GERINOTES

SECTION ON GERIATRICS, AMERICAN PHYSICAL THERAPY ASSOCIATION

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EDITOR'S MESSAGE

Welcome to the *GeriNotes* CE Focus issue. The content in this issue is excellent due to the contribution of Miriam Boelen, Guest Editor and all the authors. I encourage members to take advantage of the opportunity to earn CEU in the process of learning about PD. On this page is a recruitment ad for *GeriNotes* Editor. After 7 years I have decided to pass on the baton. Being *GeriNotes* Editor is a great opportunity; I have enjoyed it immensely and take pride in the growth of the publication. Please consider applying; contact me if you have questions.

Carol Schunk, PT, PsyD

PRESIDENT'S PERSPECTIVE: MULTIDISIPLINARY COMPETENCIES NOW READY FOR USE

John O. Barr, PT, PhD

In June 2008, the American Geriatrics Society convened a meeting of 21 organizations that represent health care professionals who care for older adults in order to address recommendations from the 2008 Institute of Medicine Report, *Retooling for an Aging America: Building the Health Care Workforce*,[1] the subject previous Perspectives[2,3] and the focus of our own Retooling Taskforce. This meeting led to the development of a loose coalition--the Partnership for Health in Aging (PHA)--that identified as its first step the development of a set of core competencies in the care of older adults that are relevant to and can be endorsed by all health professional disciplines.

A PHA workgroup of health care professionals with experience in competency development, certification, and accreditation was convened in February 2009. Workgroup members represented 10 health care disciplines: Dentistry, Medicine, Nursing, Nutrition, Occupational Therapy, Pharmacy, Physical Therapy, Physician Assistants, Psychology, and Social Work. The workgroup began with a comprehensive matrix of competencies across these 10 disciplines. Through an iterative process, the workgroup drafted a set of baseline competencies that were ultimately endorsed by 28 professional organizations, including the American Physical Therapy Association (APTA). I had the privilege of serving as a member of the workgroup involved in this exciting interdisciplinary process, while Section members Rita Wong, PT, EdD, and Dale Avers, PT, DPT, PhD, served as APTA reviewers. The workgroup reviewed all comments received, and developed the final set of competencies, listed below. This set describes essential skills in key domains that health care professionals in the above 10 disciplines should have, and necessary approaches they should master, by the time they complete their entry-level degree, in order to provide quality care for older adults, as follows:

DOMAIN #1: Health Promotion and Safety

1. Advocate to older adults and their caregivers interventions and behaviors that promote physical and mental health, nutrition, function, safety, social interactions, independence, and quality of life.
2. Identify and inform older adults and their caregivers about evidence-based approaches to screening, immunizations, health promotion, and disease prevention.
3. Assess specific risks and barriers to older adult safety, including falls, elder mistreatment, and other risks in community, home, and care environments.
4. Recognize the principles and practices of safe, appropriate, and effective medication use in older adults.
5. Apply knowledge of the indications and contraindications for, risks of, and alternatives to the use of physical and pharmacological restraints with older adults.

DOMAIN #2: Evaluation and Assessment

1. Define the purpose and components of an interdisciplinary, comprehensive geriatric assessment and the roles individual disciplines play in conducting and interpreting a comprehensive geriatric assessment.
2. Apply knowledge of the biological, physical, cognitive, psychological, and social changes commonly associated with aging.
3. Choose, administer, and interpret a validated and reliable tool/instrument appropriate for use with a given older adult to assess: (a) cognition, (b) mood, (c) physical function, (d) nutrition, and (e) pain.
4. Demonstrate knowledge of the signs and symptoms of delirium and whom to notify if an older adult exhibits these signs and symptoms.
5. Develop verbal and nonverbal communication strategies to overcome potential sensory, language, and cognitive limitations in older adults.

DOMAIN #3: Care Planning and Coordination Across the Care Spectrum (Including End-of-Life Care)

1. Develop treatment plans based on best evidence and on person-centered and directed care goals.
2. Evaluate clinical situations where standard treatment recommendations, based on best evidence, should be modified with regard to older adults' preferences and treatment/care goals, life expectancy, co-morbid conditions, and/or functional status.
3. Develop advanced care plans based on older adults' preferences and treatment/care goals, and their physical, psychological, social, and spiritual needs.
4. Recognize the need for continuity of treatment and communication across the spectrum of services and during transitions between care settings, utilizing information technology where appropriate and available.

DOMAIN #4: Interdisciplinary and Team Care

1. Distinguish among, refer to, and/or consult with any of the multiple health care professionals who work with older adults, to achieve positive outcomes.
2. Communicate and collaborate with older adults, their caregivers, health care professionals, and direct-care workers to incorporate discipline-specific information into overall team care planning and implementation.

DOMAIN #5: Caregiver Support

1. Assess caregiver knowledge and expectations of the impact of advanced age and disease on health needs, risks, and the unique manifestations and treatment of health conditions.
2. Assist caregivers to identify, access, and utilize specialized products, professional services, and support groups that can assist with care-giving responsibilities and reduce caregiver burden.
3. Know how to access and explain the availability and effectiveness of resources for older adults and caregivers that help them meet personal goals, maximize function, maintain independence, and live in their preferred and/or least restrictive environment.
4. Evaluate the continued appropriateness of care plans and services based on older adults' and caregivers' changes in age, health status, and function; assist caregivers in altering plans and actions as needed.

(continued on page 33)

CONTINUING EDUCATION MODULE

PARKINSON'S DISEASE: CONSIDERATIONS WITH AGING POPULATIONS

A Section on Geriatrics Continuing Education Module

OVERVIEW

Therapists who work with people with Parkinson's disease (PD) (the majority being 65 years old and older) must be versed not only in PD signs and symptoms but also in responses to medications. An awareness of the heterogeneity of the Parkinson's population is necessary for individualization of interventions. Increasing your knowledge in this subject matter will improve patient intervention and examination, a prime goal of all physical therapists and assistants.

MODULE CHAPTERS

I. *Guide to Physical Therapist Practice*: Preferred Practice Pattern 5 Neuromuscular by Heitzman and Staples
II. Using Motor Learning Concepts to Guide Interventions in Individuals with PD by Osborne
III. Gait and PD by Boelen
IV. Postural Control in Adults with Idiopathic PD by Smith and Batson
V. Deep Brain Stimulation by Mai
VI. Pharmacologic Management in PD by Kegelmeyer and Park
VII. Nutrition and PD by Bottomley
VIII. Effect of Group Education and Individual Rehabilitation on Patients with Idiopathic PD by Vollman
IX. Amplification of Fall Risk in PD: The Influence of Comorbidities by Foreman, Ballard and Dibble

AUTHORS

See specific biographies following each chapter

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Mary Vollman, PT

REFERENCE LIST

References can be found at the end of each chapter in the module.

OBJECTIVES

The reader will be able to:

1. Explain how each of the components of patient management apply to the patient with Parkinson's disease.
2. Explain the learning capabilities of an individual with Parkinson disease and apply motor learning concepts to intervention development for this population.
3. Increase understanding of various gait abnormalities observed in people with Parkinson's disease and offer insight to a variety of interventions for increased efficacy of patient management.
4. State the impact of postural control deficits on balance in adults with idiopathic Parkinson's disease.
5. Describe deep brain stimulation (DBS) including the procedure, candidates for DBS, advantages and disadvantages of the procedure, contraindications for DBS, precautions that need to be followed, and implications for physical therapy.
6. Understand the mechanisms of action and side effect profiles of the dopaminergic and nondopaminergic medications used in the treatment of PD, and to relate this to the physical therapy management of patients with PD.
7. Describe the role that nutrition plays in the prevention, development, evolution, and treatment of PD.
8. Discuss the effectiveness of an intervention combining Group Education and Individual Rehabilitation for people with idiopathic PD based upon the Guo et al study.
9. Increased awareness of comorbidities that may amplify fall risk in persons with PD.

TARGET AUDIENCE

Physical Therapists and Physical Therapist Assistants

CONTACT HOURS/CONTINUING EDUCATION UNITS: 4 CONTACT HOURS OR .4 CEUS

CONTINUING EDUCATION CERTIFICATE OF COMPLETION

A Continuing Education certificate will be provided to each participant after successful completion of the course requirements and payment of a processing fee. The Section on Geriatrics is a recognized component of the American Physical Therapy Association. The Section on Geriatrics has not applied to any state licensure agency for prior approval of this course. The module has all the components (content, objectives, qualified instructors, reference lists, and post test) that will allow participants to submit the certificate of completion to meet CE requirements in most chapters.

HOW TO SUBMIT CEUs

To obtain CEUs for this continuing education unit, participants must complete the post test as well as the evaluation form on pages 5 & 6. A processing fee of $40.00 for SOG members and $80.00 for nonmembers is required. To apply for CEUs send the post test and the evaluation form to the Section on Geriatrics along with payment. Applications must be postmarked no later than December 31, 2010. Upon submission of materials and a passing score of 80% or higher on the post test the Section will mail you a continuing education certificate for .4 CEUs. Those with incomplete submissions will be notified via e-mail and given the opportunity to re-take the exam.

PARKINSON'S DISEASE
CONTINUING EDUCATION UNIT POST TEST

Instructions: To obtain CEUs for this continuing education unit, participants must complete the post test as well as the evaluation form on the back of this page. See specific instructions for submission of the completed post test on next page. Please circle the correct answer for each question.

1. Patients with Parkinson's disease have impairments that cross multiple systems but should be placed in the primary practice pattern under which system?
   a. Musculoskeletal
   b. Neuromuscular
   c. Cardiovascular/pulmonary
   d. Integumentary

2. Which of the following is true regarding implicit or procedural learning?
   a. Includes the rehearsal of verbal cues
   b. Includes tasks that accumulate with repetition
   c. Involves remembering certain events
   d. Involves conscious recall

3. Which one of the following is the primary cause of slower gait velocities in people with PD?
   a. Reduction in stride length
   b. Reduced cadence
   c. Freezing
   d. Dysrhythmical gait

4. Successful intervention for increasing postural control in Parkinson's disease typically focus on:
   a. Aquatic therapy
   b. Conscious movement patterning
   c. Static movement to make gains in postural control
   d. Single system strategies

5. Ideal candidates for deep brain stimulation should:
   a. Be over 75 years old
   b. Demonstrate < 10% improvement on the UPDRS
   c. Be diagnosed with Parkinson's Plus Syndromes
   d. Respond positively to Levodopa

6. All of the medications used to manage Parkinson's disease can lead to which of the following serious side effects in the elderly?
   a. Confusion
   b. Cardiac arrhythmia
   c. Hypertension
   d. Sleep attacks

7. Dopaminergic dysregulation syndrome involves which of the following?
   a. Akinesia
   b. Compulsive behaviors
   c. Confusion
   d. Orthostatic hypotension

8. To maximize the function of persons with Parkinson's disease, Sinemet and Levodopa should be taken:
   a. With high protein foods
   b. On a full stomach
   c. Just before bedtime
   d. 30 minutes before a meal

9. What was the educational format for the intervention group described in the Guo et al trial?
   a. Internet information pertaining to medications, exercise and research
   b. Three interactive group education sessions with supplemental Internet information pertaining to the patient preferred topics of meals, mood, and moving
   c. One group lecture pertaining to home and community exercise programs
   d. Formal lectures pertaining to medications, latest advances in treatment, and mood

10. Which of the following co-morbidities is an independent risk factor for falls among people with Parkinson's disease?
    a. Psoriasis
    b. Bells Palsy
    c. Urge urinary incontinence
    d. Upper extremities osteoarthritis

CONTINUING EDUCATION MODULE

PARKINSON'S DISEASE PHYSICAL THERAPY CE UNIT EVALUATION FORM

Please rate the following questions		1= strongly disagree			5= strongly agree	
1	The course material met the stated objectives	1	2	3	4	5
2	The information will be useful in my practice	1	2	3	4	5
3	The articles were well written and informative	1	2	3	4	5
4	The authors were knowledgeable for this topic	1	2	3	4	5
5	I am satisfied with this unit as a CE course	1	2	3	4	5
6	I would like future CE courses in GeriNotes	1	2	3	4	5

Please offer any additional comments, suggestions, or topics for focused issues below: _____

_____

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Submission for Continuing Education Credits

To obtain CEUs for these continuing education participants must complete the post test as well as the evaluation form on this page. Return page 5 and 6 with a processing fee of $40.00 for SOG members and $80.00 for nonmembers. Submission must be postmarked no later than December 31, 2010. Upon submission of materials and a passing score of 80% or higher the Section will mail you a CEU certificate for .4 units. Those submitting incomplete material will be contacted via e-mail.

Name _____ APTA Number _____

Address _____

City _____ State _____ Zip _____

SOG member _____ yes _____ no

Professional designation ☐ PT ☐ PTA

E-Mail Address _____

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1111North Fairfax
Alexandria, VA 22314

GUEST EDITORIAL

Miriam Boelen, PT

It is a privilege to participate as the Guest Editor of the Parkinson's disease (PD) focus issue of *GeriNotes.* It has been well established that PD is a progressive disorder. The details of its progression, signs, symptoms, and treatment interventions, however, are not well known for those clinicians who do not specialize in PD. The articles in this issue are intended to offer those details with the purpose of improving understanding and efficacy in managing this population. Generally speaking, the *rate* of progression seen in people with PD (PwP) can vary greatly from one individual to another and is directly related to the *rate* of cell loss in the substantia nigra compacta that is responsible for dopamine production. Due to this variance in rate of progression, you may see patients who remain fully independent after having PD for 20 years or you may see someone with significant mobility restrictions after 6 years. Typically people report how many years they have had PD based on their date of diagnosis, which is normally a very memorable event. The actual onset of PD is more difficult to determine since PwP don't become symptomatic until approximately 80% of the dopamine producing cells are lost. The *pattern* of progression was published in the 1960s by Dr Hoehn and Dr Yahr. You can see from this staging system that impairment of postural reflexes in PD occurs later in disease progression. Early symptoms of postural instability, orthostatic hypotension, or dementia are indicative of Parkinson Plus disorders and not PD.

Stage 1 = Unilateral disease
Stage 2 = Bilateral disease, balance intact
Stage 3 = Mild to moderate bilateral disease, some postural instability, physically independent
Stage 4 = Severe disability; still able to walk or stand unassisted
Stage 5 = Wheelchair bound or bedridden unless aided

Hoehn and Yahr Staging of Parkinson's Progression

The Hoehn & Yahr staging system was established prior to the advent of the medication, Levodopa that replaces the deficient dopamine in the brain. Prior to Levodopa, the symptoms of Stages 2 and 3 were more predictable and definitive. Postural reflexes were either present, impaired, or absent. Currently, PwP who take Levodopa can actually move between Stages 2 and 3 or Stages 3 and 4 depending on their responses to this medication. Physical therapists should be aware of such changes in motor responses to medications so that interventions address the motor variations experienced by an individual. Although there may be commonalities regarding treatment interventions based on the Hoehn & Yahr staging system, the heterogeneity of this population dictates an individualized approach. As you sift through the articles in this issue, you will find this to hold true. In addition to an individualized approach, a comprehensive approach is also necessary when working with the PD population. Optimal mobility is typically achieved with a combination of interventions due to the limitations of each intervention in isolation. These interventions include: exercise, patient education, compensatory strategy training, medicinal adjustments, and/or a surgical procedure--deep brain stimulation (DBS).

This PD issue of *GeriNotes* is intended to offer a condensed cross section of the examination, evaluation, and interventions typically used for PwP. Examples of how co-morbidities can exacerbate balance problems in PwP is discussed. Gait deviations commonly seen in PwP and interventions and motor learning concepts to enhance efficacy of interventions are reviewed. Postural instability is the hallmark for Hoehn & Yahr Stage 3. It is necessary for therapists to understand factors contributing to postural instability and treatment strategies. The topic of exercise permeates each article only underscoring its importance. Medications have benefits but also limitations and side effects. Knowledge in these areas, in particular to motor fluctuations, can direct us in our interventions. Unwanted side effects should be discussed with the physician. Anyone who treats a person with PD who has undergone a DBS procedure, needs to be aware of precautions and contraindications to avoid harm to the patient. An overview from who is a candidate for this procedure to postoperative management is reviewed. Additionally, nutritional implications regarding etiological factors in PD, prevention, and treatment is discussed.

People with PD can benefit from therapy at any and all stages of Hoehn & Yahr. It is not unusual to see a person with PD who has never received therapy for her PD until they are in the later stages and falling. Hopefully with this newly gained knowledge we can advocate therapy for the person with PD who walks through our door with a frozen shoulder and never had therapy for her PD and feel more confident in treating the PD motor symptoms.

REFERENCE

1. Hoehn MM, Yahr MD. Parkinsonism: Onset, progression and mortality. *Neurology.* 1967;17(5):427-442.

Miriam Boelen is a physical therapist who has specialized in PD for 20 years. She is the author of a recently published book for physical therapists, *Health Professionals' Guide to Physical Management of Parkinson's Disease*. She is an ACSM certified Health Fitness Specialist.

EDITOR'S MESSAGE

I want to thank Guest Editor, Miriam Boelen; she brought her expertise on Parkinson's Disease to the issue and was a great help in the development and editing of the articles. I also want to thank Editorial Board members, Sandy Levi for editing the test questions and Ken Miller for doing the pre-read.

Carol Schunk PT PsyD,
GeriNotes Editor

USING THE *GUIDE TO PHYSICAL THERAPIST PRACTICE* FOR PATIENTS WITH PARKINSON'S DISEASE

Jill Heitzman, PT, DPT, GCS, CWS, CEEAA, FACCWS
William Staples, PT, PhD, DPT, GCS, CEEAA

THE GUIDE

The *Guide to Physical Therapist Practice* (*Guide*) has classified the Neuromuscular Practice Pattern chapter 5 into 9 subcategories; 5A-I. Each of these categories is based on the type of neuromuscular condition, age of acquisition, and progressive nonprogressive nature of the dysfunction.[1] While each neurological condition may have associated cardiovascular/pulmonary, musculoskeletal, and/or integumentary conditions, the primary condition is neurological. The first 3 of the 5 components of patient management (examination, evaluation, diagnosis) will assist the therapist in determining the proper preferred practice pattern placement as well as if any secondary preferred practice pattern(s) may be necessary. After determining the most appropriate preferred practice pattern, the therapist will be able to use the practice pattern(s) to continue with the last two components of patient management components (prognosis and intervention).

Parkinson's disease (PD) occurs when substantia nigra cells in the basal ganglia area of the midbrain degenerate at an accelerated rate, resulting in a chemical deficit of dopamine.[2] The resultant effect on the extrapyramidal system is clinically characterized by abnormalities in the: patient's posture, skeletal muscle tone, and mobility. These abnormalities could lead the clinician to begin thinking about a musculoskeletal preferred practice pattern. The decrease in the mobility of the patient with PD can result in endurance loss and subsequent effect on the cardiovascular/pulmonary system that could lead the clinician to the cardiovascular/pulmonary preferred practice patterns. The resultant pressure on the skin due to immobility could also result in the clinician considering an integumentary preferred practice pattern for risk reduction. While all these multiple body systems are affected by PD, the primary preferred practice pattern should be the Neurological Preferred Practice Pattern 5E: Impaired Motor Function and Sensory Integrity Associated with Progressive Disorders of the Central Nervous System.[1]

PRACTICE PATTERN 5E

The Preferred Practice Pattern 5E includes patients/clients with functional limitations associated with the motor and sensory impairments as a result of CNS disorders and includes any combination of the following:

- Exacerbation or remission of symptoms with treatment
- Impaired affect
- Impaired autonomic function
- Impaired cognition
- Impaired endurance
- Impaired expressive or receptive communication
- Impaired motor function
- Impaired sensory function
- Progressive loss of function[1]

Each of these impairments may be associated with the other systems (cardiovascular/pulmonary, musculoskeletal, and integumentary) that provides support that the proper primary placement for a patient with PD is this preferred practice pattern, Neuromuscular 5E. Parkinson's disease is listed in the *Guide* under this pattern as ICD-9-CM code 332.[1]

Each individual with PD has a unique course of symptoms and progression. Not all of the common symptoms of PD are present in all individuals. Therefore, a complete picture of the individual can only be obtained by following the 5 components of patient management as set forth in the *Guide to Physical Therapist Practice*.

EXAMINATION/HISTORY

The first component of the Physical Therapist Examination is the history. This is an important component in patient management for the patient with PD. The demographics of the patient include not only his age and primary language/culture, but his social life, living arrangement, and employment. This information will assist in planning the intervention program in relation to home programs and ultimate discharge needs. Adaptations to work and the home environment may need to be considered. The general health of this patient is important in relation to psychological, physical, and social function as well as any co-morbidities/health habits/pre-existing conditions of the other systems. Early education on changes may need to be implemented. The patient's history of PD, including any previous interventions, hospitalizations, and medications, will enable the clinician to determine the progression of the disease and past successes/failures. Finally, the current complaint/condition and functional status/activity needs to be explored to allow a determination of specific tests and measures needed.

EXAMINATION/SYSTEMS REVIEW

The next component of the examination is the systems review. By screening the systems of cardiovascular/pulmonary, integumentary, musculoskeletal, neuromuscular, cognition/communication, and learning styles, the clinician will be able to determine which specific tests and measures are needed to examine the patient and assist in determination of therapy needs/outcomes. This is an important step in the examination process and one that is often overlooked. Screening is especially important when considering direct access. Physical therapists must be able to recognize a possible systemic condition that is masquerading as a neuromuscular disease. The screening process is also important to uncover any *red flags* that might require medical referral or limitations for certain exercise programs.

EXAMINATION/TESTS AND MEASURES

The final step to the examination is the actual tests and measures. There are many tests and measures available to the clinician for consideration. The aerobic capacity will need to be determined with

an appropriate test that examines the vital signs and distance, and could include the 6 or 2 minute walk test or the 2 minute step test. Many functional outcome tests are available for evaluating balance, gait, and mobility and the clinician should choose ones that have been researched for specificity and sensitivity keeping in mind the history and systems review for the particular patient. These tests could include (but are not limited to) the Berg Balance Scale, Gait Speed, Timed Up and Go, and the Dynamic Gait Index. Muscle function and performance needs to be examined and should include dynamic strength testing such as the chair rise test, muscle tone determination for rigidity, or kinesthetic movements and alternating movements such as toe tap test. Posture should be measured in both sitting and standing to enable the clinician to determine if the trunk is amendable to change. Measurements including occiput to wall or rib/pelvis distance can be easily used in a clinic. Range of motion needs to be examined using goniometry or functional movements such as the back scratch test and the chair sit-and-reach. Pain can have an effect on the outcome of therapy interventions and must not be overlooked. Finally, any concerns in the home environment, work environment, and self care issues may need further examination or referrals to other professionals.

PROGNOSIS AND PLAN OF CARE

Once the examination is complete, the therapist can evaluate the results and determine the impairments and diagnosis that will lead to the prognosis and plan of care. Patients that have been placed in the Preferred Practice Pattern 5E have progressive conditions and may have advancing (with possibly increasingly severe) impairments, functional limitations, and disabilities. Therefore, the prognosis should take this into account and look to develop "optimal function within a variety of settings within the context of the impairments, functional limitations and disabilities."[2]

The *Guide to Physical Therapist Practice* can assist the clinician in developing the interventions based on the level of dysfunction currently present in patients with progressive diseases. By employing each phase of patient management, the clinician can identify the needs of the patient in relation to her caregiver, environment, and stage of disease progression. Using the *Guide*, the clinician can have supportive documentation to identify patient diagnostic codes, recommended number of visits, anticipated progress, and documented physical function that can support the need for skilled therapy services and interventions. Working with the patient and family on understanding the role of physical therapy for this progressive disease can promote a positive patient/provider relationship that is necessary due to the anticipated multiple episodes of care over the lifetime of the patient with Parkinson's disease.

REFERENCES

1. American Physical Therapy Association. *Guide to Physical Therapist Practice*. 2nd ed. Alexandria, VA: APTA; 2001.
2. Wichmann R, Hulme JB. Impaired motor function and sensory integrity associated with progressive disorders of the central nervous system (Pattern E). In: *Neuromuscular Essentials: Applying the Preferred Physical Therapist Practice Patterns*. Thorofare, NJ: Slack Incorporated; 2008.

Jill Heitzman is board certified by ABPTS in Geriatrics with recertification in 2008, and certified as a wound specialist by the American Academy of Wound Management, with recertification in 2007. She is Clinical Coordinator of Education/Therapy Services and Outpatient Program Developer for Rehabworks/East Alabama Medical Center. She also consults to companies developing programs and clinical residencies, teaches continuing education programs, writes articles and home studies on aging topics, and teaches online classes for the College of St. Scholastica. Dr Heitzman has served as the Program Chair for the SOG since 2003 and is on the faculty for the Section's CEEAA courses.

Bill Staples is an assistant professor at the Krannert School of Physical Therapy at the University of Indianapolis. He has recently completed his Doctor of Health Science (DHS) degree. He received his specialist certification in 1995. He has served on the Board of Directors as well as the Treasurer of the Section on Geriatrics. Bill has lectured nationally on a variety of geriatric PT issues. Bill maintains his clinical skills by working part-time in home health care.

CERTIFIED EXERCISE EXPERT FOR AGING ADULTS

Physical therapists with the CEEAA credential will demonstrate expert clinical decision-making skills in (1) designing and applying an effective examination and exercise prescription and (2) measuring the effectiveness and reflecting the current evidence of exercise for all aging adults. The 3 courses are designed to build on each other; however, Courses 1 and 2 can be taken out of sequence.

UPCOMING CEEAA COURSES & LOCATIONS:

Central:
Texas State University--San Marcos
Department of Physical Therapy
601 University Drive
San Marcos, TX 78666

Course 1: January 15-16, 2011
Course 2: March 12-13, 2011
Course 3: May 28-29, 2011

Mid West:
Des Moines University Physical Therapy Department
3200 Grand Avenue
Des Moines, IA 50312

Course 1: October 23-24, 2010
Course 2: March 19-20, 2011
Course 3: May 14-15, 2011

East Coast:
Nova Southeastern University
3200 S. University Drive
Ft. Lauderdale, FL 33328-2018

Course 1: April 30-May 1, 2011
Course 2: September 17-18, 2011
Course 3: November 12-13, 2011

Haywood Regional Medical Center
(Greater Asheville Area)
262 LeRoy George Dr.
Clyde, NC 28721

Course 3: September 18-19, 2010

Contact Latasha Magness at latschamagness@apta.org to register for any of these courses!

USING MOTOR LEARNING CONCEPTS TO GUIDE INTERVENTIONS IN INDIVIDUALS WITH PARKINSON'S DISEASE

Jacqueline Osborne, PT, DPT, GCS

Cognition is the ability to think. Impairment in the ability to think is impairment in the ability to learn. Learning is the acquisition of knowledge about a task with practice or exposure.[1] Therefore, *motor* learning is the acquisition of knowledge about a movement. As many as 55% of individuals with Parkinson's disease (PD) have cognitive deficits that impair movement and therefore function.[2] Among the most prominent deficits are those in executive functions such as planning and problem-solving, those requiring visual–spatial abilities, and those involved with free recall and learning.[2] Sommer et al showed that learning deficits exist in individuals with PD without *measurable* cognitive impairment.[3] Therefore, an individual with PD does not have to be diagnosed with subcortical dementia to display movement problems and memory deficits.

As physical therapists it is our obligation to consider learning concepts in each patient we treat regardless of the presence of overt clinical signs of cognitive impairment. Physical therapists are skilled at using their knowledge of aging, biomechanics, anatomy, physiology, and neuroscience to optimize a patient's function, and inherently have a working knowledge of the pathophysiology behind a disease process. However, it may not be as intuitive for physical therapists to apply their expertise to a task being taught and to instructions used to implement teaching. Thus, the purpose of this article is to educate physical therapists to identify the learning capabilities of individuals with PD and apply motor learning principles to intervention development.

NEUROLOGY OVERVIEW

Briefly, the ability to produce a movement is grossly dependent upon the pyramidal system and the extrapyramidal system in the brain. The pyramidal system extends from the cortex to the spinal cord via the brainstem. The extrapyramidal system includes nuclei in the basal ganglia and the cerebellum. Axons project from these nuclei to communicate with the brainstem and spinal cord, and to the cortex via the thalamus (Figure 1).

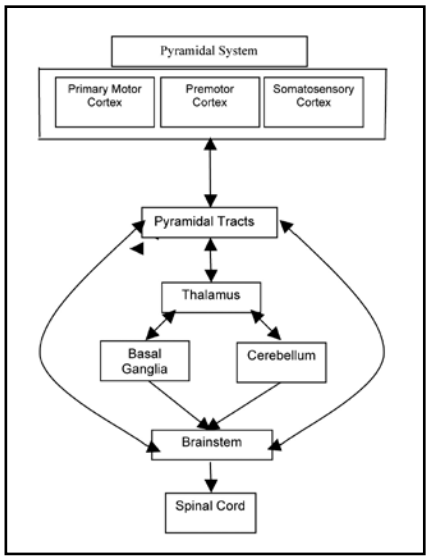

Figure 1.

The basal ganglia nuclei include the caudate, putamen, globus pallidus, substantia nigra, and subthalamic nucleus. They are highly integrated and are responsible for regulating the planning and execution of a movement. The cerebellum adjusts a movement generated by the basal ganglia making it precise and accurate. Dopamine, a neurotransmitter found largely in the substantia nigra, has both inhibitory and excitatory effects on the motor pathways in the basal ganglia. Therefore, dopamine loss results in depression of an excitatory component of movement and stimulation of an inhibitory component of movement. Since normal movement depends on the appropriate dopamine levels in the brain, we can deduce that dopamine loss contributes to a complex presentation of motor signs and symptoms in an individual with PD. For example, a person may demonstrate a hyperkinetic activity such as a festinating gait that is a progressively increasing gait speed and a simultaneous diminishing step length. The same individual may also exhibit hypokinetic activity such as bradykinesia and freezing episodes.

LEARNING IN PD

Individuals with PD have difficulty performing tasks that require automatic and often reflexive adjustments to movement as in a learned skill or habit such as walking or writing. These types of tasks accumulate with repetition and are not dependent on conscious processes.[1] They rely on an individual's implicit knowledge of the task. Therefore, the ability to learn a task that can be performed without attention or conscious thought is called procedural or implicit learning. It has been documented that the cerebellum,[1] basal ganglia,[4] and motor cortex[1] are crucially involved in learning a procedural task and that the procedural learning mechanism in an individual with PD is impaired.[3] It has also been shown that the ability to use explicit information that requires awareness and attention, and can be consciously recalled such as facts and events, is not as affected in individuals with PD.[3] This is called declarative or explicit learning. Reciting your address or multiplying two numbers together are examples of explicit tasks. The medial temporal lobe and the prefrontal cortex are among the brain regions necessary to learn or use explicit or declarative information.[1] Despite the anatomic separation of the brain regions that direct procedural and declarative learning processes, significant integration exists between these two systems.[1] The value of explicit or factual information to an individual with PD depends on the task being performed and how this knowledge is gained.[1]

As physical therapists we often expect our patients to implement verbal instructions we give regarding how to perform a task or adjust a movement. However, in the individual with PD we must understand that explicit instruction based in facts such as "pick up your leg and step over the box" or "walk in a heel-toe pattern" often interferes with the individual's ability to initiate and execute the desired movement. Thus, we cannot assume that giving explicit information is beneficial in this population for learning an implicit task such as walking. Re-

call that giving explicit instructions will likely interfere with the ability for an individual with PD to implement these instructions. Therefore, consideration of *how* explicit instruction is relayed to an individual with PD becomes important in directing intervention.

FEEDBACK

Careful application of feedback during an intervention can help a physical therapist maximize the potential for the individual with PD to relearn an implicit task such as one that requires sequencing a movement or implementing a movement pattern. Consider that an individual with PD may have to "study" to be able to perform a movement that prior to dopamine loss was automatic. This is different than robotically performing a task repeatedly. Rehabilitating an implicit task like walking may require the strategic use of intrinsic and extrinsic feedback rather than repetition of a task without focus toward a particular detail about the task.

Intrinsic feedback, signals internal to the individual, include attentional cues, visual cues, and mental practice. Extrinsic feedback, signals external to the individual, include verbal cues and tactile cues (Table 1).

Table 1.

INTRINSIC FEEDBACK	EXTRINSIC FEEDBACK
Attentional Cues	Verbal Cues
Visual Cues	Tactile Cues
Mental Practice	

For the purposes of the following discussion, feedback is applied to interventions designed to improve an individual's ability to initiate a movement. The same exercise or activity can be performed repeatedly with emphasis on distinctive aspects of the task using different types of intrinsic or extrinsic feedback.

Attentional Cues

An attentional cue is a form of intrinsic feedback useful for implementing movement initiation. If the task is to perform heel taps on a step, the attentional cue might be to clear the toe over the top of a 4 to 5 inch step, and to maintain all of the body weight on the stance limb through the entire repetition. The individual is verbally instructed to tap the heel of the foot onto the surface of the step, to immediately remove it after the tap, and place it on the floor. This is done before asking the individual to attempt the task. Demonstration may help to relay the task to the individual before the actual attempt. The important component, however, lies in the consequence of the individual's actions. If the individual does not clear the step, he is asked to stop, regroup, and voice which aspect of the movement is of interest. After the individual verbalizes understanding of the error, the task is repeated. It is feasible that an entire treatment session be spent on one specific task such as the one described above. At the next treatment session, the task is matched as specifically as possible to the individual's functional limitations. Not only will the individual have worked on a task that improves a specific function, but will also have addressed spatial awareness, posterior weight acceptance, single limb stance, and hip strengthening. It is important to note that heel taps rather than toe taps are emphasized in this population to encourage a posterior weight shift and counteract an inherently anterior center of mass.

Visual Cues

A visual cue such as colored tape placed on the floor or a laser pointer directed at the floor in front of an individual can provide the visual signal necessary to *unlock* a person's ability to initiate a step. To perform heel taps on a step, a piece of colored tape is placed on the run of a 4 to 5 inch step. The individual is instructed to touch the tape with the heel of the shoe, to maintain all of the body weight on the stance limb through the entire repetition, to immediately remove it after the tap, and place it on the floor where another piece of tape is located. Demonstration may be appropriate to relay the details of the task. If an error is made such as banging into the step with the shoe, shifting the body weight onto the stepping limb, or sliding the foot off the step, the individual is asked to stop, regroup, and verbalize which part of the task led to the error. Successful learning of the task using a visual cue occurs if the error that was made is addressed, understood, and actively avoided. Without the visual cue of the target on the floor, the amplitude of the repeated action diminishes until the movement ceases. The visual cue helps the individual maintain the appropriate speed and amplitude of movement throughout the entire movement so that a functional task such as stepping over an object on the floor or stepping through a doorway threshold can be completed.

Mental Practice

Mental practice is another form of intrinsic feedback that can be used to encourage movement initiation. To perform the heel tap exercise described above, the therapist gives explicit instructions to the individual to perform the task such as, "tap your heel on the step ten times." The therapist observes the execution of the task and identifies errors such as banging into the rise of the step with the shoe, reaching out the upper extremity to catch balance, or sliding the shoe uncontrollably off the step. Next, the therapist works with the individual to point out the errors and break down the components of the task. This may require writing the steps down for the individual to review at a subsequent time. The therapist and the individual verbalize the task step-by-step as previously identified. Before attempting the activity, the individual mentally practices the steps simultaneously verbalizing each step as he progresses thorough the activity. Then the individual performs the task. If a mistake occurs, the individual is asked to stop, regroup, and start again with mental practice. It may be helpful to perform the mental practice for the heel tap activity in a standing position with the eyes closed and holding onto a solid object for support to maximize the specificity of the task.

Mental practice can also be implemented for functional tasks such as initiating movement to get out of bed. The therapist may start the activity by observing the individual move from his sleeping position to sitting on the edge of the bed in an environment that simulates his bedroom setup as much as possible. The information is used to identify problem areas such as rolling, pushing up to sitting, or managing sheets and blankets. Next, strategies are developed to resolve these issues. The important factor is to break down the steps such that the individual can close his eyes and repeat each step aloud while he performs each component in his mind. Then, the instructions are implemented. If a mistake occurs, the individual is asked to stop and start again from the beginning with mental practice. It may be very beneficial to

reinforce this practice by including the individual's caregiver in the session.

Verbal Cues

Verbal cues, a form of extrinsic feedback, can also be used to initiate movement. To perform the heel tap exercise, the therapist repeats a word or phrase that the individual can respond to when implementing the task. For example, each time the individual places his heel on the step the therapist says "heel" or "step." It is important that no other verbal cues or instructions are given during the performance of the task as it will likely interfere with the individual's ability to successfully complete the task. It may be helpful for the individual to repeat the verbal cue systematically as he performs the task. The individual can use this cue for subsequent functional tasks such as stepping over an object during walking or stepping through a doorway. If a freezing episode occurs at a doorway threshold for example, the individual may be able to say this word aloud to assist in generating the motor plan necessary to continue walking.

Tactile Cues

A tactile cue, a form of extrinsic feedback, can also provide the signal necessary for an individual with PD to initiate a movement. For example, during the heel tap exercise described above, the physical therapist can touch an easily accessible part of the individual such as the anterior superior knee or ipsilateral shoulder prior to the movement to be initiated. It is important that no other form of feedback be given to the individual during the task. The therapist explains that the individual is to wait for the tactile cue before implementing the task. If the individual anticipates the cue and initiates the heel tap prior to the cue, the activity is stopped and the individual is asked to identify the error before attempting the task again. The individual can use this type of cue for the functional task of stepping through a doorway threshold similar to the verbal cue previously described. Rather than saying a word aloud, an individual can tap the side of his leg.

CONCLUSION

For an individual with PD to successfully use intrinsic or extrinsic feedback, the person must be engaged in some detail of the task for the ability to perform the task to be retained. Repetition without focus is similar to expecting an individual with PD to regain the ability to perform a task without attention or conscious thought of the task. Additionally, giving an extrinsic form of feedback such as verbal cues while the individual is also receiving visual cues or attentional cues to implement a movement will likely interfere with the ability to successfully perform the movement. For example, performing interventions in front of a mirror may interfere with the intervention if the individual is receiving visual information when attempting to use an alternate form of feedback. Motor learning concepts should be applied to functional tasks whenever possible rather than expecting an individual to carry these techniques over to different activities. Guidelines for implementing motor learning concepts in those with PD are listed in Figure 2.

- Define the type of task being performed
- Define the type of feedback you will use for the intended task
- Consider the principle of specificity when desigining interventions
- Avoid performing activities in front of a mirror to minimize visual interference
- Do no expect carryover; rather practice each functional task specifically
- Ask the individual to keep a diary of movement problems to help direct interventions
- Involve family members and caregivers
- Provide written instructions
- Be consisitent

Figure 2.

Consider that the signs and symptoms of PD are widespread and that no two individuals experience the effects of the disease in the same way. Therefore, not all forms of intrinsic or extrinsic feedback will be beneficial for every individual with PD. A form of feedback that was once beneficial for an individual may cease to be helpful for initiating or sustaining a movement. Once an extrinsic cue is known to the individual it becomes intrinsic since the individual can now anticipate the cue and influence the implementation of the cue.

Little is known regarding the systematic use of intrinsic or extrinsic feedback to facilitate learning a procedural task such as walking. Vidoni et al note that the impact of explicit instructions depends on many factors such as the presence or absence of a brain lesion, the location of the lesion, and the characteristics of the task being taught.[1] In practice, a combination of the implicit and explicit learning systems may be the most beneficial for learning motor skills.[1]

Further research is needed to determine the efficacy of motor learning concepts to guide motor skill planning, problem-solving, and movement initiation in individuals with Parkinson's disease. Although fall risk can be assessed pre- and postimplementation of the interventions described above, no functional performance measures or outcomes measures currently exist that quantify an individual's response to motor learning strategies.

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GAIT AND PARKINSON'S DISEASE

Miriam Boelen, PT

Addressing gait in people with Parkinson's disease (PwP) is often a dynamic, individualized, and comprehensive process. Wide arrays of treatment interventions are available that can be used selectively to address these individual needs. Overall, gait deviations in PwP can vary from one individual to another and also change over time within an individual. The dynamics of gait can be altered by changes in Parkinson's disease (PD) medications or during the medication cycle itself. It is difficult to talk about gait without considering a person's stability while walking. Some of the Parkinsonian gait deviations in and of themselves can cause instability and falls. Freezing of gait, postural instability, and cognitive impairment are considered independent fall risk factors in PD.[1] Common occurrences of reported instability are during turns, freezing, when not paying attention, rushing, when PD medications are not working 'off state', when PD meds are working 'on state.' Reports of feet catching tend to be more related to freezing. Either freezing or shuffling can cause tripping, both have reduced ground clearance. Repeated fallers tend to have etiological patterns that cause their falls.[2] Obtaining a fall history or causes of loss of balance (near falls) can offer insight into trends and interventions.

GAIT DIVERSITY

You have probably heard someone say, "That person has a typical Parkinson's gait." Immediately you envision the forward flexed posture and short shuffling steps. This is not the case for everyone who has PD. The gait deviations seen in PwP can be diverse. Someone in Hoehn & Yahr stage 1 may only exhibit reduced arm swing or a reduction of stride length unilaterally. Turning away from the affected side may be more difficult but not necessarily problematic. Hoehn & Yahr Stage 2 can exhibit a reduction of bilateral arm swing and stride length. Shuffling may or may not be present. Stride length may be asymmetrical or arrhythmical. Freezing of gait may exist in some PwP and others may never develop freezing. You may observe a festinating gait that presents as an involuntary speeding up of gait with a progressive shortening of stride length and tendency to lean forward. This type of gait can be very destabilizing. Each individual with PD has their own unique set of gait idiosyncrasies that may require a combination of various interventions to unlock the restrictiveness of their deviations and instabilities.

UNIVERSAL ATTRIBUTES

As you can see there can be much diversity in the "Parkinsonian gait" but there are also a few common attributes that spans the course of the disease.

Attention Demands

The first attribute is the increased attention demands needed to walk optimally. The need to pay more attention is due to basal ganglia dysfunction, which is normally responsible for automatic movements.[3,4] The *level* of attention needed will depend on disease severity.[5] The *ability* to pay attention under dual task conditions to maintain gait velocity and rhythmicity will depend on cognition.[6] It has been my experience that some individuals can be less safe or may walk significantly slower when walking with a cane compared to no device due to its dual tasking affects. Others may need to use a walker and bypass the thought of using a cane because of the cognitive challenge.

Gait Hypokinesia

The second universal gait attribute is gait hypokinesia or a reduction of amplitude of movement. These changes may be pronounced or very subtle yet present early on in the disease course.[7] People with PD have slower gait velocities compared to age matched norms and is primarily due to a reduction stride length.[8,9] Cadence (steps per minute) remains relatively intact.[10]

Optimizing stride length or gait velocity for functional ambulation is a common target for gait interventions. Gait velocity norms can be used as an educational tool for patients so they know how they compare to their healthy age/sex matched peers. Norms can also be helpful in establishing goals (see Tables 1 and 2). Normative values in these tables were established from a national study involving over 7000 independent living older adults ranging from 60 to 95 years old who performed the 6 minute walk test.[11]

FREEZING OF GAIT

Freezing is the inability to take steps while walking or when attempting to start walking. Attempted movement is visible *stutter stepping* but nonproductive. Freezing is not well understood since freezing can occur when dopamine levels are optimal after taking Levodopa medication; this is called *on* freezing. Freezing can also occur in other disorders that do not have dopamine deficiencies. *Off* freezing is freezing that occurs when dopamine levels in the brain are low. This is typically seen in early morning prior to taking the initial dose of dopaminergic medications or can be dependent on the medication cycle itself. The incidence of freezing increases with disease progression.[12] Freezing can be 'triggered' by various activities, environmental factors, or psychological factors. One person may be riddled with freezing triggers yet another individual may have only one or two triggers. Freezing episodes are typically less than 10 seconds but can last up to a minute.

Common Freezing Triggers

- Start hesitation (hesitation when initiating walking)
- Turning
- Walking through narrow spaces
- Destination hesitation (hesitation when approaching a destination such as a chair)
- Thresholds
- Posture (forward flex posture can cause anterior instability and trigger a freeze)
- Rushing
- Psychological factors: anxiety, stress

Table 1. Female Walking Velocity Norms by Age

Age	Meters per second (m/sec)	Meters per minute (m/min)	Miles per hour (mph)*	Kilometers per hour (km/h)*
60–64	1.52 m/sec	91.2 m/min	3.40 mph	5.47 km/h
65–69	1.43 m/sec	85.8 m/min	3.20 mph	5.15 km/h
70–74	1.38 m/sec	82.8 m/min	3.09 mph	4.97 km/h
75–79	1.29 m/sec	77.4 m/min	2.89 mph	4.65 km/h
80–84	1.17 m/sec	70.2 m/min	2.62 mph	4.22 km/h
85–89	1.08 m/sec	64.8 m/min	2.42 mph	3.89 km/h
90–94	0.90 m/sec	54.0 m/min	2.01 mph	3.23 km/h

*Rounded to the nearest hundredth.
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Table 2. Male Walking Velocity Norms by Age

Age	Meters per second (m/sec)	Meters per minute (m/min)	Miles per hour (mph)*	Kilometers per hour (km/h)*
60–64	1.70 m/sec	102.0 m/min	3.80 mph	6.11 km/h
65–69	1.59 m/sec	95.4 m/min	3.56 mph	5.73 km/h
70–74	1.55 m/sec	93.0 m/min	3.47 mph	5.58 km/h
75–79	1.40 m/sec	84.0 m/min	3.13 mph	5.04 km/h
80–84	1.32 m/sec	79.2 m/min	2.95 mph	4.75 km/h
85–89	1.20 m/sec	72.0 m/min	2.68 mph	4.31 km/h
90–94	1.02 m/sec	61.2 m/min	2.28 mph	3.67 km/h

*Rounded to the nearest hundredth.
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During a freeze, lateral weight shift is rapid and insufficient in magnitude causing the inability to unload the potential swing leg long enough to take a step. People with PD who freeze demonstrate a greater dysrhythmicity of gait than their counterparts who don't freeze.[13] Interventions for freezing focus on weight shifting, improving force production, rhythmicity of gait, and reviewing attentional compensatory 'antifreezing' strategies while targeting the freezing triggers. Antifreezing strategies are maneuvers used to 'unlock' a freezing episode. For example, during start hesitation a patient attempts to take his/her first step *backward* instead of forward and then starts walking forward with the same foot. Visual cues such as a line or pen on the floor is most effective for unlocking *off* freezing.

TURNS

People with PD exhibit greater lateral instability than people without PD that becomes more pronounced with increased disease severity. Lateral stability is needed when turning. Instability with turns is multifactoral and includes postural instability coupled with a narrow based support, and limited trunk mobility.[14] Hip abductor weakness, unrelated to PD but due to aging and deconditioning, can also affect stability with turns. Although there are many factors influencing a turn, I have found that foot placement or the stride length of the foot on the outside of the turn can play a significant role in turning ability. Typically the foot on the outside of the turn is lagging behind, which can also trigger a freezing episode. For example: if making a *left* turn, increasing the stride length of the *right* foot should be emphasized to reduce difficulties. Patient education on foot placement is often beneficial.

GAIT INTERVENTIONS

Interventions to improve gait often requires a comprehensive approach. The initial concern is balance and safety. Focus is primarily on improving stride length that encompasses not only linear walking but also turns and freezing of gait. Conditioning and flexibility training is often needed to improve gait stability and can have secondary benefits in the reduction of deviations. For those who remain unsteady even with our best efforts will require training on how to use an assist device safely.

Attention Strategies

Attention strategies may be sufficient to normalize gait for people with mild PD. The person directs their attention to the feet and thinks *take long steps* or *heel down 1st*. One phrase may be more effective than the other and should be determined on an individual basis.

Perceptual Motor Training

Sensory recalibration training can normalize amplitude of movements and is defined as "the process of teaching a patient to self-monitor & accept that what feels too big is normal."[15] For example, if an individual is walking with short (small) steps, this step length will feel normal or natural to a person with PD. When stride length is normalized, perceptually it will feel *big*. One of the hallmark symptoms of PD is bradykinesia (slowness of movement) that secondarily has affects on the amplitude of movement resulting in hypokinesia. The LSVT BIG training focuses on exercises that are of higher intensity with a single focus on amplitude and uses whole body movements. This type of training is showing significant improvements with gait velocity, balance, and dual tasking abilities when compared to controls.[16]

Rhythmic Auditory Stimulation

Rhythmic auditory stimulation (RAS) uses alternate neuronal pathways to improve rhythmicity of gait as well as force production that are both impaired in PwP. The end result is improved stride length and secondarily improved gait velocity. It has shown to be effective in Hoehn & Yahr stages 1 through 4.[17] I have found that RAS music can be effective with some individuals who have dementia and have difficulty following verbal cues. One study demonstrated

long-term benefits of 3 weeks after a 3-week training program.[18] To implement RAS you will need to know the cadence (steps per minute) of your patient. Then you need an auditory stimulus (metronome, music) that is in the approximate range (beats per minute-bpm) of your patient's cadence. From there, simply make adjustments (increments of 5 bpm up or down) until stride length is optimized and the patient is able to easily hook into the beat. A metronome such as the Seiko DM50 is small enough to fit into the palm of your hand and can be clipped onto the patients clothing to free up the clinician. I have found that only a few patients prefer the metronome since it can become somewhat annoying. The metronome can be helpful in determining the optimal beat/minute for those few individuals whose gait is so dysrhythmic that attempting to count cadence can be challenging. Walking to music has shown to facilitate rhythmicity of gait and improve motor synchronization to a greater degree than walking to a metronome.[19] Music CDs that were composed specifically for therapeutic interventions can be obtained from www.colostate.edu/dept/CBRM. Commercial CDs for people who have a walking cadence of 108 steps per minute or greater and can follow more complex musical arrangements can purchase CDs from www.workoutmusicvideo.com. Cardiovascular responses and rate of perceived exertion should be monitored.

Walkers

There continues to be scant research in the area of PD and the use of walkers. Over the past 20 years, working with PwP and making walker recommendations for approximately 400 individual cases I have found that once a patient is too unsteady or unsafe to walk unaided, the use of a walker can offer substantial benefits in fall prevention, improving stamina, independence, and reduce caregiver strain. Often patients are resistant to the idea of using a walker due to concerns of "going down hill" or fearful of "becoming dependent" on the walker. Patient education is critical regarding their misconceptions as well as the benefits of using a walker. As with any individual, if you can experience the benefits of a device, the likelihood of becoming interested in using it is much greater. Patients should be given the opportunity to "test drive" a walker to comparatively experience how it feels to walk with and without an assist device. Having various types of walkers available in the clinic is helpful regarding the evaluative process in determining the most appropriate walker. Sometimes the seat of a 4 wheeled walker can act as a freezing trigger or make a PwP walk too far behind the walker. Perceptually, patients report concerns regarding their legs bumping into the seat. Walkers that allow the seat to flip up and out of the way while they walk can facilitate safer and proper walker usage and reduce potential freezing. Gait improvements I have observed when using a walker is a reduction in freezing, improved stride length, turning stability, and gait velocity. Patients have also reported improved confidence in their walking balance.

Foot wear

If a patient reports that their feet tend to catch on carpeting or have problems with freezing, then shoe wear may be contributing to instabilities or falls. Other than having a well fitting supportive shoe, the main concern regarding foot wear is the sole of the shoe specifically the forefoot. During the stance phases of gait, PwP exhibit a reduced impact at the heel and a greater forefoot loading.[20] Wearing

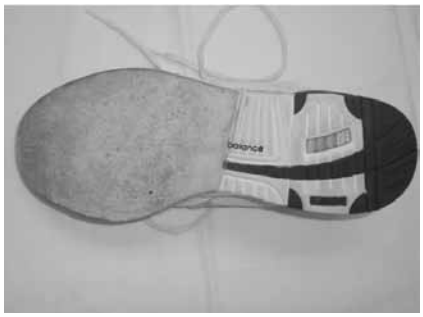

Figure 1. Resurfaced running shoe.

shoes with soft rubbery soles will create additional friction between the shoe and ground surface causing the foot to catch and cause a forward directional loss of balance or fall. Soles with ridges can act similarly on more compliant surfaces such as carpeting or grassy areas. One solution to this problem is to have the front half of the shoe sole resurfaced with a leather sole or other material with a lower friction coefficient (Figure 1). A shoe repair shop can resurface shoes but not all shoes are "surgical candidates."

It is important for therapists to recognize the individuality of the "Parkinson's gait." Interventions addressing both intrinsic factors and extrinsic factors may be necessary to optimize functional ambulation. Intrinsically, therapists have a lot to offer our patients such as: perceptual motor retraining, use of rhythmic auditory stimulation, attention strategies, visual cues, and last but not least, a comprehensive exercise program. Extrinsically, the use of an assistive device and/or adapting shoe wear may be needed.

Miriam Boelen is a physical therapist who has specialized in PD for 20 years. She is the author of a recently published book for physical therapists, *Health Professionals' Guide to Physical Management of Parkinson's Disease.* She is an ACSM certified Health Fitness Specialist.

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POSTURAL CONTROL IN ADULTS WITH IDIOPATHIC PARKINSON'S DISEASE: TREATING COMPLEX BALANCE DEFICITS WITH MULTISYSTEM APPROACHES

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INTRODUCTION – BACKGROUND AND PURPOSE

Idiopathic Parkinson's disease (PD) is common in the elderly with an estimated annual incidence rate of 114.7 (per 100,000 person-years) in people aged 50 to 99.[1] While PD is common in older individuals, a substantial number of people are diagnosed in their 30s and 40s.[2,3] Of the many and varied clinical manifestations of PD, at least 80% of people exhibit postural control (PC) deficits throughout the course of the disease. These deficits are characterized by inflexible, weak, and inappropriately timed responses (both anticipatory and reactive) to perturbation.[4] Resulting balance and gait disorders evolve with disease progression and declines in PC that increase fall risk, increase fear of falling, decrease quality of life, and contribute to subsequent losses of independence.[2,5,6]

Postural control deficits become clinically recognizable as the dopaminergic system in the basal ganglia is depleted. These deficits cannot be fully explained by basal ganglia dysfunction alone; however, PC mechanisms in PD also are influenced by non-dopaminergic pathways.[7] Clinical signs of PC deficits include: diminished dynamic trunk control, decreased postural sway with external perturbation,[4] abnormal cognitive processing and integration of visual, vestibular, and sensory input, and decreased muscle torque generation, especially when persons experience excursions outside functional limits of stability.[5,6,8-10]

Since PC deficits are common in PD, it is important to consider how biological systems interact with the environmental constraints in the evolution of the impairment profile as it changes throughout the disease process. Additionally, effective treatment strategies should address those PC and balance deficits specific to each patient with PD at each stage of the disease process. It is the aim of this paper to describe the systems leading to balance impairments and provide evidence-based treatment strategies that address the complexity of systems interaction in postural control.

REQUIREMENTS FOR NORMAL POSTURAL CONTROL

Normal postural control depends on complex interactions between multiple systems to maintain physical alignment of the body in space in order to execute strategies to maintain balance. Although PC necessitates quick-acting, global neuromuscular synergies for orientation and balance, these muscular patterns are not located in the central nervous system as a set of reflexes that trigger equilibrium responses.[11] Instead, postural control is represented by a "central set," that is, the integration of multiple systems within the body as they interact with the environment and the task.[11] Balance is achieved moment by moment by selection and integration of multiple sensory resources required by the environmental context and the specifics of task demands. These resources include a confluence of input from biomechanical systems (muscles, bones, and joints that determine mechanical limits of stability), sensory systems (visual, auditory, proprioceptive), dynamic control systems (as in control of gait and posture), systems that orient in space (vestibular, proprioception, perception), and cognitive faculties (attention, memory, and judgment). Appropriate predictive (anticipatory) and responsive (reactive) balance strategies, either voluntary or spontaneous, depends on multisystem integration.[11] If any system is deficient in this process, then deficits in postural control and balance emerge.

POSTURAL CONTROL DEFICITS IN PARKINSON'S DISEASE

Postural control deficits are present throughout the disease process even though they might not readily be observable clinically. Fall risk increases as these deficits become manifest. An estimated 90% of persons with PD fall over the course of the disease process.[5,12,13] Researchers have described early signs of PC deficits that are neither readily observable nor readily measurable (not represented on clinical measures of balance), such as inconsistency and ineffectiveness in the choice of post-perturbation recovery strategy, excessive posterior excursion of the center of mass during loss of balance, hypokinetic weight shifting in recovering balance, and multiple (repetitive) anticipatory postural adjustments, which become greater as the disease progresses.[14,15] Further study is warranted, however, to determine the exact clinical significance of these early alterations in postural stability.[15] While signs of PC deficits exist in the early stages of PD, no direct link has been established to the magnitude of fall incidence or fall risk.

As PD progresses from earlier to later stages, increased cognitive impairments are noted, indicating dysfunctions in higher levels of motor control that subserve PC.[16] These cognitive changes manifest clinically as changes in visuospatial processing (shifts in the central set or from one type of feedback to another), diminished working memory (difficulty with interfering stimuli), diminished long term memory (where organization or association between tasks is required), decreased procedural learning (related to attention), and decreased executive dysfunction (decreased response to new environmental situations/context), even in the absence of PD-related dementias.[16]

More specifically, during the course of PD progression, defective attention mechanisms develop from deficient procedural memory, negatively impacting on motor planning, and thus, on postural control.[2,16] In the disease process, deficient attentional strategies have been associated with increased fall risk.[7] As the disease progresses, people lose the abil-

ity to move spontaneously and must "attend" to stabilizing their balance during simple functional movements, making multi-tasking challenging. The ability to split attention or perform dual task activities during motor tasks such as gait becomes extremely difficult and increases risk for fall.[2,17]

Other impairments affecting postural stability are neuromusculoskeletal dysfunctions that evolve secondarily to the longer-term basal ganglia dysfunction. Tonal impairments, for example, manifest as bradykinesia (a slowed ability to start movements) or akinesia (inability to start movements).[18] Abnormal tone (eg, rigidity) negatively manifests in PC as an "ineffective stiffening response and inability to modify postural responses for changing postural demands related to direction of perturbation and initial stance posture."[19] Due to the inflexibility and poor timing of postural strategies, bradykinesia alone can increase fall risk.[20]

Hypokinesia (abnormally decreased movement amplitude) is another impairment that manifests as part of the disease process, and may well be a compensation for decreased balance and postural control. People with PD tend to move more slowly when the task calls for more dynamic balance, such as in multi-tasking (such as walking, dialing the phone, and speaking on the phone concurrently) or with a more complex sequence of movements (walking on an unstable surface with varying base of support).[2] Slowed movement coupled with the neuromusculoskeletal problems of axial- and stooped postural rigidity combine to create balance problems independent of strength or range of motion losses.[21]

As balance challenges increase in PD, resulting from deficits in PC, patients present with increased dependence on external cueing—visual, auditory, and verbal (cognitive)—to normalize movement patterns.[2] Cueing cannot compensate, however, for the progression of somato-sensory and visual deficits within the course of the disease. People may experience a decline in vertical orientation as postural changes occur, decreased responsiveness of stretch reflexes, decreased proprioceptive feedback of position and movement, and inability to orient the head and trunk when visual and vestibular information is diminished or absent.[22] Problems in proprioceptive integration deficits result in an inability to orient the body in space, and an increased reliance on vision for accurate movement.[22] Increased reliance on vision itself is a problem, because problems in visual processing and visual focus jeopardize balance. For example, one of the visual processing problems in PD is decreased vestibulocollic reflexes.[23] Declines in both the vestibulocollic and vestibulo-ocular reflexes can result in reduced gaze stability that in turn directly impact balance by decreasing the reliability of visual information gained during dynamic tasks.

INTERVENTIONS TO IMPROVE POSTURAL CONTROL STABILITY IN PD

To capture the scope of PC deficits in PD, clinicians should aim to address as many impairments as possible within one treatment in order to maximize outcomes efficiently. This treatment strategy enlists the functional movement patterns of the whole person within the context of meaningful activities. Such a comprehensive, functional approach also is more likely to be more enjoyable, thus ensuring compliance and retention.[24] Among the evidence-based treatment approaches, task-specific training has been shown to be robust in different treatment management approaches for persons with PD.[25] As cognitive and perceptuo-motor strategies are embedded within task specific strategies and into task specific training, motor learning increases.[25] Once such program, as developed by King et al, reported positive outcomes consisting of improved postural control using a balance program that focused on augmented sensorimotor (perceptuo-motor) and cognitive elements accompanying agility exercises.[25]

A number of complementary approaches to balance training are emerging. While evidence is limited, these approaches integrate complex movement tasks with "mindful" (ie, augmented cognitive and perceptual awareness) to normalize PC through influencing the central set.[26] These interventions include: Tai Chi, the Alexander Technique, several styles of solo and partner dance, aquatic therapy, and cognitive focused interventions. All of these interventions require active awareness and conscious attention to whole body movement or sustained, focused attention to stimuli.

Tai Chi in Parkinson's Disease

Tai Chi as an ancient *soft* martial art, whose exercises (usually done standing) incorporate slow, rhythmic, physical movements involving whole body rotation and weight shifting, with flexion and extension of the hips and knees, coordinated arm movements, and attention to rhythmic breathing and spinal alignment. Persons attend to balance while transitioning from positions of stability towards positions of greater mobility. In addition to its focus on movement, the interaction between conscious sensory awareness and thought elicits responses in somatosensory and neuromuscular control pathways that lead to improved PC.[27] With practice of this conscious movement method, studies have demonstrated increased postural control, increased well-being, and balance in normal elderly individuals. In a study by Tsang et al, Tai Chi was shown to improve balance and postural control in individuals as measured by dynamic posturography, by improving vestibular control, sensory organization, proprioception, weight shifting, and limits of stability.[28] Similar effects were observed in studies on Tai Chi and balance in participants with PD, as well as improved perceptions of postural control and balance leading to improved quality of life.[27,29,30]

Alexander Technique in Parkinson's Disease

The Alexander Technique is an approach to training postural coordination through light touch and verbal cueing during the performance of daily and skilled activities. The Alexander Technique particularly aims to improve balance by bringing to conscious awareness faulty postural habits and unnecessary tension and effort.[31] The Technique redirects the person towards a more active state of postural readiness through promoting a combination of focused awareness in action, enhanced tonic muscular activity, and balanced mechanical support. Limited, but promising evidence of its effectiveness on balance has been shown in a variety of studies with the elderly,[32,33] as well as on mobility, mood, and quality of life in persons with PD. In a randomized controlled trial, 93 subjects with PD were divided into 2 groups, Alexander Technique and massage, and given equal treatment doses. Results were significant for the Alexander group on the Secondary Parkinson 's Disease Dis-

ability Scale, Beck Depression Inventory, and the Attitudes to Self Scale, with improvements sustained through a 6-month follow-up.[34]

Dance in Parkinson's Disease

Because of rhythmic enhancement, regulated stepping patterns, and emphasis on whole body coordination, various styles of dance have been advocated to improve balance in persons with PD. Rhythmic auditory cueing, as well as verbal and visual cueing by the dance teacher, help facilitate movement. Simple-to-complex movement sequences and stepping and locomotor patterns, both alone in a group and with partners, requires quick reaction time and variability in balance strategies, as well as promotes strength and flexibility.[35] In a 2009 study by Hackney et al, on the effects of tango, waltz, and foxtrot on balance in persons with PD, significant increases in the Berg balance scale, 6 minute walk distance, and backward walking stride length were found after 13 weeks of instruction, compared with a control group, who suffered declines in these areas.[36] The researchers related the functional gains in this study to the ability of dance to incorporate dynamic weight shifting, backwards walking, dynamic perturbations, and single limb support, all of which tend to be deficient in PD.[36] Dance also requires complex cognitive processing (stimulating memory, prediction, and judgment), as well as augments sensory integration to enhance the body schema for improved body awareness in space. Finally, dance is an enjoyable activity that is feasible in terms of cost-effectiveness, compliance, and sociability.

Aquatic Therapy in Parkinson's Disease

Aquatic exercise is a technique that can incorporate central processing and address individual impairments, due to the unique nature of water. The properties of buoyancy, density, and viscosity interact with the body, providing forces and input to the body. For example, as the patient exercises in water, the patient is supported by the buoyancy of the water allowing for more upright posture.[37] Muscular effort can be modulated by either moving with or against (resisting) the water.[38] As the patient moves, proprioception is enhanced by water viscosity and density. Patients can move at their own selected comfortable pace in water with more time to detect balance perturbations. Persons can recovery from near falls and falls without injury.[38]

Using these properties of the water, aquatic exercises and aquatic therapy protocols are well evolved. Ai Chi, for example, incorporates elements of Tai Chi and is performed standing in shoulder-depth water using a combination of deep breathing and slow, broad movements of the arms, legs, and torso.[39] The Halliwick protocol also is popular, which incorporates breathing, mental practice, and trunk rotational components to facilitate control in the water.[33] Due to the possible impact of these techniques on systems needed for increased postural control, the effects of these methods on postural control have been studied.

Despite the popularity of aquatherapy for many patient populations, evidence on the effectiveness of aquatherapy for persons with PD is limited. While Halliwick, Ai Chi and other forms of aquatic exercise have shown positive outcomes on postural control in non-neurologically impaired populations,[38,42] only one study showed improvement in trunk control in persons with PD using the Halliwick technique. As with other treatments listed here, results appear promising, but are limited in scope with this population, and more research is warranted to demonstrate effectiveness in affecting PC impairments with PD.

Attention Focused Interventions in Parkinson's Disease

Enhanced attentional focus appears to have a positive effect on balance when recruited in motor learning. Just how and in what way a patient focuses his or her attention makes a difference in balance abilities. The focus of attention may be directed intrinsically (towards the individual) or extrinsically (towards the environment). Wulf et al demonstrated that persons with PD achieved increased postural stability (as measured by decreased postural sway) emerged when attentional strategies were focused on extrinsic cues (the environmental effects of the task) instead of intrinsic, body-based cues.[43] Future research bears on whether motor learning through external focus interventions is permanent and generalizable to dual task situations.

SUMMARY

Postural control deficits are common in adults with PD throughout the course of the disease. The nature of these deficits is multifaceted and includes central system impairments and those impairments brought about by the disease process. These impairments lead to higher fall risk, higher fall incidence, loss of independence, and decreased quality of life. Interventions for improvements in postural stability should focus not only on meaningful tasks, but also on engaging active, conscious, sensory awareness, and cognitive problem solving to improve postural control and balance. Increasingly, the literature supports interventions that address remediation of these deficits through whole body movement in complex environments. These interventions allow the person's cognitive and perceptual faculties to be challenged to promote sensory integration and normalization of the motor plan that helps to decrease fall risk and improve quality of life, while allowing for an enjoyable therapeutic experience by the patient.

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DEEP BRAIN STIMULATION

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OVERVIEW

As discussed in previous articles in this issue, the cardinal signs of Parkinson's disease (PD) include resting tremor, rigidity, bradykinesia, and postural instability. Traditional medical treatment includes pharmacological management using Levodopa and other medications.[1,2] With long term use of Levodopa, patients with PD may develop dyskinesias and fluctuations in motor control.[1] During *on* phases of medication, motor control is good; however, mobility can deteriorate during *off* phases.[1] The off phases occur when the effects of the medication has worn off prior to the time of the next dose or can occur abruptly regardless of medication cycle. Patients who have symptoms that are not adequately controlled by medications may be a candidate for deep brain stimulation (DBS). Okun[3] estimates that 10% to 20% of patients with PD may benefit from DBS. The purpose of this article is to describe DBS including the procedure, candidates for DBS, advantages and disadvantages of the procedure, contraindications for DBS, precautions that need to be followed, and implications for physical therapy.

DBS DEVICE AND PROCEDURE

Deep brain stimulation is an FDA-approved surgical technique used for treatment of PD.[3-6] The stimulator unit consists of 3 components: the lead, the extension, and the neurostimulator.[2,4,7] The lead is a thin wire with 4 electrodes that is placed in the brain through a small burr hole in the skull.[4] The extension is an insulated wire that passes under the skin and connects the lead to the neurostimulator. In most cases, the neurostimulator is surgically implanted near the clavicle.[2,4,7] The neurostimulator, also referred to as an implantable pulse generator (IPG), holds the battery.[2]

There are several different types of neurostimulator units available for deep brain stimulation. Medtronic, Inc. produces the Activa PC Neurostimulator (dual channel), Activa RC Neurostimulator (dual channel, rechargeable), Kinetra Neurostimulator (dual channel), and Soletra Stimulator (single channel). [4] Components of the Activa PC Neurostimulator can be found in Figure 1. Most surgical centers use dual channel neurostimulators, although some will complete a staged technique where the electrode is implanted on one side of the brain and then the second electrode is placed at a later date if necessary.[3,4,6] Refer to Figure 2 for placement of the Activa PC Neurostimulator.

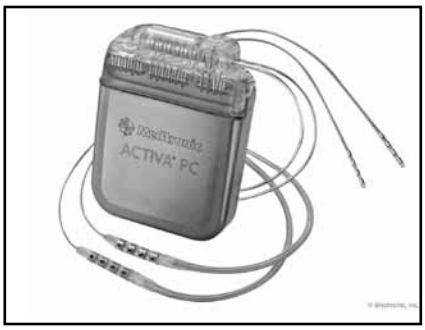

Figure 1. The Activa PC Neurostimulator. Medtronics, Inc. Minneapolis, MN. Reprinted with permission from Medtronic, Inc.

Figure 2. Placement of the Activa PC Neurostimulator unit in a Patient. Reprinted with permission from Medtronic, Inc.

Identifying the Target

The target for the electrode placement can be located using radiology tests including computed tomography (CT) or magnetic resonance imaging (MRI). Another technique that can be used is microelectrode recording (MER), during which a wire monitors the activity of the target area. In addition, the MER is used to confirm accurate target location intraoperatively by identifying the firing patterns of the intended target site.[8] Neuroimaging techniques facilitate location of the structures, but MER provides increased precision of placement. The Deep Brain Stimulation Study Group[1] suggest that MER may increase the risk of adverse events, such as intracranial bleeding. Despite increased risk of adverse events, the accuracy of placement associated with the MER is chosen for optimal outcome related to the resolution of the signs and symptoms of PD.[8] In a study by Johnsen and colleagues,[9] notes concerns of brain shift or electrode displacement during surgery when using MER. Of interest, Lozano and associates[8] note this possibility of brain shift as a reason to choose MER over MRI so that placement can be adjusted for optimal outcomes.

In patients with PD, DBS typically targets the subthalamic nucleus (STN) or the pars interna of the globus pallidus (GPi).[1-6,10-12] Refer to Figure 3 for electrode placement. The STN is targeted for patients with symptoms of tremor, bradykinesia, and rigidity, whereas the GPi is targeted for patients with symptoms of Levodopa induced dyskinesias.[2] The STN appears to be the target of choice.[1,13,14] In a study by The Deep Brain Stimulation for Parkinson's Disease Study Group,[1] the target site was determined based on the experience and preference of the investigator. A total of 96 patients received stimulation to the STN, compared to only 38 patients who received stimulation to the GPi.[1] The GPi is a larger area compared to the size of the STN, resulting in the need for greater stimulation. Follett and colleagues[14]report that the STN is used more frequently despite a lack of evidence showing that the stimulation to the STN is superior to the GPi. Okun and Foote[13] suggest that more adverse effects may be seen in patients with stimulation to the STN because of a pos-

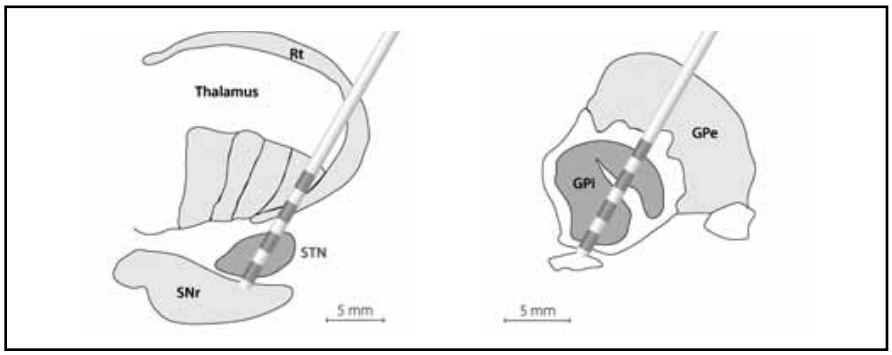

Figure 3. DBS electrode placement into the Subthalmic Nucleus (STN) on Left; DBS electrode placement into the Globus Pallidus (GPi) on Right. Reprinted with Permission from Medtronic, Inc.

sibility of overflow of current to different areas of the brain. Studies have shown a reduction in medications following DBS placement in the STN.[3,7,13]

Moro and colleagues[15] examined long term effects of STN and GPi stimulation. Both groups had sustained improvements after 5 to 6 years. They noted patients with STN stimulation had greater improvements in their Unified Parkinson Disease Rating Scale (UPDRS) scores, but also had more adverse effects compared to the GPi group. However, the study did not directly compare STN to GPi. Moro and colleagues[15] followed the subjects from the study completed by the Deep Brain Stimulation Group, so randomization of the procedure (STN vs. GPi placement) was not completed.

Few studies have compared stimulation of the STN to stimulation of the GPi.[10,13,14,16] In studies directly comparing the areas, improvements have been noted with stimulation in both groups.[10,14,16] Studies by Anderson et al[16] and Alberts et al[10] found no significant differences between STN and GPi stimulation in motor functioning as measured by the UPDRS. Follett and colleagues[14] completed a large, randomized comparison of STN to GPi stimulation with almost 300 subjects included in the 2 year study. Their findings supported that there was no differences in motor functioning based on placement of the DBS. They found that depression and visual processing speed was worse in patients with STN stimulation compared to the GPi. Medication reductions was greater in the group with STN stimulation.[14] They suggest that future target choice may depend on nonmotor factors.[14]

Once the electrode has been placed, the DBS unit will be tested with the patient awake to determine therapeutic effects.[4] The neurostimulator may be placed at the time of electrode placement or it may be done on an outpatient basis.[4] The DBS unit is programmed for the first time approximately 4 weeks after surgery. This allows for the resolution of any swelling.[2]

Prior to the advent of DBS, patients may have undergone pallidotomy or subthalamotomy procedures to reduce the effects of PD. These surgeries are irreversible and may induce further neurological deficits.[1] Deep brain stimulation to the STN and GPi can also improve the signs and symptoms of PD. The Deep-Brain Stimulation Study Group[1] found that magnitude of benefit was greater using DBS compared to other studies using pallidotomy or subthalamotomy.

CANDIDATES FOR SURGERY

Patients who have fluctuations of symptoms using medications should consider DBS. Okun[3] and Ngoh[2] suggests that the best candidates have idiopathic PD, are younger than 69 years old, and have little to no cognitive dysfunction. Patients may also be tested using the UPDRS following 12 hours of being *off-medication* and then again after a larger dose of medications. Good surgical candidates have at least a 30% improvement in their score on the UPDRS with medication. Symptoms that are relieved using Levadopa typically respond well to DBS.[2-5] The features of PD that do not respond well to medication likely will not be improved using the DBS.

Okun[3] describes the Florida Surgical Questionnaire for Parkinson's Disease (FLASQ-PD) as a valid screening tool for DBS. The questionnaire includes 5 sections: criteria for the diagnosis of probable PD, potential contraindications to PD surgery, general patient characteristics, favorable/unfavorable characteristics, and medication trial information. (Questionnaire is available at: http://mdc.mbi.ufl.edu/)

Patients that have dementia or other cognitive decline are typically excluded from DBS surgery.[2] Patients older than 69 may be considered for surgery, but Okun[3] notes an increased risk of hemorrhage in this age group. Other concerns for patients in this age group include the possibility of additional brain atrophy compared to younger patients. The possibility of thinner skin could result in exposure of leads that could increase the risk for infection.[3] Other comorbidities may have an effect on outcome. For example, patients with swallowing disorders are at higher risk for aspiration, patients with known incontinence may be at a higher risk for infection, and patients with cognitive or affective disorders may worsen their cognitive symptoms[3].

DBS ADVANTAGES

Deep brain stimulation can improve the symptoms of patients with PD.[1-6,10] In one study, patients with DBS have significantly improved mobility during their *off-medication* state resulting in significant improvement in tremor, rigidity, brakdykinesia, gait, postural stability, and activities of daily living.[1] Deep brain stimulation allows for precise target acquisition. Other advantages to this technique include fewer complications compared to pallidotomy and thalamomotomy, as well as the flexibility to adjust, turn off, and/or reverse the technique as new technology is developed.[2,3,6,17] Stimulation of the STN often reduces the need for medications.[3,7]

RISKS/DISADVANTAGES

As with any surgery, there are risks associated with the procedure. Risks related to the surgery include a slight risk of seizure, hemorrhage, weakness, numbness, paralysis, speech deficits, cognitive deficits, and misplaced leads.[3-5] There is a slightly higher risk for infection.[3,5] Risks related to the neurostimulator unit include device malfunction, lead migration, lead disconnection, lead erosion, breaking leads or extensions, and the need for battery replacement (average every 5 years).[2-4] Complications related to the stimulation including, but not limited to, paresthesias, muscle contractions, dysarthria, diplopia, depression, suicide, obsessive-compulsive

thoughts, and aggressive behaviors.[2-4] These complications may be resolved by changing the programmed settings.[5]

Some disadvantages to the procedure include cost, travel, and time commitment. Insertion of the DBS unit is more expensive than pallidotomy or subthalamomotomy, but a reduction in medication costs is possible so cost may not be a factor.[3] Deep brain stimulation requires more follow up appointments compared to pallidotomy or subthalamotomy.[3]

As noted previously, the first programming session occurs approximately 4 weeks after surgery.[2] The programming sessions for DBS may take 4 to 5 hours.[2] Programming sessions occur frequently during the first 6 months with follow up appointments as often as every 6 months.[3] Refer to Box 1 for parameters for DBS. This time commitment can be a disadvantage if the patient lives at a great distance from the care center. In the United Kingdom, nurse specialists monitor and adjust parameters of DBS locally to decrease travel to specialty centers.[18]

- Amplitude
- Range: 0-10.5 Volts (V)[4]
- Typical setting: 2.5-3 V, >3.7 V doubles the amount of battery usage[2]
- Rate
- Range: 0-250 Hz[4]
- Typical setting:100-185 Hz[2]
- Pulse width
- Range: 60-450 microseconds[4]
- Typical setting: 60-120 microseconds[2]

Note: High amplitudes and wide pulse widths may cause tissue damage.[4]

Box 1. Parameter Settings for Activa DBS unit

CONTRAINDICATIONS

Contraindications for DBS include[4]:

- Magnetic Resonance Imaging – MRI may heat the lead electrodes which can result in injury, coma, paralysis, or death. According to Medtronic, Inc.,[4] MRI can be performed safely with the Activa System. However, MRI may result in jolting from the stimulator or may turn the device off. This could lead to poor image quality if the patient's tremor returns during the test.
- Diathermy–the diathermy unit may cause tissue damage or death when used in conjunction with the DBS unit.
- Patient who cannot operate the neurostimulator.
- Patients who do not respond to test stimulation.

PRECAUTIONS

Patients should be educated that strong magnetic fields such as theft detectors and security screening devices may switch the device on or off. Some patients may notice increased stimulation when this occurs or may demonstrate an increase in Parkinson symptoms.[2,4,6] Some physicians suggest carrying an identification card so that such devices may be avoided. According to Keith Kroner, a DBS representative for Medtronic, Inc., "any therapy utilizing magnetic energy should be minimized, as most DBS generators can be spontaneously turned on or off with relatively low strength magnets. Patients should bring their remote control device to all therapy sessions and be familiar with its functioning so they are able to turn a device back on if needed" (written communication, May, 2010).

Manipulations of the neck as well as aggressive stretching techniques may cause damage to the leads and/or extensions. With these techniques, the wires in the extensions can break resulting in the need for additional surgery. Physical therapists should be cautious with aggressive range of motion activities in the neck following DBS implantation (Keith Kroner, written communication, May, 2010).

Patients should take caution to avoid damage to the neurostimulator case. It may result in severe burns if ruptured or pierced.[4] Medtronic, Inc.[4] reports, "The Activa System may be affected by or adversely affect medical equipment such as cardiac pacemakers or therapies, cardioverter/ defibrillators, external defibrillators, ultrasonic equipment, electrocautery, or radiation therapy."

IMPLICATIONS FOR PHYSICAL THERAPY

Deep brain stimulation can have a positive effect on patients with PD. Physical therapy services may be used following DBS implantation. Physical therapists must be mindful of contraindications and precautions discussed in this article. As noted previously, the signs and symptoms that do not respond to Levodopa will not respond to DBS.[2-5] Tremor, bradykinesia, and rigidity typically respond well to DBS. Studies by Alberts et al,[10] Follett et al,[14] and Anderson et al[16] showed significant improvements in motor symptoms as measured by the UPDRS when the DBS was *on* stimulation.

Postural instability and gait may or may not respond well to DBS.[11] For patients that do show improvements in gait, velocity increases have been noted. Velocity improvements were noted due to increased stride length in patients with the STN stimulated, whereas patients with GPi stimulation showed increases in stride length and cadence.[11] A case report by Farris and Giroux[19] showed that gait was worse initially after implantation of the DBS due to the presence of a spastic gait pattern preoperatively. They noted that gait significantly improved in this patient 8 months following DBS due to 6 months of intensive physical therapy.[19] Johnsen et al[20] found that gait symmetry improved following bilateral DBS implantation. Tassorelli and associates[21] found a significant improvement in the UPDRS-ME (motor examination subsection) and in the Functional Independence Measure (FIM) for patients undergoing physical therapy following DBS implantation.

MISCELLANEOUS ISSUES

This article has addressed how DBS impacts the cardinal signs of PD. There are other features of the disease that have been investigated following DBS. Hypophonia and other speech issues can be a common complaint in patients with PD.[22] Farrell and colleagues[22] found no change in speech following implantation of DBS. It should be noted that their sample size was small and compared speech following thalamotomy, pallidotomy, or DBS to patients who had not undergone surgery. They proposed that the surgical patients may have had minimal speech involvement prior to surgery as a reason for their results.[22] In contrast, Walker et al[23] showed improvement in stuttering during the *on* phase of DBS compared to the *off* phase following unilateral left STN stimulation (language dominant hemisphere).[23]

Patients with cognitive deficits are not considered ideal candidates for DBS implantation. A review article by Williams-Gray et al[24] reveals that DBS may affect cognitive functioning. Some research has noted improvement in executive functioning, but a decline in the ability to change behaviors in new situations. Overall no benefit to cognition was noted and DBS may actually worsen symptoms.[24] Furthermore, Gal-

lagher[25] reports a higher incidence of compulsive gambling and impulse control disorders in patients with DBS.

CONCLUSION

In conclusion, DBS is a surgical option for some patients with PD. Candidates must have a good response to symptoms using Levodopa, along with fluctuations in motor function due to *on/off* cycles of the medications. With DBS, patients with PD can have improvement in management of symptoms. Physical therapists should be aware of contraindications and precautions when working with this population. Patients with DBS can continue to benefit from physical therapy services.

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PHARMACOLOGIC MANAGEMENT IN PARKINSON'S DISEASE FOR THE PHYSICAL THERAPIST

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Physical therapy treatment of the individual with Parkinson's disease (PD) has a two-way interaction with medication regimen. The exercise we prescribe can impact the effects of medication, and at the same time medications can have a profound effect on our patients' response to therapeutic exercise. Exercise improves function and slows physical decline,[1,2] thus helping to forestall the need for symptom control through medication. In fact, recent data indicates that exercise may actually improve the hyper-excitability in the basal ganglia normally seen in the Parkinsonian state.[3] Additionally, there is emerging evidence in animal models that exercise may provide a neuroprotective effect[4-6] furthering its ability to forestall the need to initiate pharmacologic therapy.

An understanding of the pharmacologic management of PD is important for the physical therapist working with this population. Medication on/off cycles can have a profound impact on function and therefore must be taken into account when planning treatment sessions. In order to get optimal benefit from programs designed to work on underlying impairments such as strength, balance, and ROM, individuals should undergo therapy when he or she are feeling best. Therefore, timing interventions when the patient is *on* is recommended. However, when specifically addressing functional deficits, it is best to work with individuals at a time when they are experiencing limited function, and are *off*. Unfortunately, all medications have the potential to cause unwanted side effects as well as some serious adverse events. Knowledge of the side effect profiles of the medications your patient is on can help you better prescribe effective therapy, recognize and assist in managing expected side effects, and know when to refer back to the physician if the patient is experiencing adverse effects of medication (Box 1). At this time pharmacologic treatment is aimed at symptomatic relief. The decision to begin pharmacologic treatment is based on level of functional impairment including:[7]

- Effect of PD on the dominant hand
- Degree to which PD is interfering with work, activities of daily living, or social and leisure function
- Presence of significant bradykinesia or gait disturbance
- The individual's philosophy regarding the use of drugs

- Appearance of bothersome dyskinesias
- Significant periods of "off" time
- Appearance of frightening or troublesome hallucinations
- Significant confusion, somnolence

Box 1. When to contact the physician about possible medication related issues:

Once the decision is made to initiate therapy, the physician has two categories of medications to choose from, dopaminergic or nondopaminergic (Appendix). It is important to note that PD is a very heterogeneous disease with symptoms ranging from tremor dominant, akinetic-rigid, and postural instability gait disorder (PIGD) forms. Thus, the approach to treatment for each individual patient is tailored to his or her specific symptomatic profile and stage of disease. Given this, the lack of a stereotypic prescribing regimen in PD is appropriate. Typically, the dosing schedules for many of the medications are based on the patient's daily schedule of activities and the need to optimize function. Successful pharmacological management requires not only expert knowledge of PD, but also open communication between the physician and patient regarding symptomatic response and the individual needs of the patient. Therapists can assist in this process by encouraging patients to communicate any issues they are having with their physician and to remain compliant with their medication plan.

DOPAMINERGIC MEDICATIONS

The gold standard for symptomatic relief of PD is levodopa (L-dopa). L-Dopa is the immediate precursor of dopamine, and when taken orally, it crosses the blood-brain barrier and is converted to dopamine, the neurotransmitter that is depleted in PD. It is typically given in the carbidopa + L-dopa formulation. Carbidopa is an aromatic acid decarboxylase inhibitor that does not cross the blood brain barrier, and prevents the peripheral metabolism of L-Dopa. Using this combined formulation allows lower doses of L-dopa to be used, thus minimizing peripheral side effects such as nausea/vomiting. Medications that are most typically used are Sinemet and Stalevo. There are now long acting and short acting forms of these medications, helping to provide a longer duration of effective therapy with fewer motor fluctuations. In general, bradykinesia and rigidity respond best, while tremor can be resistant.

Individuals on dopaminergic therapy may complain of motor fluctuations. Some patients may have *wearing off*, during which they have return of their PD symptoms before their next dose is due. Patients may also experience *on/off* phenomena, where there is shifting between functioning and nonfunctioning states that may or may not predictably correlate to dosing of medication. Others may develop dyskinesias, or unwanted involuntary movements involving the head, shoulders, or limbs. These motor fluctuations are more typical after longer duration of use; and after 5 years of L-dopa therapy, up to 75% of patients have troublesome complications. It is not that L-dopa loses its effectiveness, but rather, since PD is a progressive neurodegenerative illness that gets worse with time, higher dosages are required to alleviate worsening symptoms. Of note, while L-dopa is typically the strongest and most effective medication for PD, there are no universally effective medications to treat balance and gait problems. This is where physical therapy is an invaluable resource. In clients who have significant motor impairments while *off*, the thera-

pist should help to identify strategies for safe mobility (ie, raised seat height, use of a rollator walker, use of auditory cues to facilitate movement). L-dopa does have side effects (Table 1), and in particular, therapists should be aware of its potential to cause orthostatic hypotension that can lead to falls. Blood pressure should be monitored during position changes. Therapists can play a key role in educating these patients about orthostatic hypotension and how to safely manage it and prevent falls. The elderly are more likely to suffer from the psychiatric side effects such as confusion and hallucinations. If you note any signs of new confusion in a client on this medication, you should immediately notify the physician. It should also be noted that patients with PD are at an increased risk for osteoporosis. This is particularly troubling given the high incidence of falls in this population. Extra diligence in providing therapy for fall prevention is recommended.

As Table 2 demonstrates, the time to onset and the duration of effect vary based upon the formulation of Sinemet. Therapists should be aware of this when scheduling therapy sessions as clients typically are better able to participate in therapeutic exercise when their medications are at peak effectiveness. If an individual is having difficulty with transfers, gait, or any activities of daily living due to wearing off, it is recommended that the therapist address these issues during the time when these problems are occurring. Compensatory strategies may be the only effective therapy, and choice of strategy is best determined if the patient is assessed during the time that they are exhibiting functional deficits.

Table 1. Side Effects of Dopaminergic Medications

Common	Adverse or Troubling
Dyskinesias	Agitation
Nausea	Confusion
Orthostatic hypotension	Delusions
Somnolence	Hallucinations
Vivid dreams	Psychosis
Can induce increase in homocysteine levels which can lead to osteoporosis	

Dopamine agonists (pramipexole, ropinirole, apomorphine, bromocriptine) work by directly stimulating the postsynaptic D2 receptors in the striatum. These medications can be used alone (monotherapy) or in conjunction with levodopa. They are often added to the therapeutic regimen to reduce the dosage of levodopa needed or to overcome the adverse effects of long-term levodopa therapy. Dopamine agonists (DA), when used as a monotherapy, have a low incidence of dyskinesias. There is also evidence that early use of the DA reduces time to develop motor complications and dyskinesias from chronic levodopa therapy.[8] Dopamine agonists are associated with fewer motor fluctuations, but generally provide less symptomatic relief than levodopa. Dopamine agonists can be effective in patients with advanced PD complicated by motor fluctuations and dyskinesia, but are usually ineffective in individuals who have no therapeutic response to levodopa. Given the higher incidence of dyskinesias in young onset PD, some experts suggest initiating DA in young onset patients, but starting older individuals on levodopa. Side effects are similar to levodopa though DA can cause peripheral edema, which is a rare side effect of levodopa (Appendix).

Dopaminergic dysregulation syndrome (DDS) is a problem that has been noted with dopaminergic agent use and appears to be more common with the use of DA. In one study, 3.4% of subjects with PD were found to have DDS.[9] This syndrome involves the compulsive use of dopaminergic drugs and is more common in young, male patients. These individuals will seek increasing doses of dopamine therapy and will be very reluctant to decrease their dosage, even in the face of significant dyskinesias or other side effects. Dopaminergic dysregulation syndrome can be associated with hypomania or manic psychosis and also with impulse control disorders. Impulse control disorders typically involve compulsive shopping, gambling, and hypersexuality. This side effect can have an extremely negative impact on quality of life and relationships with family members, especially as these patients can have a lack of insight into these problems. Therapists should consider asking questions in the history that would help to elicit information regarding the possible existence of impulse control disorders. If patients or their family members disclose the presence of this disorder, the therapist should acknowledge that this may be a medication side effect and notify the physician for follow up. Dopaminergic dysregulation syndrome and impulse control disorders are managed by striving to optimize symptom control with the lowest possible dose of dopamine.

Monoamine oxidase type B inhibitors (MAO-B) (selegiline and rasagiline) are mildly effective for symptomatic treatment of PD, but recent data suggests that rasagiline *may* be neuroprotective. Monoamine oxidase metabolizes dopamine, so the selective inhibition of monoamine oxidase allows for elevated levels of dopamine in the brain. The MAO-B inhibitors are not as effective as levodopa, but may delay time to when levodopa is needed and can help reduce the dosage of levodopa. Confusion is a frequent side effect in the elderly. Other common side effects are nausea, headache, and insomnia, particularly with selegiline.

Catechol –O–methyl transferase (COMT) inhibitors (entacapone) block the peripheral degradation of levodopa, thus prolonging its action. These medications are used to reduce *off* time and increase *on* time in levodopa treated patients, but are ineffective when given alone. Thus, they are given with each dose of levadopa. To simplify this, Stalevo is available, which is a formulation of carbidopa+levadopa+entacapone. Side effects are similar to the dopaminergic medications, however, 40% of

Table 2. Sinemet and Levodopa Formulations: Time to Onset and Duration of Effect

	Onset	Duration
Immediate Release (IR) 10 mg carbidopa/100 mg levodopa, 25/100, 25/250	20-40 mins.	2-4 hours
Controlled Release (CR) 25/100, 50/200	30-60 mins.	3-6 hours
"Liquid Levodopa" (dissolved tablets)	10-20 mins.	0.5-1 hour

patients will experience an orange urine discoloration.

NONDOPAMINERGIC MEDICATIONS

Prior to the discovery of levodopa the only available medications to treat PD were anticholinergics (benzatropine and trihexiphenidyl). Acetylcholine and dopamine are usually in electrochemical balance in the basal ganglia. In PD, dopamine is depleted; therefore, the action of acetylcholine becomes exaggerated. Anticholinergics decrease the activity of acetylcholine, thus helping to achieve the critical dopamine-acetylcholine balance. They can be particularly effective in the treatment of tremor, thus mild tremor dominant PD may be initially treated with an anticholinergic. Side effects include blurred vision, dry mouth, sedation, constipation, cognitive decline, and urinary retention. Anticholinergics may be contraindicated in older patients because they can cause confusion and hallucinations. They may have negative interactions with anti-histamines, anti-psychotics, amantadine, and alcohol.

Another nondopaminergic option is amantadine. This drug was originally introduced as an antiviral agent against the flu, and was surreptitiously noted to be useful in relieving PD symptoms in 1969. The mechanism of action in PD is unclear, but likely it augments dopamine release and may block reuptake. It is has a mild symptomatic effect, however, it is the only anti-parkinsonian drug that can improve levodopa induced dyskinesia. This effect is usually short-lived. Confusion, constipation, ankle edema, and livedo reticularis (lace-like purplish discoloration of the knees and ankles) are common side effects. Patients on both DA and amantadine may develop

Appendix. Medications Used to Manage Parkinson's Disease

Class	Drugs	Treatment Mechanism	Comments	Side Effects	More serious side effects and Adverse events
Levadopa	Carbidopa-levodopa (Sinemet, Parcopa) Carbidopa, entacapone, and levodopa (Stalevo)	Replaces dopamine in the brain	There is no evidence that chronic administration of levodopa exacerbates the degenerative process in PD.	Dizziness Dyskinesias Headache Nausea Orthostatic hypotension Somnolence	Agitation Confusion Delusions Hallucinations Psychosis Can increase homocysteine levels that can lead to osteoporosis
Dopamine Agonists	Pramipexole (Mirapex, Mirapex ER) Ropinirole (Requip, Requip XL) Apomorphine Bromocriptine	Directly stimulates dopamine receptors	Associated with fewer motor fluctuations	Nausea Orthostatic hypotension Peripheral edema Sleep attacks Somnolence Vomiting	Impulse control disorders Confusion Hallucinations Bromocriptine and pergolide can lead to valvular heart disease
MAO B inhibitors	Selegiline (eldepryl) Rasagiline (azilect)	Slows breakdown of dopamine	Rasagiline may be neuro-protective thus is often prescribed in early disease	Headache Insomnia Nausea	Confusion (elderly) Rarely concomitant use of tricyclic antidepressants or SSRIs has led to serious adverse reactions
COMT inhibitors	Tolcapone (tasmar) Entacapone (Comtan)	Levadopa extenders Reduce methylation of levodopa and dopamine that increases plasma half life of levodopa	Used to treat patients with motor fluctuations who are experiencing end of dose wearing off periods	Dyskinesia Nausea Orthostatic hypotension	Confusion Hallucinations
Anticholinergics	Benzatropine (Cogentin) Trihexiphenidyl (Artane)	Reduce acetylcholine and help restore balance between dopamine and acetylcholine	Symptomatic relief of tremors	Blurred vision Constipation Dry mouth Sedation Urinary retention	Cognitive decline Hallucinations
Anti-viral agent	Amantadine	Increases dopamine release, inhibits uptake and stimulates dopamine receptors	Mild antiparkinsonian effect; can help with dyskinesias	Constipation, Dry mouth, **constipation** Livedo reticularis (knees, ankles) Peripheral edema	Confusion Glaucoma

peripheral edema (Appendix). Therapists should monitor patients' ankles and calves and note the appearance of this edema. We cannot ameliorate the edema, but can help clients understand its origin and teach strategies such as elevation and pressure stockings, and look for any adverse effects on function or the integumentary system. The physician should be notified if the edema is significant or having a negative impact on the patient.

A great deal of research efforts are now being placed on finding neuroprotective agents for PD. Currently, there are no medications that can slow down or halt the progression of this disease. Clinical trial data has suggested that rasagiline 1 mg daily may be neuroprotective, however this is not conclusive.[10] Ideally, dopaminergic cells in the substantia nigra can be protected from the degenerative processes associated with PD; however, this is proving to be complicated as the mechanisms involved in dopaminergic neuron loss and the presence of Lewy Bodies (both pathologic hallmarks of PD) are not well-understood. Once effective neuroprotective agents are identified they could be used in patients with early disease to prevent the onset of debilitating symptoms, assuming they are well-tolerated with minimal side effects. Some agents that are currently being investigated for neuroprotective effect include CoQ10, MAO-B inhibitors, glutamate antagonists, creatine, and isradipine. CoQ10 and creatine are being investigated due to their impact on mitochondrial dysfunction.[8]

SUMMARY

Pharmacologic management of PD is complex and requires careful communication between the patient and the physician. Physical therapists play a key role in facilitating this process by helping to monitor response to medications as well as the occurrence of unwanted side effects. The results of physical therapy based assessments of motor impairments and function should be communicated to the physician in a timely manner so that they can be used in making treatment decisions. Physical therapists who work with the PD population need to have an understanding of the pharmacological management of this complex disease in order to provide safe and effective care.

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NUTRITION AND PARKINSON'S DISEASE

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As the population ages there are an ever increasing number of older people afflicted with neurodegenerative diseases such as Parkinson's disease (PD). The pathogenesis of PD is unknown, although many environmental and lifestyle factors may play a causal role in the development of this devastating malady. More and more research suggests that nutrition may be a means of preventing PD. Evidence promotes various nutrients as a means of treating PD. Though the epidemiological evidence for an association between dietary agents and lifelong exposure to specific food elements is not conclusive, recent data supports the association between nutrient components and the prevention, etiology, and treatment of PD.[1]

A number of different theories have been developed involving the role of nutrients in the prevention and symptom management of this disease. Nutritional requisites have been found to be helpful in managing symptoms such as tremors and rigidity. The purpose of this article, written for this special issue of *GeriNotes*, is to explore the literature currently available specific to nutrition and Parkinson's disease. The intent of this review is to examine the literature to determine the role that nutrition plays in the prevention, development, evolution, and treatment of PD.

THE ROLE OF NUTRITION IN NEUROPROTECTION

The etiology of PD, the second most common movement disorder in older adults, is unclear. A genetic vulnerability, even in idiopathic PD seems likely. Additional factors like endo- and exotoxins are proposed to contribute to the induction and in some cases possibly the acceleration of the pathology. Among the epidemiological risk factors, dietary components are broadly discussed. Moreover, there is a growing awareness concerning the possibility of preventive dietary habits.[2] However, dietary factors are difficult to assess. This review gives an overview on epidemiological studies addressing a possible relation of dietary compounds and the risk for PD.

Omega-3 (n-3) polyunsaturated fatty acids may exert a neuroprotective effect in PD. Recent work in animal models of PD supports the contention that n-3 polyunsaturated fatty acids found in fatty fish are neuroprotective. Moreover, a few epidemiological reports suggest that a high intake of fish is associated with a lower risk of developing PD.[3] Other data suggest that n-3 polyunsaturated fatty acids prevent the decrease in dopamine associated with the development of PD.[4]

Amino acids derived from protein sources are converted into dopamine. There is evidence that, due to nutritional deficits--particularly the ***antioxidants***, that the provision and breakdown of these proteins is not adequate. This leads to a decrease in dopamine production over a long period of time. It is thought that the lack of dopamine in the brain results in the destructive free radical damage seen in this disease and that antioxidants may play a protective role in correcting this metabolic deficit and ultimately in the prevention and treatment of PD. In combination with low intake of omega-3 polyunsaturated fatty acids, the amalgamation of these two dietary situations sets the stage for neurodegeneration. With the direct effect on dopamine production, PD is the likely consequence.

Certain antioxidants are important in the maintenance of neurological cell health. ***Coenzyme Q10*** allows cells to produce energy, ***Vitamin B3*** is important in the creation and transfer of chemical energy, ***Vitamin B6*** is essential for normal brain and nervous system functioning. It has been determined that concentrations of B6 may result in improved production of essential neurotransmitters. ***Coenzyme Q-10, Vitamin E,*** and ***Vitamin C*** are considered powerful antioxidants that help protect brain cells. ***L-Methionine*** supplies sulfur to the body for the synthesis of ***glutathione***, another important antioxidant in the body. ***L-Tyrosine*** has effects on neurotransmitters that may affect several health conditions including PD. ***Vitamin A*** and ***Beta-carotene*** contributes to the production of the neurotransmitter acetylcholine, which is required for normal neurological function and neurotransmission.

Oxidative stress, selective neuronal loss, and diminished activity of thiamine-dependent enzymes play a role in many neurodegenerative diseases, including PD. ***Thiamine*** deficiency was found to reduce the voltage-dependent K+ membrane conductance, leading to decrease in functional efficiency, disuse, and ultimately neuronal death.[5] Thiamine appears to be a powerful neuroprotector.

Herbal substances, such as ***Ginkgo Biloba*** have been found to have the potential of maintaining circulation to the brain. Increased ***Phosphatidyleserine*** (soy) plays a role in neurotransmission, may support brain function and boosts memory.

NUTRIENT INTAKE: A RISK FACTOR FOR PARKINSON'S DISEASE?

There is a suggested association of PD with high intake of ***total fat, saturated fats, cholesterol, lutein*** (related to beta carotene and vitamin A) and ***iron***.[6] Powers study evaluated PD risk and dietary intake of fats, cholesterol, and iron. A low intake of cholesterol, particularly in the presence of high iron, was associated with an increased risk for PD.[7]

Oxidative stress is an important mechanism of cell death in PD and brain ischemia. ***Vitamins C, E,*** and ***A*** are important antioxidants and deficiency of these agents has been implicated in the mechanisms of atherosclerosis. Vitamins C and E were found to be deficient in PD leading to vascular changes and increasing the rate to decline of functional abilities.[8] This clearly emphasizes the necessity of maintaining sufficient dietary intake of these powerful antioxidants.

Manganese (Mn) is an essential mineral. It is essential for smooth functioning of the brain as it helps to balance the neuroendocrines and is needed in the production of enzymes. Mn is present in virtually all diets at low concentrations. Sources of Mn include: Tea, wheatgerm, spinach, split peas, nuts, oatgerm, oat-

meal, pineapple, and green leafy vegetables. The principle route of intake is through food consumption, though inhalation exposure may occur in various occupational cohorts. For the most part, humans maintain stable tissue levels of Mn. Excessive levels of Mn results in adverse neurological effects which resemble PD symptoms of tremor, rigidity, excessive sweating, and the like. The research in this area suggests that high concentrations of Mn resulting from excessive dietary exposure may increase the risk for PD.[9]

Calcium has been implicated in the development of PD. Core motor symptoms in PD are attributable to the degeneration of dopamine neurons in the substantia nigra. Recent work has revealed that there is an inflammatory process that occurs in the ***L-type calcium [Ca(2+)]*** channels in the substantia nigra making dopamine neurons susceptible to mitochondrial toxins. There is an increased reliance on Ca(2+) that leads to sustained metabolic stress, inflammation, and death. This indicates that homeostatic calcium stress could be a determinant of the selective vulnerability of these neurons in PD. The influx of calcium into the neuron leads to a metabolic imbalance that renders the cell unable to produce dopamine and exacerbates cellular death.[10] Providing a calcium channel blocker has been found to decrease these negative forces on dopaminergic neurons and provide a neuroprotective strategy for the treatment of PD.

Diary product consumption was positively associated with risk of PD, particularly in men.[11] The mechanism for this are not clearly understood, though it is postulated that the combination of increased ***saturated fats, cholesterol,*** and excessive amounts of ***calcium*** may explain this risk.

Interestingly, ***caffeine*** consumption was inversely associated with PD in men, but not women. In other words, these results suggest that caffeine reduces the risk of PD but that this hypothetical beneficial effect may be prevented by the presence of estrogen, especially in women on hormone replacement therapies.[12] Data from Asian populations on dietary and lifestyle factors associated with PD have examined various risks in individuals with PD.[13] Current versus never smokers exhibited a reduced risk of PD. Total caffeine intake was inversely related to PD risk. Green tea drinking was unrelated to PD risk and ingredients of black tea other than caffeine were suggested to be responsible for the beverage's inverse association with PD in the Singapore Chinese Health Study. In other words, the jury is still out relative to the role that caffeine plays.

NUTRITIONAL CONSIDERATIONS IN THE TREATMENT OF PARKINSON'S DISEASE

The early treatment of PD consists of nonpharmacologic treatment, consideration of neuroprotective therapy, and initial symptomatic treatment. Education for the patient and family, access to support groups, regular exercise, and good nutrition are essential to the overall management of PD. Disease-modifying therapies, such as agents that provide neuro-rescue or neuroprotection, will provide a major advance in the treatment of PD.[14] Interventions at the genetic/environmental level or that affects the cascade of pathophysiologic events, protein aggregation, or apoptosis could result in neuroprotection. Many nutritional and pharmacological agents are being investigated for neuroprotective potential. A major paradigm shift has recently occurred because of the recent basic and clinical data indicating that dopamine agonists, rather than levodopa, should be the initial symptomatic therapy in PD. Nonetheless, levodopa may be started first in some patients because of patients' age, cognitive status, or cost of drugs.

Diet is particularly important for people who are on Sinemet or levodopa. Sinemet or levodopa can interact with food at two locations, the gut and the brain. Food in the stomach can delay absorption of levodopa into the blood. If levodopa is taken on a full stomach, the food must first be digested before it enters the intestines. Levodopa is absorbed across the intestinal wall into the bloodstream. Ideally, levodopa or sinemet should be taken on an empty stomach. The drugs may actually be ineffective if taken with food. At the level of the brain, protein can interfere with levodopa. This happens because levodopa uses the same molecules as protein to be carried into the brain. Amino acids of protein take the places that would otherwise be occupied by levodopa on the molecule. A low protein diet is recommended. The RDA for protein is .8 grams/kg or .4 grams/pound of body weight. The recommendation is to maintain this RDA, but not to exceed it.

The interest in a protein redistribution diet, also called daytime protein restriction diet, has increased among patients with PD. Since certain amino acids compete with L-dopa in the intestine and at the blood-brain barrier, daytime protein restriction may improve fluctuations in motor ability.[15] However, this diet can contribute to weight loss, nutrient deficiencies and cause cognitive disabilities if the diet is not correctly observed. Studies are currently in process to clarify how medication with levadopa in combination with different diets (relative contributions of protein, fat and carbohydrate) may affect motor fluctuations, nutritional status, and cognitive ability.

Individuals with PD appear to be at greater nutritional risk for malnutrition than a matched population.[16] It appears that PD leads to poor metabolism, absorption, and utilization of specific nutrients leading to deficits and cachexia (loss of appetite, weight loss, muscular wasting, and general mental and physical debilitation). Simple screening and assessment tools can be used to detect nutritional risk for malnutrition and other nutrient deficits. Registered dieticians play a key role in helping patients with PD to optimize their nutritional status and manage various nutrition-related symptoms and medication side-effects. It is important that the physical therapist recognize nutritional deficits and their consequences and refer the patient to the dietician for strategies in managing a variety of nutrition-related symptoms.[17,18]

To monitor the nutritional status of patients with tools that are easily employed is crucial as a comprehensive approach to treatment in PD. The Mini Nutritional Assessment (MNA) questionnaire is a valid and reliable tool for the evaluation of nutritional status.[19]

Parkinson's patients are at a higher risk for nutritional deficits as a result of a swallowing dysfunction manifested by dysphagia and delayed gastric emptying problems.[20] As PD progresses, patients experience variable levels of dysphagia with or without aspiration. Etiological factors include the manifestation of the underlying neurologic disease, a gap in anti-parkinsonian medications, the use of metoclopramide (an antiemetic and gastroprokinetic agent - primarily

used to treat nausea and vomiting, and to facilitate gastric emptying in patients with gastroparesis), and/or postoperative medical complications leading to a debilitated clinical state.

Low body mass index (BMI) is associated with low bone mineral density increasing the risk for osteoporosis. Possible determinants of weight loss in PD include poor metabolic efficiency with decreased movement/activity, impaired hand-month coordination, difficulty chewing, dysphagia, intestinal hypomotility, depression, decreased reward processing of dopaminergic mesolimbic (intermediate areas) regions, nausea, and anorexia as the side effects of medication, and increased energy requirements due to muscular rigidity and involuntary movements.[21] Patients should be provided with sufficient amounts (RDA) of vitamin D and calcium to reduce the risk of hip fractures and strengthen bone density.

Nutritional status was assessed in patients with PD.[22] Weight loss since the onset of disease occurred in 52% of the patients and 22% lost more than 12.8kg. Although 67% experienced eating difficulties of some kind, dietary intakes of protein and energy were adequate. Plasma levels of albumin, vitamin A, vitamin E, iron and zinc were found to be significantly lower compared with healthy age-matched controls. The significance of low levels of the antioxidants and zinc are most likely related to oxidative stress in the pathogenesis of this disease.

Aggressive nutritional intervention has become a cornerstone of treatment for many patients with neuromuscular disease. Malnutrition is a common problem among patients with PD. Recognition of nutrition as an independent, prognostic factor for survival and disease complications in PD has illustrated the importance of individualized nutritional management in symptomatic treatment. Common practice includes the inclusion of dietary supplements and the use of nutraceuticals and functional foods in the treatment and prevention of PD and other neuromuscular diseases in the early stages.[23] Nutritional treatment includes caloric supplementation, the diagnosis/ treatment of dysphagia and supplements to enhance neuromuscular functioning. Evidence supporting the efficacy of dietary supplements in PD continues to support the notion that nutrition may be a way to intervene without the side effects inherent in drug use.[24]

Omega-3 polyunsaturated fatty acids were found to normalize cerebral excitability and reduce the psychological side effects of anxiousness, psychosis, and dementia in PD. It is suggested in this study that the priority in nutritional care and food provision in neurodegenerative diseases should be to achieve a high n-3 protein, nutrient rich diet to facilitate the production of dopamine and reduce cognitive symptoms.[25]

The free radical (eg, oxidative stress) process has been linked to the death of dopaminergic cells in PD.[26] The ability of the ***antioxidants curcumin*** and ***naringenin*** to exhibit neuroprotection may be related to their antioxidant capabilities and their capability to penetrate in the brain (eg, passing the blood-brain barrier).

Vitamin E is essential for neurological function.[27] This fact, together with a growing body of evidence indicating that neurodegenerative processes are associated with oxidative stress, lead to the convincing idea that PD and some forms of parkinsonism syndromes may be prevented and/or treated by the antioxidant properties of vitamin E. In a study the efficacy of supplementation with vitamin E determined the number of cells in the substanita nigra with lesions dropped significantly with intramuscular injection of this nutrient. In addition, functional tests indicated a reduction in contraversive and ipsiversive rigidity and resulting characteristic motor patterns. Repeated intramuscular administration of vitamin E exerted a rapid protective effect on the dopaminergic neurons in early PD.[28]

Inflammatory processes and vascular dysfunctions appear to play important roles in the pathogenesis of age-associated pathologies such as PD. A large body of evidence shows that both ***vitamin E and C*** are important in the central nervous system and that a decrease in their concentrations causes structural and functional damage to the cells. Several studies link diets rich in fruits and vegetables containing generous amounts of vitamins E and C and a lower incidence of PD.[29]

Coenzyme Q10 is a powerful antioxidant that boosts energy and enhances the immune system. In PD it is thought to repair a defect in the powerhouse of the cell (mitochondria). The safety and tolerability of high dosages of coenzyme Q10 has been studied in patients with PD. High doses (ie, 1000-3000 mg/day) of Q10 were found to be well tolerated as long as vitamin E was also provided as a complimentary antioxidant.[30] Coenzyme Q10 has also been shown to slow the functional decline at lower dosages.[31] At dosages of up to 1200 mg/day compared with a placebo group, Shults and colleagues demonstrated the Parkinson's subjects on the supplement developed less disability and the benefit was greatest in those receiving the highest dose. Coenzyme Q10 appears to slow the progressive deterioration of function in PD.

There is no cure or proven strategy for slowing the progression of PD. Although there are signs of pathology in many brain regions, the core symptoms of PD are attributable to the degeneration of dopaminergic neurons. This is a potential clue to the vulnerability of these neurons in their increasing reliance on ***Ca(2+)*** channels to maintain autonomous activity with age.[32] This reliance produces a sustained metabolic stress on mitochondria that accelerate cellular aging and death. The management of calcium's effects may be instrumental in the interventions for neuroprotection against the symptoms and progression of PD. The recommendation is to maintain the RDA of 1200 – 1500 mg/day of calcium in the diet or via supplement, but not to exceed this level due to calcium's effects on dopaminergic neurons.

NUTRITIONAL CONCERNS FOR PEOPLE WITH PARKINSON'S DISEASE

Navigating the maze of nutrition information and advice available to the public is challenging, even for a healthy consumer or medical professional. Add PD to the mix and the challenges spiral higher. The nutritional issues faced by people with PD are complex and diverse, and many of the issues do not have clear answers. When it comes to nutrition, what matters most?

It is difficult for a person to feel well and maintain energy when he or she is not eating properly. Eating properly involves eating regularly (no meal skipping), eating a variety of foods from all of the food groups (grains, vegetables, fruit, milk/ dairy, meat/beans), and eating prudently to maintain a healthy weight. Although this sounds like simple advice,

implementing it can be a challenge, particularly if life is hectic or if the symptoms of Parkinsons affect the ability to shop, prepare food, and eat. Depression can interfere with appetite and is also a consideration that needs to be addressed in rehabilitation. Poor nutrition will lead to poor functional abilities and reduced stamina. Unexplained weight loss may also occur and may be considered a nutritional risk factor if weight loss of 10% or more of usual body weight occurs.

Should PD patients take supplements? Dieticians recommend that people with PD take a well balanced multivitamin and eat a balanced diet. Good nutrition can be difficult in light of the functional problems inherent in PD, so a multivitamin is protective for nutritional deficits. The other supplement recommendation is calcium. Both women and men should have 1200 – 1500 mg/day of calcium.

Constipation, urinary tract infections (UTIs), thinning bones, and unexplained weight loss are all common in persons with PD. Preventing or managing these conditions can be accomplished through proper hydration and nutritional intake.

Constipation is common in PD. This can be an embarrassing issue to raise with health care providers and physical therapists do not typically ask PD patients about bowel or bladder function. Prevention and treatment of constipation is critical, as severe constipation can lead to bowel obstruction, a potentially life-threatening condition. Although the constipation observed in PD is due in large part to the disease itself, lifestyle measures can be useful for managing it. These include eating foods high in fiber (whole grain bread, bran cereals or muffins, fruits and vegetables, beans and legumes, and prunes) and drinking plenty of fluid. Then there is exercise, which helps maintain bone density as well as eases constipation.

Proper hydration, which is achieved by drinking plenty of fluids, is important in the prevention of constipation and UTIs. Fluid replacement is important, especially when participating in physical activity. The patient should be instructed to drink fluids throughout the day--ideally water. Consuming fruits and vegetables, foods that are naturally high in water content, can also increase daily fluid intake. Side effects of anti-Parkinson medications or anticholinergic agents (ie, Cogentin, Artane) may include dry mouth, feelings of thirst, thick or sticky saliva, dry eyes, and constipation. It is important to consume adequate fluids; again, preferably water, when taking medications. The goal of 8 glasses per day (~2 liters/day) of water in addition to any other fluids normally consumed within the course of the day (such as juice, milk, or coffee) is a good aim. Adequate fiber in any healthy diet in combination with adequate water helps to prevent the risk of constipation and dehydration.

Persons with PD are also at risk for thinning bones and need to consider adequate nutritional intake to promote strengthening of bones and maintenance of bone density. This intake should include foods containing micronutrients such as calcium, magnesium, vitamin D, and vitamin K. A variety of foods need to be consumed, and incorporate foods high in micronutrients, such as dairy products (ie, low-fat versions of milk, cheese, and/or yogurt), beans, grains, and nuts. Vitamin D maintains calcium blood levels in the body within normal limits, and is crucial for adequate absorption of calcium from the blood stream. If adequate calcium is not available, the body begins to break down bone in order to supply the needed nutrient. Often, vitamin D needs are not met by dietary methods alone. Recent research indicates that limited exposure to the sun during the spring, summer, and fall of 5 to 15 minutes per day (between 10 a.m. and 3 p.m.) will provide the body with the current recommended amount of vitamin D. Allow some sun exposure to hands, arms, and face for a few minutes each day to total 60 minutes per week. In the absence of any sun exposure, it is important to increase daily intake of vitamin D-fortified foods such as milk and orange juice (products fortified in vitamin D indicate this on their containers). Foods that naturally contain vitamin D include liver, eggs, and fatty fish (i.e., salmon). Healthy sun exposure and adequate nutritional intake may maximize a person's vitamin D status to promote good health.[33]

People with PD are prone to osteoporosis due to low bone-mineral density. Risk factors for osteoporosis include older age, low body weight, smoking, excessive alcohol intake, limited exposure to sunlight, inadequate intake of vitamin D and calcium, and lack of weight-bearing exercise. Osteoporosis can be especially worrisome to a person with PD who faces an increased risk of falling. The inevitable result is an increased risk of fractures. Bone-mineral density needs to be monitored on a regular basis.

The medications used for PD can themselves cause nutrition-related side-effects, such as nausea and poor appetite. Typically these side-effects are most severe when a medication is first prescribed but some individuals have continuing problems with them. Taking a small snack (such as ginger ale and a few crackers) along with medications may help to control these side-effects. If nausea or poor appetite persist, these symptoms can lead to weight loss.

Amino acids (from dietary protein) can interfere with the uptake of levodopa into the brain. Though not everyone experiences this, eating high-protein food (such as meat, fish, poultry and dairy products) decreases the effectiveness of levodopa. Dieticians recommend keeping the meat portion of a meal to about the size of a deck of cards and take levodopa or Sinemet® prior to a protein-containing meal (at least 30 minutes before a meal). Usually it is the timing of protein intake, not its total quantity over the course of the day that is the problem, so though restricted protein diets are sometimes recommended, most dieticians suggest that this may only lead to an increased risk of undesired weight loss.

To summarize – what is important for PD and nutrition?

- A balanced diet with all of the daily nutritional requirements.
- Maintenance of bowel regularity.
- Drink plenty of fluids during the day to keep hydrated, which helps to prevent constipation.
- Maintenance of bone health.
- Balance medications and food.
- Adjust nutritional priorities for each patient's situation and stage of disease.
- If progressive weight loss occurs, special attention to adequate calorie consumption is very important.
- Extra fresh fruits and vegetables provide fiber that will help or prevent constipation.
- Limiting protein intake or stagger-

ing the levodopa dosing to avoid conflicts with meals can help solve this problem.
- Take medications before mealtimes on an empty stomach.
- For people who have swallowing difficulties, a diet of soft foods may be recommended.

Do "wonder" foods or supplements delay progression of Parkinson disease?

Supplements (both nutritional and herbal) and dietary therapies are high on the list of complementary therapies used by people with PD. In spite of compelling theories about the effectiveness of various supplements or dietary factors in delaying progression of the disease, we lack definitive, evidence-based answers. Some therapies have been studied only in test tubes or with laboratory animals. Few human trials have been done (eg, those examining antioxidant vitamin supplements), and most have produced disappointing results. Coenzyme Q10 is one nutritional supplement that is of considerable interest to the scientific community and is under study to determine if it has any potential benefit in PD.

Some foods that are in the "won't hurt and might help" (at least in theory) category include coffee (several population studies have suggested that coffee may be protective against Parkinson's, particularly in men), green tea, a variety of fruits and vegetables, foods rich in vitamin E such as wheat germ, nuts and seeds, and vegetable oil. If the antioxidants present do not help with PD symptoms, they may help with some other aspect of health so there is certainly no reason not to use them.

When thinking about the potential value of using this or that supplement, consider the factors of cost, safety, and effectiveness and be sure not to be "taken in" by hyped headlines. For example, a recent headline read, *Vitamin B6 May Cut Risk of Parkinson's Disease*. Behind the headline: this study finding, while interesting, was only observed among smokers and the study addressed only the onset, and not the progression, of PD.

Parkinson's disease symptoms vary from person to person and by stage of disease. Each person must set nutritional priorities based on the issues they face. In early PD, we should all emphasize eating well and maintaining a healthy weight. As the disease progresses, adjustment of a patient's diets to manage specific new symptoms as they emerge (such as swallowing difficulties, medication side-effects, bowel issues and eating challenges) needs to be considered. The goal of thoughtful nutrition is not just to ease PD symptoms; it is also to allow the PD patient to continue to use food as a source of pleasure in life.

SUMMARY

As discussion turns to the prevention of disease, how early stages of life predispose people to adult diseases has emerged as a research focus. Recent research of epidemiology of disease factors looks at early-life behaviors such as poor nutrition, reduced activity levels, the way we handle stress, exposure to infection, and environmental factors, such as chemical and pesticide exposure, to determine their roles in neurodenenerative disease development? Could nutrition be linked to PD and if so, is it possible to modify the diet to influence the course of this pathology? The importance of saturated fats, cholesterol, obesity, inactivity, and other health behaviors have been established as risk factors that result in the increase incidence of all disease. In PD research now leans toward determining the relative contributions of genetic and environmental factors, determining when during the life course that a given exposure has its greatest effect and how exposures may accumulate over the life span.[34] The data for PD suggest that a number of insults related to nutrition over time may contribute to establishing this disease.

Approximately 1.5 million Americans have PD, and more than 60% of them use nutritional supplements and alternative therapies. Conventional management of PD is limited. The pharmaceutical and surgical options that are available have significant side effects and only correct symptoms for a limited period of time. Even with the best conventional treatment, the disease progresses and becomes severely disabling. No existing conventional therapy halts the progress of the disease; available medicines only treat symptoms temporarily. Conventional medicine views the course of the disease as *progressive* and *irreversible*. Nutrition has the potential to be an alternative, complementary option in an attempt to prevent, slow, stop, or reverse the PD process. More and more research on the relationship between nutrition and Parkinson's disease provides much hope for the future.

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(President's Perspective continued from page 3)

DOMAIN #6: Health Care Systems and Benefits

1. Serve as an advocate for older adults and caregivers within various health care systems and settings.
2. Know how to access, and share with older adults and their caregivers, information about the health care benefits of programs such as Medicare, Medicaid, Veterans' Services, Social Security, and other public programs.
3. Provide information to older adults and their caregivers about the continuum of long-term care services and supports--such as community resources, home care, assisted living facilities, hospitals, nursing facilities, subacute care facilities, and hospice care.

The Section's Retooling Taskforce will be doing further work on implementation of these competencies by the physical therapy profession, including the development of additional physical therapy-specific competencies. However, both new graduates and experienced physical therapists and physical therapist assistants are encouraged to review these competencies as a means of self-assessment and as a stimulus for professional development. The complete PHA competencies document, including further background information and related definitions, can be found at www.geriatricspt.org

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EFFECT OF GROUP EDUCATION AND INDIVIDUAL REHABILITATION ON PATIENTS WITH IDIOPATHIC PARKINSON'S DISEASE

Mary Vollman, PT

INTRODUCTION

The purpose of this article is to review literature pertaining to the effect of group education and individual rehabilitation on patients with idiopathic Parkinson's disease (PD).

METHOD

A search was conducted in Pub Med under clinical queries for therapy using the search terms "Parkinson's disease AND physical therapy AND patient education" with a narrow/specific filter. This yielded two studies, the first of which was the Guo[11] article. The APTA Hooked on Evidence database was also searched under the neuromuscular clinical scenario, specific to the condition of PD, and further narrowed to older adult, Hoehn and Yahr Stage 2, randomized controlled trials only. This yielded the same Guo et al article.

RESULTS

The Guo et al article was selected for review because it is a randomized controlled trial in which authors sought to determine the effect of group education and individual rehabilitation on heath related quality of life issues in people with idiopathic PD.

The study design was a randomized controlled trial, with masked assessors and intention-to-treat analysis was not provided.

The Participants

The authors originally identified 68 potential participants from an electronic database in the Specialized Idiopathic Parkinson's Disease Outpatient Clinic in Houshan Hospital affiliated with Fudan University. The inclusion criteria for participation was: a diagnosis of idiopathic PD in Hoehn and Yahr Stages 1, 2, or 3 (confirmed by two neurologists), stable drug usage without long-lasting or significant off periods, and an ability to independently keep a diary and visit the hospital using public transportation. Participants were excluded for the following reasons: secondary PD; multiple system atrophy, corticobasal degeneration, significant cognitive impairment (Mini-Mental State Examination of 26 or less), psychiatric drug reactions, chronic heart disease, osteoporosis, other significant co-morbidities, or refusal of a visit. Forty-five individuals were found to be eligible to participate in the study and signed consent forms; however, one individual was disqualified because of a family emergency. This resulted in 44 participants for randomization. Participants were randomized (N=44) via a computer generated series to either intervention (N=23) or control (N=21) groups after enrollment, informed consent, and baseline assessment.

The intervention group (N=23) received 3 group lectures and up to 24 individual rehabilitation sessions over the course of 8 weeks. Group lectures were based on participant preference and determined by telephone interview of the original 68 potential participants. Topic choices included diet, rehabilitation, mental health, and advances in medicine. The 3 participant preferred topics for group education were moving (rehabilitation), meals (diet), and mood (mental health). The group education intervention consisted of a total of 3 sessions, with one session for each domain. The sessions were scheduled to last for approximately 45 minutes each, and were designed to be interactive rather than a formal lecture format. The educational information was supplemented with Internet information and support was provided for participants to access the Internet. Participants experiencing difficulty with tremor or tone were instructed in methods of resetting the mouse, stylus, and keyboard as needed.

Individual rehabilitation sessions consisted of 24 one-half hour sessions over 8 weeks. Sessions were designed to address specific movement deficiencies and were conducted by physical and occupational therapists. Some of the treatments included cued (visual, auditory, and tactile) exercises, balance training, high intensity resistance training, body weight supported treadmill training, and active music therapy.

The control group (N=21) received baseline assessment and final assessment. At the end of the trial the control group received an educational lecture and a comprehensive rehabilitation session.

Validity authors protected from threats to validity through randomization and reported baseline homogeneity of the study sample. Assessors were masked to protect from ascertainment bias. Some threats to validity include no intention-to-treat analysis for drop-outs, and treating professionals were not blinded, possibly resulting in supplemental care bias. Authors also report a lack of a true control group and no long term follow up to be limitations of the study. Additional studies addressing these issues are needed. Overall these threats seem minor and pose minimal risk to validity.

RESULTS

Authors reported statistically significant differences between the control and intervention groups for the outcome variables of the PDQ-39, the UPDRS II, UPDRS III, and patient reported mood status at 8 weeks. The difference between the means for the PDQ-39 at 8 weeks was 17 (95% CI=14.47-19.92). Because the bands of the confidence interval are narrow and do not cross 0, we can be confident in the results. It seems probable that a client with idiopathic PD would consider a 17-point improvement in the PDQ-39 score to be clinically important.

CONCLUSION

The results of this study demonstrate the value of a collaborative and client centered approach to a tertiary preventative intervention for clients with idiopathic PD. Group education combined with individual rehabilitation is an efficient and effective intervention that resulted in statistically and clinically important differences in health-related quality of life issues for clients with idiopathic Parkinson's disease.

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Table 1. Group Education Sessions

TOPIC	INSTRUCTOR	SPECIFIC INTERVENTION TOPICS
Meals	Registered Dietician	Protein drinks, fiber, calcium, vitamin D, H_2O
Mood	Psychologist	Evaluations, medications, talk therapy
Moving	Physical and Occupational Therapists	Standard and tailored programs with preparation for home and community exercise

Table 2. The Outcome

OUTCOME	MEASURE	SCORE
HR-QOL PDQ-39	Mobility, ADLs, emotional well being, communication, cognition, stigma, bodily discomfort, social support	Lower scores=better perceived health status
UPDRS II ADL	Dependency for ADLs	Higher scores =greater disability
UPDRS III motor exam	Progression of motor symptoms	Higher scores =more severe symptoms
SEAD (Schwab and England Activities of Daily Living)	Self-rating ADL status	Higher scores =greater independence
Zung Self-Rating Depression Scale (SDS)	Self-report related to symptoms associated with depression	Higher scores indicate more severe depression
Patient Mood Status (PMS)	Self assessment of mood	Higher scores indicate more optimistic mood

Table 3. The Evidence at 8 Weeks

Outcome	Intervention Group	Control Group	P value
PDQ-39	25.4 (SD=4.0)	42.5 (SD=4.2)	.001
UPDRS II ADL	5.5 (SD=1.3)	13.1 (SD=1.3)	.001
UPDRS III Motor	15.8(SD=2.5)	29.3 (SD=2.7)	.001
SEADL	.8(SD=. 2)	.8(SD=.2)	Not significant
Zung (SDS)	24.8(SD=1.2)	24.8(SD=1.6)	Not significant
Patient Mood Status (PMS)	58.4(SD=2.4)	39.3(SD=2.5)	.001

Table 4. PDQ-39 Difference of the Means at 8 Weeks

Measure @8 weeks	Control N=19		Experimental N=2l		Difference of unadjusted means	95%CI
PDQ-39 (Lower scores indicate better perceived health status)	Mean	SD	Mean	SD	17.1	14.47-19.73
	42.5	4.2	25.4	4		

AMPLIFICATION OF FALL RISK IN PARKINSON'S DISEASE: THE INFLUENCE OF COMORBIDITIES

K. Bo Foreman, PT, PhD
D. James Ballard, PT, DPT
Leland E. Dibble, PT, PhD, ATC

Long term studies examining the progression of Parkinson's disease (PD) report the mean age of diagnosis to be in the mid sixties.[1] The diagnosis of PD typically occurs after the presentation of motor deficits such as resting tremor, rigidity, hypokinesia, or postural instability.[2] Parkinson's disease in isolation results in worsening of motor deficits and progressive functional decline. While these problems are primarily the result of the degenerative process, underlying comorbidities may amplify or contribute to these deficits. In addition, given the typical age of onset, persons with PD will often present with a host of concurrent medical conditions (comorbidities). This is confirmed by research that estimated over 50% of Medicare beneficiaries had multiple comorbidities at the turn of the century.[3]

The consequences of comorbidities include, but are not limited to, impaired mobility, depression, reduced quality of life, and increased health care expenditures relative to cohorts without comorbidities.[4] In an on-going longitudinal study, we have demonstrated that the majority of persons with PD have one or more comorbidities, and that the accumulation of these comorbidities is related to a decline in gait, balance, and quality of life as well as an increase in disease severity as measured by the Movement Disorders Society Unified PD Rating Scale (UPDRS).

While the overall number of diagnoses appears to have some influence on mobility, balance, and overall quality of life, the relationship is only of moderate strength. In our clinical experience, the specific type of comorbidity present has a profound impact on mobility and balance function in persons with PD.[5] While this observation has yet to be directly examined in research studies, based on related evidence, we feel it is critical for physical therapists (PTs) to focus on PD as well as the non-neurologic medical history of their clients. To illustrate the importance of this issue, we present two clinical cases of comorbidities with particular relevance to mobility and balance in one person with PD with urinary incontinence (case 1) and another person with PD with peripheral neuropathy (case 2).

Case 1: Urinary Incontinence

Lower urinary tract dysfunction may be present in many individuals with PD. The presence of this dysfunction may arise from alteration of peripheral urologic structures (for example from prostate surgery in males or from child birth in females) or from autonomic disturbances associated directly with the neurologic problems of PD. Urge urinary incontinence (UI), the complaint of involuntary leakage accompanied by or immediately preceded by urgency, occurs in 25% to 28% of persons with PD[6,7] and can be seen early in the course of PD.

While urge UI is certainly a social concern for individuals experiencing it, it also appears to be an independent risk factor for falls in neurologically healthy individuals and those with PD. In women without PD that experienced at least one urge UI episode each week, fall risk was increased 26% and the risk of fractures was increased 34%. In persons with PD, urinary incontinence increased the probability of falling by almost 6 times (adjusted OR = 5.9, 95% CI: 1.4-24.6).[8] Regardless of the presence of a neurologic diagnosis such as PD, urinary frequency, nocturia, and rushing to the bathroom to avoid urinary incontinence have all been hypothesized to increase fall risk.[9]

The initial referral for client 1 (Male; age: 78 years; Hoehn and Yahr stage: 3; UPDRS motor subsection: 11; history of falls: >5 in the past year) (Table) came from his neurologist with the diagnoses of PD and UI with a request to evaluate and treat for increasing balance problems and fall occurrences. Client 1 had been diagnosed with PD for 16 years prior to the referral to PT and he was currently taking dopamine replacement medications (Sinemet 6 times per day). In addition to PD, client 1's non-neurologic medical history was significant for a transurethral resection of the prostate for benign prostatic hypertrophy. Clinical examinations of balance and mobility were performed using the Functional Gait Assessment (FGA), Timed Up and Go (TUG), and Six Minute Walk (6MW). Outcomes of these examinations are presented in the Table. Further questioning regarding his fall history revealed multiple near falls or falls during efforts to get to the bathroom. History of incontinence episodes were only elicited following explicit questions targeted at UI ("Do you experience such a strong and sudden urge to void that you leak before reaching the toilet?").[10] Client 1 reported a frequency of UI occurrences 4 times per day and a frequency of voiding every 50 minutes.

After findings consistent with urge UI, client 1 was referred to a urologist for medical evaluation. Urologic examination confirmed the diagnosis of urge UI and client 1 was referred back to physical therapy. The initial treatment involved combined fall risk reduction interventions and education on home pelvic floor muscle training. Although pelvic floor muscle training is an effective treatment for urge urinary incontinence,[11,12] client 1 experienced only a moderate reduction in UI episodes as a result of the training. It was hypothesized that the reduced efficacy relative to neurologically healthy individuals was due to the autonomic dysfunction associated with PD. Treatment strategies targeted at fall reduction included practice of compensatory step-

Table. Demographics and Clinical Examination Findings.

	Age/gender	Years w/ PD	H&Y	UPDRS	History of Falls (per yr.)	FGA	TUG (sec)	6MW (m)
Client 1 (PD and UI)	78/M	16	3	11	>5/year	26	7.2	484.80
Client 2 (PD and PN)	76/M	6	3	26	>10/year	17	16.77	452.48

ping, practice of balance challenges in altered sensory situations, and education about the adverse effect of urinary urgency on his fall risk. Most importantly, client 1 did experience a reduction in his self–reported near fall and fall frequency in response to therapy.

Case 2: Peripheral Neuropathy

Individuals with lower extremity peripheral neuropathy (PN) experience similar fall related risks as those with PD.[13] In both disease processes sensory integration is affected[14] resulting in increased reliance on other sensory stimulus such as vision.[15] In addition, authors have postulated that the diminished sensory integration is caused by impaired proprioception.[16] While sensory integration has been shown to play an important role in postural stability, to our knowledge there are no reports in the literature documenting the effects of peripheral sensory deficits in persons with PD in regards to balance and mobility. Despite this lack of research evidence, in our clinical experience, the sensory deficits associated with PN interact with the sensory integration and motor deficits associated with PD to create profound fall risk above and beyond that which could be ascribed to PD alone.

The initial prescription from his treating neurologist was a diagnosis of PD and the request to evaluate and treat. Client 2 had been diagnosed with PD 6 years prior to the referral to PT, and he was currently taking dopamine replacement medications (Sinemet 4 times per day) as well as a dopamine agonist. The mobility and fall history was significant for almost daily near falls and greater than 10 falls in the past year. In addition to PD, client 2 reported a diagnosis of idiopathic neuropathy that primarily affected his distal lower extremities. His PD and fall history were as follows. (Male; age: 76 years; Hoehn and Yahr stage: 3; UPDRS motor subsection: 26; history of falls: >10 in the past year) (Table). Sensory testing revealed moderately impaired light touch and sharp/dull sensation inferior to the bilateral malleoli. Following sensory testing, clinical examinations of balance and mobility were performed using the FGA, TUG, and 6MW while on his medications. Performance on the FGA, TUG, and 6MW were severely affected (Table). When considered relative to published PD specific fall risk cut-off scores for the TUG,[17] his performance reflected a profound fall risk even when on medications. Furthermore, FGA scores were significantly lower when compared to scores for similar aged healthy elderly (70-79 y/o: 24.9).[18] During FGA testing, client 2 manifested particular difficulty with items that required reductions or alterations in visual or vestibular function (eyes closed, head turns will walking).

Based on his examination findings, the clinical hypothesis was that this client's poor balance performance and fall risk were due to the interaction of impaired ankle somatosensation with the motor deficits consistent with PD. Treatment strategies targeted at fall reduction were implemented and included lower extremity strengthening, use of vision as a compensation for lower extremity sensory deficits, and practice of balance challenges in altered sensory situations. Given the irreversible nature of his PN, a large component of treatment involved education of the client and his caregiver regarding the use of an assistive device (a 4-wheeled walker with brakes) and the need for guarding during balance challenges. In addition, floor to chair/floor to stand transfer training was initiated to confirm independence and insure that the client would not be stranded once he had fallen. Unfortunately, client 2 did not experience improvements in his clinical balance test performance over the course of treatment. However there was a mild reduction in his self–reported near fall and fall frequency. The client and caregiver attributed this reduction to the assistive device and guarding.

DISCUSSION

The diagnosis of PD alone carries an increased risk for falls and fall related injury. However, the mobility and balance performance of persons with PD is generally heterogeneous and therefore it is unclear what characteristics beyond PD amplify a persons balance and mobility deficits. In the presented cases, we summarize the clinical findings of two persons with specific comorbidities, which in our experience are particularly relevant to PTs treating persons with PD. These cases vividly illustrate the potential amplification of balance and mobility deficits in the context of PD with either urinary incontinence or peripheral neuropathy.

CLINICAL RELEVANCE AND CONCLUSIONS

As part of the examination process, PTs must consider the effect of co-morbidities on motor deficits such as gait and balance. Physical therapists examining persons with concurrent PD and UI or PN should anticipate the potential for repetitive falls and plan treatment accordingly. Only with treatments addressed at their Parkinsonian deficits as well as their co-morbidities will fall risk reduction treatment be optimized.

While both types of co-morbidities may worsen function, the origin of their effect appears to be different. The threat of UI will often induce rushing to the bathroom, which in the context of PD may produce freezing or tripping. The near fall or fall events in individuals with PD and UI may be reversible with the implementation of a regular voiding schedule and pelvic floor muscle training. In contrast, PN in the context of PD, creates a sensory motor situation that may be a "recipe for disaster." Persons with PD and PN may not improve their balance performance in response to treatment. However, reductions in fall risk may be achieved through the use of assistive devices and client and caregiver training.

The objective of this paper was to raise the awareness of PTs regarding factors that may amplify fall risk in persons with PD. The cases presented provide only observations of the interaction of PD with comorbidities. Further research is needed to determine the effects of comorbidities on selective aspects of balance and mobility function as well as the effects of co-

morbidity management in conjunction with PD rehabilitation.

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CANDIDATES FOR 2010 SOG ELECTION

The Section on Geriatrics is pleased to announce candidates for 2010 elections. Candidate statements will appear in the September issue of *GeriNotes* with electronic voting to follow in October. The Nominating Committee would like to thank all candidates for their willingness to serve.

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Andrea Saevoon, SOG Section Executive, resigned her position effective July 2nd. Andrea plans to attend graduate school for a Master of Social Work which has been her professional goal. As Section Executive Andrea's responsibilities have been varied, but she has always been helpful and cheerful in assisting the SOG Board and members to the benefit of the Section. Thank you and best of luck to Andrea in this new passage of her life.

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- Franz Kafka

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