Clinical Neurology: Parkinson’s Disease

Javier Benitez SPT

Hunter College
Overview

Parkinson’s disease (PD) is a chronic and progressive neurodegenerative disorder. First named by Parkinson's in 1807, this disease is considered the second-most common neurodegenerative disease in the US with 1 million affected, surpassed only by Alzheimer’s within the US. Sixty thousand more people are diagnosed annually. This number is expected to substantially increase, with the the baby boomer generation turning 60 - the age when many people present with PD. This disease effects motor and autonomic function, sleep, and may cause neuropsychiatric issues, as well as dementia. PD will effect a patient’s posture, movement speed and amplitude, speech, gait, facial expression, and upper extremity (UE) dexterity. The most notable effect on a patient’s daily living is bradykinesia - slowed movement, which shows to have the greatest impact of a patient’s quality of life (Farley, B, 2008). A patient’s movement amplitude is also vastly decreased, resulting in increased falls risk and decreased balance. Patients also lose the ability to show spontaneous facial expression. In the latter stages, rigidity inhibits respiration, and swallowing, which increases the risk of pneumonia. While numerous motor and autonomic functions can be greatly impaired, physical therapy coupled with medicinal intervention has shown to impede the progression of this neurodegenerative disease.

Pathology

It is still unclear what causes PD, but it is widely believed to be multifactorial. The substantia nigra suffers a decrease in stored dopamine (DA). Loss of DA affects both the direct & indirect substantia nigra pathways of the basal ganglia. This results in less excitatory thalamic
input to the cortex, which may impede the inhibitory motor response. Some recent evidence considers this disease to be caused by environmental factors, coupled with aging, which lead to diminished DA stores. Postencephalatic parkinsonism may be due to a slowly progressing virus, or long-term effects from a proliferating infection. Some evidence shows a correlation between pesticide and insecticide exposure, as well as particular elements/contaminants found in well water. Excitatory nerve cell death, the increase of free radicals, and nigral mitochondria dysfunction have all been connected to the pathology of the disease.

**Clinical Manifestation & Differential Diagnosis**

Rigidity is characterized as an increased resistance during the full range of passive movement. Pt’s with PD in the form of “cogwheel” or “lead pipe” rigidity. In cogwheel rigidity, the resisted movement is accompanied by tremor. Patients with PD do suffer from rigidity - their muscles still fire on a musculoskeletal level. However, the synaptic firing is not large enough to generate a muscle contraction, preventing patients from firing agonist muscles to stop a movement that has already been initiated. This also plays a role in patients not being able to counteract a misstep to avoid a loss of balance, as well as the appearance of loss of strength. Procaine injections have been utilized to inhibit the rigidity, without affecting spontaneous movement. Deep tendon reflexes also seem to remain intact in persons with PD. Research by Tatton has found evidence that long loop reflexes are compromised in drug-induced Parkinsonian monkeys (Tatton, 1982). This creates a similar motor neuron excitability for different environmental demands. This gives the patients a similar perceived exertion rate for movements that are of varied demand (writing compared to lifting a heavy object). This presents
as an issue especially during gait, whereby patients diminish their arm swing, trunk rotation, gait stride length, and are unaware of it. Rigidity may also enhance energy expenditure, possibly causing the patient to mistaken feel they are exerting themselves more than they actually are, which is especially present in postexercise fatigue.

Tremors in PD present at rest, and typically diminish or stop during movement. Some patients do have postural tremors, as well. Studies have found that tremors are due to a combination of depleted DA stores, as well as lesions in the basal ganglia and cerebellar red-nucleus pathways. While the tremors may be distracting or noticeable to others, they rarely affect a patient’s ability to perform their activities of daily living.

Postural instability increases the rate of falls, and leads to numerous falls - with some patients falling more than once a week. Patients with PD are nine times more likely to fall than control groups of their same age(Bloem, 2001). Patients with PD are also more prone to falling as the disease progresses. Drug treatment typically cannot reduce their falls risk, but exercise and deep brain stimulation has been shown to minimize the occurrence of falls. While it is unclear as to a definitive reason for postural instability, some hypotheses include diminished proprioceptive and kinesthetic processing - particularly, a delayed labyrinthine equilibrium. Other theories dealt with the patient’s ability to process vestibular input from the perturbation and react with appropriate long- and middle-latency reflex responses to maintain postural balance. Patients with PD also showed strategy selection defects when losing their balance, as well. People will typically utilize a hip or ankle maneuver to regain balance, but patients with Parkinson's do not utilize a specific one, which prevents them from reacting in time to avoid falls. Falls risk for patients with PD seems to be a combination of lack of movement, lack of reaction, rigidity, and the lack of coordination in central sensory processing.
Parkinsonism gait is characterized by a slow gait with a short stride length. Decreased foot clearance is also apparent, which also increases the risk of falling. Festination, the act of quickly shuffling one’s feet in a certain direction, attempting to catch up with one’s center of gravity, is also present. Festination occurs either forward (known as propulsion) and backwards (retropulsion). The theories behind festination involve either a patient’s slow or inconsistent motor response associated with bradykinesia, which cause the short stride length, or other gait kinematic differences. Gait kinematics do change in patients with PD. Patients may develop a flat-foot or toe-heel progression. A lack of plantar-flexion during terminal stance, as well as decreased dorsiflexion and hip flexion, also play a role in increasing the falls risk. Gait and postural dysfunction are considered the greatest challenges for patients with Parkinson’s to adapt to home and work activities.

Dysfunction in perception, attention, and cognition are considered to be due to the decrease in cortical excitation from the caudate nucleus. Frontal lobe deficits prevent the patient from being able to shift their attention away from irrelevant observations, properly accessing and utilizing their short term memory of their visuospatial observations. Patients also experience difficulty learning by means of selective attention toward specific stimuli - they show difficulty in choosing an appropriate motor response for a particular stimulus. Procedural learning, defined by Saint-Cyr research as acquiring a motor skill or a cognitive routine through repeated exposure of a particular activity within consistent restraints (Saint-Cyr, 1988), may also prove difficult with patients. Research by Pascual-Leone found that patients were able to acquire procedural learning, but required more practice than their control subjects (Pascual, 1993). Their findings also showed a benefit to acquiring procedural learning with visual input alone, as opposed to coupling it with a motor task. These factors may rationalize the benefit of more therapy, with a portion of
the therapy being focused on the demonstration of their lack of movement, or a visual demonstration of proper movement.

Other nonmotor symptoms include loss of smell, bowel and bladder dysfunction, and orthostatic hypotension. Dizziness may be a result of orthostatic hypotension (dizziness when standing up). While orthostatic hypotension is not an indicator of PD, the combination of these nonmotor symptoms may prove to be a sign of the disorder. Quality of life (QOL) concerns arise from these symptoms, as well as urinary incontinence, sexual dysfunction, excessive salivation, weight changes, skin problems, fatigue, fear, anxiety, depression, and sleep disorders. Rapid-eye movement [REM] behavior disorder (vivid dreams), daytime drowsiness (possibly due to medication), and decreased sleep at night (which may be due to the onset of restless leg syndrome) also may negatively affect QOL. Medication may also impair other cognitive symptoms, including dementia, memory loss, and confusion - all possibly affecting QOL.

Parkinson's disease comes on in stages. The Hoehn and Yahr Staging Scale for Parkinson's Disease has a five stage scale, with 2 sub-stages added in recent years (These subcategories have not been validated but are commonly utilized). Stage 0 shows no signs of the disease. Unilateral symptoms (resting tremor or bradykinesia in the upper extremity) occur in stage 1, and axial involvement (along the body’s mid-line) comes on at sub-stage 1.5. Bilateral bradykinesia and rigidity develop at Stage 2. Sub-stage 2.5 shows mild bilateral impairment with independent recover in the Retropulsion Test (aka Pull Test), indicating that a patient still may recover from loss of balance in Stage 2. Stage 3 shows balance impairment, and mild to moderate disease, while the patient still is independent. Flexion in the neck, trunk, and hip are present, along with a decreased ability in postural righting and balance responses. Testing the righting response (eyes closed, feet together, and pushing from standing position) is the specific
indicator for the onset of Stage 3. These factors alter their ability to maintain their center of
gravity over their base of support, in turn, affecting a patient's ability to maintain their balance.
Rigidity comes on bilaterally, initially affecting their ability to rotate their trunk and swing their
arms during gait. The initiation of movement becomes more challenging, requiring much
concentration, possibly being initiated by cortical pathways, as opposed to via the lesioned basal
ganglia pathways. While requiring more time to perform ADL, patients with Stage 3
Parkinson’s still maintain their independence. In Stage 4 Parkinson’s is fully developed. While
the patient may still ambulate independently, the patient requires assistance for ADL, which is
what distinguishes Stage 3 from Stage 4. Parkinsonian Stage 5 has the patient unable to stand or
transfer without risk of falling. Patients are confined to a wheelchair or bed, with high concern
for respiratory dysfunction.

Medical Intervention

Parkinsonism is treated with a pre-cursor of DA, levo-dihydroxyphenylalaline (L-dopa),
which can penetrate the blood-brain barrier. To increase the storage of L-dopa within the basal
ganglia, the L-dopa must be converted once it has crossed the blood-brain barrier. The amino
acid, carbidopa, is administered with L-dopa to prevent the conversion of L-dopa into DA
outside of the blood-brain barrier. The L-dopa greatly reduces bradykinesia and rigidity, and is
less effective at controlling tremor and postural instability. Nigral neurons are able to remain
intact due to the receptors and the nerve cells being post synaptic to dopaminergic neurons. This
allows the Nigral neurons to be, at least initially, responsive to DA. The effectiveness of L-dopa
diminishes after prolonged use (10 years or more), with dyskinesia (involuntary movements)
developing. A “on-off” phenomenon also develops, whereby the patient experiences a short-lived improvement, followed by a rapid decline digression, and accompanied dyskinesia as well as possibly dystonia. The “on” phase progressively becomes shorter in duration. Slow-release and controlled release forms of L-dopa have been used to minimize these long-term effects of this drug. DA rector D2 agonists have been developed to minimize the effectiveness of L-dopa due to long-term use. Some of these D2 agonists include: ropinirole, pramipexole, pergolide, and bromocriptine. Medication that prevents the breakdown of DA is also utilized. Known as catecholamine-O-methyltransferase [COMT], drugs like entacapone prevent DA break down and/or its reuptake. Deprenyl, rasagiline, and selegiline act as monoamine oxidase inhibitors and are also utilized in combination with L-dopa and carbidopa.

Due to the lack of DA storage, L-Dopa and DA agonists may lower blood pressure, which indicates a need to reassess these medications once antiparkinsonian drugs have been incorporated into the medical treatment. Medication may also result in hallucinations, with patients reporting of seeing ugly creatures or monsters, including during therapy sessions. This may hinder the patient’s ability to utilize alternative treatments during therapy like computer games and virtual reality simulators. Keep in mind that medicinal intervention does not thoroughly address all symptoms of PD, as the disease progress.

**Surgical Intervention**

Deep-brain stimulation (DBS) stimulates the global pallidus internal segment or the subthalamic nucleus which, in turn, reduces dyskinesia, rigidity, bradykinesia, akinesia, improves gait and lessens the dosage of medication necessary. DBS has been shown to improve
movement velocity and the speed that muscles are recruited for activity(cite). DBS is also reversible and safer for bilateral surgeries. Thalamic stimulation has been shown to decrease tremors. There is also evidence of intense therapy immediately after surgery utilizing the process of neuroplasticity(cite).

**PT Evaluation**

Patient Examination should include observed levels of rigidity, bradykinesia, balance impairments, gait abnormalities, and their effect on ADL. Objective measures of these impairments should be recorded when possible to better communicate with health care professionals, even if scales may not be as useful for therapeutic goals and interventions. For therapy-focused goals, it would be beneficial to assess and, eventually, reevaluate functional activities for treatment planning. Consider a patient’s gait speed, distance, and quality during forward, backward walking, and braiding. Be sure to include a patient’s ability to quickly and abruptly change speeds, as well as perform cognitive tasks during these observations.

The Hoehn and Yahr Scale is utilized to objectively evaluate the disease’s progression. The United Parkinson’s Disease Rating Scale (UPDRS) is a widely used assessment tool to measure cognitive and emotional status, ADL capabilities, motor function, and side effects from medication. The Core Assessment Program for Intercerebral Transplantation (CAPIT) is utilized for patients who have had surgical interventions (CAPIT is utilized more in research than clinic, as it is lengthy). There are numerous gait, functional assessment, and balance tools, including Timed-Up-and-Go Test (TUG), 10-meter walk test (10MWT), the 5 or 10 Time Sit-to-Stand Test, the Dynamic Gait Index (DGI), and the Berg Balance Scale (BBS). Carefully analyzing
how a patient performs a test (or a portion of the more lengthy tests) will be beneficial when determining a patient’s treatment plan and goals. Assessments should be done on a patient’s abilities with and without vision. Early stage Parkinsonism will show difficulty in tandem walking, as well as standing on unstable surfaces. Posturography measures are considered good indicators of postural instability, especially in Stages 1 and 2 of Hoehn and Yahr’s parkinsonian scale. Dynamic balance is easily tested using the functional reach test, and a more in-depth assessment may be done using the Balance Evaluation Systems Test (BESTest). A falls history is also a good indicator of future falls.

Chest expansion and vital capacity is useful, as well, due to the concern for its effect if a patient were to acquire a pneumonia. When rigidity is being assessed, it is important to assess respiratory, extremity, and trunk strength, as well as periodically assessing active and passive range of motion.

**Implication of PT intervention**

One current successful approach to PD care has been the use of physical therapy during the earlier stages of Parkinson’s diagnosis. The success of LSVT BIG has tested the intervention efficacy of amplitude, volume, and movements in patients with parkinsonism (Sanjay, 2013). Further research is being done to find the least amount of effective intervention to benefit the patient. Every aspect of movement may effect a patient with PD. Due to the affected basal ganglia, patients present with a flexed posture, have a difficult time regaining their balance (especially the labyrinthine equilibrium reactions), and also have diminished trunk rotation. Patients with PD suffer from a decrease in motion (bradykinesia) and an inability to initiate the movement or perform purposeful movement (akinesia). Patients will also hold fixed postures,
will not be able to change their direction as easily, including stopping movement once it is initiated. During gait, patients do not swing their arms - one of the distinguishing gait characteristics. Patients with PD have difficulty with movements involved with timing, sequencing, or two movements simultaneously. This prevents patients from being able to perform simultaneous tasks, for example, while walking. The more steps in a task sequence, the harder it is for patients with PD to perform. Transitioning between different types of movements also proves to be more difficult than doing one repetitive movement continuously (i.e. Stationary bike pedaling).

**Treatment**

Treatment should be based on measurements assessed, the patient’s functional requirements and his goals. Improving movement and range of motion (ROM) in the trunk and extremities will diminish a patient’s effort necessary to ambulate and respire, as well as allow for easier maintenance of balance and executing ADL. Although L-dopa will diminish the bradykinesia, extensive therapy in the earlier stages will improve movement and decrease the likelihood of the development of pneumonia. Therapy is more beneficial when started at the first sign of the disease, but have still shown advantageous when began during Hoehn and Yahr stages 1 to 3. Treatment should involve complex, sequential movements that involve numerous senses, and mimic functional activities necessary in the patient’s everyday life. These movements should focus on large amplitude moving, which challenge balance, and are
enjoyable. Numerous treatment approaches, like Lee Silverman Voice TherapyBIG (LSVT BIG) incorporate large-amplitude movements that are recommended for numerous hours per week for each day of the week. It is important to realize that if a patient does not enjoy the movements, they will be less likely to exercise as consistently and as effectively, which will damper the treatment and minimize efficacy. Dual motor-cognitive tasks and complex movements that force patients to quickly change directions in response to environmental obstacles have shown improved reassessment scores for tests including: TUG, UPDRS, 10MWT, and numerous balance tests.

Functional treatments should revolve around sitting and standing, bed mobility, and postural awareness. In the latter stages, emphasis should be placed in chest expansion and any treatments and exercise that improve respiration. Besides LSVT BIG and sensory attention focused exercise (PD SAFEx), alternative activities include ballroom dance, Zumba, martial arts (karate and tai chi), and alpine hiking. To minimize rigidity, treatments should strategize moving muscles through its full range – emphasizing the lengthening of the flexor muscles, and the contraction of the extensor muscles to avoid the flexed posture. While aerobic exercise was beneficial for improving gait and respiratory function, it did not minimize symptoms.

Body-weight support treadmill training has been utilized with the benefit of allowing the patient to feel safe from falls and able to take part in dual motor-cognitive tasks with less concern for falling. It has shown to be helpful to minimize rigidity during the earlier portion of treatment sessions and to time the medication so it is peaking during treatment sessions.

Any physical activity and movement seems to improve the patient’s mood, encourage initiative in their treatment, and enhance their quality of life. Low-impact aerobic group classes also help create a support group for patients and their spouses. Music, as well as metronome use,
may be utilized to help participants with the timing and rhythm of the movements, which would translate to ambulation. Rhythmic exercise also decreases rigidity and bradykinesia, as well as improves gait. Visual cues (i.e. parallel lines on the floor or distant elevated visual object/structure) may prevent patients from freezing (Martens, 2014). It is for this reason that patients with PD also have an easier time negotiating stairs, due to the stairs providing visual cues. Cool-downs are also a good opportunity to work on fine motor hand activities. In regards to improving strength in functional activities, function-focused exercises are more beneficial than weightlifting. Due to the difficulty patients have with complex, sequential, varying tasks, treatment should involve these tasks to help them practice executing complex movements.

**Case Scenario**

Patient is a 63 yo female with Stage 3 Parkinson’s (Hoehn and Yahy Staging Scale). Observation includes significant slowing of body movements, early impairment of equilibrium on walking or standing, and generalized function that is moderately severe. Assessments show the following:

**Initial Observation:** Patient presents seated in waiting room with flexed head, neck, and trunk posture. Resting tremor in bilateral distal arms is apparent. Patient appears wide-eyed with minimal facial expression. Patient begins to speak at a normal audible level, but progressively speaks quieter toward the end of her breath. Patient experiences threshold freezing when moving into the evaluation area, and has a diminished gait speed and arm swing during ambulation.

**Cognition:** A&Ox3
Strength:

MMT: Bilateral (B) LE: generally 4+/5 with Hip EXT 3+/5; B UE generally 4/5; Trunk Flexors generally 4+/5; Trunk Extensors generally 3+/5

Flexibility:

ROM: B UE within functional limits, except limited elbow extension: 120 - 10 degrees; B LE limited Hip Extension (120-5 degrees) and limited Knee extension (120-7 degrees)

Forced Vital Capacity: 2,324 cm^3

Rigidity: (UPDRS scale) 2, apparent in trunk mobility

Tremor at rest: Bilateral distal arms at level 2 (UPDRS scale) displaying moderate amplitude and intermittent.

Balance: impaired righting response

Gait:

TUG: 15 seconds

10MWT: 19.4 seconds

5 Time Sit to Stand:16.3 seconds

DGI:18/24

BBS: 47

ADL effect: patient complains of not being able to stand up from bed without feeling a lack of balance. Patient feels extremely fatigued when walking just a few blocks, which was never the case. Patient also has a challenging time walking from kitchen to tv room with her glass of iced tea, without spilling some of it on the floor.
**Initial Treatment Phase**

**Warm-up:**

1. (standing in front of mirror) Large-amplitude reciprocating arm swings with trunk rotation.
2. Supine bilateral bridges
3. Supine Terminal Knee Extension

**Treatment**

2. Sit 2 Stand from elevated table 1 set x 10 reps.
3. Step forward and reverse: patient takes a forward step with large-amplitude arm swinging and stride length, then steps back to original standing position. Repeat 10 x/side.
4. Doorway ambulation: patient crosses through doorway, utilizing a distant stationary visual to progress through doorway to minimize freezing threshold. 1 set of 10 doorway ambulations.
5. Posture Press: pressing vertebrae against wall, while maintaining upright posture, and tucking in chin throughout. 2 sets of 20 seconds.

**Cool-down**

1. Fine-motor tasks. Sign the alphabet once with each hand.

**Education:**
1. Avoiding flexed forward posture.

2. Exaggerating arm swinging and long leg stride during ambulation.

3. Deeper breaths and louder speech.

4. Fix eyes on a distant object to avoid freezing threshold.

5. Time rigidity and bradykinesia medication within an hour prior to therapy session, HEP, and/or ADL.

HEP

Warm Up:

1. Supine Bilateral Bridging 2 sets x 10 reps with 3 sec hold at peak

2. (seated) Reach down toward ground by flexing trunk and then sitting tall in chair, extending arms overhead as high as possible. 2 sets x 10 reps with 2 sec hold at elevated arms position.

Exercises:

1. Standing Terminal Knee Extension with Theraband 2 sets of 10 reps with 3 sec hold for each leg.

2. Standing Elbow extension with theraband 2 sets of 10 reps with 3 sec hold

3. Sit 2 Stand from elevated seat 10 reps x 2 sets with 5 sec hold at standing position.

4. Ambulation: walk through doorway with large-amplitude arm swing and longer stride length. Turn around and return to starting position for one complete lap. Perform 5 laps of 10 meters.
5. Dual motor cognitive task: Walk 10 meters while holding tennis ball in slightly cupped hands. 10 meters x 2 sets.

Cool-down

1. Hip Flexor stretch 30 seconds x 2/leg
2. Elbow Flexor stretch: 30 seconds x 2/arm
3. Fine-Motor Task: sign alphabet with each hand x 1 set.

References


Tatton, L. (1982) Long latency reflexes to imposed displacements of the human wrist:


