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Management of Balance and Gait in Older Individuals with Parkinson Disease

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Summary

Difficulties with walking and balance are common among people with Parkinson disease (PD). Effective management of these walking and balance problems is critical, as these issues can contribute to decreased mobility, falls, social isolation, reduced quality of life, and increased mortality. This review provides an overview of the gait and balance disorders associated with PD and discusses the methods currently utilized to manage gait and balance problems among people with PD. These methods include pharmacological, surgical, and rehabilitative approaches. Remaining challenges and future directions, including the need for comprehensive, multidisciplinary, patient-centered care provided by qualified movement disorders experts, are also highlighted.

Keywords: gait, balance, Parkinson disease, deep brain stimulation, exercise, levodopa, physical rehabilitation

What is Parkinson Disease?

Parkinson disease (PD) is a progressive neurodegenerative disorder that affects between 1-2% of individuals over 65 years of age and 3-5% of those 85 or older [1]. Age appears to be the greatest risk factor for PD, but many other contributing factors have also been identified [2-4]. At present, PD affects 1-1.5 million people in the United States and an estimated 40 million people worldwide. The number of individuals with PD will increase in coming decades, as the percentage of the U.S. population over age 65 rises from its current level of 12% to over 20% by 2030 [5].

PD is characterized by four cardinal features: tremor, bradykinesia, rigidity, and postural instability. The symptoms of PD arise only after substantial loss of dopaminergic neurons in the substantia nigra pars compacta. In fact, PD was originally attributed only to neuronal loss within the substantia nigra pars compacta, and a concomitant loss of dopamine. Pharmacologic replacement of dopamine is the most common, and arguably most effective, treatment for PD. However, PD is now thought to involve not only the dopaminergic system, but also other neurotransmitter systems whose role may become more prominent as the disease progresses [6]. These non-dopaminergic systems may be particularly implicated in balance and gait disorders, the management of which is the focus of this paper. Balance and gait impairments are particularly problematic for individuals with PD, as they can lead to falls, injury, and fear of future falls. As a result of injury and/or fear of falling, individuals may limit their physical activity which may result in social isolation as well as subsequent muscle weakness and osteoporosis. These in turn may contribute to increased risk of future falls and fall-related injuries [7]. The purpose of this paper is to provide an overview of the gait and balance disorders associated with PD and discusses the methods currently utilized to manage gait and balance problems among people with PD. The ideal combination and prioritization of these approaches needs to be determined on an case by case basis.

Gait, Balance, and Falls in Parkinson Disease

Gait Impairments

Many individuals with PD have gait difficulties that greatly limit functional mobility. The majority of studies focusing on Parkinsonian gait examined walking in a straight line. These studies showed that straight ahead walking in PD is characterized by: 1) a flexed posture, 2) short, shuffling steps, 3) deficits in stride length regulation, 4) reduced foot clearance during swing, 5) increased cadence, 6) limited extension at the hip and knee and reduced ankle plantarflexion, 7) reduced transverse rotation of the trunk and pelvis, 8) inappropriate coactivation of muscle agonist-antagonist pairs, and 9) reduced muscle activation amplitudes [8-14]. The cause of these gait impairments is thought to be a disturbance in the motor set function of the basal ganglia, which is involved in regulation of movement amplitude [15]. It has also been hypothesized that the basal ganglia contribute to the regulation of postural alignment and motor control, which may play a role in gait dysfunction in PD [16,17].

People with PD often have even more difficulty walking in challenging situations than they do walking forward. For example, individuals with PD experience exacerbated gait impairments when required to perform a concurrent task. Specifically, during dual task walking, gait speed and step length are reduced and stride to stride variability is increased relative to single task walking [18-23]. In backward walking, gait speed and stride length are also reduced relative to forward walking in people with PD compared to people without PD [24,25]. Both dual task walking and backward walking have been recommended as important tasks to evaluate when assessing gait in people with PD.

In addition to difficulties with dual task walking and backward walking, many people with PD have more difficulty turning while walking than they do walking in a straight line. Individuals with PD, particularly those with a history of freezing or falls, use an increased number of steps to turn as compared to people without PD [26,27]. People with PD also tend to turn en bloc, i.e. rotating the head and trunk simultaneously rather than in sequence, and require greater time to

turn [28-30]. The work of Crenna et al. [28] is particularly interesting because it reveals that turning deficits are present even in individuals with mild PD who as yet have no alteration or impairment in their straight walking. This suggests that turning difficulty may affect individuals with PD even from a very early stage of the disease when other motor symptoms are not yet apparent.

Turning is particularly problematic because it is one of the primary triggers for freezing of gait (FOG). FOG, “an episodic inability (lasting seconds) to generate effective stepping”, affects more than 50% of patients who have had PD for over 5 years [31,32]. FOG commonly occurs during turning, but may also occur at gait initiation or when walking through doorways or other narrow spaces. Those who experience FOG are more negatively affected than those without FOG by challenging conditions like dual task walking and backward walking [25]. FOG is a prominent cause of falls, particularly during turning [33-35]. Falls that occur during turning are eight times more likely to result in hip fracture than falls during straight line walking [36].

Balance Impairments

Postural instability is one of the cardinal symptoms of PD. Though it is often associated with mid-to-late disease progression, recent studies have shown subclinical manifestations of postural instability in the early stages of PD as well [37-40]. Many different factors are believed to contribute to postural instability including impaired peripheral sensation and somatosensory integration, decreased strength, axial rigidity, and impaired postural responses [41-46]. Disturbed motor programming within the basal ganglia as well as abnormally processed proprioceptive signals are thought to be underlying causes of postural instability in PD [47]. Recent studies have also consistently shown that postural instability is not relieved with dopaminergic medication, therefore, non-dopaminergic pathways have been hypothesized to contribute to postural instability [48, 41].

Many different modes have been used to quantify instability in PD. In a clinical setting, quick screening of postural instability may include the UPDRS Retropulsion/Pull test [49] or the Push and Release test [50]. Assessments of static postural control have found that individuals with PD have increased sway area and abnormal shifts in the center of pressure [38, 39, 51]. Dynamic posture, such as during gait and functional activities, has also been shown to be impaired with slower velocities of movement and decreased ability to move the center of mass appropriately [40, 42, 46, 52, 53].

Both anticipatory postural adjustments and reactive postural adjustments are impaired in individuals with PD, with decreased amplitude and coordination of these movements [40, 54, 55]. An association exists between fear of falling and postural instability, though whether it is a causative relationship or a correlation is unclear [56]. However, postural instability is also related to falls in individuals with PD [57-59].

Falls

Individuals with PD are five to nine times more likely to sustain recurrent falls than age-matched individuals without PD [60, 61]. Falls and fractures are the primary reason for hospital admissions in individuals with PD [62, 63]. Studies have shown that 25-32% of falls result in injury [58, 64] with at least 3% [65] resulting in serious injury requiring medical attention. These are substantial numbers considering in prospective studies, 45-68% of individuals with PD will fall during a 12 month period with 29-51% experiencing two or more falls [58, 61, 64, 66]. In a given six-month period 29% of individuals with PD without a history of falling will experience their first fall [43]. Furthermore, individuals with PD have a 3.2 fold greater risk of hip fracture than age-matched individuals without PD [67]. Falls and subsequent hip fractures carry substantial personal costs, such as fear of falling, withdrawal from social activities, and a decreased quality of life [7, 68]. Hip fractures also represent a substantial financial burden to society, with the cost of hip fracture care in individuals with PD totaling approximately \$192

million per year in the United States alone [69, 67]. One study suggests that frequent falls in an individual with PD can be used as a marker to predict life expectancy [70].

The majority of falls in this population occur in the home and have intrinsic causes (i.e. disease processes such as postural instability, freezing, loss of concentration, etc.) as opposed to extrinsic causes (i.e. a wet floor, obstacle, etc.) [60, 64, 65]. Falls often occur during transfers, when bending or reaching, while walking especially in dual task situations, and during turning as well as due to freezing and tripping/accidental causes [7, 60, 64, 65]. It is interesting to note that 2/3 of individuals who fell said their signs and symptoms were under control when they fell [60].

Many studies have compared individuals with PD who fall and those who do not fall. Those who fall tend to have longer disease duration, increased disease severity, a history of previous falls, increased levodopa replacement medication use, and increased freezing and dyskinesia [64, 71, 43, 58, 59]. They perform more poorly in balance and functional mobility measures, have increased fear of falling, have increased sway in standing, and have decreased lower extremity strength [60, 71, 43, 72, 59]. Cognitive factors such as decreased memory, power of attention, and reaction time have also been shown to be associated with falling [64, 58]. Though cardiovascular issues such as orthostatic hypotension and postprandial hypotension have been shown to be present in individuals with PD, their contribution to falls has been debated [64, 43, 58, 73].

Therapeutic Approaches to Management of Gait and Balance in Parkinson Disease

Pharmacological Treatment

Levodopa (L-DOPA) is often the drug of choice in the pharmacologic management of motor symptoms in PD as it replaces endogenous dopamine which is reduced in individuals with PD [74]. Bradykinesia and rigidity often respond well to treatment with L-DOPA, while there is increased variability in response with respect to tremor [75]. The introduction of L-DOPA also has proven to be beneficial in improving gait disturbances. Several gait parameters are

negatively influenced by withdrawal of L-DOPA therapy [26]. The parameters most consistently impacted when patients are *off* L-DOPA are stride length and speed [76-84]. Additional features noted in individuals *off* L-DOPA include decreased arm swing and reduced trunk rotation, decreased ground reaction forces and reduced range of motion at the knee [85], and reduced plantarflexor power [84]. Many studies have demonstrated that stride length and gait speed are responsive to L-DOPA, with one study showing an increase of 0.25 meters/sec from *off* to *on* medication [86, 80], while other parameters such as stance duration and arm swing are dopa-resistant [8, 76-78, 87]. These dopa-resistant aspects of gait support the idea that the pathology of PD extends further than just the nigrostriatal dopaminergic system [75].

Freezing of gait (FOG) is one of the most debilitating and least understood gait disturbances in PD [88]. Treatment of freezing is often extremely difficult, and specifically regarding L-DOPA, results are mixed. Those patients who experience FOG during the *off* state often experience improvements in freezing after taking L-DOPA [89]. Schaafsma and colleagues found that among those participants who experience primarily *off* state freezing, FOG episodes occurred more frequently, had a longer duration, and also involved significant akinesia. In the same study, the observation of FOG in the *on* state was quite different as the episodes occurred less frequently, had a shorter duration, and did not involve complete blocks in movement [90]. Individuals with PD experiencing *on* state freezing present a much more challenging problem as their FOG is not responsive to L-DOPA. This problem is considered to be quite rare [88], and it has been suggested that dopaminergic agents be reduced in individuals with *on* period freezing [91]. For patients experiencing FOG that is resistant to L-DOPA, other therapeutic options may be considered [92].

Some alternative pharmacologic approaches aimed at treating FOG have been somewhat effective, while others have not. Selegiline, an MAO-B inhibitor, was shown to be effective in reducing the development of FOG in patients with early PD [93]. Shoulson and colleagues found similar results in individuals in the advanced stages of PD [94, 95], and it is

suggested that this may be of greater clinical significance as FOG and falls are highly related in the later stages of PD [92]. Rasagiline, another MAO-B inhibitor, has also been shown to have a positive effect on FOG, however, the clinical significance of this improvement is not yet understood [92]. Amantadine has been shown to have conflicting results with respect to FOG. Giladi and colleagues reported that those treated with amantadine were less likely to develop FOG [96], while Macht and colleagues reported an association between treatment with L-DOPA and amantadine and increased FOG frequency [97]. Botulinum toxin was originally found to have a positive effect on FOG [98, 99]; however, Gurevich and colleagues found dissimilar results and also found that botulinum toxin increased fall risk [100]. Methylphenidate has also been examined as a treatment for FOG [101, 102]. In a study examining apomorphine, an alternative option for those with advanced PD with motor complications, researchers found that FOG was not improved following a single dose of this medication [103]. In summary, FOG remains a substantial problem that is not well addressed by current pharmacologic approaches.

Deep Brain Stimulation

Surgical intervention, in the form of deep brain stimulation (DBS), is an increasingly common treatment method often employed in advanced stages of PD where pharmacological therapy does not adequately control parkinsonian symptoms and/or complications such as wearing off of medication and levodopa-induced dyskinesias have developed. With the intent of improving the predominant symptoms in PD, including gait disturbances and postural instability, the favored target for stimulation is most often the subthalamic nucleus (STN) [104]; however, there is conflicting evidence regarding gait improvement following STN stimulation in those with PD [105-110]. It is important to note that stimulation of the internal globus pallidus (GPi) has been a target for DBS in the past, but STN stimulation is now preferred by many because the antiparkinson medications can typically be reduced more following STN as compared to GPi surgery, and less stimulation energy is needed [111]. Recently, support for stimulation of the

pedunculopontine nucleus (PPN) [112-114] has gained traction among surgeons, but again, evidence supporting this technique without pairing it with STN stimulation is controversial [115]. It is imperative that healthcare professionals understand these surgical techniques and how they may affect gait and balance in individuals with PD so that intervention plans may be designed properly.

Using quantitative measures, STN stimulation has been shown to improve gait parameters such as stride length and gait velocity [109]. In a meta-analysis conducted by Bakker et al., bilateral STN stimulation was found to improve gait disturbances and postural instability, as assessed by the UPDRS, in the *off* medication state [108]. When examining anti-Parkinson medication and STN DBS and their effects on gait and gait stability, Hausdorff et al. [110] noted that combined effects of medication and DBS on gait speed, stride length, and gait stability were larger than effects of DBS alone. Krack et al. [107] reported that STN stimulation provided an initial improvement in gait and postural instability, but performance declined with time, and by five years, patients' gait disturbances and postural instability were more impaired than prior to surgery. For those patients experiencing freezing of gait (FOG) that is responsive to levodopa (L-DOPA), STN stimulation has been shown to fully replicate the L-DOPA induced improvement seen prior to surgery [107, 116-119]. However, regarding L-DOPA resistant FOG, STN stimulation has proven to be ineffective [120, 121, 116, 118]. As such, new surgical targets are necessary to determine if DBS surgery is effective at improving FOG in individuals with PD.

It has been proposed by previous reports that the PPN plays a role in the origination of PD motor symptoms, particularly gait disturbances and postural instability [122, 123]. Moro and colleagues examined UPDRS II, III, and IV scores as well as sub-scores in six subjects at three and 12 months post unilateral PPN stimulation [114]. At three months post-surgery, UPDRS II scores significantly improved, predominantly due to marked decreases in sub-scores for falling and freezing [114]. The UPDRS III scores (including gait, postural stability) demonstrated a trend toward improvement; however the scores did not reach significance [114]. UPDRS II sub-

scores for falling at 12 months were similar to those at three months, and UPDRS III scores trended toward significant improvement from three to 12 months post-implantation [114]. In contrast, Stefani et al. found that in the *off* medication state, PPN DBS alone did not improve UPDRS III scores [115]. It was also found that PPN DBS + STN DBS when *on* medication improved UPDRS II scores [115]. In both *on* and *off* medication states, dramatic improvements in gait and balance were found in two patients post-PPN stimulation. It is important to note that these differences were found 42 (patient 1) and 16 (patient 2) days post-surgery and as such micro-lesion effects from the surgery might still have been present. Repeated clinical observations at time points more than 3 months post-surgery are necessary to avoid micro-lesion and placebo-like effects following this type of procedure [124]. In summary, while the PPN, especially when paired with STN DBS, has recently been proposed as a surgical target for improving gait and balance in those with PD, it is not yet recommended as a substitute for STN DBS [115]. Current evidence is limited by questions regarding electrode targeting and in some studies it is not clear that electrodes were successfully placed within the PPN. More data are needed to confirm or refute PPN DBS efficacy on gait and balance deficits in those with PD.

Physical Rehabilitation

Treadmill Training

Recently, the effects of treadmill training (TT) on gait parameters, balance, quality of life (QOL), and functional tasks have been examined to determine efficacy of this intervention for people with PD. Mehrholz et al. conducted a systematic review of studies investigating the effects of TT on specific gait parameters in those with PD [125]. Eight small studies were included and improvements were noted in gait speed, stride length, and walking distance when comparing individuals who underwent TT and controls [125]. With regards to training intensity, Fisher et al. reported that high-intensity TT yielded significant improvements in self-selected gait speed, stride length, step length, and gait kinematics [126]. When compared with conventional

gait training, structured TT led to significant improvements in over-ground walking speed (1.37 m/s pre to 1.56 m/s post-training) and stride length (0.72 m pre to 0.78 m post-training), as well as double stance duration [127]. Miyai et al. studied body weight-supported treadmill training (BWSTT) versus conventional physical therapy in individuals with PD and found the BWSTT group maintained improvements in gait speed and stride length at one and four months post intervention [128]. Multi-directional TT (forward, backward, sideways) and step-perturbation training have also been shown to improve cadence and gait speed in men with PD [129].

While gait parameters are commonly measured when investigating the effects of TT, other studies have examined its effects on a variety of other outcome measures. Studies by Cakit et al. and Protas et al. have shown that different protocols for TT can improve balance [130], decrease fear of falling [130], and decrease number of falls [129]. A six week TT program in which participants with PD walked at 70%-80% of maximal heart rate led to improvements in lower extremity functional tasks (20-m walking time, timed U-turn, turning around a chair, ascending/descending stairs, standing from a chair, and standing on one foot) [131]. In addition to improvements in lower extremity capabilities and balance, Hermann et al. found that TT significantly improved QOL [132].

The mechanisms by which TT improves gait in individuals with PD are not yet fully understood. In a recent study by Bello and colleagues examining these mechanisms, the authors theorized that gait improvements following TT are related simply to the belt movement on the treadmill, which forces stepping through stretch facilitation of hip flexors and plantarflexors and the termination of the stance phase [133]. Other studies support the idea that TT forces step lengthening and may be an influential factor in gait improvements [9, 134, 135]. Protas et al. [129] hypothesized that visual and auditory cues, which have been shown to positively impact gait and balance in those with PD [136], provided by the treadmill could have lead to improvements in gait and balance, respectively. Herman et al. theorized that perhaps the treadmill provides an external rhythm that compensates for the ineffective internal rhythm of the

basal ganglia in those with PD. Another idea proposed by Herman et al. acknowledges that the intense, repetitive TT may add to motor learning which may explain the lasting improvements in gait five to six weeks post TT [132].

Although limited, there is good evidence supporting the short-term benefits of TT for people with PD. While the mechanisms behind the gait and balance improvements are not yet fully understood, the literature documenting such improvements should not go unnoticed. As such, rehabilitation professionals should consider TT as an adjunct to over-ground gait training for people with PD.

Use of External Cues and Assistive Devices

External cues are thought to benefit individuals with PD by bypassing the degenerated basal ganglia and utilizing other neural pathways. It is hypothesized that external cues act via the parieto-premotor pathways or the cerebellar circuits [22, 137]. Auditory, somatosensory, and visual cues have all been studied.

Auditory cueing has been shown to improve speed, cadence, stride length, and gait variability, though some studies have shown it has less effect on stride length and gait initiation than visual cueing [22, 138-141]. Auditory and somatosensory cues are comparable for improving turning velocity and in dual task situations in individuals with PD [142, 143]. Visual cueing can increase push off and improve gait initiation, and it has been shown to be most effective at increasing stride length [22, 138]. Some studies have demonstrated, though, that visual cues may decrease cadence and can interfere with dual task items [22, 140].

Some studies have attempted to combine different cueing types to see if they have an additive effect. When visual and auditory are combined for gait, it is less effective than either separately [140]. However, when an audiovisual system was used to cue sit-to-stand transfers, speed and torque of the transfer were better than without cueing [144]. An attentional strategy and auditory cueing combined are effective in improving gait [143, 145, 146].

315 In a recent review of cueing for individuals with FOG, Nieuwbower [147] concluded that
316 visual cueing may benefit movement initiation while auditory may be more effective to maintain
317 gait symmetry and timing, though neither was able to consistently prevent FOG. Cueing should
318 be individually altered, as some cueing can induce freezing in some individuals, and on-FOG
319 and off-FOG may respond differently [147]. Freezers tend to benefit more from cueing during
320 turns and at -10% of their preferred pace for normal walking [141, 142].

321 Bryant et al. [148] sent individuals home with an auditory stimulator that had been
322 programmed during an initial evaluation. After one week of individuals practicing at home for 30
323 minutes a day, gait speed and stride length showed improvement, and 90% of participants
324 thought the device was helpful and easy to use [148]. Self-cueing techniques can be utilized as
325 well. Counting out loud, saying standard phrases, and singing are sometimes used [149].
326 Teaching individuals to sing in their heads while performing activities has been shown to
327 improve gait and turning in individuals with mild to moderate PD [150].

328 Dual task walking seems to benefit more from cueing than single task walking in
329 individuals with mild to moderate PD. In theory, the cueing allows an individual to decrease the
330 amount of attention necessary for gait, freeing up more attention for the secondary task [139,
331 143].

332 Recent studies have shown short-term carry over after training, though it is believed that
333 the cue must continue to be present to be effective long-term. Training with auditory cues for
334 eight weeks, three times a week while increasing the tempo allowed a plateau in progress to be
335 met, with a benefit in gait parameters (stride length, velocity, and cadence) being sustained six
336 weeks later [151]. Other studies of shorter cue-training duration have shown shorter carryover in
337 turning, dual task benefits, and fear of falling [142, 143].

338 Long-term delivery of cues may be accomplished via specialized assistive devices [152].
339 Assistive devices such as canes and walkers are frequently prescribed for people with PD and
340 can be modified to include a laser that projects lines on the floor that serve as visual cues to

facilitate gait. Other devices may offer additional postural support beyond that offered by traditional devices [153]. Limited studies have been conducted to date to determine which assistive devices are optimal, but some guidance is available in the literature [154, 155]. Emerging devices may also hold promise for the treatment of FOG [156].

Resistance Training

Evidence suggests that muscle force production is reduced in individuals with PD, even in early stages of the disease, when compared to age-matched controls [157]. In fact, muscle weakness has been described as a primary symptom of PD [158]. Resistance training has been shown to be an effective intervention for improving strength, muscle mass, and function in healthy older adults [159]; however, evidence supporting the efficacy of this type of intervention for individuals with PD is currently limited. Falvo and colleagues suggest that resistive exercise may reduce the presence of PD sequelae such as impaired motor function, muscle and bone weakness, as well as reduced quality of life (QoL) and fear of falling, and as such stress the need for well-controlled clinical trials examining high-intensity resistance training in those with PD [160].

Recently, Dibble et al. studied the effects of a 12 week high-intensity, eccentric resistance training program on a variety of outcome measures for individuals with PD [161]. The investigation compared an active control group and an experimental group, both of which partook in the same resistance traditional resistance training program; in addition, the experimental group completed high-intensity eccentric training, while the control group did not. The experimental group had significant improvements in lower extremity muscle force, bradykinesia (measured by functional walking tasks), and QoL [161]. Similar results were found in a study examining eccentric resistance training and its effects on muscle structure, stair descent, and six minute walk test (6MWT) in individuals with PD [162]. Regarding specific gait parameters, Scandalis et al. found that an eight week strengthening program improved stride

length (0.83 m pre to 0.95 m post-training) and walking velocity (1.0 m/sec change pre to post-training) in individuals with PD [163]. Examining the effects of resistance training on balance, investigators have found that those with PD undergoing both balance and resistance training demonstrated significant improvements in balance and muscle strength [164, 165].

Balance Training

Balance training is a priority for individuals with PD to address postural instability. A recent systematic review by Dibble et al.[166] found moderate evidence supporting exercise-induced improvements in balance performance in mild to moderate PD. Keus et al.[167] recommend balance training as one of the top four recommendations for physical therapy, and the majority of exercise programs include some form of balance training [149, 166, 168-171]. Many mixed exercise programs (not balance training exclusively) have shown improvement in balance assessments (i.e. TUG, BBS, 5-step test speed) and during functional activities (i.e. gait parameters, turning, possible decrease in falls) after treatment [166, 169, 172]. Each of these treatment protocols has been studied for different durations and frequencies, most without specifying the length of balance training specifically. Many different modes of balance training can be utilized including, but not limited to, perturbations on a treadmill, calisthenic/balance exercises, practicing functional tasks that require increased balance, and balance specific training on a force plate [166, 171-173]. Further details on activities included in the balance exercise portion of treatments are not specifically described in most studies and, therefore, cannot be compared between studies.

An individual's confidence in his balance should also be addressed during balance training, as decreased balance confidence and fear of falling can negatively impact quality of life and are associated with falls [167]. Whole body vibration was thought to increase balance in individuals with PD, however, more recent studies show no added benefit using this form of therapy [174, 175]. Some individuals with PD might also have vestibular deficits that contribute

to their balance impairment; some studies show vestibular rehabilitation techniques may be beneficial [176, 177].

Flexibility Exercises

Since rigidity is known to be one of the cardinal symptoms of PD, stretching is often recommended to be included in a rehabilitation program for this population [167, 170, 149]. Many exercise protocols that have shown improvement in individuals with PD include stretching or flexibility as one component of the program [57, 169, 172, 171]. Increased rigidity in the hip and trunk musculature in individuals with PD has been focused on as it is thought that this increased axial rigidity may contribute to poor posture and abnormal coordination during walking and turning [42, 178, 179]. Individuals with PD may have decreased axial flexibility, which can contribute to decreased functional reach [180, 181].

Very few studies have truly examined the outcomes of a stretching or flexibility program in individuals with PD. Since studies have shown that axial rigidity is not affected by levodopa replacement therapy, a stretching program may be an effective alternative [182, 179]. Flexibility training has been shown to improve functional reach with subjective improvements in daily tasks such as driving, scratching their back, and rolling in bed but not in other functional tasks such as stand to supine time or six-minute walk distance [180]. The flexibility training focused on decreasing overall activation, relaxation into stretches with emphasis on axial structures, and performing functional task [180]. A promising study by Bartolo et al.[183] evaluated a rehabilitation protocol for individuals with PD who also had lateral trunk deviations. During this exercise program, which included stretching and relaxation exercises, functional strengthening, and gait and balance training, emphasis was placed on correcting postural trunk deviations and improving trunk control. They found improved posture, flexibility, and trunk mobility with gains lasting up to six months [183].

In a recent article presenting a new exercise program framework, King and Horak [170] summarize that rigidity impacts posture by increasing flexor tone, decreasing trunk range of motion during functional tasks and walking, and increasing muscular co-contraction which is unfavorable during automatic postural responses. They suggest that activities be performed that have increased emphasis on trunk and head rotation, postural awareness/erect alignment, flexor muscle lengthening, and increasing limits of stability [170]. As of now, this exercise program has not been evaluated for its effectiveness.

Neck rigidity, which is also greatly increased in individuals with PD, has recently been shown to correlate with posture and gait as well as have significant impact on functional mobility and balance [182]. At this time, no studies have addressed the efficacy or effectiveness of stretching or neck flexibility exercises on neck rigidity or functional tasks. In summary, there is very little research that supports stretching in individuals with PD; however, expert opinion continues to prioritize the inclusion of stretching and flexibility exercises in rehabilitation and prevention exercise regimens for individuals with PD [169, 172, 167, 170, 149, 171].

Alternative Exercise Programs

Alternative modes of exercise such as dance and Tai Chi have also been shown to be effective for improving balance and gait function in individuals with PD. Dance may be ideally suited to those with PD, as it is a social activity that is inherently motivating [184]. Dance is also performed to music which can provide an external cue to facilitate movement. In addition, dance involves the teaching and practice of specific movement strategies and incorporates dynamic balance challenges. Dance can also result in improved cardiovascular function, a testament to the fact that, if done with sufficient intensity, dance is an excellent form of aerobic exercise [185, 186]. Several studies have reported significant improvements in balance, six minute walk distance, and gait velocity in groups with PD who participated in Argentine tango or waltz/foxtrot classes [187-191]. For a comprehensive review of the literature regarding dance

and PD please see Earhart [192]. For recommendations on implementing dance classes for individuals with PD see Hackney & Earhart [193].

Tai Chi is another alternative exercise approach that has been examined in several studies with mixed results (for review see 194). Two studies have specifically reported balance outcomes, and both noted reduced falls or improved balance [195, 196]. Many of the Tai Chi studies are limited by a small sample size and some also lack a control group. It is also unclear whether Tai Chi is superior to traditional exercise, as one study found no differences between a conventional exercise group and a Tai Chi group [197].

Multidisciplinary Care Approaches

Most care for individuals with PD is currently performed on an outpatient basis using monodisciplinary care [198]. Few studies have assessed the effectiveness of the use of collaborative, intense inpatient rehabilitation for individuals with PD. One observational study found that individuals who participated in an inpatient rehabilitation program post-DBS placement had expedited optimization of their DBS settings and medications due to the 24-hour monitoring, reducing the process from a period of months to a period of weeks. The intense therapy post-surgery also allowed functional changes and gains to be immediately utilized, balance and gait training optimized, psychological changes to be monitored, and a home exercise program to be created to hopefully maintain gains [199].

Ellis et al.[200] further suggests that the comprehensive, multidisciplinary environment might be effective for individuals with PD who are experiencing a decline in function, falling more frequently, or are having more complex symptoms. After an average of 21 days of rehab, 71% of the patients in this cohort, including patients who did and did not require medication alterations, showed a clinically important improvement according to the Functional Independence Measure [200]. The inpatient rehabilitation team has been suggested to include some combination of neurosurgeons, neurologists, physical therapists, occupational therapists,

speech-language pathologists, nurses, case managers, nutritionists, sexologists, psychiatrists, and physiatrists, as is necessary for the individual patient [198-200]. As of now, however, no comparison of outcomes to standard care or cost analysis has been made. Effects on quality of life, carryover of training into the home environment, long term outcomes after inpatient rehabilitation, and characteristics of individuals who are most appropriate for inpatient rehabilitation are still unknown at this time.

Multidisciplinary care has also been advocated for outpatient settings, though research is again limited [198]. Utilization of both medication and physical therapy approaches combined was superior to medication alone [201]. Multidisciplinary care can result in both short-term [202] and long-term benefits, and can help most individuals to maintain or improve function for up to three years [203].

Remaining Challenges & Future Directions

While there are multiple approaches to the treatment of gait and balance disorders in PD, and multidisciplinary approaches offer promise for the best overall management of these conditions, several areas of challenge remain. One major issue is the fact that many aspects of gait and balance are unresponsive to levodopa and likely represent non-dopaminergic features of disease. Freezing of gait is an example of a very disabling symptom that is currently not well addressed by any approach. Another area of challenge is that of motor complications, i.e. dyskinesias and wearing off phenomena, that arise after long-term treatment with levodopa. In addition to the lack of effective treatments and the presence of treatment side effects, there is also challenge related to a lack of medical professionals with the specialized training and knowledge necessary to deliver the most up to date and evidence-based care for people with PD.

Future directions for the field of gait and balance management in PD are many and will likely include a shift from monodisciplinary care, where the focus has been on treating the

symptoms, to a multidisciplinary approach that is patient-centered and focuses not only on symptoms and impairments but also emphasizes quality of life. Future work is needed to develop new medications, advance the targeting and application of DBS, and implement more uniform and informed approaches to physical rehabilitation using evidence-based guidelines. Several studies are currently underway that will provide new evidence regarding the effectiveness of exercise-based approaches in the management of PD and should serve to inform future care models for individuals with PD.

EXECUTIVE SUMMARY

• Gait, Balance and Falls in Parkinson Disease

Gait and balance difficulties are a major problem for people with Parkinson disease. Gait is characterized by shuffling steps, decreased trunk rotation, flexed posture, decreased speed, and in some cases freezing. Postural instability is one of the four cardinal features of PD and is associated with increased fall rates.

• Therapeutic Approaches to Management of Gait and Balance Problems

Medical management of PD has, until relatively recently, focused primarily on pharmacological approaches but many aspects of gait and balance impairments are not adequately addressed by medication. More recently, deep brain stimulation has been added to the arsenal of treatment tools but again does not fully address gait and balance issues. As such, physical rehabilitation approaches are a key aspect of managing gait and balance in PD and can include specific gait and balance training, resistance training to improve muscle strength, as well as prescription of assistive devices and use of external cueing.

521○ No current treatments effectively deal with all aspects of gait and balance impairments, with
522 freezing of gait being particularly resistant to treatment.

523

524● **Remaining Challenges and Future Directions**

525○ Multidisciplinary, patient-centered care models are emerging and hold promise for improving
526 quality of care for individuals with PD.

527○ Such approaches will become critical in the management of gait and balance in PD using a
528 combination of medications, surgery, and rehabilitation.

529○ Management of gait and balance in PD is essential as impairments in gait and balance have a
530 major impact on quality of life, and quality of life may become the main outcome measure for
531 evaluating the effectiveness of PD management approaches.

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References

1. Fahn S: Description of Parkinson's disease as a clinical syndrome. *Annals of the New York Academy of Science*. 991, 1-14 (2003). **** Very informative and easy to understand overview of the major signs and symptoms of PD.**
2. Chade AR, Kasten M, Tanner CM: Nongenetic causes of Parkinson's disease. *J. Neural Transm.* 70: 147-151 (2006).
3. Biskup S, Gerlach M, Kupsch A, et al.: Genes associated with Parkinson syndrome. *J. Neurol.* 255, 8-17 (2008).
4. Taylor KS, Cook JA, Counsell CE: Heterogeneity in male to female risk for Parkinson's disease. *J Neurol. Neurosurg. Psychiatry* 78, 905-906 (2007).
5. Spillman BC, Lubitz J: New estimates of lifetime nursing home use: have patterns of use changed? *Med. Care.* 40, 965-975 (2002).
6. Perry, EK, et al.: Topography, extent, and clinical relevance of neurochemical deficits in dementia of Lewy body type, Parkinson's disease, and Alzheimer's disease. *Annals of the New York Academy of Science*. 640, 197-202 (1991).
7. Bloem BR, Hausdorff JM, Visser JE, et al.: Falls and freezing of gait in Parkinson's disease: a review of two interconnected, episodic phenomena. *Mov. Disord.* 19(8), 871-884 (2004). **** Includes excellent model of how gait and balance impairments are part of a vicious cycle that includes falling and reduced quality of life.**
8. Morris ME, Iansek R, Matyas TA et al.: Ability to modulate walking cadence remains intact in Parkinson's disease. *Journal of Neurology, Neurosurgery and Psychiatry.* 57(12), 1532-4 (1994).
9. Morris ME, Iansek R, Matyas TA et al.: The pathogenesis of gait hypokinesia in Parkinson's disease. *Brain.* 117(Pt.5), 1169-1181 (1994).
10. Morris ME, Iansek R, Matyas TA, et al: Stride length regulation in Parkinson's disease: normalization strategies and underlying mechanisms. *Brain.* 119, 551-568 (1996).
11. Morris ME, McGinley J, Huxham F, et al: Constraints on the kinetic, kinematic, and spatiotemporal parameters of gait in Parkinson's disease. *Hum. Mov. Sci.*, 18, 461-483 (1999).
12. Morris ME, Huxham FE, McGinley J, et al.: Gait disorders and gait rehabilitation in Parkinson's disease. *Adv. Neurol.*, 87, 347-361 (2001).
13. Pedersen SW, Öberg B, Larsson LE, et al.: Gait analysis, isokinetic muscle strength measurement in patients with Parkinson's disease. *Scand. J. Rehab. Med.* 29, 67-74 (1997).
14. Rogers MW. Disorders of posture, balance, and gait in Parkinson's disease. *Clin. Geriatr. Med.* 12, 825-845 (1996).
15. Lewis GN, Byblow WD, Walt SE. Stride length regulation in Parkinson's disease: the use of extrinsic, visual cues. *Brain.* 123(Pt. 10), 2077-2090, (2000).

16. Grasso R, Peppe A, Stratta F, et al.: Basal ganglia and gait control: apomorphine administration and internal pallidum stimulation in Parkinson's disease. *Exp. Brain Res.* 126(2), 139-148, (1999).
17. Steiger MJ, Thompson PD, Marsden CD. Disordered axial movement in Parkinson's disease. *J. Neurol. Neurosurg. Psychiatry.* 61(6), 645-648, (1996).
18. Camicioli R, Oken BS, Sexton G, et al.: Verbal fluency task affects gait in Parkinson's disease with motor freezing. *J. Geriatr. Psychiatry Neurol.* 11(4), 181-185, (1998).
19. O'Shea S, Morris ME, Iansek R. Dual task interference during gait in people with Parkinson disease: effects of motor versus cognitive secondary tasks. *Phys. Ther.* 82(9), 888-897 (2002).
20. Rochester L, Hetherington V, Jones D, et al.: Attending to the task: interference effects of functional tasks on walking in Parkinson's disease and the roles of cognition, depression, fatigue, and balance. *Arch. Phys. Med. Rehabil.* 85(10), 1578-1585 (2004).
21. Rochester L, Hetherington V, Jones D, et al.: The effect of external rhythmic cues (auditory and visual) on walking during a functional task in homes of people with Parkinson's disease. *Arch. Phys. Med. Rehabil.* 86(5), 999-1006 (2005).
22. Rochester L, Nieuwboer A, Baker K, et al.: The attentional cost of external rhythmical cues and their impact on gait in Parkinson's disease: effect of cue modality and task complexity. *J. Neural Transm.* 114(10), 1243-1248 (2007).
23. Plotnik M, Giladi N, Hausdorff JM. Bilateral coordination of gait and Parkinson's disease: the effects of dual tasking. *J. Neurol. Neurosurg. Psychiatry.* 80(3), 347-50 (2009).
24. Hackney M, Earhart GM. Backward walking in Parkinson disease. *Mov. Disord.* 24(2), 218-223 (2009).
25. Hackney ME, Earhart GM. The effects of a secondary task on forward and backward walking in Parkinson disease. *Neurorehabil. Neural Repair*, in press.
26. Morris ME, Huxham F, McGinley J et al.: The biomechanics and motor control of gait in Parkinson disease. *Clinical Biomechanics.* 16(6) 459-470 (2001).
- * **Good description of gait in PD and effects of medication.**
27. Stack EL, Ashburn AM, Jupp KE: Strategies used by people with Parkinson's disease who report difficulty turning. *Parkinsonism Relat. Disord.* 12, 87-92 (2006).
28. Crenna P, Carpinella I, Rabuffetti M, et al.: The association between impaired turning and normal straight walking in Parkinson's disease. *Gait & Posture.* 26, 172-178 (2006).
29. Visser JE, Voermans NC, Oude Nijhuis LB, et al.: Quantification of trunk rotations during turning and walking in Parkinson's disease. *Clin. Neurophysiol.* 118, 1602-1606 (2007).
30. Hong M, Perlmuter JS, Earhart GM: A kinematic and electromyographic analysis of turning in people with Parkinson disease. *Neurorehabil. Neural Repair.* 23(2), 166-176 (2009).

31. Nieuwboer A, Dom R, De Weerd W, et al.: Abnormalities of the spatiotemporal characteristics of gait at the onset of freezing in Parkinson's disease. *Mov. Disord.* 16, 1066-1075 (2001).
32. Giladi N, Nieuwboer A: Understanding and treating freezing of gait in parkinsonism, proposed working definition, and setting the stage. *Mov. Disord.* 23, S423-S425 (2008).
33. Michałowska M, Fischer U, Krygowska-Wajs A, et al.: Falls in Parkinson's disease. Causes and impact on patients' quality of life. *Funct. Neurol.* 20(4), 163-8 (2005).
34. Robinson K, Dennison A, Roalf D, et al.: Falling risk factors in Parkinson's disease. *NeuroRehabilitation.* 20(3), 169-82 (2005).
35. Moore ST, MacDougall HG, Ondo WG: Ambulatory monitoring of freezing of gait in Parkinson's disease. *J Neurosci Methods*, 167, 340-348 (2008).
36. Cumming R, Klineberg R: Fall frequency and characteristics and the risk of hip fractures. *J. Am. Ger. Soc.* 42, 774-778 (1994).
37. Benatru I, Vaugoyeau M, Azulay JP: Postural disorders in Parkinson's disease. *Neurophysiologie Clinique.* 38(6), 459-465 (2008).
38. Chastan N, Debono B, Maltete D, Weber J: Discordance between measured postural instability and absence of clinical symptoms in Parkinson's disease patients in the early stages of the disease. *Movement Disorders.* 23(3), 366-372 (2008).
39. Frenklach A, Louie S, Koop MM, Bronte-Stewart H: Excessive postural sway and the risk of falls at different stages of Parkinson's disease. *Movement Disorders.* 24(3), 377-385 (2009).
40. Mancini M, Zampieri C, Carlson-Kuhta P, Chiari L, Horak FB: Anticipatory postural adjustments prior to step initiation are hypometric in untreated Parkinson's disease: an accelerometer-based approach. *European Journal of Neurology.* 16(9), 1028-1034 (2009).
41. Grimbergen YA, Langston JW, Roos RA, Bloem BR: Postural instability in Parkinson's disease: the adrenergic hypothesis and the locus coeruleus. *Expert Review of Neurotherapeutics.* 9(2), 279-290 (2009).
42. Horak FB, Dimitrova D, Nutt JG: Direction-specific postural instability in subjects with Parkinson's disease. *Exp. Neurol.* 193(2), 504-521 (2005).
43. Kerr GK, Worringham CJ, Cole MH, Lacherez PF, Wood JM, Silburn PA: Predictors of future falls in Parkinson disease. *Neurology.* 75(2), 116-124 (2010).
44. Kim S, Horak FB, Carlson-Kuhta P, Park S: Postural feedback scaling deficits in Parkinson's disease. *J. Neurophysiol.* 102(5), 2910-2920 (2009).
45. King LA, St George RJ, Carlson-Kuhta P, Nutt JG, Horak FB: Preparation for Compensatory Forward Stepping in Parkinson's Disease. *Arch. Phys. Med. Rehabil.* 91(9), 1332-1338 (2010).
46. McVey MA, Stylianou AP, Luchies CW, et al.: Early biomechanical markers of postural instability in Parkinson's disease. *Gait Posture.* 30(4), 538-542 (2009).

47. Grimbergen YA, Munneke M, Bloem BR. Falls in Parkinson's disease. *Curr. Opin. Neurol.* 17(4), 405-415, (2004).
48. Bohnen NI, Cham R: Postural control, gait, and dopamine functions in parkinsonian movement disorders. *Clin. Geriatr. Med.* 22(4), 797-812, vi (2006).
49. Visser M, Marinus J, Bloem BR, Kijes H, van den Berg BM, van Hilten JJ: Clinical tests for the evaluation of postural instability in patients with parkinson's disease. *Arch. Phys. Med. Rehabil.* 84(11), 1669-1674 (2003).
50. Valkovic P, Brozova H, Botzel K, Ruzicka E, Benetin J: Push-and-release test predicts Parkinson fallers and nonfallers better than the pull test: comparison in OFF and ON medication states. *Mov. Disord.* 23(10), 1453-1457 (2008).
51. Blaszczyk JW, Orawiec R, Duda-Klodowska D, Opala G: Assessment of postural instability in patients with Parkinson's disease. *Exp. Brain Res.* 183(1), 107-114 (2007).
52. Buckley TA, Pitsikoulis C, Hass CJ: Dynamic postural stability during sit-to-walk transitions in Parkinson disease patients. *Mov. Disord.* 23(9), 1274-1280 (2008).
53. Merello M, Fantacone N, Balej J: Kinematic study of whole body center of mass position during gait in Parkinson's disease patients with and without festination. *Mov. Disord.* 25(6), 739-746 (2010).
54. Bleuse S, Cassim F, Blatt JL, et al.: Anticipatory postural adjustments associated with arm movement in Parkinson's disease: a biomechanical analysis. *J. Neurol. Neurosurg. Psychiatry.* 79(8), 881-887 (2008).
55. Dimitrova D, Horak FB, Nutt JG: Postural muscle responses to multidirectional translations in patients with Parkinson's disease. *J. Neurophysiol.* 91(1), 489-501 (2004).
56. Adkin AL, Frank JS, Jog MS: Fear of falling and postural control in Parkinson's disease. *Mov. Disord.* 18(5), 496-502 (2003).
57. Ashburn A, Fazakarley L, Ballinger C, Pickering R, McLellan LD, Fitton C: A randomised controlled trial of a home based exercise programme to reduce the risk of falling among people with Parkinson's disease. *Journal of Neurology, Neurosurgery & Psychiatry.* 78(7), 678-684 (2007).
58. Latt MD, Lord SR, Morris JG, Fung VS: Clinical and physiological assessments for elucidating falls risk in Parkinson's disease. *Mov. Disord.* 24(9), 1280-1289 (2009).
59. Matinolli M, Korpelainen JT, Korpelainen R, Sotaniemi KA, Virranniemi M, Myllyla VV: Postural sway and falls in Parkinson's disease: a regression approach. *Mov. Disord.* 22(13), 1927-1935 (2007).
60. Bloem BR, Grimbergen YA, Cramer M, Willemsen M, Zwinderman AH: Prospective assessment of falls in Parkinson's disease. *J. Neurol.* 248(11), 950-958 (2001).
61. Fink HA, Kuskowski MA, Orwoll ES, Cauley JA, Ensrud KE, Osteoporotic Fractures in Men (MrOS) Study Group: Association between Parkinson's disease and low bone density and falls in

- older men: the osteoporotic fractures in men study. *J. Am. Geriatr. Soc.* 53(9), 1559-1564 (2005).
62. Temlett JA, Thompson PD: Reasons for admission to hospital for Parkinson's disease. *Intern. Med. J.* 36(8), 524-526 (2006).
63. Woodford H, Walker R: Emergency hospital admissions in idiopathic Parkinson's disease. *Movement Disorders.* 20(9), 1104-1108 (2005).
64. Allcock LM, Rowan EN, Steen IN, Wesnes K, Kenny RA, Burn DJ: Impaired attention predicts falling in Parkinson's disease. *Parkinsonism Relat. Disord.* 15(2), 110-115 (2009).
65. Ashburn A, Stack E, Ballinger C, Fazakarley L, Fitton C: The circumstances of falls among people with Parkinson's disease and the use of Falls Diaries to facilitate reporting. *Disabil. Rehabil.* 30(16), 1205-1212 (2008).
66. Wood BH, Bilclough JA, Bowron A, Walker RW: Incidence and prediction of falls in Parkinson's disease: a prospective multidisciplinary study. *J. Neurol. Neurosurg. Psychiatry.* 72(6), 721-725 (2002).
67. Melton LJ III, Leibson CL, Achenbach SJ, Bower JH, Maraganore DM, Ober AL, Rocca WA. Fracture risk after the diagnosis of Parkinson's disease: influence of concomitant dementia. *Mov Disord.* 21(9): 1361-1367, 2006.
68. Rahman S, Griffin HJ, Quinn NP, Jahanshahi M: Quality of life in Parkinson's disease: The relative importance of the symptoms. *Movement Disorders.* 23(10), 1428-1434 (2008).
69. Bacon WE: Secular trends in hip fracture occurrence and survival: age and sex differences. *J. Aging Health.* 8(4), 538-553 (1996).
70. Kempster PA, O'Sullivan SS, Holton JL, Revesz T, Lees AJ: Relationships between age and late progression of Parkinson's disease: a clinico-pathological study. *Brain.* 133(Pt 6), 1755-1762 (2010).
71. Dennison AC, Noorigian JV, Robinson KM, *et al.*: Falling in Parkinson disease: identifying and prioritizing risk factors in recurrent fallers. *Am. J. Phys. Med. Rehabil.* 86(8), 621-632 (2007).
72. Mak MK, Pang MY: Fear of falling is independently associated with recurrent falls in patients with Parkinson's disease: a 1-year prospective study. *J. Neurol.* 256(10), 1689-1695 (2009).
73. Martignoni E, Tassorelli C, Nappi G: Cardiovascular dysautonomia as a cause of falls in Parkinson's disease. *Parkinsonism Relat. Disord.* 12(4), 195-204 (2006).
74. Moore ST, MacDougall HG, Gracies JM *et al.*: Locomotor response to levodopa in fluctuating Parkinson's Disease. *Experimental Brain Research.* 184(4), 469-478 (2008).
75. Sethi K.: Levodopa unresponsive symptoms in Parkinson disease. *Movement Disorders.* 23(Suppl 3), S521-S533 (2008).

76. Blin O, Fernandez AM, Pailhous J et al.: Dopa-sensitive and dopa-resistance gait parameters in Parkinson's disease. *Journal of the Neurological Sciences*. 103(1), 51-4 (1991).
77. Blin O, Fernandez AM, Serratrice G.: Quantitative analysis of gait in Parkinson patients: increased variability of stride length. *Journal of the Neurological Sciences*. 98(1), 91-7 (1990).
78. Bowes SG, Clark PK, Leeman AL et al.: Determinants of gait in the elderly parkinsonian on maintenance levodopa/carbidopa therapy. *British Journal of Clinical Pharmacology*. 30(1), 13-24 (1990).
79. Pedersen SW, Eriksson T, Oberg B. Effects of withdrawal of antiparkinson medication on gait and clinical score in the Parkinson patient. *Acta Neurologica Scandinavica*. 84(1), 7-13 (1991).
80. McIntosh GC, Brown SH, Rice RR et al.: Rhythmic auditory-motor facilitation of gait patterns in patients with Parkinson's disease. *Journal of Neurology, Neurosurgery and Psychiatry*. 62(1), 22-6 (1997).
81. Azulay JP, Mesure S, Amblard B et al.: Visual control of locomotion in Parkinson's disease. *Brain*. 122(Pt.1), 111-120 (1999).
82. Hanakawa T, Katsumi Y, Fukuyama H et al.: Enhanced lateral premotor activity during paradoxical gait in Parkinson's disease. *Annals of Neurology*. 45(3), 329-336 (1999).
83. Ebersbach G, Sojer M, Valldeoriola F et al.: Comparative analysis of gait in Parkinson's disease, cerebellar ataxia and subcortical arteriosclerotic encephalopathy. *Brain*. 122(Pt.7) 1349-1355 (1999).
84. Morris ME, Matyas TA, Iansek R et al.: Temporal stability of gait in Parkinson's disease. *Physical Therapy*. 76(7), 763-777 (1996).
85. Koozekanani SH, Balmaseda MT Jr, Fatehi MT et al.: Ground reaction forces during ambulation in parkinsonism: pilot study. *Archives of Physical Medicine & Rehabilitation*. 68(1), 28-30 (1987).
86. O'Sullivan JD, Said CM, Dillon LC et al.: Gait analysis in patients with Parkinson's disease and motor fluctuations: influence of levodopa and comparison with other measures of motor function. *Movement Disorders*. 13(6), 900-6 (1998).
87. Morris M, Iansek R, Matyas T et al.: Abnormalities in the stride length-cadence relation in parkinsonian gait. *Movement Disorders*. 13(1), 61-9 (1998).
88. Okuma Y.: Freezing of gait in Parkinson's disease. *Journal of Neurology*. 253(Suppl 7), VII27-32 (2006).
89. Fahn S.: The freezing phenomenon in parkinsonism. *Advances in Neurology*. 67, 53-63 (1995).
90. Schaafsma JD, Balash Y, Gurevich T et al.: Characterization of freezing of gait subtypes and the response of each to levodopa in Parkinson's disease. *European Journal of Neurology*. 10(4), 391-8 (2003).

91. Giladi N.: Freezing of gait. Clinical overview. *Advances in Neurology*. 87, 191-7 (2001).
92. Giladi N.: Medical treatment of freezing of gait. *Movement Disorders*. 23 (Supl 2), S482-8 (2008).
93. Giladi N, McDermott MP, Fahn S et al.: Freezing of gait in PD: prospective assessment in the DATATOP cohort. *Neurology*. 56(12), 1712-1721 (2001).
94. Shoulson I.: DATATOP: a decade of neuroprotective inquiry. Parkinson Study Group. Deprenyl and tocopherol antioxidative therapy of parkinsonism. *Annals of Neurology*. 44(3 Suppl 1), S160-6 (1998).
95. Shoulson I, Oakes D, Fahn S et al.: Impact of sustained deprenyl (selegiline) in levodopa-treated Parkinson's disease: a randomized placebo-controlled extension of the deprenyl and tocopherol antioxidative therapy of parkinsonism trial. *Annals of Neurology*. 51(5), 604-612 (2002).
96. Giladi N, Treves TA, Simon ES et al.: Freezing of gait in patients with advances Parkinson's disease. *Journal of Neural Transmission*. 108(1), 53-61 (2001).
97. Macht M, Kaussner Y, Moller JC et al.: Predictors of freezing in Parkinson's disease: a survey of 6,620 patients. *Movement Disorders*. 22(7), 953-6 (2007).
98. Giladi N and Honigman S.: Botulinum toxin injections to one leg alleviate freezing of gait in a patient with Parkinson's disease. *Movement Disorders*. 12(6), 1085-6 (1997).
99. Giladi N, Gurevich T, Shabtai H et al.: The effect of botulinum toxin injections to the calf muscles on freezing of gait in parkinsonism: a pilot study. *Journal of Neurology*. 248(7), 572-6 (2001).
100. Gurevich T, Peretz C, Moore O et al.: The effect of injecting botulinum toxin type a into the calf muscles on freezing of gait in Parkinson's disease: a double blind placebo-controlled pilot study. *Movement Disorders*. 22(6), 880-3 (2007).
101. Pollak L, Dobronevsky Y, Prohorov T et al.: Low dose methylphenidate improves freezing in advanced Parkinson's disease during off-state. *Journal of Neural Transmission Supplementum*. 72, 145-8 (2007).
102. Auriel E, Hausdorff JM, Herman T et al.: Effects of methylphenidate on cognitive function and gait in patients with Parkinson's disease: a pilot study. *Clinical Neuropharmacology*. 29(1), 15-7 (2006).
103. Vaamonde Gamo J, Cabello JP, Gallardo Alcañiz MJ, et al.: Freezing of gait unresponsive to dopaminergic stimulation in patients with severe Parkinsonism. *Neurologia*. 25(1), 27-31 (2010).
104. Clarke CE, Worth P, Grosset D et al.: Systematic review of apomorphine infusion, levodopa infusion and deep brain stimulation in advanced Parkinson's disease. *Parkinsonism & Related Disorders*. 15(10), 728-741 (2009).

105. Tagliati M. Fine-tuning gait in Parkinson disease. *Neurology*. 71(2), 76-7 (2008).
106. Van Nuenen BF, Esselink RA, Munneke M et al.: Postoperative gait deterioration after bilateral subthalamic nucleus stimulation in Parkinson's disease. *Movement Disorders*. 23(16), 2404-6 (2008).
107. Krack P, Batir A, Blercom N et al.: Five-year follow-up of bilateral stimulation of the subthalamic nucleus in advanced Parkinson's disease. *The New England Journal of Medicine*. 349(20), 1925-1934 (2003).
108. Bakker M, Esselink RA, Munneke M et al.: Effects of stereotactic neurosurgery on postural instability and gait in Parkinson's disease. *Movement Disorders*. 19(9), 1092-9 (2004).
109. Johnsen EL, Mogensen PH, Sunde NA et al.: Improved asymmetry of gait in Parkinson's disease with DBS: gait and postural instability in Parkinson's disease treated with bilateral stimulation in the subthalamic nucleus. *Movement Disorders*. 24(4), 590-7 (2009).
110. Hausdorff JM, Gruendlinger L, Scollins L et al.: Deep brain stimulation effects on gait variability in Parkinson's disease. *Movement Disorders*. 24(11), 1688-1692 (2009).
111. Volkmann J.: Deep brain stimulation for the treatment of Parkinson's disease. *Journal of Clinical Neurophysiology*. 21(1), 6-17 (2004).
112. Mazzone P, Lozano A, Stanzione P et al.: Implantation of human pedunculo pontine nucleus: a safe and clinically relevant target in Parkinson's disease. *Neuroreport*. 16(17), 1877-1881 (2005).
113. Plaha P and Gill SS.: Bilateral deep brain stimulation of the pedunculo pontine nucleus for Parkinson's disease. *Neuroreport*. 16(17), 1883-7 (2005).
114. Moro E, Hamani C, Poon YY et al.: Unilateral pedunculo pontine stimulation improves fall in Parkinson's disease. *Brain*. 133(Pt.1), 215-224 (2010).
115. Stefani A, Lozano AM, Peppe A et al.: Bilateral deep brain stimulation of the pedunculo pontine and subthalamic nuclei in severe Parkinson's disease. *Brain*. 130(Pt.6), 1596-1607 (2007).
116. Davis JT, Lyons KE, Pahwa R.: Freezing of gait after bilateral subthalamic nucleus stimulation for Parkinson's disease. *Clinical Neurology and Neurosurgery*. 108(5), 461-4 (2006).
117. Limousin P, Krack P, Pollak P et al.: Electrical stimulation of the subthalamic nucleus in advanced Parkinson's disease. *The New England Journal of Medicine*. 339(16), 1105-1111 (1998).
118. Stolze H, Klebe S, Poepping M et al.: Effects of bilateral subthalamic nucleus stimulation on parkinsonian gait. *Neurology*. 57(1), 144-6 (2001).
119. Yokoyama T, Sugiyama K, Nishizawa S et al.: Subthalamic nucleus stimulation for gait disturbance in Parkinson's disease. *Neurosurgery*. 45(1), 41-7 (1999).

120. Rodriguez-Oroz MC, Obeso JA, Lang AE et al.: Bilateral deep brain stimulation in Parkinson's disease: a multicentre study with 4 years follow-up. *Brain*. 128(Pt.10), 2240-9 (2005).
121. Bejjani BP, Gervais D, Arnulf I et al.: Axial parkinsonian symptoms can be improved: the role of levodopa and bilateral subthalamic stimulation. *Journal of Neurology, Neurosurgery and Psychiatry*. 68(5), 595-600 (2000)
122. Nandi D, Aziz TZ, Giladi N et al.: Reversal of akinesia in experimental parkinsonism by GABA antagonist microinjections in the pedunculo pontine nucleus. *Brain*. 125(Pt.11), 2418-2430 (2002).
123. Munro-Davies LE, Winter J, Aziz TZ et al.: The role of the pedunculo pontine region in basal-ganglia mechanisms of akinesia. *Experimental Brain Research*. 129(4), 511-7 (1999).
124. Benedetti F, Pollo A, Lopiano L et al.: Conscious expectation and unconscious conditioning in analgesic, motor, and hormonal placebo/nocebo responses. *The Journal of Neuroscience*. 23(10), 4315-4323 (2003).
125. Mehrholz J, Friis R, Kugler J et al.: Treadmill training for patients with Parkinson's disease (Review). *Cochrane Database Systematic Review*. (1), 1-31 (2010).
126. Fisher BE, Wu AD, Salem GJ et al.: The effect of exercise training in improving motor performance and corticomotor excitability in people with early Parkinson's disease. *Archives of Physical Medicine & Rehabilitation*. 89(7), 1221-9 (2008).
127. Pohl M, Rockstroh G, Ruckriem S et al.: Immediate effects of speed-dependent treadmill training on gait parameters in early Parkinson's disease. *Archives of Physical Medicine and Rehabilitation*. 84(12), 1760-6 (2003).
128. Miyai I, Fujimoto Y, Ueda Y et al.: Treadmill training with body weight support: its effect on Parkinson's disease. *Archives of Physical Medicine and Rehabilitation*. 81(7), 849-852 (2000).
129. Protas EJ, Mitchell K, Williams A et al.: Gait and step training to reduce falls in Parkinson's disease. *NeuroRehabilitation*. 20(3), 183-190 (2005).
130. Cakit BD, Saracoglu M, Genc H et al.: The effects of incremental speed-dependent treadmill training on postural instability and fear of falling in Parkinson's disease. *Clinical Rehabilitation*. 21(8), 698-705 (2007).
131. Kurtais Y, Kutlay S, Tur BS et al.: Does treadmill training improve lower-extremity tasks in Parkinson disease? A randomized controlled trial. *Clinical Journal of Sports Medicine*. 18(3), 289-291 (2008).
132. Herman T, Giladi N, Gruendlinger L et al.: Six weeks of intensive treadmill training improves gait and quality of life in patients with Parkinson's disease: A pilot study. *Archives of Physical Medicine and Rehabilitation*. 88(9), 1154-8 (2007).
133. Bello O, Marquez G, Camblor M et al.: Mechanisms involved in treadmill walking improvements in Parkinson's disease. *Gait and Posture*. 32, 118-123 (2010).

134. Rubinstein TC, Giladi N, Hausdorff JM.: The power of cueing to circumvent dopamine deficits: a review of physical therapy treatment of gait disturbances in Parkinson's disease. *Movement Disorders*. 17(6), 1148-1160 (2002).
135. Murray MP, Sepic SB, Gardner GM et al.: Walking patterns of men with parkinsonism. *American Journal of Physical Medicine*. 57(6), 278-294 (1978).
136. Morris ME.: Movement disorders in people with Parkinson disease: a model for physical therapy. *Physical Therapy*. 80(6), 578-597 (2000).
137. Rochester L, Baker K, Hetherington V, *et al.*:Evidence for motor learning in Parkinson's disease: acquisition, automaticity and retention of cued gait performance after training with external rhythmical cues. *Brain Res*. 1319 103-111 (2010).
138. Jiang Y, Norman KE:Effects of visual and auditory cues on gait initiation in people with Parkinson's disease. *Clin. Rehabil*. 20(1), 36-45 (2006).
139. Rochester L, Burn DJ, Woods G, Godwin J, Nieuwboer A:Does auditory rhythmical cueing improve gait in people with Parkinson's disease and cognitive impairment? A Feasibility study. *Movement Disorders*. 24(6), 839-845 (2009).
140. Suteerawattananon M, Morris GS, Etnyre BR, Jankovic J, Protas EJ:Effects of visual and auditory cues on gait in individuals with Parkinson's disease. *J. Neurol. Sci*. 219(1-2), 63-69 (2004).
141. Willems AM, Nieuwboer A, Chavret F, *et al.*:Turning in Parkinson's disease patients and controls: the effect of auditory cues. *Mov. Disord*. 22(13), 1871-1878 (2007).
142. Nieuwboer A, Baker K, Willems AM, *et al.*:The short-term effects of different cueing modalities on turn speed in people with Parkinson's disease. *Neurorehabil. Neural Repair*. 23(8), 831-836 (2009).
143. Rochester L, Rafferty D, Dotchin C, Msuya O, Minde V, Walker RW:The effect of cueing therapy on single and dual-task gait in a drug naive population of people with Parkinson's disease in northern Tanzania. *Mov. Disord*. 25(7), 906-911 (2010).
144. Mak MK, Hui-Chan CW:Cued task-specific training is better than exercise in improving sit-to-stand in patients with Parkinson's disease: A randomized controlled trial. *Mov. Disord*. 23(4), 501-509 (2008).
145. Baker K, Rochester L, Nieuwboer A:The effect of cues on gait variability--reducing the attentional cost of walking in people with Parkinson's disease. *Parkinsonism Relat. Disord*. 14(4), 314-320 (2008).
146. Baker K, Rochester L, Nieuwboer A:The Immediate Effect of Attentional, Auditory, and a Combined Cue Strategy on Gait During Single and Dual Tasks in Parkinson's Disease. *Arch. Phys. Med. Rehabil*. 88(12), 1593-1600 (2007).
147. Nieuwboer A: Cueing for freezing of gait in patients with Parkinson's disease: a rehabilitation perspective. *Mov. Disord*. 23 Suppl 2 S475-81 (2008).

148. Bryant MS, Rintala DH, Lai EC, Protas EJ: An evaluation of self-administration of auditory cueing to improve gait in people with Parkinson's disease. *Clin. Rehabil.* 23(12), 1078-1085 (2009).
149. Morris ME, Martin CL, Schenkman ML: Striding out with Parkinson disease: evidence-based physical therapy for gait disorders. *Phys. Ther.* 90(2), 280-288 (2010).
- * Review of current evidence to support specific physical therapy approaches to management of gait in PD.**
150. Satoh M, Kuzuhara S: Training in mental singing while walking improves gait disturbance in Parkinson's disease patients. *Eur. Neurol.* 60(5), 237-243 (2008).
151. Ford MP, Malone LA, Nyikos I, Yelisetty R, Bickel CS: Gait training with progressive external auditory cueing in persons with Parkinson's disease. *Arch. Phys. Med. Rehabil.* 91(8), 1255-1261 (2010).
152. Nieuwboer A, Kwakkel G, Rochester L, et al.: Cueing training in the home improves gait-related mobility in Parkinson's disease: the RESCUE trial. *J. Neurol. Neurosurg. Psychiatry.* 78(2), 134-140, (2007).
153. Bryant MS, Rintala DH, Lai EC, et al.: Evaluation of a new device to prevent falls in persons with Parkinson's disease. *Disabil Rehabil Assist Technol.* 4(5), 357-63 (2010).
154. Cubo E, Moore CG, Leugran S, et al.: Wheeled and standard walkers in Parkinson's disease patients with gait freezing. *Parkinsonism Relat. Disord.* 10(1), 9-14 (2003).
155. Constantinescu R, Leonard C, Deeley C, et al.: Assistive devices for gait in Parkinson's disease. *Parkinsonism Relat. Disord.* 13(3), 133-8 (2007).
156. Bächlin M, Plotnik M, Roggen D, et al.: A wearable system to assist walking of Parkinson's disease patients. *Methods Inf. Med.* 49(1), 88-95 (2010).
157. Cano-de-la-Cuerda R, Perez-de-Heredia M, Miangolarra-Page JC et al.: Is there muscular weakness in Parkinson's disease? *American Journal of Physical Medicine & Rehabilitation.* 89(1), 70-6 (2010).
158. Koller W and Kase S. Muscle strength testing in Parkinson's disease. *European Neurology.* 25(2), 130-3 (1986).
159. Macaluso A and De Vito G.: Muscle strength, power and adaptations to resistance training in older people. *European Journal of Applied Physiology.* 91(4), 450-472 (2004).
160. Falvo MJ, Schilling BK, Earhart GM.: Parkinson's disease and resistive exercise: rationale, review and recommendations. *Movement Disorders.* 23(1), 1-11 (2008).
161. Dibble LE, Hale TF, Marcus RL et al.: High intensity eccentric resistance training decreases bradykinesia and improves quality of life in persons with Parkinson's disease: a preliminary study. *Parkinsonism & Related Disorders.* 15(10), 752-7 (2009).

- 1089 162. Dibble LE, Hale TF, Marcus RL et al.: High-intensity resistance training amplifies muscle
1090 hypertrophy and functional gains in persons with Parkinson's disease. *Movement Disorders*.
1091 21(9), 1444-1452 (2006).
1092
- 1093 163. Scandalis TA, Bosak A, Berliner JC et al.: Resistance training and gait function in patients
1094 with Parkinson's disease. *American Journal of Physical Medicine & Rehabilitation*. 80(1), 38-43
1095 (2001).
1096
- 1097 164. Hirsch MA, Toole T, Matiland CG et al.: The effects of balance training and high-intensity
1098 resistance training on persons with idiopathic Parkinson's disease. *Archives of Physical*
1099 *Medicine and Rehabilitation*. 84(8), 1109-1117 (2003).
1100
- 1101 165. Toole T, Hirsch MA, Forkink A et al.: The effects of a balance and strength training
1102 program on equilibrium in Parkinsonism: a preliminary study. *NeuroRehabilitation*. 14(3), 165-
1103 174 (2000).
1104
- 1105 166. Dibble LE, Addison O, Papa E: The effects of exercise on balance in persons with
1106 Parkinson's disease: a systematic review across the disability spectrum. *Journal of Neurologic*
1107 *Physical Therapy*. 33(1), 14-26 (2009). **** Systematic review of latest evidence on balance**
1108 **training in PD.**
1109
- 1110 167. Keus SH, Bloem BR, Hendriks EJ, Bredero-Cohen AB, Munneke M, Practice
1111 Recommendations Development Group: Evidence-based analysis of physical therapy in
1112 Parkinson's disease with recommendations for practice and research. *Mov. Disord*. 22(4), 451-
1113 60; quiz 600 (2007).
1114 **** Establishes evidence-based guidelines for physical therapy practice in PD.**
1115
- 1116 168. Canning CG, Sherrington C, Lord SR, et al.: Exercise therapy for prevention of falls in
1117 people with Parkinson's disease: a protocol for a randomised controlled trial and economic
1118 evaluation. *BMC Neurol*. 9 4 (2009).
1119
- 1120 169. Frazzitta G, Maestri R, Bertotti G, et al.: Rehabilitation in Parkinson's disease: assessing
1121 the outcome using objective metabolic measurements. *Mov. Disord*. 25(5), 609-614 (2010).
1122
- 1123 170. King LA, Horak FB: Delaying mobility disability in people with Parkinson disease using a
1124 sensorimotor agility exercise program. *Phys. Ther*. 89(4), 384-393 (2009).
1125
- 1126 171. Tamir R, Dickstein R, Huberman M: Integration of motor imagery and physical practice in
1127 group treatment applied to subjects with Parkinson's disease. *Neurorehabil. Neural Repair*.
1128 21(1), 68-75 (2007).
1129
- 1130 172. Gobbi LTB, Oliveira-Ferreira MDT, Caetano MJD, et al.: Exercise programs improve
1131 mobility and balance in people with Parkinson's disease. *Parkinsonism Relat. Disord*.
1132 15(Supplement 3), S49-S52 (2009).
1133
- 1134 173. Jessop RT, Horowicz C, Dibble LE: Motor learning and Parkinson disease: Refinement of
1135 movement velocity and endpoint excursion in a limits of stability balance task. *Neurorehabil.*
1136 *Neural Repair*. 20(4), 459-467 (2006).
1137
- 1138 174. Arias P, Chouza M, Vivas J, Cudeiro J: Effect of whole body vibration in Parkinson's
1139 disease: A controlled study. *Movement Disorders*. 24(6), 891-898 (2009).

- 1140
1141 175. Ebersbach G, Edler D, Kaufhold O, Wissel J: Whole Body Vibration Versus Conventional
1142 Physiotherapy to Improve Balance and Gait in Parkinson's Disease. *Arch. Phys. Med. Rehabil.*
1143 89(3), 399-403 (2008).
1144
1145 176. Rossi-Izquierdo M, Soto-Varela A, Santos-Perez S, *et al.*: Vestibular rehabilitation with
1146 computerised dynamic posturography in patients with Parkinson's disease: improving balance
1147 impairment. *Disability & Rehabilitation.* 31(23), 1907-1916 (2009).
1148
1149 177. Zeigelboim BS, Klagenberg KF, Teive HA, Munhoz RP, Martins-Bassetto J: Vestibular
1150 rehabilitation: clinical benefits to patients with Parkinson's disease. *Arq. Neuropsiquiatr.* 67(2A),
1151 219-223 (2009).
1152
1153 178. Winogrodzka A, Wagenaar RC, Booij J, Wolters EC: Rigidity and bradykinesia reduce
1154 interlimb coordination in Parkinsonian gait. *Arch. Phys. Med. Rehabil.* 86(2), 183-189 (2005).
1155
1156 179. Wright WG, Gurfinkel VS, Nutt J, Horak FB, Cordo PJ: Axial hypertonicity in Parkinson's
1157 disease: Direct measurements of trunk and hip torque. *Exp. Neurol.* 208(1), 38-46 (2007).
1158
1159 180. Schenkman M, Cutson TM, Kuchibhatla M, *et al.*: Exercise to improve spinal flexibility and
1160 function for people with Parkinson's disease: a randomized, controlled trial. *J. Am. Geriatr. Soc.*
1161 46(10), 1207-1216 (1998).
1162
1163 181. Schenkman M, Morey M, Kuchibhatla M: Spinal flexibility and balance control among
1164 community-dwelling adults with and without Parkinson's disease. *J. Gerontol. A Biol. Sci. Med.*
1165 *Sci.* 55(8), M441-5 (2000).
1166
1167 182. Franzén E, Paquette C, Gurfinkel VS, Cordo PJ, Nutt JG, Horak FB: Reduced performance
1168 in balance, walking and turning tasks is associated with increased neck tone in Parkinson's
1169 disease. *Exp. Neurol.* 219(2), 430-438 (2009).
1170
1171 183. Bartolo M, Serrao M, Tassorelli C, *et al.*: Four-week trunk-specific rehabilitation treatment
1172 improves lateral trunk flexion in Parkinson's disease. *Movement Disorders.* 25(3), 325-331
1173 (2010).
1174
1175 184. Palo-Bengtsson L, Ekman SL: Social dancing in the care of persons with dementia in a
1176 nursing home setting: a phenomenological study. *Sch. Inq. Nurs. Pract.* 11(2), 101-8 (1997).
1177
1178 185. Belardinelli, R *et al.*: Waltz dancing in patients with chronic heart failure: a new form of
1179 exercise training. *Circulation: Heart Failure.* 1, 107-114 (2008).
1180
1181 186. Peidro RM *et al.*: Tango: modificaciones cardiorrespiratorias durante el baile. *Revista*
1182 *Argentina de Cardiología*, 70, 358-363 (2002).
1183
1184 187. Hackney ME, Kantorovich S, Levin R, *et al.*: Effects of tango on functional mobility in
1185 Parkinson disease: A preliminary study. *J. Neurol. Phys. Ther.* 31(4), 173-179 (2007).
1186
1187 188. Hackney ME, Earhart GM: Short Duration, Intensive Tango Dancing for Parkinson
1188 Disease: An Uncontrolled Pilot Study. *Complement. Ther. Med.* 17, 203-207 (2009).
1189

- 1190 189. Hackney ME, Earhart GM: Effects of dance on movement control in Parkinson disease: a
1191 comparison of Argentine Tango and American Ballroom. *J. Rehab. Med.* 41, 475-481 (2009).
1192
- 1193 190. Hackney ME, Earhart GM: Effects of dance on gait and balance in Parkinson disease: a
1194 comparison of partnered and non-partnered dance movement. *Neurorehabil. Neural Repair.*
1195 24(4), 384-392 (2010).
1196
- 1197 191. Hackney ME, Earhart GM: Effects of dance on balance and gait in severe Parkinson
1198 disease: A case study. *Disabil. Rehab.* 32(8), 679-684 (2010).
1199
- 1200 192. Earhart GM: Dance as therapy for individuals with Parkinson disease. (2009) *Eur. J. Phys.*
1201 *Med. Rehabil.* 45(2), 231-238 (2009).
1202
- 1203 193. Hackney ME, Earhart GM: Recommendations for implementing partnered tango classes
1204 for persons with Parkinson disease. *Amer. J. Dance Ther.* 32(1), 41 (2010).
1205
- 1206 194. Lee MS, Lam P, Ernst E: Effectiveness of tai chi for Parkinson's disease: A critical review.
1207 *Parkinsonism & Related Disorders.* 14, 589-594 (2008).
1208
- 1209 195. Marjama-Lyons J et al.: Tai Chi and reduced rate of falling in Parkinson's disease: a single
1210 blinded pilot study. *Movement Disorders.* 17, S70-S71 (2002).
1211
- 1212 196. Hackney ME, Earhart GM: Tai Chi improves balance and mobility in people with Parkinson
1213 disease. *Gait & Posture.* 28, 456-460 (2008).
1214
- 1215 197. Cheon SM, Sung HR, Ha MS, et al.: Tai Chi for physical and mental health of patients with
1216 Parkinson's disease. In: *Proceedings of the first international conference of tai chi for health.*
1217 Seoul, Republic of Korea, 68 (abstract) (2006).
1218
- 1219 198. van der Marck MA, Kalf JG, Sturkenboom IH, Nijkrake MJ, Munneke M, Bloem
1220 BR:Multidisciplinary care for patients with Parkinson's disease. *Parkinsonism Relat. Disord.* 15
1221 Suppl 3 S219-23 (2009).
1222
- 1223 199. Cohen DB, Oh MY, Baser SM, et al.:Fast-Track Programming and Rehabilitation Model: A
1224 Novel Approach to Postoperative Deep Brain Stimulation Patient Care. *Arch. Phys. Med.*
1225 *Rehabil.* 88(10), 1320-1324 (2007).
1226
- 1227 200. Ellis T, Katz DI, White DK, DePiero TJ, Hohler AD, Saint-Hilaire M:Effectiveness of an
1228 inpatient multidisciplinary rehabilitation program for people with Parkinson disease. *Phys. Ther.*
1229 88(7), 812-819 (2008). * **Presents effects of a multidisciplinary inpatient program in a large**
1230 **sample of people with PD.**
1231
- 1232 201. Ellis T, de Goede CJ, Feldman RG, et al.: Efficacy of a physical therapy program in
1233 patients with Parkinson's disease: a randomized controlled trial. *Arch. Phys. Med. Rehabil.*
1234 86(4), 626-32 (2005).
1235
- 1236 202. Trend P, Kaye J, Gage H, et al.: Short-term effectiveness of intensive multidisciplinary
1237 rehabilitation for people with Parkinson's disease and their carers. *Clin. Rehabil.*16(7), 717-25
1238 (2002).
1239

1240 203. Carne W, Cifu DX, Marcinko P, et al.: Efficacy of multidisciplinary treatment program on
1241 long-term outcomes of individuals with Parkinson's disease. J. Rehabil. Res. Dev. 42(6), 779-86
1242 (2005).
1243