterized by feelings of guilt, lack of esteem, helplessness, remorse, and sadness. Its causes may be endogenous due to monoamine deficiency, or exogenous due to the experience with a chronic, progressive neurodegenerative disease, or both. *Hallucination and delirium* may occur often and their incidences increase with age and the degree of cognitive impairment. They may be induced by psychotropic drugs, environmental stimuli, or medical illnesses. *Anxiety, panic attack, and agitation* disturb a major of patients, and they can occur in association with depression and delirium. Treatments include 1) reducing or discontinuing non-essential psychotropic medications, 2) treating concomitant medical illnesses, 3) encouraging physical activities, 4) patient and caregiver education, and 5) judicious use of antidepressants, anxiolytics, and atypical neuroleptics.

Cognitive impairment. Up to 40% of patients will eventually manifest overt dementia and they are at particular risk for drug-induced psychosis, more rapid progression of disability, and reduced survival. *Parkinson's disease dementia (PDD)* needs to be distinguished from concomitant Alzheimer's disease, vascular dementia, and dementia with Lewy bodies. PDD is typically characterized by executive dysfunction as well as impairments of visuospatial performance, temporal ordering, memory and attention, with a relative sparing of language and social behavior. At this time, there is no proven medication for the therapeutic intervention or prevention of PDD. Management should include 1) treating underlying medical problems, 2) discontinuing unnecessary or offending medications, 3) reducing antiparkinsonian medications, 4) patient and caregiver education, and 5) a trial of cholinesterase inhibitor.

Sleep disturbances. More than 75% of patients have some form of sleep abnormalities. They include *insomnia, REM behavior disorder, nightmare, sleep apnea, fragmented sleep, excessive daytime sleepiness, and sleep attack*. Management include 1) initiating sleep hygiene program, 2) treating depression and anxiety, 3) minimizing nocturia, 4) treating REM behavior disorder, 5) discontinuing offending medications, 5) using controlled-release levodopa and short-acting hypnotics, 6) sleep study, and 7) judicious use of stimulant for excessive daytime sleepiness.

Autonomic dysfunction. Autonomic disturbances in Parkinson's disease can manifest as *constipation, urinary problems, incontinence, orthostatic hypotension, sexual dysfunction,* impaired thermoregulation, sensory phenomena, and dysphagia.

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Assessments and Outcome Measures for Parkinson's Disease

by Karon Cook, PhD

Parkinson's disease (PD) imposes both motor (e.g., gait dysfunction, postural instability) and non-motor symptoms (e. g., depression, anxiety), and its pharmacological treatment is associated with life-impacting side-effects. PD outcomes can be evaluated using a range of assessment strategies including elemental assessments (e.g., magnetic resolution imaging of the brain following deep brain stimulation), functional evaluations (e.g., activities of daily living), and measurement of QOL variables (e.g., well-being). Outcomes data may be obtained by clinician or other expert rating, self-report, or proxy report. In this article, we examine 4 measures frequently used for assessing persons with PD: (1) the Unified Parkinson's Disease Rating Scale (UPDRS),[1] (2) the Hoehn and Yahr (HY) scale, (3) the Schwab and England (SE) Scale,[2] and (4) the 39-item Parkinson's Disease Quality of Life scale (PDQ-39).[3] We briefly describe, for each measure, the instrument's purpose, strengths, limitations, and, when they exist, available alternatives.

UPDRS

After its development in 1987,[1] the UPDRS became widely used as an outcome variable in trials evaluating new treatments for PD. The UPDRS is a rating tool designed for following the clinical course of PD over time. It consists of subscales that measure: (1) mentation, behavior, and mood (e. g., cognition, motivation), (2) activities of daily living (ADLs) (e.g., swallowing, dressing), and (3) motor skills (e.g., tremor, rigidity). Though the motor section of the UPDRS must be completed by a trained clinician, some investigators have patients complete the mentation, behavior, and mood scale and the ADL scale (17 items total), thus reducing the amount of clinical time dedicated to completing the UPDRS. Louis and colleagues[4] found moderate to excellent agreement (weighted kappa values = 0.63 to 1.0) between clinician and self-ratings on the 17 items of these 2 subscales. The authors of the scale[1] state that the UPDRS can be completed in 10-20 minutes. However, for their study population, Martinez-Martin[5] report an upper range of 40 minutes to complete the UPDRS.

There are portions of the UPDRS that are problematic. The ADL scale of the UPDRS has been critiqued as "conceptually unclear" because it contains not only items that measure ADLs, but also items that measure impairment (e.g., salivation, falling, and sensory complaints).[6] The items measuring depression, motivation/initiative, and tremor have been shown to poorly associate with other domains measured by the UPDRS.[5] Martinez-Martin and colleagues recommend that these aspects of PD (depression, motivation/initiation, and tremor) are better evaluated with scales developed specifically to measure them.

Multidimensional scales that are alternatives to the UPDRS include the New York University Parkinson's Disease Evaluation, the Short Parkinson's Evaluation Scale, and the University of California Los Angeles scale.[6] None of these alternatives, however, have been as widely employed nor as extensively evaluated as the UPDRS. Studies evaluating the UPDRS support its reliability and validity as an outcome measure.

HOEHN AND YAHR

Hoehn and Yahr[7] developed their scale after collecting and analyzing data on 802 patients with confirmed PD. The scale classifies PD into 6 stages: 0 = "no clinical signs evident," 1 = "unilateral involvement only," 2 = "bilateral involvement only," 3 = "first evidence of impaired postural and righting reflexes by examination or a history of poor balance, falls, etc; disability is mild to moderate," 4 = "fully developed severe disease; disability marked," and 5 = "confinement to bed or wheelchair." The psychometric and/or clinimetric properties of the HY scale have not been well assessed.[6] However, it is the staging instrument most often used to quantify the severity of PD.

SCHWAB AND ENGLAND SCALE

The Schwab and England scale is a disability scale commonly used in study populations with PD. In their review of clinical evaluation tools for PD, Ramaker, et al.[6] note that there have not been many studies to evaluate the characteristics of the Schwab and England scale. However, based on their review of existing studies, they concluded that there was evidence that the Schwab and England scale has "good reliability" and "substantial validity." Alternatives to the Schwab and England scale include the Northwestern University Disability Scale and the Intermediate Scale for Assessment of Parkinson's disease.[6] There is evidence for the validity and reliability of both of these, but neither has been used as extensively as the Schwab and England in study populations with PD.

PDQ-39

Quality of life (QOL) is a multidimensional construct that reflects the individual's subjective assessment of valued life domains. Marinus and colleagues[8] recently published a systematic review and quality assessment of existing PD-specific measures. Of the measures they examined, the Parkinson's Disease Questionnaire-39 (PDQ-39)[9] had undergone the most extensive psychometric evaluation. The PDQ-39 is comprised of 39 items and 8 subscales that measure quality of life dimensions related to PD (mobility, activities of daily living, emotional well-being, stigma, social support, cognitions, communication, and bodily discomfort). These quality of life domains were identified through in-depth interviews with patients who had PD. The content and construct validity of the PDQ-39 scores used as measures of therapeutic outcomes have been well established. High inter-item consistency estimates were found, not only for the subscale scores, but for the total score or the PDQ-39 "summary index" (PDQ39-SI) as well (0.84).[10] Peto and colleagues[11] conducted a higher-order factor analysis of the dimension scores and cross-validated their results in a second sample. A single factor accounted for over half the variance in the scores. These results suggest that the PDQ39-SI is an appropriate summary index of PD quality of life dimensions.

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Parkinson's Disease and Gait

by Elizabeth J. Protas, PT, PhD, FACSM

Although Parkinson's disease (PD) is classically associated with the symptoms of resting tremor, bradykinsesia, rigidity, and gait and postural abnormalities, these symptoms are not necessarily equivalent in each patient. In fact, some classifications of PD describe patients as either tremor predominant or postural instability gait difficulty predominant (PIGD).[1] The patient exhibiting PIGD is more often referred to physical therapy (PT) for treatment. An individual who is rated on the Hoehn & Yahr disability scale as 3 or higher on the 0-5 scale, has compromised postural righting reflexes, and is unable to recover balance on a pull test. At this stage, falls can become a recurring problem. Patients have difficulty walking sideways or backwards. Gait is described as slow and shuffling, with shortened, festinating steps (involuntary hurrying). Arm swinging and trunk rotation is decreased while walking, and individuals often have difficulty taking a step, also known as start hesitation. Episodes of decreased movement or freezing occur more frequently during walking. People with PD have difficulty turning since turns are a series of step initiations. Freezing can be triggered by doorways, leaving elevators, or narrow hallways.

Although limited research is available on the outcomes of PT treatment for patients with PD, some interventions are promising. A patient who has recurrent falls can benefit from an examination of the problems and from specific safety training. Rearranging the furniture to reduce the likelihood of tripping or walking sideways (for example, moving the coffee table away from the front of the couch) can also reduce falls as can gait and balance training that challenge the patient's stability.[2-4] Flexibility exercises to improve axial mobility have been demonstrated to be beneficial. Cueing strategies also have benefits for the patient. Visual cues such as brightly colored tape or pointer flashlights improve step and stride lengths. In contrast, auditory cues set at a pace 25% faster than the patient's usual gait speed improve walking speed.

Many patients and their families think of PT as a one-time event for the patient; however, the therapist needs to determine on the examination if there are any new concerns that would make an intervention by the physical therapist appropriate. If the patient and family notice significant changes in function or gait or if the patient begins to have injuries from falls, a referral and examination for PT is indicated. Substantial improvements in function, balance, and gait can occur.

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including medications, surgical procedures, and non-pharmacologic interventions, such as exercise, education and support. Care of the patient requires experienced clinicians, patient cooperation, and utilization of all available treatment options.

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Non-Motor Complications (Continued from page 2)

Treatment needs to be individualized and with an understanding of the patient's neurological, physical and emotional conditions.

Sensory phenomena. Patients often complain of sensory distresses, including *rigidity, cramps, dystonic pain, musculoskeletal aching associated with immobility, heaviness in limbs, and restless leg symptoms*. They occur more frequently in 'off' periods. Management strategies may consist of 1) ruling out and treating concomitant rheumatologic, orthopedic, radicular, and neuropathic problems, 2) stretching exercises, 3) physical therapy and increasing activity, 4) medication adjustment to prevent 'off' periods, 5) prescribing appropriate medications, including baclofen, NSAIDs, etc., 6) botulinum toxin injection.

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Physical Therapy and Occupational Therapy Treatment of PD: Models of Care

by Marilyn Trail, MOT, OTR, BCN and Trianna Warkentin, MS, PT, NCS

Though there is general agreement in the literature that physical therapy (PT) and occupational therapy (OT) are important adjunctive treatments for patients with Parkinson's disease (PD), there is also agreement that the efficacy of exercise and PT/OT treatment for PD patients has not been adequately studied. Better-designed research studies are needed. However, Carr and Shepherd,[1] Morris, [2] and Shenkman [3] have presented models of care that serve as comprehensive treatment guidelines. The following is a brief summary of the work of these four theorists and others.

Shenkman offers a model that differentiates between impairments that occur as direct effects of central nervous system (CNS) pathology (that may not be improved by PT/OT intervention), those that occur indirectly because of musculoskeletal alterations, and those that result from a composite of CNS and non-CNS impairment. Shenkman draws upon treatment techniques based upon Bobath (NDT), Feldenkrais, and Knott and Voss (PNF). Shenkman proposes that musculoskeletal impairments contribute to the PD patient's total disability and restricts the ability to perform activities of daily living. She emphasizes the importance of exercises for spinal flexibility, suggesting that tightness may contribute to poor balance and functional limitations. She implicates hip rotator and lumbar lateral flexor musculature, hip and knee flexor tightness as contributing to shortened step length. She places emphasis upon techniques to decrease rigidity, including relaxation.[4]

Carr and Shepherd and Morris base their models upon motor control or motor learning theory. They believe that slowness of movement and difficulty initiating muscle activation, rather than rigidity, are the major deficits underlying the poverty of motor performance. Musculoskeletal changes (forward head, flexed posture) also contribute to reduced speed and difficulty in initiation of gait. All three have suggested that rigidity may be in part an adaptation to hypokinesia and de-emphasize its importance on movement. They stress the need to teach the biomechanical features of an action and the use of cognitive strategies to help alter ineffective movement patterns.

Carr and Shepherd[1] site lack of evidence in the motor learning literature that passive or therapist controlled movement or active non-specific exercise will carry over to improve performance of specific tasks. Tasks should always be practiced in the environment where they take place.

Morris further bases her model of treatment on the pathogenesis of PD and an evaluation of the evidence for therapeutic intervention. Normal movement can be obtained by teaching patients to bypass the basal ganglion pathology and compensate with cortical control mechanisms. External cues (visual, auditory, and proprioceptive) can assist patients with cognitive deficits. Morris advocates task specific training regimes. Patients should be taught to do one thing at a time and to avoid dual activities such as talking or carrying a cup of water when walking. [5] Long movement sequences should be broken into steps. Patients can focus on learning and performing one step at a time. Exercise and activity training should be undertaken when patients are at their peak medication dose.

The environment can be structured to facilitate motor performance. For instance, chair heights can be raised and gradually lowered. Visual cues to normalize step length and to overcome freezing can be helpful as can auditory cuing such as moving to music, or counting. A recent study found that assistive devices based on visual cues are not consistently beneficial in overcoming freezing in most patients. [6]

All authors agree that PD patients benefit from activity and exercise programs and that therapies should begin early in the disease course to help preserve musculoskeletal flexibility (and thoracic expansion), prevent deconditioning, minimalize mental decline, and find solutions to functional problems. Exercise in the early stages of PD should involve walking, swimming, and general exercises for flexibility, breathing, strength, and cardiovascular fitness.

The authors have had good outcomes with motor neuron disease patients who have assumed the forward head, flexed posture, using general spinal mobilizations and stretching to the shoulder complex and anterior chest musculature, incorporated with a good seating system that provides lumbar support. We have also had success with passive positional stretching with a towel roll to the cervical and lumbar spine with the patient in supine.[7] Chin tucks and scapular reactions and depressions are also indicated for PD patients. Aggressive stretching of the plantar flexors in sitting and standing are beneficial for assisting with increased ease of sit to stand and improved balance reactions.

Resting tremor does not play a significant role during purposeful movement and does not appreciably interfere with function. It is responsive to medication. It might be beneficial for patients to repetitively practice fine motor activities such as buttoning and picking up small objects, but there is no direct evidence in the literature for this. PD patients commonly complain of fatigue, and energy conservation/motion economy techniques can help alleviate symptoms.

We have successfully used visualization techniques and task/activity analysis to assist PD patients perform functional activities such as showering, dressing, and meal preparation. Each phase of the task/movement can be analyzed and broken into steps and the biomechanics of each movement examined. Unnecessary steps and movements should be eliminated. Other steps and movements can be modified and the patient can practice the biomechanics of the movements involved. Morris suggests making stick drawings of each action and teaching preparatory movements before the endeavor takes place and avoiding long action sequences.

Gait and balance deficits place patients at risk for falls. A safety assessment can identify problem areas in the home or workplace. Patients benefit from grab bars, shower seats, and

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Depression in Persons with Parkinson's Disease

by Naomi Nelson, PhD

Although the methods of diagnosis and treatment of Parkinson's disease (PD) are well documented in the literature, much less is known about the psychosocial impact of the illness. The attempts to explore the psychosocial aspects of PD have been limited to pharmaceutical trials when examining quality of life and by conducting studies that use small samples of PD patients which may take liberty with randomization of subjects and the use of control groups.[1] Only a few studies specifically focus on psychosocial intervention strategies that have the potential of improving the quality of life and the development of coping skills so essential for those living with chronic illness.

Depression, the most common psychosocial concern in PD, occurs in about 50% of patients [2] and while the cause is still unknown, experts believe that the dopamine system plays a significant role. Both norepinephrine and serotonin, two neurotransmitters related to dopamine regulation, are critical for mood regulation but are deficient in PD. [3] This reduction in neurotransmitters may contribute to a major mood disorder that is characterized by decreased energy and motivation; feelings of sadness, helplessness, hopelessness; changes in weight, sleep and appetite; irritability; and thoughts of suicide. [4] These depressive symptoms may predate the development of motor symptoms and may fluctuate over time. The patient might alternate between a normal affect and a depressive state. The episodes occur more frequently occur when the patient is "off" the medications and they may sometimes improve when the motor symptoms are better controlled. Treating the patient with conventional antidepressants aimed at alleviating apathy or lack of sleep may be helpful for these signs of depression. [5]

Another form of depression, reactive depression, is less responsive to antidepressants.[4] Reactive depression may be experienced by those newly diagnosed with PD and others with more advanced disease who are losing independence and control because of changes in motor functioning and feelings of helplessness.[6] Some patients may be responding to a job loss with subsequent changes in income and loss of identity whereas other patients may be grieving the death of a family member, changes in living arrangements, or social isolation.

Depression in PD may also co-exist with subtle cognitive symptoms such as forgetfulness, attention problems, and problems in tracking the conversations of several individuals at one time.[2] It may be difficult for the allied health professional to sort out what is depression and what is cognitive decline; therefore, consultation with family members and the physician may be necessary. Increased emotional stress can also intensify depression, memory decline, and motor symptoms.[5] The emergence of increased stress enhances the critical need for a balance in all spheres of the patient's life – home, work, rehabilitation therapy, and social/volunteer activities.

Regardless of the form of depression, the experience of mood changes can profoundly affect those with PD and their families as well as the patient's progress in therapy. At times an allied health professional may be the first to notice depression in the person with PD, notably when progress in therapy has reached a plateau or progress declines because of changes in motor functioning. Therapists might also misinterpret the flat affect or masked facial expression of a PD patient to be symptoms of depression.

Allied health professionals may also discover that depression coincides with increased postural instability[7] and concerns about finances. The presence of both features begins to interfere with the prescribed treatment program. If the patient is worrying about the financial cost of therapy or whether therapy is helping with instability, the therapy may be stopped prematurely and the maximum benefit of improved mobility and balance is not attained. Sometimes the effectiveness of therapy may be compromised while the patient is receiving new medications or a change in dosage of medications. The therapist can, within the rubrics of professional judgment, also recommend or reinforce the need for nonpharmacological methods for treating depression. This may include, but is not limited to, exercise (walking, tai chi, yoga and water therapy), community education/support groups, and behavioral/ cognitive counseling for individuals or families. The team approach used by health care professionals for support, encouragement, and expert clinical care can be very therapeutic for the depressed person with PD.

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The Changing Communicative Needs of Patients with Parkinson's Disease

by Jean M. Whitehead, MA, CCC/SLP

As Parkinson's disease (PD) progresses, so do changes within the speech and vocal production system. Most patients eventually exhibit hypokinetic dysarthria with associated respiratory, laryngeal and articulatory dysfunction.[1] Hypokinetic dysarthria is characterized by reduced loudness or hypophonia, reduced pitch changes, reduced range of articulatory movements, and changes in rate such as short rushes of speech and at times repetitive stutter like dysfluencies.[2] These can impair communication and adversely affect quality of life. Concurrent dementia can reduce ability to compensate for the disordered speech production.[3]

Pharmacologic and neurosurgical interventions alone have had limited results in improving the disordered speech and vocal systems of PD patients.[4] The best clinical response in speech treatment is usually seen when a patient is receiving maximum benefit from the medication.[3] Speech treatment aims to strengthen muscles involved with volume production and articulation. Conscious retraining of timing for speech production and attention to clarity are included in some therapies. There are no large studies that examine issues of central neurotransmitter changes or anatomical-functional correlates of improved speech in PD.[5]

With the advancing communication disorder comes increasing frustration on the part of the patient as well as family members and caregivers. The patient finds himself excluded in conversational situations and social activities. They often find it easier to withdraw and to limit social contacts rather than struggle with the verbal exchange. Patients often stop using the telephone and are further cut off from others. This exclusion can happen when a patient comes for a medical appointment. Because of the time involved and the slow effortful communication, questions about health issues and conversation are frequently directed to the family member rather than the patient.

Patients with PD should be considered for possible use of augmentative communication devices. These may be very simple amplification systems if the primary problem is that of reduced loudness. However, since most PD patients have associated deficits of articulation, phonation, and rate, making their speech louder does not improve the situation and can actually complicate it.

Some patients may benefit from using nonelectronic communication boards or notebooks. They can be purchased or custom made. Communication boards often have an alphabet in large type for spelling out words. Though this appears to be an elementary form of communication, it can be very effective. Other boards include common pictures, words, or word phrases. Notebooks usually include similar items but expand on the number and type of entries and are divided into groups with a common theme.

Some patients can effectively use computerized equipment. These devices can produce an electronic display, hard copy printout, verbal output or combinations of these. The cost of computerized equipment varies, and devices costing thousands of dollars may not be financially accessible to many.

A speech/language pathologist should evaluate the patient's communication disorder and assess for augmentative communication equipment. Some families spend large sums to buy the latest "miracle" machine for a patient and then discover it is not appropriate when motor skills and communicative needs change. Equipment can be shared through a loaner program when it is no longer appropriate for a particular patient.

Consider he changing ability of the patient to access the equipment. Ease of access is critical for the patient to functionally use the device. Various switches and scanning devices should be compatible with the electronic equipment so patients can use simple movements to activate it. Caregivers should be trained with the device so they can assist .

Communicative needs of patients with PD change as the disease progresses. PD patients should be evaluated by speech/ language pathologists and assisted with these changing needs to help find the effective and efficient methods of communication.

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PT/OT Treatment *(Continued from page 5)*

raised toilet seats. Other items of equipment such as reachers, button hooks, computer arm supports, and selected writing devices can facilitate ADL and IADL.

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There are several viable alternatives to the PDQ-39. These include Parkinson's Disease Quality of Life (PDQL) questionnaire,[12] and the Parkinson's Impact Scale (PIMS).[13] Of these 2 alternatives, the PDQL is a better choice because there is more evidence demonstrating the reliability of this measure compared to the PIMS.[6]

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PT/OT Evaluation Tools

ROM (include cervical, axial)
Muscle Strength
Hand-grip Dynamometer
9-hole Peg Test or Purdue Pegboard
Functional Reach
Timed Get-Up and Go (TUG)
Pull Test
360˚ Turn
2, 6, or 10-minute walk
Visual analogue scale for fatigue
Supine to stand time
Timed ADL tasks

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